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EDITORIAL

MAJOR REVIEW

Keratoconus
Dr. Anil Radhakrishnan

ORIGINAL ARTICLES

Efficacy of Internal Limiting Membrane Removal in Recalcitrant Diabetic Macular Oedema - Comparative Analysis of Macular Thickness by Optical Coherence Tomography
Dr. Meena Chakrabarti, Dr. Valsa Stephen, Dr. Sonia Rani John, Dr. Arup Chakrabarti

Intravitreal Bevacizumab in Diabetic Retinopathy
Dr Gopal S Pillai, Dr Abhijith Khake, Dr Niranjan Pehere, Dr Rajasree Nambyar, Dr Lilan Bhat, Dr Anuradha Rao, Dr Meenakshi Dhar

Peribulbar Anaesthesia For Vitreoretinal Procedures
Dr. Savita Bhat, Dr. Mahesh Gopalakrishnan, Dr. Pushpa Isaac, Dr. Anantharaman Giridhar

Comparison Of Results Of Optic Disc Analysis Using Stereoscopic Biomicroscopy, Stereo Fundus Photography and Optical Coherence Tomography
Dr. Meenakshi Dhar, Dr. Indu Jayachandran, Dr. Biju Raju, Ms. Deepa P.A.

OCULAR PHARMACOLOGY

Povidone–Iodine in Ophthalmology
Dr. Arup Chakrabarti, Dr. Sonia Rani John, Dr. Valsa Stephen, Dr. Meena Chakrabarti

OPHTHALMIC INSTRUMENTATION

Combined Endoscopic, Laser Assisted DacryoCystoRhinostomy (ECLAD)
Dr. Abraham Kurien

Jacksons Cross Cylinder (JCC)
Dr. R. Nirupama Balaji, Dr. K.S. Chandrakanth, Dr. Sheeja, Dr. Ramakrishnan, Dr. Tresa, Dr. Preetha

OPHTHALMIC SURGERY

Releasable and Adjustable Sutures for Safe and Predictable Outcome Following Glaucoma Filtration Surgery
Dr. Chockalingam, M., Dr. Anup Chirayath

CURRENT CONCEPTS

An Update on Eales’ Disease
Dr. Jyothirmay Biswas, Dr. Aditya Verma
CASE REPORTS

308 A Rare Case Of Subfoveal Choroidal Neovascular Membrane In Radiation Retinopathy- Combination Therapy Works…
Dr Gopal S Pillai, Dr Abhijith Khake, Dr Lakshmi Nisha Menon, Dr Meenakshi Dhar, Dr Anuradha Rao, Dr Lilan Bhat

311 Subhyaloid Haemorrhage Following Dengue
Dr. Valsa Stephen, Dr. Meena Chakrabarti, Dr. Arup Chakrabarti, Dr. Sonia Rani John

314 Acute Posterior Multifocal Placoid Pigment Epitheliopathy- A Case Report
Dr Bini S T, Dr Biju John; Dr Pravada, N.

PHOTO ESSAY

317 Idiopathic Choroidal Neovascular Membrane in a 9-year-old child
Dr. Ramkumar G., Dr. Archis Shedbale, Dr. Mahesh, G, Dr. A. Giridhar

CONSULTATION SECTION

319 Dr. Rasik B. Vajpayee, Dr. Virender S. Sangwan, Dr. Rajesh Fogla, Dr. Queresh B. Maskati, Dr. Anthrayose C.V., Dr. Freddy T. Simon, Dr. Noel Moniz

COMMUNITY OPHTHALMOLOGY

323 What is Computer Vision Syndrome ?
Dr. Meena Chakrabarti

JOURNAL REVIEW

329

BOOK REVIEW

331

CME PROGRAMMES

335

PG TEAR SHEET

337
Plagiarism

“Indian writer apologises for plagiarism” was one of the hottest news in the first half of 2006. when ‘The Harvard Crimson’ an independently run newspaper published by students at Harvard University, reported that the best seller by the Chennai-born 17 year old writer Kaavya Vishwanathan was plagiarised in parts. This news forced the writer to acknowledge that she had borrowed language and passage from two popular novels by Megan Mc Cafferty and that being a fan of this author she “may have internalised her words”!

Current English dictionaries contend that the word “Plagiarism” is of Latin origin and comes from the word “Plagiarius” meaning kidnapper. Plagiarism has been there since time immemorial as evidenced by the fact that giants such as Ptolemy, Galileo, Newton and Mendel have all been accused of plagiarism by modern scientists who reexamined their data. In our modern academic world this dishonest deed of “Plagiarism” is prompted by the ‘publish – or – perish’ hysteria. Plagiarism is much more easily performed in this era of e-literature as relevant paragraphs from any reference source can be just cut-and pasted, obviating the need to even spell check the text, as it has already been checked and published.

The theft of someone’s words or thoughts – ‘Plagiarism’ has been a matter of great concern in medical literature. This term applies to the following:

a. Unreferenced use of other’s published and unpublished ideas.
b. Submission of another’s work under ‘new ‘authorship sometimes in a different language.
c. ‘Blanket references’: numerous references given together usually cut and pasted from a published reference source without proper citations in the present article.
d. Second generation reference.
e. Duplicate or repetitive publication of one’s own previously published work (self plagiarism) 2,3

Overt plagiarism in medical publications is rare. More frequently what is seen is passive ‘plagiarism’ or attribution failure. Most authors who plagiarize do not usually do it deliberately. This is probably because of the lack of understanding as to what actually constitute ‘plagiarism’. Although detailed, the above definition is not complete. It is evident that there are more expressions of plagiarism in more innocent terms – presenting slides for a lecture without copyright permission and plagiarism of illustrations are all glaring example 5.

In the previous era plagiarism could more easily escape detection. Today by the simple process of ‘searching’ a string of words in an electronic database makes plagiarism more obvious. Copying more than 30 letters, 7-10 words without a quotation mark and without proper citations in your articles can constitute plagiarism 6, 7
Deliberate Plagiarism amounts to cheating as it involves deliberately copying the work of another and presenting it as your own. However more common is the accidental plagiarism where the author fails to cite his reference sources completely and correctly.

The two most common forms of accidental plagiarisms are

1) Paraphrases with no citations.
2) Misplaced citations.

Any fact that is stated in the text of an article other than what is of ‘common knowledge’ requires a specific reference.

A paraphrase accurately states all the relevant information from a reference source in your own words (without using the authors words or his sentence structure). At the end of the paraphrase, the citation number should be included. Any quoted, paraphrased or summarized material that is presented after the citation number is plagiarized.

Here are certain tips to avoid accidental plagiarism

- Cite every piece of information (with the exception of the ones that are a result of your own research or is of common knowledge).
- Use quotation marks every time you use the author’s words.
- At the beginning of the first sentence in which you quote, paraphrase or summarizes another person’s work, make it clear that it is someone else’s idea.

According to Cleary and Ryan......
Charles Schepens says..............
In their study in 1972, Cleary and Ryan proved........

- At the end of the last sentence containing the quoted, paraphrased or summarized material insert a parenthetical citation to show where the material comes from.

Overt Plagiarism is considered worse than theft and amounts to academic suicide. It is not only worse than theft but even less clever as the stolen material is not concealed in a secret place, but, is kept exposed to the eyes of everyone. There may be reviewers and even readers who get the experience of the opportunity to read both the original and plagiarized article and to be sure it does give you a dé ja-vu phenomena!!

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Introduction: Keratoconus is a non-inflammatory ectatic corneal disorder of uncertain etiology which is usually bilateral and progressive. Progressive corneal thinning results in irregular astigmatism, myopia and later, conical protrusion of cornea when it is evident on clinical examination.

Prevalence and distribution: Among the various corneal ectasias, it is the commonest with a prevalence of 54.5 per 100,000 and an incidence of 1 in 2000 per year. The true incidence, as demonstrated by corneal topography however is likely to be within 1 in 600 to 1 in 420. It is bilateral in 96% of cases. Abortive form or ‘forme fruste keratoconus’ is often seen in the family members or the fellow eye.

Etiological factors: Despite extensive study, we are indeed far from understanding the exact underlying pathological mechanism of keratoconus. It is believed that corneal thinning may be due to defective formation or destruction of extracellular matrix and abnormal collagenase activity as evidenced by altered levels of fibronectin and type 4 collagen. Biochemical and immunohistological studies have shown increased levels of proteases and other catabolic enzymes in the basal epithelial cells of keratoconic eyes. Decreased levels of proteinase inhibitors, α1 proteinase inhibitor and α2 macroglobulin have also been noted in the corneal epithelium. Excessive eye rubbing or atopic eye disease might induce keratoconus by inducing epithelial damage. Epithelial stress can lead to increased keratocyte apoptosis through an interleukin-1 dependant mechanism and can cause changes in stromal matrix.

Over twenty publications are there in literature supporting the alteration in protease activity. Teng postulates that keratoconus is primarily a disease of the ectodermal layer of the cornea [epithelium] and corneal stroma is only secondarily affected when disruption of basal epithelial cells occurs. But this theory fails to explain the low recurrence rate after corneal transplantation.

In about 6–15% of patients with keratoconus, family history is present. High astigmatism, mildly irregular mires, inferior corneal steepening and substantial asymmetry in the central dioptric power between the two eyes are seen in family members of patients with keratoconus and have been suggested to represent variable forms of expression of the gene. Pedigree analysis in these families suggests an autosomal dominant mode of inheritance. However, discordance seen between monozygotic twins suggests highly variable expression. Though the genetic basis for most forms of keratoconus remain poorly defined, about seven loci have been mapped. In the keratoconic cornea, a possible genetic predisposition to increased sensitivity to apoptotic mediators by keratocytes has also been hypothesized.

Keratoconus is seen in association with other systemic and ocular diseases. Atopy and eye rubbing has long been associated. Harrison RJ et al, in a clinic-based study of 67 patients found that atopic disease, either present or past was found in 56.7% and further 11.9% had evidence of atopy in the form of highly elevated IgE without clinical disease. But keratoconus patients with or without atopy did not differ significantly with regard to sex, age of onset or rate of keratoplasty. They also found that atopy was more common in bilateral.
disease and keratoconus occurred more frequently on the side of the dominant hand.

Collaborative Longitudinal Evaluation of Keratoconus (CLEK) study found that at baseline, 53% of patients had a history of atopy. Rahi et al. also found a definite history of atopy in 35% compared with 12% in the matched control group in a large controlled study. Keratoconus is also associated with non-inflammatory connective tissue disorders like Ehler-Danlos syndrome and osteogenesis imperfecta. An increased prevalence of hypermobility of joints, mitral valve prolapse and false chordae tendinae in left ventricle has also been reported in keratoconus patients. Also there is association with Down’s syndrome, congenital hip dysplasia, floppy eye lid syndrome, Marfan’s syndrome, Turner’s syndrome and Apert’s syndrome.

Association has also been found with ocular pathology like retinitis pigmentosa and Leber’s congenital amaurosis, probably related to eye rubbing. Literature also describes association with Fuch’s endothelial dystrophy, posterior polymorphous dystrophy and stromal dystrophies like granular and lattice.

Among the various incriminated etiological factors and associations - atopy, age, sex, race, eye rubbing, mitral valve prolapse, handedness, collagen vascular disease, ocular trauma, pigmentary retinopathy, Marfan’s syndrome, Down’s syndrome and history of contact lens wear, Bawazeer AM et al. found that only eye rubbing is a significant predictor of keratoconus in a multivariate analysis. In the same case-control study comprising 120 subjects, in addition, atopy and family history of keratoconus showed an association in univariate analysis.

Pathogenesis: It is important to review the normal corneal structure for better understanding. X-ray diffraction studies have revealed that in the normal cornea, there exist significant differences between anterior one third and posterior two-thirds. In the posterior two-third, lamellae lie in the plane of the cornea, arranged in a parallel fashion and run without interruption from limbus to limbus with minimal interweaving between lamellae. The preferred direction of the posterior lamellae is in the inferior-superior or nasal-temporal direction while no such preference is seen in anterior stroma. At the limbus, collagen fibrils change their course to pursue a circular or pseudocircular course. In the anterior stroma, there is extensive anteroposterior interweave and a portion of lamellae that arise in the limbus insert into the region of Bowman’s membrane. This arrangement is believed to be essential for maintenance of corneal shape. Also, the anterior lamellae often split in both lateral and anterior-posterior direction, which may fuse with lamellae running in a different direction. The points at which lamellae split are potentially weak and rely on interfibrillar forces to maintain cohesion. In addition to anteroposterior interweave, there are other elements that bind collagen lamellae together. These are interactions between collagen fibrils and other matrix proteins like proteoglycans, type 6 collagen and keratoepithelin. The interfibrillar space also harbour keratocytes which interact with each other via long processes.

If this interfibrillar glue is weakened, collagen lamellae would have the tendency to tear apart with minimal trauma. Also this would result in displacement of lamellae and thinning of stromal tissue locally. The central and inferior portion is more likely to be affected as interlamellar strength is at a minimum in that area in a normal cornea. When it occurs in the anterior stroma, due to an ‘unknown primary event’, probably on exposure to proteases, or with minimal trauma or under genetic influence, lamellar sliding and redistribution of stromal mass occurs resulting in ectasia.

Collagen is arranged in lamellae of uniform diameter fibrils. It is formed from pro-collagen, consisting of three α chains with additional amino and carboxy terminal extensions (pro-α chains). Fibrils are constructed from aggregation of collagen molecules after cleaving off pro-collagen peptides. Collagen also undergoes a series of post-translational modifications one of which is the formation of cross-links via enzymatic oxidation of lysine and hydroxyl-lysine residues to their respective aldehydes. These aldehydes condense with other aldehydes or condense with lysine and hydroxyl-lysine residues to form intramolecular or intermolecular covalent cross-links between collagen peptide chains. In keratoconus there is reduced level of lysine hydroxylation and reduced cross linking involving hydroxyl-lysine. The stiffness of keratoconic cornea is only 60% of normal cornea and undergoes pepsin digestion twice as much due to decrease in
The new treatment modality, collagen cross linking with riboflavin and ultraviolet-A radiation stiffens the cornea and improves its biomechanical properties by restoring it to a good extent. The clinical features: Progressive loss of vision is the usual presenting complaint. Glare, aversion to light and monocular diploplia can also occur. High astigmatism with scissoring reflex on retinoscopy is typical. Prominent corneal nerves are seen frequently. The characteristic finding is an eccentrically located conical protrusion of the cornea. At the apex of the cone, cornea is thinner. Two types of cones are described – [1] round/nipple-shaped cone which is central in location and [2] oval/sagging cone which may extend to the limbus and is more prone for contact lens fitting problems. Fleischer’s ring, a partial or complete annular line seen at the level of epithelium marks the base of the cone and provides a landmark for the peripheral edge of the cone. The ring is formed from hemosiderin pigment deposited in the basal epithelium due to altering the conical protrusion. When faint it is better appreciated with Cobalt blue light by tangential illumination (Fig. 1). As the ectasia progresses the ring becomes narrower and prominent. Vogt’s striae are fine vertical posterior stromal folds found near the apex of the cone. They disappear on application of pressure to the globe. Fine linear anterior stromal scars, which develop within the cone due to rupture in Bowman’s layer may be seen. Subtle clear spaces in the anterior stroma have also been described. In more advanced cases deeper opacities can be seen at the apex of cone resulting from dehiscence in Descemet’s membrane (Fig. 2). Corneal hydrops result from stromal imbibition of aqueous through these defects, when sudden drop in vision occurs. Angulation of the lower lid in down gaze may be seen in advanced keratoconus and is called Munson’s sign. A conical reflection on nasal cornea, when light is shined from temporal side – Rizutti’s sign may also be seen. ‘Oil-drop reflex’ may be seen on distant direct ophthalmoscopy. None of the clinical findings may be present in early keratoconus. Inability to superimpose the central keratometric rings suggests irregular astigmatism as in keratoconus. Placido disk may show crowding of rings inferiorly which indicates corneal steepening. Corneal Topography: Rabinowitz and McDonnell developed algorithms for the detection of keratoconus based on 3 observations – I/S value [dioptric power difference between superior and inferior paracentral corneal region] > 1.9 D, central corneal power > 48.7 D and difference in progression of corneal steepening.
steepening between two eyes. The method yields a positive result for keratoconus suspect if I/S value is >1.4 D and central corneal power is >47.2 D. Maeda et al have devised a system for autodiagnosis of keratoconus called Klyce-Maeda software based on ten topographic indices with high sensitivity and specificity (Fig. 4).

With Orbscan Topography System, Auffarth et al found that tangential curvature [instantaneous curvature maps] provide better information about the morphology of keratoconus. Also apex of the cone and thinnest point were found to be located separately.

Histopathology: Thinning of corneal epithelium with degeneration of basal cells occur early. Disruption of basement membrane with epithelium growing posteriorly into Bowman's layer and stromal collagen growing anteriorly into the epithelium forming Z-shaped interruptions at the level of Bowman's layer, is typical of keratoconus.

Fleischer's ring, a hallmark of keratoconus is found at the base of the cone. Light and electron microscopy reveal that ferritin particles accumulate within and between the cells, particularly in the basal epithelium. Anterior clear spaces and breaks in Bowman's layer are also seen. Within the cone, collagen fibrils are normal-sized but the number of lamellae is abnormally low. By electron microscopy, FLS [Fibrous Long Spacing] collagen with a periodicity of 100 to 110 nm, in contrast to a periodicity of 60 to 64 nm found in normal collagen is seen within the area of corneal thinning.

Course: The onset of keratoconus occurs at about puberty which typically progresses over a period of 10 to 20 years after which it stops, though the degree can be highly variable. The rate of progression is also uneven and there can be periods of quiescence in between.

Collaborative Longitudinal Evaluation of Keratoconus (CLEK) study is a 8 year multi center observational study, largest to date, of 1209 keratoconus patients, followed up annually for 8 years. Its goals were to prospectively characterize changes in vision, corneal curvature, corneal status and vision specific quality of life. It found that over 7 years of follow-up, CLEK subjects showed a slow but clear increase in corneal curvature and drop in BCVA under high contrast and low contrast conditions more so in the latter. Also, keratoconus patients are generally RGP CL wearers with moderately steep corneas. Contact lens wear increased the risk of incident scarring in keratoconus more than two-fold. After controlling for disease severity in the form of corneal curvature, a keratoconic eye fitted with a RGP lens resulting in an apical touch fluorescein pattern did not have an increased risk of central scarring at base line. At baseline CLEK [at the onset of study] 13.5% had a family history of keratoconus and 53% had a history of atopy. The incidence of connective tissue diseases was not higher in keratoconic individuals. Younger age at baseline and poor high contrast BCVA at presentation predicted the rate of change in corneal curvature. Multivariate analyses of 5-year prospective data from the CLEK Study cohort showed that baseline corneal curvature, contact lens wear, corneal staining, and younger age were predictive of the development of corneal scarring.

Management: depends on the stage of the disease. The various modalities are

1) Spectacles
2) Contact lenses – RGP CL
   Scleral lenses – Boston lenses
3) Collagen cross-linking
4) Intra corneal rings
5) Keratoplasty procedures
6) Phakic IOLs
7) Refractive lens exchange
8) Combinations - Intracorneal rings + collagen cross-linking
Intracorneal rings + Phakic IOLs
PTK + Intracorneal rings + Collagen cross-linking

**Contact lenses** – Rigid Gas Permeable Contact Lenses [RGP-CL] correct irregular astigmatism produced by the abnormal corneal shape and significantly improves the best corrected visual acuity.

Keratoconus is typically managed by a variety of rigid contact lens fitting techniques and lens designs. The two most fundamental fitting techniques are apical corneal touch (including divided or three-point touch) and apical clearance. The information provided by corneal topography can help in selecting appropriate initial trial contact lenses.

A standardized keratoconus fitting protocol which was developed by the CLEK study can simplify contact lens management in patients with mild to moderate keratoconus. All contact lens parameter options are uniform except for base curve and secondary curve radii, which are determined by interpretation of fluorescein patterns. The initial trial lens's base curve is the average keratometric reading; sequentially steeper lenses are applied until definite apical clearance is observed. Despite the potential risk for corneal scarring imposed by flat-fitting [apical touch] rigid contact lenses, most patients wear flat-fitting lenses as was demonstrated in the CLEK Study.

Piggy back lenses [RGP-CL over a soft CL] can be used in patients who are uncomfortable with RGP wear, more so in those who are prone for epithelial erosion at the apex of cone.

Rose-K design RGP lenses are specially designed for keratoconic eyes with a diagnostic set comprising of 26 lenses with base curves ranging from 5.1 to 7.6 mm in 0.1mm increments, a standard lens diameter of 8.7 mm. It is among the most popular custom-made lenses for keratoconus and provides a better fit and visual performance. Jain AK and Sukhija J in a study of 38 eyes found that Rose-K design lenses are successful in visually rehabilitating 100 % of moderate and 96 % of severe keratoconus eyes. In their series on Indian eyes, most patients (90 %) maintained contact lens wear comfort. Also, corneal curvature on axial maps of videokeratography is a better predictor of base curve of final fit contact lens.

Soper lens is another custom made lens which has two zones in the central posterior curvature. The central zone is designed to vault steep central area and is of varying steepness dependent on the patient's cornea. The peripheral zone is always manufactured with a 45 D curvature designed to vault slightly the relatively normal midperiphery and limbal cornea.

**Boston Scleral Lens Prosthetic Device (BSLPD)** provides clear vision and comfort to most patients who are intolerant to traditional lenses especially in advanced keratoconus. It is a fluid ventilated scleral lens designed to enclose a bubble free reservoir of fluid over the corneal surface. A series of breaches created between the haptic bearing surface of the lens and the underlying sclera facilitates the aspiration of surface tears into the reservoir so that intrusion of air bubbles during blink is prevented. The shape of haptic confirms exactly to that of underlying sclera to maintain functionality and prevents intrusion of air bubbles. It has the disadvantage that it is highly expensive [ costs $ 5,000 for one and $ 7,600 for a pair ] and requires a time-consuming care regimen. It is considered an option before surgery for those who can afford it.

**Collagen cross-linking by Riboflavin and UV-A:** Collagen cross-linking consists of photopolymerisation of collagen fibres by the combined action of a photosensitizing substance (riboflavin or vitamin B2) and ultraviolet type A rays from a solid state UV-A source. Photopolymerisation increases the rigidity of corneal collagen and its resistance to ectasia.

Under topical anaesthesia, a 7 mm circle is marked on the cornea using a Thornton marker and epithelium of the marked area scraped off using a blunt spatula. A few drops of a solution containing freshly prepared 0.1 % riboflavin and 20 % dextran is put on the cornea and left in place for 5 minutes. UV lamp is then turned on, after making sure that it is focused on the apex of cornea at a distance of 10-12 mm, to obtain a radiant energy of 5.4 J/cm2 for 5 minutes. The lamp is then turned off, riboflavin-dextran solution again instilled, and 5 minute exposure repeated 5 times (total exposure 25 minutes, total treatment time 30 minutes). After treatment, eye is washed with Balanced Salt Solution, a drop of antibiotic and cycloplegic instilled and a Balanced Contact Lens applied.

After cross-linking, biomechanical studies have found an increase in corneal rigidity of 328.9 % in human...
corneas, mostly in the anterior stroma. There is an increase in collagen-fibre diameter and resistance to enzymatic digestion by collagenases. The UV-A light also produces apoptosis of ‘unhealthy’ activated keratocytes, in addition to being absorbed by riboflavin to strengthen the collagen. However, at 6 months repopulation of keratocytes occur in the anterior stroma. A mean reduction in Keratometry by 2.5D is noted after treatment. Though there are several studies which have found favourable outcome with this mode of treatment, Dresden Clinical Study has the maximum data base and longest follow-up. The 3 and 5-year results of the Dresden clinical study have shown that in all treated 60 eyes the progression of keratoconus was at least stopped. In 31 eyes there also was a slight reversal and flattening of the keratoconus by up to 2.87 diopters. Best corrected visual acuity improved slightly by 1.4 lines. So far, over 150 keratoconus patients have received crosslinking treatment in Dresden. Laboratory studies have revealed that the maximum effect of the treatment is in the anterior 300 mm of the cornea. As for the corneal endothelium, a cytotoxic level for endothelium was found to be 0.36 mW/cm which would be reached in human corneas with a stromal thickness of less than 400 microns, which signifies the importance of preoperative pachymetry. Collagen crosslinking has the potential to become the standard therapy for progressive keratoconus in the future, diminishing significantly the need for corneal transplantation.

**Intracorneal rings**: Intracorneal rings act as passive spacing agents which flatten the central cornea. These crescentic inserts made of PMMA placed circumferentially in the cornea were used initially for the treatment of low myopia. The amount of correction achieved is dependent on ring thickness, thicker the ring more the correction. On insertion they shorten the arc length of the anterior corneal surface, iron out gross irregularities and in effect create a ‘second limbus’. They are placed within the cornea at 65–70 % depth to lift the superior or inferior ectasia. Patients who are contact lens intolerant with central clear cornea [stages 2 and 3] are ideal candidates. Intracorneal rings namely Kerarings, INTACS and Ferrara rings, INTACS [Addition technology] has been most extensively studied.

In the pre-operative assessment, in addition to routine work-up, ultrasonic pachymetry at multiple locations and corneal topography is done preferably using Pentacam or Orbscan. After preparation as in routine anterior segment surgery under topical anesthesia, a small radial corneal incision (−1.0 mm in length) is made at 70 % depth, the outermost part of it, 1 mm central to the temporal limbus after ensuring centration. Two intrastromal tunnels (clockwise and counterclockwise) are created using specialized instruments. For INTACS insertion, tunneling is done after ensuring that vacuum is built up. The segments are then inserted and a suture is put. The selection of segment is based on standard nomograms. In a global or central cone two rings of same thickness are inserted while in an asymmetrical cone, one thin segment in the flatter area which is usually superior and one thicker segment in the steeper area which is usually inferior is done. In peripherally located cones, inferior segment alone may be more beneficial than 2 segments.

In the largest series by Colin J et al, a prospective study of 100 eyes with clear central corneas which underwent INTACS insertion found that, at two years UCVA and BCVA improved in 80.5 % and 68.3 % of eyes respectively. Preoperatively 22 % had a BCVA of 20/40 or better while postoperatively 53.7 % had the same. The mean keratometry readings decreased from 50.1±5.6 D preoperatively to 46.8±4.9 D at two years follow-up. Contact lens tolerance was restored in over 80 % of cases. In 4 eyes INTACS were removed without complications due to dissatisfaction with visual symptoms related to ring edges.

Keratoplasty procedures: As recipient pathology, keratoconus has the distinction of enjoying excellent results following penetrating keratoplasty. Kirkness et al \(^5\) reported 97% graft clarity at 4 years in 1990 and Beckingsale P et al \(^5\) reported 98% graft clarity at 5 years. Using same-sized trephine for both donor and recipient can reduce postoperative myopia in patients with highly myopic refraction \(^58\), \(^59\).

Anterior deep lamellar keratoplasty (DALK) has the advantage that it is an extra ocular procedure. There is less endothelial damage and minimal chance of rejection and so lesser steroid requirement. The globe is tectonically stronger and earlier stable refraction can be expected. In their large series of 181 eyes, Anwar M \(^60\) et al found that 89% achieved 20/40 or better vision, while only 10% achieved 20/20 at 6 months follow-up after Anterior Deep Lamellar Keratoplasty. Watson SL et al \(^61\) in a retrospective case-control study of 47 eyes, of which 27 underwent DLK and 25 underwent PK for keratoconus, found that at 55 months follow-up, mean BCVA was 6/6 in PK group while it was 6/9 in DLK group. Funnell CL \(^62\) also found similar results though two eyes in the PK group experienced rejection. Thus, with visual outcome almost similar to full thickness procedure, DALK has probably become the procedure of choice in young keratoconus patients as the risk of immunological rejection is minimal. However, this cannot be performed when there is a dehiscence in Descemet's membrane as in an eye which had hydrops.

Excimer laser assisted anterior lamellar keratoplasty uses excimer laser for ablating corneal stroma to the desired depth in both donor and recipient \(^63\).

Procedures like phakic IOL or refractive lens exchange could be considered for refractive correction once refractive stability is achieved. However ultrasound pachymetry can be inaccurate in up to one-third of cases \(^64\).

A variety of combination treatments are being tried, aimed at refractive correction, but with little long term follow-up. Combination of collagen cross linking and Intacs can give better results than Intacs insertion alone \(^65\). The same may be combined with phototherapeutic keratectomy in cases with prominent apical scarring. Similarly, phakic IOL may be combined with Intacs to better deal with ametropia seen in keratoconus \(^66\).

Conclusion

The last decade has witnessed revolutionary advances in the field of keratoconus. Intracorneal ring to a good extent is capable of dealing with gross corneal asymmetry, so that contact lens wear can be resumed, though not universally applicable. With the resurgence of Deep Anterior Lamellar Keratoplasty the risk of immunological rejection has become minimal, prompting surgeons to offer this option early so that patients with advanced disease have better long term visual outcome and better quality of life.

Years of painstaking basic research has found clinical application in the form of collagen cross linking using riboflavin and ultraviolet-A radiation, which is seen to arrest disease progression, at least for a few years and to some extent improve the mean keratometry values. If long term refractive stability is assured, the future is likely to see an explosive increase in refractive surgical procedures like phakic IOLs or refractive lens exchange.

References


Efficacy of Internal Limiting Membrane Removal in Recalcitrant Diabetic Macular Oedema - Comparative Analysis of Macular Thickness by Optical Coherence Tomography

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Abstract

Aim: To evaluate the efficacy of Internal limiting membrane peeling during pars plana vitrectomy for recalcitrant diabetic macular oedema on the macular thickness and final visual outcome.

Method: 30 eyes with recalcitrant diabetic macular oedema were studied. During parsplana vitrectomy, ILM peeling (ILM Peeled: group I) was performed in 15 eyes and ILM was not removed in 15 eyes (ILM Preserved: group II). Main outcome measures were assessed by Optical Coherence Tomography determined macular thickness reduction and visual acuity. Follow up periods were longer than 6 months in all cases.

Results: Reduction in retinal thickness was significantly higher in ILM peeled group (Mean reduction of 400 μm on 7th post operative day was present in 80% of group I patients and 40% of group II patients.) Improvement of Snellen’s visual acuity by one line was present in 60% of patients in group I and 40% of patients in group II.

Conclusion: These results indicate that removal of ILM contributes significantly to decrease in macular thickness and better functional outcomes.

Recalcitrant Diabetic Maculopathy is one of the most important causes for significant visual loss in a diabetic patient and is unresponsive to laser photocoagulation. In the presence of associated hypertension, hyperlipidemia and chronic renal dysfunction, the degree of fluid and hard exudate accumulation under the macula is aggravated leading to significant visual impairment. The degree of retinal thickening is gross, with accumulation of plaques of hard exudates under the fovea, making response to laser photocoagulation poor. The treatment options available in this situation are (1) use of intravitreal injection of Triamcinolone acetonide, (2) Pars plana vitrectomy with or without internal limiting membrane deroofing, (3) intravitreal injection of Anti VEGF agents and (4) various combinations of the above. Intra vitreal injection of Triamcinolone acetonide (TA) has been in use for several years now. Triamcinolone acetonide acts by decreasing the growth factor, stabilizing endothelial tight junctions and decreasing the permeability of water as well as solutes. Intravitreal injection of Triamcinolone is associated with complications such
as endophthalmitis (1.4 %), glaucoma (25–30 %), cataract (54 %), uveitis, retinal detachment and intraocular haemorrhage. Moreover the benefits achieved with this modality of therapy is transient. A visual improvement of 2.4 lines and a 55 % reduction in central macular thickness in 38 % of patients wears off with in 3 months of therapy.

Surgical options in the treatment of recalcitrant diabetic macular oedema includes pars plana vitrectomy combined with peeling of epiretinal membranes and or internal limiting membranes and removal of subretinal hard exudates.

Triamcinolone assisted pars plana vitrectomy is preferred by many surgeons as the TA helps delineate the posterior cortical vitreous, epiretinal membrane and the internal limiting membrane. The half life of Triamcinolone acetonide in the vitreous cavity in a vitrectomised eye is only 1-6 days. The small amount of Triamcinolone crystals sequestered in the vitreous cavity is not significant enough to cause a postoperative intraocular pressure spike, but may be just enough to prevent postoperative intraocular inflammation.

The concept of denuding the inner retinal surface of the internal limiting membrane promotes migration of cells, egress of extra cellular fluid and blood out of the retina and towards the vitreous cavity. Reduction in retinal thickening and improvement in oxygenation should theoretically improve the visual acuity.

The use of indocyanine green (ICG) to assist internal limiting membrane peeling has been associated with reports of retinal and optic nerve toxicity. The adverse effects of indocyanine green assisted internal limiting membrane peeling has been reported in 46.7% of subjects who developed slowly progressive onset of optic atrophy with in six months of undergoing surgery, associated with irreversible peripheral visual field defects predominantly in the nasal field. Other reports on vitrectomy with internal limiting membrane peeling for diabetic macular oedema or macular hole surgery does not show any intraoperative or postoperative complication attributed to the use of indocyanine green or any clinical or angiographic evidence of indocyanine green toxicity.

The present study aims to evaluate the efficacy of internal limiting membrane peeling during pars plana vitrectomy for recalcitrant diabetic macular oedema, on optical coherence tomographically determined macular thickness and final visual outcome.

**Materials and Methods**

A prospective randomised interventional study of 30 eyes with recalcitrant diabetic macular oedema was performed at our centre during a study period of 2005 January to 2006 August.

Eyes were included if they are unresponsive to conventional laser treatment defined as at least two focal laser application by a retinal specialist at least within or less than six months prior to enrollment into the study. Inclusion criterias were (1) diabetic age >10 years with tolerably good control (HbA1C <7), (2) presence of clinically significant macular oedema demonstrated clinically and angiographically, (3) clear media with absence of significant lens opacity, (4) post laser status with a history of atleast 2 focal lasers by a retinal specialist at ≥ 6 months prior to enrollment, (5) Stable lipid profile, (6) Absence of macular ischemia, (7) OCT evidence of central retinal thickness ≥ 500 μm and (8) Absence of evidence of vitreomacular traction on OCT.

Thus only eyes with recalcitrant diabetic macular oedema that have been refractory to standard laser treatment, without a taut posterior hyaloid on OCT were included in this study.

Patients with poor diabetic control, overt nephropathy, dyslipidemia, significant cataract and presence of macular ischaemia on FFA were excluded from the study.

Preoperative assessment of the patient included 1) Assessment of degree of diabetic control by HbA1C, 2) presence of associated hypertension, 3) Serum lipid profile, 4) Best corrected visual acuity, 5) Fluorescein angiography, 6) Optical coherence tomography of the macula and 7) Visual fields.

The patients were randomised to undergo parsplana vitrectomy with or without internal limiting membrane peeling. During vitrectomy the internal limiting membrane was peeled in 15 eyes (ILM Peeled Group I) while in the remaining 15 eyes ILM was not peeled (ILM preserved Group II). During vitrectomy an observation of whether a PVD was present or not, and
the degree of adherence of posterior cortical vitreous to the macula was noted. Triamcinolone acetonide was injected into the vitreous cavity (4 mg / 0.1 ml of preservative free Triamcinolone acetonide) to delineate the posterior cortical vitreous and aid in induction of a posterior vitreous detachment. Our technique of internal limiting membrane peeling was as follows: Dye assisted (ICG: 5 eyes and Trypan blue: 10 eyes) staining of the internal limiting membrane was carried out and internal limiting membrane peeling was initiated by creating a tear in internal limiting membrane in the inferotemporal area close to the disc. By the technique of maculorhexis internal limiting membrane peeling was completed and the membrane removed with intraocular forceps.

Intraoperative endolaser photocoagulation was carried out for eyes with proliferative diabetic retinopathy, however the macular area was left untreated in all the patients.

Postoperative follow up was carried out weekly for the first month and at monthly intervals for one year. At every follow up the best corrected visual acuity and OCT evaluation was performed. Fluorescein angiography was repeated after one month, at three months and twelvth month following the procedure. The degree of diabetic control, control of hyperlipidemia, and renal status were also monitored regularly at monthly intervals. Postoperative visual field charting was attempted in 15 eyes who underwent internal limiting membrane peeling.

Results

30 patients who were enrolled into the study were of the age group ranging from 45 years to 71 years (mean aged of 58 years) 14 were males and 16 were females giving a M: F ratio of 1:1.5. The diabetic ages of the patients ranged from 7 years to 20 years (mean duration of 13.5 years). The degree of diabetic control as assessed by measurement of glycosylated hemoglobin varied from 5.9 to 7.5 (mean 6.7). Associated co-morbid conditions included 1) hypertension alone in 8 patients, (2) hyperlipidemias in 7 patients, (3) Hypertension and hyperlipidemias in 5 patients, (4) Chronic renal failure in 3 and (5) no associated disease in 6 patients.

15 eyes were diagnosed as proliferative diabetic retinopathy (50 %) and 15 (50 %) had background diabetic retinopathy with maculopathy and recalcitrant diabetic macular oedema.

The preoperative best corrected visual acuity ranged from CF 2M to 6/36. 26 patients (86.6 %) had a preoperative visual acuity <6/60 while 4 eyes has a vision of 6/60 or above.

Preoperative Fluorescein angiographic findings included (1) Diffuse leak in all 30 eyes, (2) ERM in 1 eye, (3) increase in size of foveal avascular zone in one patient, (4) Cystoid Macular oedema 5 eyes and (5) Subfoveal serous RD in 4 eyes.

Preoperative OCT findings included (1) retinal thickness of 490 μm - 600 μm in 14 eyes, (2) 600-700 μm in 10 and (3) > 700 μm in 6 eyes. Cystoid Macular Oedema was present in 5 eyes and subfoveal serous RD in 4 eyes. Majority of the patients (21 eyes 70 %) had diffuse spongy type of retinal oedema. Presence of an epiretinal membrane was identified in one eye.

All patients underwent triamcinolone assisted vitrectomy where 0.5 ml of preservative free triamcinolone acetonide was used to delineate the posterior cortical vitreous and induce a posterior vitreous detachment. No eye had a spontaneous posterior vitreous detachment noticed intraoperatively. In all patients, the posterior hyaloid was found adherent to the macular area and the posterior vitreous detachment was induced by passive aspiration using a soft tipped flute needle. 15 eyes underwent denuding of the inner retinal surface by peeling of the internal limiting membrane. Dye assisted internal limiting

Fig. 1. Demonstrating reduction in macular oedema and resorption of hard exudates (by serial fundus photo and FFA) following vitrectomy with ILM Peel
membrane peeling was performed in all 15 eyes (Group I ILM peeled group) using indocyanine green to stain the internal limiting membrane in 6 eyes and trypan blue to stain the internal limiting membrane in 9 eyes. In the Group II eyes internal limiting membrane peeling was not attempted and this group of 15 eyes underwent a triamcinolone assisted vitrectomy alone.

Post operative visual acuity, intraocular pressure, optical coherence tomography, fluorescein angiography and visual fields were repeated at predetermined intervals. (Fig. 1).

Optical coherence tomography determined macular thickness analysis was performed after 1 month, on the 3rd and 12th months following the procedure. (Fig. 2 and 3)

The rate of reduction in retinal thickness varied between the two groups and a significant and dramatic reduction in retinal thickness was observed in eyes belonging to group I [(250 μm – 400 μm) vs (150 μm- 250 μm) in group II]. At each postoperative visit the best corrected visual acuity was checked. A slow but steady improvement in visual acuity was observed in group-I eyes. In comparison, the improvement in visual acuity in group II eyes was very slow and in 50 % of the patients the visual acuity stabilized at 6/60 by twelth postoperative month. (Fig. 4 and Fig. 5).

In both the groups, failure in visual improvement was noticeable in eyes with CME and sub foveal serous retinal detachment both of which persisted to a certain degree at 12 months follow up. Patients with higher Hb A1C at base line were found to have slower reduction in retinal thickness with a final retinal thickness varying between 290 μm and 360 μm pointing towards the need for continued rigorous diabetic control post operatively to sustain the good effects of the surgery.
Table 1. Details of study patients who underwent vitrectomy with or without ILM peeling for recalcitrant diabetic macular oedema

<table>
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<th>Sl No</th>
<th>Age / Sex</th>
<th>Duration of Diabetic Type of DR</th>
<th>Associated HT/HL</th>
<th>Hb A, C</th>
<th>Preoperative Demographic</th>
<th>TA+ PPV ILM Peel+1/</th>
<th>Post OP Demographics</th>
<th>OCT</th>
<th>Visual acuity</th>
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<td>58/F</td>
<td>20yrs</td>
<td>PKD</td>
<td></td>
<td>5.8</td>
<td>5.8</td>
<td></td>
<td>- 538 μm</td>
<td>423 μm</td>
</tr>
</tbody>
</table>
Fluorescein fundus angiography was performed after 1 month, 6th month and at 12th month following the procedure and showed progressively decreasing leak. However CME which was present in 5 eyes persisted at 12th postoperative month.

The fundus picture looked better steadily with progressively reducing retinal oedema, fragmentation and resorption of hard exudates.

Group I eyes (10 eyes), with visual acuity better than 6/60 underwent visual field charting on the 6th postoperative month. Analysis of visual field did not show any field defects in the nasal quadrant as observed in previous studies in patients in whom ICG assisted internal limiting membrane peeling was performed.

**Discussion**

The role of vitrectomy with ILM peeling in the management of eyes with diabetic macular oedema without a taut posterior hyaloid, refractory to standard laser treatment has been extensively studied. Most of these studies (Table 1) have shown that the results of parsplana vitrectomy with internal limiting membrane peeling lead to resolution of DME, but was not always associated with visual improvement. In diabetic eyes, CME and subfoveal serous retinal detachment were poor prognostic indicators for visual recovery.

Various other groups of workers have conclusively shown that pars plana vitrectomy with ILM peeling leads to expedited resolution of diffuse diabetic macular oedema and visual improvement without subsequent epiretinal membrane formation. Quantitative assessment of OCT images at the end of follow up revealed that retinal thickness in the macula appeared nearly normal with or without reappearance of foveal pit in 73.3%. The effects of resolution of macular oedema, reduction in retinal thickness and improved visual function were longer lasting than following intravitreal triamcinolone acetonide injection. The results of our series of 30 patients with recalcitrant DME also shows excellent outcomes with internal limiting membrane peeling. However contrary to other studies which have demonstrated slowly developing optic atrophy in 46.5% of eyes following use of indocyanine green for internal limiting membrane peeling, our study did not reveal any intraoperative or post operative complication attributable to the use of indocyanine green or any clinical or anaigraphic evidence of indocyanine green toxicity.

The visual improvement following parsplana vitrectomy with internal limiting membrane peel was gradual and occurred within 6 months to one year following the surgery.

**References**


Intravitreal Bevacizumab in Diabetic Retinopathy

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Abstract

To study the effect of Intravitreal Bevacizumab in proliferative diabetic retinopathy (PDR), the worse eye of 44 cases of diabetic retinopathy underwent intravitreal bevacizumab injection. Focal macular laser or PRP was performed when required for this eye and the fellow eye. Visual acuity, Optical coherence tomography and fluorescein angiographic changes over 9 months were studied. The results were compared with the fellow eye, which underwent laser treatment only.

An improvement in visual acuity and reduction in angiographic leakage were observed in 39 eyes at 9 months follow up. Serial OCT follow up showed decrease of edema in 32 cases of clinically significant macular oedema.

Bevacizumab is very useful and safe as an adjuvant in controlling neovascularisation and macular edema in diabetic retinopathy.

Introduction

Advanced proliferative diabetic retinopathy (PDR) is one of the most important causes of loss of vision in the diabetic patient. Advances in vitreoretinal surgical techniques including laser photocoagulation and vitrectomy systems like wide-angle visualization and silicone oil injections have no doubt increased the results in tractional and combined rhegmatogenous retinal detachments. Different treatment methods, including the perioperative use of heparin and 5-fluorouracil in irrigating fluids during pars plana vitrectomy (PPV), have been tried in an attempt to reduce the incidence of PVR in high-risk proliferative states, but with only limited success. However these have not prevented the occurrence of blindness and these treatment modalities require technology and infrastructure that may not be available or accessible to millions of diabetics living in our subcontinent.

An inflammatory component in retinal neovascular proliferation in PDR and proliferative vitreoretinopathy (PVR) was noted. Machemer and other researchers showed that intravitreal steroid injections, particularly triamcinolone acetonide, were not toxic to the eye and may potentially be important in reducing the intraocular inflammation and vitreoretinopathy caused by fibroblastic proliferation. It is therefore intuitive that intravitreal steroid injections could be beneficial both in PDR with TRD and in PVR. Search for newer agents like targeted monoclonal antibodies were always on.

Bevacizumab targets and blocks vascular endothelial growth factor (VEGF) and VEGF’s binding to its receptor on the vascular endothelium. Since VEGF is released by many cancers to stimulate proliferation of new blood
vessels, the combination of bevacizumab and chemotherapy was found to increase objective responses, median time to progression, and survival in patients with metastatic colorectal cancer, compared with chemotherapy alone. The ocular use of this drug has a potential in all the proliferative retinopathies that express VEGF. So it is now being increasingly used in proliferative diabetic retinopathies as well as in many other diseases like vascular blocks and age related macular degeneration. Laser photocoagulation has a lot of disadvantages including decreased visual acuity, reduced contrast sensitivity and constricted fields. Our attempt in this study is to underline the effect of chemotherapeutic agents in PDR.

Methodology

In a prospective, nonrandomized, interventional case series, 44 eyes of 44 diabetic individuals were diagnosed with proliferative diabetic retinopathy. All patients received a detailed counseling of the study design and aims, and were provided with written informed consent. All patients were evaluated and treated by a single retina specialist. Inclusion criteria were patients with type II diabetes, PDR, visual acuity loss, and neovascular leakage shown by fluorescein angiography (Zeiss FF 450 plus, Germany). Eyes with history of glaucoma, cataract extraction, or other intraocular surgery were excluded from the study. Eyes with an epiretinal membrane, posterior hyaloid traction, ischemic maculopathy, and diabetic papillopathy were also excluded. The risks and benefits of the procedure were discussed with each patient before injection, and all patients were provided written informed consent. Baseline parameters were documented including best-corrected visual acuity, central macular thickness, intraocular pressure (IOP) and lenticular status. The best-corrected visual acuity was determined from the Early Treatment Diabetic Retinopathy Study (ETDRS) chart and calculated as logarithm of minimal angle of resolution (logMAR). Central macular thickness was measured by optical coherence tomography (OCT4, Zeiss). Optical coherence tomography (OCT) was performed by acquiring six radial scans, 5 mm long, centered on the fovea, and then analyzed with retinal map protocol. Intraocular pressure was measured by applanation tonometer. Care was taken to see that systemic conditions of the patients were under control (blood glucose, blood pressure, and general condition), and they were receiving oral hypoglycemic agents or insulin for glycemic control.

All patients underwent fluorescein angiography and optical coherence tomography and the eye with more angiographic leakage or vitreous hemorrhage was designated to the bevacizumab arm of the study. These patients were informed regarding the drug and its side effects; and an informed consent was obtained from all of them. The worse eye received bevacizumab intravitreal injection and panretinal photocoagulation, while the better eye received panretinal photocoagulation only.

All 44 eyes meeting these eligibility criteria received a single intravitreal injection containing 1.25 mg of commercially available bevacizumab in 0.05 mL. Data analyzed was collected for each patient at the preoperative visit and at 1 month, and 9 months post injection. A total of 44 eyes (28 right eyes, 16 left eyes) were identified, of 44 patients (27 males, 17 females). The mean age was 52.3 years (range of 22–72 years) and the mean follow-up time was 10.3 months (range of 9–12 months). All operations were performed by a single surgeon (G.S.P.) using the same surgical techniques. An unaltered, commercially available 0.05-mL solution containing 1.25 mg of bevacizumab was injected into all eyes through a sclerotomy towards the 6 o’clock position. Subconjunctival injections of antibiotics and steroids was also given in all patients. The outcome measures were studied at 1 month and 9 months, which included visual acuity, angiographic leakage on FFA, retinal thickness on OCT and any side effect profile that we thought to be due to intravitreal bevacizumab.

Angiographic leakage was graded as absent (grade1), present mildly (grade2) and gross leakage (grade3). Central macular thickness was used for statistical analysis in the OCT.

Results

All 44 patients had a diagnosis of proliferative diabetic retinopathy. 25 patients had coexisting hypertension. Nineteen eyes had some areas of vitreous hemorrhage to start with. Seven eyes were pseudophakic prior to
this intervention, 4 eyes had cataract, and 33 eyes had clear lenses.

None of the injections were associated with any significant pain or morbidity; there was mild subconjunctival hemorrhage in 6 patients, which resolved within a week. The IOP was normal on the first postoperative day for all patients.

The mean logMAR visual acuity significantly improved from 0.8 ± 0.16 at baseline to 0.6 ± 0.14 at 1 week, 0.6 ± 0.13 at 1 month, and 0.6 ± 0.11 at 9 months in the bevacizumab arm of the study. (p<0.05)

In the other eye, the mean visual acuity remained stabilized without any statistical change from 0.5 ± 0.13 to 0.5 ± 0.13 at 1 month, and 0.5 ± 0.14 at 9 months. (p=0.95)

Angiographic leakage reduced significantly in all patients who underwent the bevacizumab injection from grade 3 to grade 1 at the end of 1 month, but increased to grade 2 by the end of the study. In the PRP arm, the angiographic leakage reduced from grade 3 to grade 2 in 23 eyes, but in all the others the leakage persisted at grade 3. (Fig. 1 a & b).

OCT thickness reduced significantly by 112 ± 36 microns in all patients who underwent bevacizumab (Fig. 2) injection at the end of 1 month and by 136± 23 by the end of the study. (p<0.05). (Fig. 2 a & b).

OCT thickness had stabilized without statistical change in the PRP arm of the study. The change in macular thickness at 1 month was 21 ± 17 microns and at 9 months was 27 ± 21 microns (p=0.98)
No side effects were noted in any of the patients receiving bevacizumab.

The mean baseline IOP was $14.7 \pm 2.0$ mmHg (range 12–18 mmHg), and at 1 week, 1 month, and 9 months after injection were $15.9 \pm 2.1$ mmHg (range 12–20), $15.7 \pm 2.4$ mmHg (range 12–20), and $15.3 \pm 2.3$ mmHg (range 12–18) respectively.

**Discussion**

Bevacizumab is a monoclonal antibody directed against vascular endothelial growth factor (VEGF) initially described for metastatic colorectal cancer. VEGF is implicated in the genesis of neovascularisation in the retina in almost all vasculopathies of the retina including diabetic retinopathy, age related macular degeneration etc. Since bevacizumab was found to give good results in these cases in the eye, it has been used for a variety of retinal diseases.

The visual acuity in all eyes, which underwent bevacizumab injection, showed a significant increase. The angiographic leakage disappeared to almost negligible levels by the end of one month and remained like that through out the study. Taking into consideration the fact that the worse affected eye was enrolled into the study it is of importance to note that none of the 44 eyes, which received bevacizumab, needed to be operated upon for vitreous hemorrhage or traction retinal detachment.

Macular edema subsided significantly with bevacizumab. A change of central macular thickness of 112 microns was documented by the first month and that drop in thickness increased to 136 microns by the end of the study. There was no statistical change of the macular thickness in the PRP arm of the study. Thus macular edema occurring in PDR in this study was taken care of by bevacizumab.

Bevacizumab is a versatile monoclonal antibody, which may be complimentary to laser photocoagulation and surgery in advanced cases of proliferative diabetic retinopathy. The ease of the injection procedure and the availability and affordability makes it a very useful drug in the management of diabetic retinopathy.

The other anti VEGF agents used in the eye are pegaptanib sodium and ranibizumab, both of which have undergone large multicentered clinical trials in the management of age related macular degeneration. Clinical trials to evaluate whether their use in diabetic retinopathy can give better results than bevacizumab may be of great help. Till then, bevacizumab will remain a significant barrier between diabetic retinopathy and blindness.

Advances in nano medical and pharmaceutical technology is making definite steps to improve our surgical results in diabetic retinopathy and in many other ocular diseases.

**References**

Peribulbar Anaesthesia For Vitreoretinal Procedures

Dr. Savita Bhat MS, Dr. Mahesh Gopalakrishnan FRCS Ed, Dr. Pushpa Isaac DA, Dr. Anantharaman Giridhar MS

Abstract

Purpose: To evaluate patient comfort and experience during vitreoretinal (VR) surgery under modified peribulbar anaesthesia.

Methods: 141 patients who underwent VR surgery under peribulbar anaesthesia were included in the study. Pain was assessed using the visual analog scale.

Results: Akinesia was achieved in 130 (92%) patients. Average duration of procedure was 91.79+/-.25.37 minutes. 90% patients claimed to be comfortable throughout the procedure. 62.4% patients experienced pain lasting for less than 5 minutes. 86% of patients preferred the same anaesthesia if needed in future.

Conclusion: Peribulbar anaesthesia can be used comfortably and effectively in long duration VR surgeries.

Introduction

Primary vitreoretinal procedures (VR) are performed under local anaesthesia (LA), general anaesthesia (GA), or a combination of both. The long hours of surgery required for VR procedures necessitates GA. Several studies in the past have proven the efficacy of and safety of local anaesthesia in VR procedures, viz, both peribulbar and retrobulbar anaesthesia. New anaesthetics such as ropivacaine, etidocaine, mepivacaine and modified anaesthetic agents have been tried in a number of case series with the intention to reduce toxicity and prolong duration of anaesthesia in VR procedures. Long duration of anaesthesia can be achieved by supplementation using sub-tenon’s or indwelling catheter technique. Different routes of administration has been advocated to achieve adequate anaesthesia during these procedures. We evaluated the efficacy of the anaesthetic drug viz Lidocaine with bupivacaine in VR surgeries. Also, we studied the patient’s comfort and experience following VR procedure under peribulbar anaesthesia with supplemental subtenon’s – modified peribulbar anaesthesia.

Materials and Methods

This was a prospective, non-comparative, hospital based study conducted between March 2004 to May 2005. A total of 141 VR surgeries were included and 101 vitrectomies and 40 scleral buckling procedures were performed. The exclusion criteria included vitreoretinal surgery done in the same or the fellow eye, active ocular infection, mental retardation in patient’s refusing local anaesthesia, acute respiratory disease, when the decubitus position was impossible and when the surgeon expected prolonged surgery (more than 3 hrs). All patients were subjected to a detailed pre-anaesthetic
work up including laboratory investigations. The weight, blood sugar, blood pressure and electrocardiogram were evaluated for all patients. All patients received pre operative medications, viz Ketorolac trometamol 10mg oral, Alprazolam 0.25 mg oral, Cefuroxime 750 mg intravenous one hour before surgery. All surgeries were performed in the presence of an anaesthesiologist. A short-acting drug for anxiolysis Midazolam 0.5 or 1 mg was administered 5 minutes before peribulbar anesthesia in 18 patients who seemed very anxious. Assessment of anxiety level was completely subjective because we did not use any scale. The administration depended on the judgment of the anaesthesiologist. In addition, few drugs were given during surgery to selected patients as shown in Table 1. Peribulbar anaesthetic injection was performed using Hamilton’s technique by two surgeons experienced in that technique. The first percutaneous insertion of the needle (24 gauge, 35 mm long) was parallel to the orbital floor at the lateral aspect of the inferior orbital rim (maximal depth 25 mm), and the second (maximal depth 25 mm) at the level of the medial periconal site. The injection was immediately stopped when the globe became subjectively tense. As soon as the globe became soft, the injection was started again until the globe was again tense. A mixed anaesthetic solution of equal quantity (1:1) of plain lidocaine 2 % with hyaluronidase (1:1500) and bupivacaine 0.5 % with hyaluronidase (1:1500) was administered in all patients. An ocular cuff applied a pressure of 30 mm Hg for 15 minutes. The surgeon assessed akinesia and analgesia 15 minutes later. Analgesia was considered to be perfect when the patient did not notice any pain on holding bulbar conjunctiva and lateral rectus muscle insertion. Akinesia was considered perfect when no movement was observed in all cardinal directions of gaze. Patients were encouraged to inform the surgeon if they experienced pain during the surgery. Supplemental lidocaine 2 % (5-8 ml) was administered when needed by sub-Tenon infiltration by the operating surgeon. A buttonhole was fashioned through the conjunctiva and the Tenon’s capsule 10 mm posterior to the limbus in the temporal superior quadrant. The lidocaine was then delivered to the posterior sub-Tenon space using a blunt cannula. Supplemental injections were given only to patients who failed to develop analgesia.

Analgesia was graded during the procedure by the surgeon as follows: grade 1-adequate analgesia throughout surgery without any supplementation; grade 2-adequate analgesia with two sub-tenon injection; and grade 3-inadequate analgesia despite more than two sub-tenon injection.

Analgesia was assessed subjectively using the Visual Analog Scale (VAS). Patients were asked to rate their pain with the VAS. The VAS consisted of a 10-cm line anchored by two extremes of pain (Figure 1). Patients were asked to make a mark on the line that represented their level of perceived pain intensity. The intensity on a numerical scale from 0 to 10, with the zero representing one extreme (e.g. no pain) and the 10 representing the other extreme (e.g. “the worst pain possible”). Akinesia was considered as follows: complete - no eye movement in all directions; mild some movements in one or several directions; and absent - complete mobility in all directions. All patients received nasal oxygen throughout the procedure and were supervised by cardiac monitoring and pulse oxymeter. The other recorded variables were vital signs pulse, respiration and blood pressure before the onset of surgery, during and after the surgery. Also, volume of anaesthetic used, ocular and general complications and duration of surgery were recorded. All patients received the following drugs after the procedure, viz intravenous Ketorolac Trometamol 30mg and Cefuroxime 750 mg every 12 hourly. A questionnaire was presented to all the patients 12 to 24 hours after the surgery and their responses were noted and tabulated. Each patient was also interviewed individually.

Results

The mean age of patients (male 48 % and female 51%) was $58.78 \pm 7.07$ years (range 29-87 years). All 141 surgical procedures are listed in Figure 2.

Local anaesthesia

The average dose of Lignocaine 2% given was $139.9 \pm 46.8$ mg. It exceeded the safe permissible limits in 36 (25.3 %) patients. The average dose of bupivacaine 0.5% given was $31.7 \pm 11.9$ mg. It did not exceed safe permissible limits in any patient. The absolute volume of the anaesthetic mixture was $12.9$ ml $\pm 2.9$ (range 7 - 24 ml). Obviously the volume related to body weight.
was 0.10ml/kg ± 0.04. The volume seems high but the concentration of bupivacaine (0.5 %) is lower than that used in the other countries (0.75 %). The efficacy of a drug is due to its concentration so if the concentration decreases, the volume must be increased to obtain the same effect. The mixture was made up of an equal quantity of bupivacaine 0.5 % and lidocaine 2 %. Supplementary Sub-Tenon infiltration was needed in 29 (20.5 %) patients for analgesia.

**Analgesia and Akinesia**

Efficacy of analgesia and akinesia are shown in Table 2. Adequate analgesia was obtained with peribulbar anaesthesia alone in 79 % of patients 15 minutes after injection and with a sub-Tenon infiltration in 97 %. A total of 29 patients (20.54 %) required additional sub tenon’s infiltration. But four patients experienced mild pain throughout the surgery in spite of a sub-Tenon infiltration. Surgeons observed that the pain during cryotherapy for scleral buckling procedure often necessitated supplemental anaesthesia. Movement following peribulbar injection was observed in 6 % and akinesia was totally absent in 1 % of the patients. Complete akinesia was obtained in 94 % of patients.

**Surgery**

The duration of surgery was 91.79 ± 25.37 minutes (Range 50–165). The shortest duration was for vitrectomy done for Terson’s syndrome and the longest was for rhegmatogenous retinal detachment in a myope undergoing vitrectomy with belt buckling, cryotherapy, endolaser, fluid gas exchange and silicone oil injection.

**Complications**

None of the patients required conversion to general anaesthesia. Neurological complications were not encountered in this series. In 9 patients the systolic blood pressure during procedure was recorded to be equal to or more than 180 mm of Hg. There was no incidence of ocular complications such as retinal ischaemia or optic nerve neuropathy.

**The patient’s perspective**

According to the responses to the questionnaire, 127 (90 %) claimed to be comfortable during the surgery. Nine patients could even sleep during the procedure. However, some patients complained of discomfort and the various reasons are tabulated (Table 3). Some (16 patients) complained of other causes such as backache, decubitus, neck pain as a reason for discomfort. The stage during the vitreoretinal surgery at which pain was experienced by the patient and duration is shown in Table 4a and 4b. Only one patient graded pain intensity to be the worst ever experienced. A total of 86 (61 %) patients said the duration of surgery were as they had anticipated. However, 44 (31.2 %) felt it was longer and 11(7.8 %) felt it lasted shorter than their anticipation. A total of 121 (86 %) patients said they would prefer the same anaesthesia if required for a repeat procedure in the same or other eye. Amongst the patients preferring general anaesthesia for a future procedure, the reasons quoted were anxiety in 7 patients, pain in 3, posture in 2 and discomfort in 3. Majority (94 %) of the patients claimed they would recommend this procedure to other patients undergoing a similar surgery.

**Discussion**

Peribulbar anaesthesia has increased in popularity because it provides the same anaesthetic effect as a retrobulbar injection, but with a lower rate of complications. For posterior segment surgery, it appears to be necessary to choose an anesthetic mixture and a sufficient volume for the most potent and prolonged effect. In our study a combination of Lidocaine, bupivacaine and hyaluronidase was used. Though many new and modified drugs have been tried for anaesthesia, we used the most popular combination of lidocaine and bupivacaine which produces faster onset than bupivacaine with etidocaine. Hyaluronidase is known to improve onset time and akinesia of extraocular muscles. We injected high volume 12.9 ml ± 2.9 (range 7-24 ml). This was much higher than in many previous studies of peribulbar used for other anterior segment surgery. However, as a precaution we injected the mixture very slowly and in several stages. As the injection is stopped when the globe becomes tense, the volume of anaesthetic depends on the size of the orbit and differs from one patient to another. Adequate analgesia was obtained with peribulbar anaesthesia alone in 79 % of patients and with a sub-Tenon infiltration in 97 %. Complete akinesia was
obtained in 94% of patients. Sub tenon's infiltration helped to achieve this in 20%. Candela et al had re-injection of rates of 15% in a series of 300 cases. They concluded that the reinjection rate required is more than that for cataract surgery. Analgesia failed in 4 patients (3%) and akinesia in 1 patient (1%). In this study the block failure rate was 3%. Nicholson et al reported 21% in a series of 33 patients. Demediuk reported a failure rate of 33%3. Sharma et al reported block failure rate in their series of 100 cases as 31%13. We attribute our success rate to the higher volume of anaesthetic used compared to these studies. Sharma et al used 1.5–2 ml in Subtenons space and Demediuk et al 5 ml in peribulbar space. Our block failure rates our comparable to the series, reported by Candela et al who reported 1% block failure with usage of high volume of anaesthetic (17 ± 4.5 ml)10. Pain during cryo therapy was noticed to be the most frequent time for supplementation of anaesthetic agents in our series which is similar to that observed by Sharma et al13. The same authors have also advised repetition of block after approximately 70 to 90 min to prevent pain. The average duration of surgery was 92 minutes (range 50-165). In one patient the procedure took 165 minutes, but the patient did not complain of pain and claimed to be comfortable when interviewed later in the study. This was possibly because the patient was young (Age 41 years) and with no other systemic illness.

None of the patients in this series required conversion to general anaesthesia. Nine patients (6.3%) had high systolic pressure during the procedure, which could be controlled with drugs. There was no inadequate block of the oculocardiac reflex. Severe systemic effects such as this has been reported previously by other authors20. We did not encounter any ocular or serious systemic complications related to the block despite usage of high volume. Higher volume of the mixture is supposed to be responsible for severe complications although not proven10.

The efficacy of peribulbar block was studied in this series before presenting the questionnaire to the patient. The efficacy and safety in this series is comparable to that in other vitreoretinal procedure10,13,17. Ninety percent of patients in this series claimed to be comfortable during the surgery. Thirty five percent experienced pain and only one patient graded pain intensity to be the worst ever experienced on the visual analog scale. A total of 86 (61%) patients said the duration of surgery was as they had anticipated. However, 44 (31.2%) felt it was longer than their anticipation. In this series, 86% said they would prefer the same anaesthesia if needed for a repeat procedure in the same or other eye. This indicates the patient satisfaction with the procedure under local anaesthesia. However, few patients preferred general anaesthesia for a future procedure, the most often quoted reason was anxiety. This could largely be overcome by adequate counseling before the procedure and also appropriate pre-medication for every individual. Majority (94%) of the patients claimed they would recommend this procedure to other patients undergoing a similar surgery. Several authors in the past have studied efficacy of different local anaesthetics for vitreoretinal procedures2-4, 10. Few studies have evaluated patient reaction21,22. Newsom et al, in their large series of 1221 patients studied patient satisfaction by pain as the yardstick21. Knight et al measured the perioperative pain score and in their series 97% said they would choose local anaesthesia again as compared to our 86%.

### Conclusion

Modified peribulbar anaesthesia is effective and safe for vitreoretinal procedures, which needs more time for completion. Most of the patients are comfortable throughout the surgery. This can be used in primary vitreoretinal surgeries and also in those with contraindication to general anaesthesia.

#### Table 1. Additional intravenous per-operative medications administered to selected patients

<table>
<thead>
<tr>
<th>Generic name (Manufacturer)</th>
<th>No. of patients</th>
<th>Dosage (mg)</th>
<th>Purpose</th>
</tr>
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<tbody>
<tr>
<td>Midzolam (Manufacturer)</td>
<td>18</td>
<td>0.5-1</td>
<td>Anxiolysis</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>4</td>
<td>20-40</td>
<td>Anti-arrhythmia</td>
</tr>
<tr>
<td>Atropine sulphate</td>
<td>2</td>
<td>0.15</td>
<td>Correct bradycardia</td>
</tr>
<tr>
<td>Theophylline</td>
<td>2</td>
<td>200</td>
<td>Bronchodilatation</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>1</td>
<td>8</td>
<td>Antiemetic</td>
</tr>
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</table>

![Fig. 1. VAS (Visual Analog Scale)](image)
Table 2. Efficacy of analgesia and akinesia.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Analgesia</th>
<th>Akinesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>112 (79.43)</td>
<td>133 (94.32)</td>
</tr>
<tr>
<td>II</td>
<td>25 (17.73)</td>
<td>7 (4.96)</td>
</tr>
<tr>
<td>III</td>
<td>4 (2.83)</td>
<td>2 (1.41)</td>
</tr>
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</table>

Table 3. Cause of discomfiture in patients during VR procedure under LA

<table>
<thead>
<tr>
<th>Cause</th>
<th>Number of patients( % )</th>
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<tbody>
<tr>
<td>Pain</td>
<td>50 (35.46)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>16 (11.34)</td>
</tr>
<tr>
<td>Drapes during surgery</td>
<td>6 (4.25)</td>
</tr>
<tr>
<td>Others</td>
<td>16 (11.34)</td>
</tr>
</tbody>
</table>

Table 4a. Stage at which patient experienced pain

<table>
<thead>
<tr>
<th>Time period</th>
<th>Number of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>During local anaesthesia</td>
<td>73</td>
</tr>
<tr>
<td>During Procedure</td>
<td>15</td>
</tr>
<tr>
<td>After the procedure</td>
<td>1</td>
</tr>
<tr>
<td>During anaesthesia and procedure</td>
<td>4</td>
</tr>
<tr>
<td>During anaesthesia and after surgery</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 4b. Duration of pain

<table>
<thead>
<tr>
<th>Duration</th>
<th>Number of patients (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Throughout the procedure</td>
<td>80 (56.73)</td>
</tr>
<tr>
<td>Short (&lt; 5 minutes)</td>
<td>7 (4.96)</td>
</tr>
<tr>
<td>Long (&gt; 5 minutes)</td>
<td>4(2.83)</td>
</tr>
</tbody>
</table>

References

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Comparison Of Results Of Optic Disc Analysis Using Stereoscopic Biomicroscopy, Stereo Fundus Photography and Optical Coherence Tomography

Prof. [Dr.] Meenakshi Dhar, Dr. Indu Jayachandran, Dr. Biju Raju, Ms. Deepa PA

Abstract
Aim – To compare the results of optic disc analysis using optical coherence tomography, fundus photography and stereoscopic biomicroscopy.

Materials & Methods – A comparative, observational case series and interobserver variability study in which 2 groups of patients were analysed by all the 3 methods. One group had primary open angle glaucoma patients and the other group were patients undergoing retinal evaluation. Stereo fundus photography and stereo biomicroscopy were performed using 90D lens while optical coherence tomography was performed using stratus OCT after dilating the pupil.

Results – 50% cases showed good correlation between all the 3 methods. Optical coherence tomography showed a higher value indicating the requirement to do optical coherence tomography in all patients to detect the actual cup disc ratio which will help us to detect glaucoma cases earlier and to treat them, well before axonal loss occurs. The optic disc analysis results were confirmed by two people who assessed the optic disc separately to avoid the inter observer bias.

Key words: Cup-disc [CD]ratio, Vertical CD Ratio [VCDR], Optical Coherence Tomography [OCT], Slitlamp biomicroscopy, glaucomatous optic neuropathy.

Introduction
Optic nerve head evaluation is the hallmark of glaucoma diagnosis. Structural alteration of optic nerve head precedes the development of reproducible glaucomatous visual field defects 1. Retinal ganglion cell population at any retinal locus may be reduced by 50% before this can be detected using conventional perimetric techniques 2. Therefore identification of these changes is important in early diagnosis of glaucoma.

Fundus biomicroscopy enables more accurate, less variable Vertical Cup Disc Ratio (VCDR) estimation than direct ophthalmoscopy 3. Until recently clinical assessment of optic nerve head with biomicroscopy has been the standard practice for diagnosis of glaucomatous optic neuropathy. It varied between experienced observers and subtle changes over time are difficult to detect clinically. It is best done stereoscopically either by the indirect method with +78 or +90 D lens, the latter being more popular or with contact lenses like the Central part of Goldmann or Zeiss 4 mirror gonioscope.
Monochromatic photographs of retinal nerve fibre layer using the red free light have been also used to detect the retinal nerve fibre loss. Digital photography of the optic nerve head also is useful. Various methods to quantitatively and objectively assess the optic nerve head have evolved in the last decade for e.g. confocal scanning laser ophthalmoscopy (CSLO), scanning laser polarimetry (SLP), optical coherence tomography (OCT) and stereoscopic colour photographs (ONHPs).

In this study we are comparing the optic nerve head evaluation by stereoscopic colour photographs (ONHPs), stereo biomicroscopy (SBM) and optical coherence tomography to find the accuracy, reliability and usefulness of each in glaucoma practice. Optical coherence tomography has wider uses in retinal diseases as well, and is more likely to be available in a clinic than the others which will be available in institutional practices, thus has been chosen as the quantitative method for optic nerve head evaluation in the study.

As of now although the measurements of the various optic nerve head parameters is available but normative data has not been made available in the Stratus IV optical coherence tomography, but the normative data for retinal nerve fibre layer measurements is available.

Stereophotography uses a specialized camera to take a pair of photographs of the optic nerve, which when seen with a specialized viewer, create a three dimensional (3-D) image. It may be slightly modified to take make it easier to see the optic nerve fibers.

OCT is a non-contact, noninvasive diagnostic technique showing a cross sectional living histology of retina. It is a high resolution reproducible imaging technology, which is increasingly being used in evaluation of glaucoma patients. It uses low coherence near infrared light (850 nm) from a super luminescent diode and subsequent back scattering of the retina. It generates, posterior segment measurements with an axial resolution of 8-10 microns.

**Patients and Methods**

This is a prospective observational study conducted at Amrita Institute of Medical Sciences wherein we compared two groups of patients. **Group I** included patients of primary open angle glaucoma who came for their regular follow up. **Group II** included nonglaucoma patients who came for routine ophthalmology check up for minor refractive errors.

We evaluated both eyes of 15 patients in each group. An informed written consent was taken from each subject.

All patients with a best corrected visual acuity better than 20/30 were chosen. Their refractive errors were between –3 and +3 dioptres of sphere or between –3 and +3 dioptres of cylinder, with no ocular disease other than glaucoma.

The Cup-Disc(CD) ratio was noted in both vertical and horizontal meridians for each patient by each of the three methods. Optic nerve head assessment was done after dilating all the patients with 0.8 % tropicamide and 5 % phenylephrine eye drops. We assessed optic nerve head by [A]slit lamp biomicroscopy using a +90 D lens. The horizontal and vertical cup disc ratios were assessed. [B] A fundus non stereo digital image using Zeiss VISUPAC fundus camera FF 450 was taken for each patient. The margins of optic cup and the optic disc were marked after loading in 1-image split view mode. The cup and disc were circumscribed as two separate areas using the **Closed Polygon** or **Free-hand contour** function. Then using the measure mode in the VISUPAC software, the cup-disc ratio in the horizontal and vertical meridians were noted.

This was done by clicking on the end of line and holding the mouse button depressed and dragging it in the desired position. Additionally, the following parameters are displayed: cup and disc area in mm$^2$ and the linear C/D ratio for the total papillae in percent. For vertical meridian, an average of C:D ratio at 90 and 270$^\circ$ were taken, and for horizontal meridian an average of CD at 0$^\circ$ and 180$^\circ$ were taken. The corresponding C/D ratio data are displayed at the intersecting points of the radial lines. With change in the size of the drawn figures, the data are automatically updated.

To do this, click on the end of line and holding the mouse button depressed drag it in the desired position. Additionally, the following parameters are displayed: cup and disc area in mm$^2$ and the linear C/D ratio for the total papilla in percent.

[C]Evaluation on OCT was done using the Fast Optic Disc Protocol on the Zeiss Stratus OCT Model 3000. It analyses optic disc with a circle of 4 mm diameter.
around optic nerve head. Six radially acquired cross sectional line scans spaced 30° apart were done that were passing through the centre of optic nerve head.\(^2\) The horizontal and vertical cup disc ratio was given by the software in this method.

Optic disc analysis by fundus photograph, OCT and biomicroscopy were done by two experienced observers to avoid the interobserver bias.

Statistical methods (Pearsons Correlation and Kappa) were applied to study the significance of the difference in the CD ratio as calculated by the 3 methods.

**Results**

In Group I the age was between 48 to 76 years [Mean 59.6 years], and in Group II between 20-65 years [Mean 52.3 years]. Group I had 7 males and 8 females and Group II had 6 males and 9 females. For each of the patients the Cup Disc ratio was noted by all the three ways in both the eyes. In three patients of Group I the optic disc could not be assessed due to media opacity. Fig.1[A] depicts the CD ratio in both the vertical and horizontal meridian for each of the patients in Group I with fundus photography; Fig.1[B] depicts the CD ratio in both the vertical and horizontal meridian for each of the patients in Group I with Slitlamp biomicroscopy; and Fig.1[C] depicts the CD ratio in both the vertical and horizontal meridian for each of the patients in Group I with OCT.

Vertical CD ratio calculated by OCT was the highest in 90% of glaucoma patients and in 60% of the control group. In the normal population CD ratio as calculated by slitlamp biomicroscopy was the least.

Relationship between CD ratio as calculated by the 3 methods was compared both for horizontal & vertical CD ratios separately taking 2 modalities at a time by Pearson correlation i.e. OCT and SBM; OCT and ONHPs; then ONHPs and SBM.

ONHPs and OCT did not correlate well with each other both for the vertical & horizontal CD ratio[Fig. 2]. However, horizontal CD ratio by OCT and biomicroscopy correlated better. SBM had a good correlation with OCT & ONHPs in the Vertical axis. Vertical CD ratio is either way more clinically relevant for glaucomatous damage \(^7\). Visualisation of vertical cup is affected less by disc vessels and their branching and thus is more accurately calculated. Intra class correlation which was used to assess the relationship of all the 3 methods together was 84% for vertical & 69% for horizontal axis. Inter observer correlation was done with kappa for ONHPs and SBM and showed 60% correlation. [Fig. 3a and 3b].

**Discussion**

Glaucoma is now recognized as an optic neuropathy and not merely a disease with raised intraocular pressure. Assessment of the optic nerve head is essential for diagnosis, management and progression of the disease. With a plethora of modalities available for assessing the optic nerve head we need to know which is more reliable, which has better sensitivity and specificity and which of them is more reproducible. Various studies have tried to find the most appropriate
Fig. 2: Shows correlation between various groups

Table 1. Pearsons Correlation

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<tr>
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<th>OCT Horizontal</th>
<th>OCT Vertical</th>
<th>ONHPs Horizontal</th>
<th>ONHPs Vertical</th>
<th>SBM Horizontal</th>
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<td>OCT</td>
<td>0.38</td>
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Fig.3a Depicts the interobserver variability in Group I [glaucoma patients]

Fig.3b Depicts the interobserver variability in Group I [normal controls]

approach to assessment of the optic nerve head. The qualitative aspects noted are Contour of the Neuroretinal rim, Optic Disc Haemorrhages, Cup Asymmetry, Parapapillary atrophy, Bared circumlinear vessels and appearance of RNF layer. The quantitative parameters are Optic disc size (Vertical disc diameter), Cup disc Ratio (C:D), Rim/disc ratio. Cupping is symmetrical in both eyes in 96% of the population.

Direct Ophthalmoscopy using parallax – Spot size less than size of the disc. Disc shape is normally slightly vertically oval and the Contour depends on the shape of the optic disc canal. It is important to judge how healthy the neuroretinal rim is.

The normal distribution of the Neuroretinal rim follows the ISNT rule being the thickest at the Inferotemporal sector [83% eyes], followed in decreasing order by Supero temporal, Nasal and temporal. It is the vertical cup that correlates best with glaucomatous damage, we should record the cup/disc ratio in horizontal and vertical meridian. The large physiological cups in large discs appear round.

Clinical evaluation by SBM & ONHPs are subjective methods. They have an interobserver bias and it may not be reproducible at a later date. OCT on the other hand is objective; it picks up the measurements depending on the reflectivity change at the RPE level corresponding to the scleral margin of the disc and the optic cup is picked up by the change in reflectivity at retinal nerve fiber layer. Both these are morphological determinants for ganglion cell loss which is reflected by the CD ratio. On reliability analysis scale, CD ratio...
for the same patient was higher with OCT than with the other two methods.

We need to follow up the normal patients with high CD ratio on OCT to see if they convert to glaucoma as evidenced by visual field changes or high intraocular pressure. We also need to exclude nonglaucomatous reasons of optic disc cupping like ONH coloboma, optic pit.

ONHPs is a tedious method. OCT may overestimate the CD ratio, but is user friendly with easily interpretable and reproducible data. We also need to compare the RNFL loss in OCT for the same patient.

Variability in size & appearance of ONH of normal eyes accounts for the difficulty in detecting the presence of early glaucomatous optic nerve damage.

Accurate measures of CD ratio may be derived from OCT images of images the ONH. The disc width was defined as the separation of the terminations of the retinal pigment epithelium on each side of the image. Estimates of clinical CD ratios in normal subjects appear to be less than those with OCT.

50 % cases showed good correlation between all the 3 methods. Optical coherence tomography was showing a higher value indicating the requirement to do optical coherence tomography in all patients to detect the actual cup disc ratio, this will help us to detect glaucoma cases earlier & to treat them well before axonal loss occurs.

In one study of Hynchak, where the CD ratios by both digital Photography and stereobiomicroscopy were compared CD ratio estimation on photos showed a lesser value in normal subjects. This study suggested that caution should be exercised when using stereoscopic and non-stereo digital evaluations of CD ratio interchangeably to assess longitudinal progression in a multi-clinician setting.

The mean optic disc parameters with the 95 % confidence intervals for the distribution of normal optic disc parameters in South Indian population as suggested by a study by Sekhar et al were: disc area 3.37 mm² (2.04 - 4.7), vertical disc diameter 2.12 mm (1.67 - 2.57), vertical cup to disc ratio 0.37 (0.19 -0.55) and neuroretinal rim area 2.8 mm² (1.76 - 3.84).

The optic disc analysis results were confirmed by 2 people who assessed the optic disc separately to avoid the inter observer bias. Some studies suggest that VCDR adjusted for vertical optic disc diameter may assist in the diagnosis of glaucoma in clinical practice. Correnti et al suggested that there was no statistically significant difference between techniques with respect to sensitivity and specificity of glaucoma detection, as far as optic nerve head (ONH) measurements and glaucoma status is concerned.

Fundus biomicroscopy enables more accurate, less variable VCDR estimation than direct ophthamcopy.

**Conclusion**

Optic nerve head evaluation is paramount for glaucoma diagnosis and it should be recorded separately in the vertical and horizontal meridian. The clinician should record which of the methods they used to examine the optic nerve head i.e. slitlamp biomicroscopy, ophthalmoscopy, OCT, HRT or digital fundus photograph so that subsequent clinical decisions are not influenced by apparent VCDR changes. In our study 50 % cases showed good correlation between all the 3 methods. Optical coherence tomography was showing a higher value.

**References**


Povidone – Iodine in Ophthalmology

Dr. Arup Chakrabarti MS, Dr. Sonia Rani John DNB, Dr. Valsa Stephen MS DNB, Dr. Meena Chakrabarti MS DNB

Povidone-Iodine (PVI) is routinely used in cataract surgery for postoperative infection prophylaxis. It has also found applications in other fields of ophthalmology. In view of the important and diverse applications of povidone-iodine, it is important for us to be familiar with the chemistry and mechanism of action of this widely used drug. The aim of this paper is to throw light in the diverse uses of povidone-iodine and its applications in ophthalmology.

What is Povidone-Iodine?

The possible effect of iodine on the eye was first appreciated in 1951 when a reduction in ocular flora was reported following the application of iodine solution to the skin. Iodophors were reported to reduce skin flora around the eye in 1970, and only later was the specific combination of povidone and iodine utilized for direct ophthalmic use.

Iodophors are developed by complexing iodine with surfactants like nonionic detergents, quaternaries and macromolecules. The detergents act as stabilizers and carriers combining detergent property with antibacterial activity. Iodophors are non-irritating, non-staining and water miscible in all dilutions.

Povidone - Iodine (Betadine) is a complex polymer of iodine with polyvinyl pyrrolidone (a high-molecular weight, water soluble polymer), a complex that enhances the bactericidal activity of iodine. Iodine is slowly released from the complex, providing antimicrobial action. Most commercially available solutions of povidone-iodine have 1% available iodine.

Povidone-iodine is readily available world wide, either as a powder or as a 10 percent solution.

The spectrum of antimicrobial activity of povidone-iodine covers bacteria, viruses, and even spores. Most bacteria are killed within 30 seconds. Povidone-iodine has been shown to be a superior antiseptic agent for surgical preparation of skin. It has also been shown to be effective on the cervix and vagina in gynecological cases.

Depending on the type of application for ophthalmic use, the solution must be diluted to the appropriate strength. The diluents may be a balanced salt solution or other appropriate diluent. In the appropriate concentration, povidone-iodine is not toxic to the eye as are other iodine bearing compounds. It is a strong antiseptic with minimal secondary effects (i.e. red eye, allergic reactions) in healthy conjunctivas. It stains the area of application and rarely causes reactions like fever and generalized skin eruptions. It is important to avoid the detergent version of povidone-iodine generally used as a skin antiseptic, since this solution will adversely affect the cornea.

How Does Povidone - Iodine Work?

Povidone-iodine interacts strongly to the double bonds of saturated fatty acids in the bacterial cell wall and cell organelle membranes and also oxidizes amino acid and nucleotides. It causes pore formation and solid-liquid interfaces at the lipid membrane level of cell walls to loose cytosol material.

Bacteria generally adhere to the surfaces of infected tissue in biofilms on a glycocalyx material. Povidone-iodine can reach the biofilm and has been shown to
cause the outer layers of the biomass to slough and be killed. It not only destroys a wide range of bacteria, but also inactivates and inhibits the release of bacterial exotoxins. The superficial location of bacteria may provide susceptibility to the effects of povidone-iodine.

**Role of Povidone-Iodine in Ophthalmology:**

Povidone-iodine has found several applications in ophthalmology. It has been employed preoperatively in an attempt to reduce the incidence of postoperative infections, including endophthalmitis. Other than its use in the prophylaxis of endophthalmitis, povidone-iodine used as a topical antimicrobial agent has been reported to be effective in treating conjunctivitis and keratoconjunctivitis. It has been used in the prevention of ophthalmia neonatorum.

Povidone-iodine has also been used as a means for decontamination of donor corneas. New investigations are underway to evaluate the effectiveness of povidone-iodine to treat corneal infections. Iodophors are used as hand washes, for preoperative skin preparation, as local antiseptics, in ringworm and in oral and vaginal moniliasis.

**Preoperative Infection Prophylaxis**

The best preoperative prophylaxis has not yet been found. However, povidone-iodine has played a very important role in the prophylaxis of post-cataract surgery endophthalmitis. In fact, till the results of ESCR Endophthalmitis Study were made available povidone-iodine was the only prophylactic agent that had been shown to reduce the rate of post-cataract surgery endophthalmitis. Antiseptics like povidone-iodine lower preoperative bacterial colony counts and decrease incidence of postoperative endophthalmitis.

Postoperative endophthalmitis, which is a rare, but sometimes devastating complication of cataract surgery, remains one of the most feared problems after intraocular surgery. Literature review suggests that there had been a gradual decline in the incidence of postoperative endophthalmitis till the early 1990s. However many publications seem to indicate that the incidence of post-cataract surgery endophthalmitis is on the rise with some studies reporting the occurrences between 0.07% and 0.3%. Most surgeons believe that this increase in frequency of endophthalmitis is due to increasing popularity of sutureless clear corneal surgery. Nagaki et al reported that the relative risk for endophthalmitis was 4.6 times higher with temporal clear corneal incisions than with sclerocorneal incisions. Schmitz et al found a 0.1% incidence of endophthalmitis with clear corneal incisions and 0.07% with scleral tunnel incisions.

Bacteria causing postoperative endophthalmitis most likely originate from the normal bacterial flora of the patient’s own conjunctiva and eyelid. In 75% to 95% of reported cases, the causative organisms are gram-positive cocci. Normal conjunctival flora comprises predominantly of coagulase negative staphylococci, i.e., Staphylococcus epidermidis (95.4%). Less frequent bacteria are Staphylococcus aureus (14.8%), anaerobes (44%), (Corynebacterium species), Streptococcus species (4.4%) and gram-negative rods (7.8%). (i.e. E Coli, Pseudomonas aeruginosa). The Endophthalmitis Vitrectomy Study determined that, of 69% of patients with confirmed microbiology growth, 70% were infected with coagulase negative micrococcus, mostly Staphylococcus epidermidis, 10% with Staphylococcus aureus, 9% with streptococci, 2% with enterococci, 3% with other gram positive species and 6% with gram negative species. Anaerobic or microaerophilic organisms such as Propionibacterium acnes are more commonly found in chronic and late intraocular inflammations. Because gram-positive cocci are the main cause of acute postoperative endophthalmitis, methods intended to reduce conjunctival bacterial flora should be effective against these bacteria.

The goal of preoperative chemical preparation of the eye (e.g. with povidone-iodine) is to minimize the number of microorganisms on the skin and eye immediately before the first incision and it is also desirable to reduce the bacterial flora in the conjunctival sac. Several studies have been conducted to reduce preoperative bacterial load on the conjunctiva. In 1984, Apt and Isenberg et al showed that povidone-iodine dilutions decreased the number of colonies by 91% and decreased the number of species by 50%. These findings were statistically significant compared to the untreated fellow control eyes. In another German study, povidone-iodine showed a significantly lower
incidence of culture positive endophthalmitis, compared with silver protein solution. Speaker and Menikoff et al. 25 found evidence for an association between prophylaxis with povidone-iodine and lower incidence of postoperative endophthalmitis. Povidone-iodine decreases the conjunctival load of propionibacterium acne also, which is a common cause of chronic postoperative endophthalmitis.

For skin asepsis, a 10 percent povidone-iodine solution is widely used. In the periorbital region with many sebaceous glands the antiseptic should be administered about 10 minutes before surgery to act sufficiently. 26 There is a wide variation in the technique and concentration of povidone-iodine application in the conjunctival sac.

Povidone-iodine may be applied a) directly, flushing the upper and lower fornices of the conjunctival sac, or b) as instillation of eye drops before an intraocular procedure. Irrigation of conjunctival sac may be more effective in reducing the conjunctival bacterial load and possibly decreased susceptibility to endophthalmitis. This was suggested in a prospective, randomized, controlled trial of 200 eyes undergoing anterior segment surgery treated with topical ofloxacin. 27 The study group that underwent irrigation of the fornices with 10 ml of povidone-iodine had fewer positive conjunctival cultures than the control group that received two drops of povidone-iodine preoperatively. In this study 26 % of study eyes had positive conjunctival cultures immediately before surgery and 18 % had positive cultures at the end of surgery as opposed to 43 % before surgery and 32 % after surgery in the control eyes.

Preoperative conjunctival fornix irrigation with 5 % rather than 1 % povidone-iodine results in greater decrease in colony forming units, especially with heavier initial bacterial load (greater than 100 colony-forming units before irrigation with povidone-iodine). This was demonstrated in a prospective, randomized, double blind study of 105 patients in the United Kingdom where a statistically significant drop of 96.7 % colony-forming units was seen in the 5 % povidone-iodine group as compared with the 40 % decrease in the 1 % povidone-iodine group when there was heavier initial bacterial load. 28 The efficacy of povidone-iodine in reducing conjunctival contamination is comparable to a 3-day course of topical antibiotics. 24, 29

**Postoperative Infection Prophylaxis:**

When the timing of the bacterial penetration of the eye is considered, there are two main intervals during which the eye is at risk. The first is during the actual operation, when the ocular tissues have been incised. The second important interval during which the eye is at risk is in the immediate postoperative period. During that time the surgical wound is somewhat exposed to the environment. Five percent povidone-iodine solution applied at the conclusion of surgery significantly decreased the number of colony-forming units immediately postoperatively and at 24 hours following surgery, thereby decreasing bacteria that may enter the surgical wound postoperatively. 30 This may be a particularly significant prophylactic measure since compromised clear corneal wound architecture have been implicated in the recent spurt in the incidence of post-cataract surgery endophthalmitis. 31, 32

**Prevention of Ophthalmia Neonatorum**

Povidone-iodine has been recommended for prophylaxis against ophthalmia neonatorum, especially in developing countries. In a study by Isenberg et al. 13, 14 in 1995, it was demonstrated that prophylaxis with a 2.5 percent ophthalmic solution of povidone-iodine resulted in fewer cases of ophthalmia neonatorum over all and notably fewer cases of chlamydial conjunctivitis than prophylactic treatment with either erythromycin or silver nitrate. Povidone-iodine has many potential advantages over these currently used drugs, including a broader antibacterial spectrum. In a concentration as low as 0.1 percent, povidone-iodine is effective against Neisseria gonorrhea; in a concentration as low as 1 percent, it is effective against C. trachomatis; and in a concentration of 0.5 percent or lower, its antiviral spectrum includes the human immunodeficiency virus and herpes simplex virus.

Povidone-iodine turns the surface of the eye brown for a few minutes, a characteristic that can serve as an indicator that it has been properly applied. The possibility of misapplication is greater with the other two agents because they are colourless. With povidone-iodine, unlike antibiotics, bacterial resistance has not been encountered. Finally, it is cheaper than the other agents. In many developing countries, where
ophthalmia neonatorum is more common, no prophylaxis is used mainly because of the expense and lack of availability.

**Treatment of Ongoing Ocular Infections:**

In addition to the role of povidone-iodine in prevention of infections, its role has also been explored in the treatment of ongoing ocular infections.

A study was conducted by Isenberg S J. et al in 2002 to investigate the use of povidone-iodine in the treatment of paediatric conjunctivitis in 459 children in Manila, Philippines. Povidone-iodine 1.25 percent ophthalmic solution, applied 4 times daily, was compared with the effect of an antibiotic combination (neomycin – polymyxin B-gramicidin) and was found to be as effective in the treatment of bacterial conjunctivitis, marginally more effective against chlamydial conjunctivitis (P = 0.057) but equally ineffective against viral conjunctivitis. Povidone-iodine still continues to be highly germicidal when used at the lower concentration of 1.25 percent than 2.5 percent. Because povidone-iodine ophthalmic solution can be prepared from powder and stock solutions meant for other antiseptic purposes, it is not only inexpensive but also widely available in developing countries. Povidone-iodine 1.25 percent ophthalmic solution can therefore be considered as treatment for bacterial and chlamydial conjunctivitis, especially in developing countries where topical antibiotics are often unavailable or costly.

**Conclusion**

Povidone-iodine is frequently chosen as an antimicrobial agent in view of its various advantages. In the appropriate concentration, it is not toxic to the eye as are other iodine bearing compounds. It has a very broad antimicrobial spectrum, including bacteria, viruses, and fungi, given enough contact time in vitro. Resistance to bacteria is rare. The medication turns the eye brown for a few minutes proving that it has been applied. It is widely available as a solution or powder. Since it is not expensive, it is widely used even in developing countries.

Povidone-iodine is an effective disinfectant significantly reducing both the contamination levels of the conjunctival sac as well as the incidence of post-cataract surgery infection. Investigations of its use in treating other types of ophthalmic infections are continuing. The use of povidone-iodine in ophthalmic practice continues to reduce the incidence of blindness in children and adults throughout the world.

**References**


Combined Endoscopic, Laser Assisted Dacryocystorhinostomy (ECLAD)

Dr. Abraham Kurien MS

From the time that Toti (in 1904) first described the operation of creating a direct fistula between the lacrimal sac mucosa and the mucosa of the middle meatus of the nose, in order to relieve a blockage of the nasolacrimal duct that occurs in chronic dacryocystitis, various innovations have been tried in order to improve the efficiency of the procedure and to cut down on the morbidity associated with the operation. This external approach in dacryocystorhinostomy (DCR) surgery underwent several modifications such as suturing of the anterior and posterior mucosal flaps and intubation with tubes and stents made from various materials.

Another major innovation in this surgery was the introduction of the endonasal approach to DCR. This was first done in 1911. This later was modified by the use of the YAG laser to effect fracture of the bone and to cut the nasal and sac mucosa.

The main advantages of the endonasal procedure over the external approach are:
- better cosmesis due to absence of an external scar and the medial palpebral ligament remaining intact,
- less bleeding,
- intact lacrimal pump mechanism due to the orbicularis muscle not being tampered with, and
- associated nasal anomalies can be corrected in the same sitting,

However the endonasal procedure has its own problems:
- Most studies show that the success rate is inferior to that of the external procedure.
- Endonasal approach is in the domain of an ENT surgeon rather than the ophthalmologist who is more aware of related problems.

In 1993, a revolutionary innovation was made in the field of DCR surgery by the introduction of the use of a YAG laser which when passed through the lacrimal canaliculi, could cut the medial wall of the lacrimal sac, the bony wall of the lacrimal fossa as well as the nasal mucosa, creating a large enough fistula to overcome the blockage of tears due to nasolacrimal duct obstruction. A diode laser was later substituted for the YAG laser.

Instrumentation

ECLAD is a mode of doing Dacryocystorhinostomy through a combined approach viz. transcanalicular with nasal endoscopic monitoring.

The laser used is a diode with a wavelength of 980 nm with 10 Watts of power. This machine is an improvement on previous machines which had only 4 Watts of power and hence were not capable of going through dense bone.

980 nm wavelength has high absorption in water, oxyhaemoglobin and haemoglobin. It has concurrent vaporization of both hard and soft tissue with optical coagulation.

The preoperative check list for a transcanalicular Laser DCR is the same as for any conventional DCR.
In addition to routine tests, bleeding and clotting times are checked. Lacrimal sac tumors and chronic infections like tuberculosis of the sac have to be ruled out. A detailed examination of the middle meatus of the nose, where the fistula is to open, is done. Polyps, deviated septa, tumors and other disorders are addressed before the surgery is done.

The procedure is fairly simple and short, the operation itself taking only about ten minutes.

Operative Steps

- A syringing is done before surgery to confirm the diagnosis of Nasolacrimal duct block.
- A preoperative packing of the nose with xylocaine 2%( with adrenaline,) for shrinkage of the nasal mucosa and better visualization of the structures in the nose is done about 30 minutes before the surgery which is removed just prior to the procedure.
- Xylocaine 2% is infiltrated above and below the medial canthus upto periosteum level and a lacrimal probe is passed through the canaliculus into the sac to test the patency.
- A nasal endoscope with either 0 degree or 30 degree angulation, which is connected to a TV monitor through a video camera, is used to visualize the middle meatus, above the inferior turbinate.
- The laser probe can be passed into the sac in two ways.
  A) Either it is passed alone in the case of the more rigid fibreoptic probe or
  B) Through a 23 gauge metal cannula in case of the thinner, more flexible probe.
- The direction of the canula or the probe is first horizontally, till one encounters a hard stop and then downwards, medially and slightly anteriorly after withdrawing the tip from the bone.
- If the bony wall of the lacrimal fossa is thin, as is usually the case in female patients and in those of either sex belonging to older age groups, the pilot
indicator lamp can be visualized through the endoscope in the nose and laser applications can be made under direct vision.

- In patients with thicker bones the pilot light may not be seen through the nose and the first laser application would be done blindly and subsequent shots applied after adjusting for proper position in relation to the first burn.

- Laser burns are seen by the appearance of smoke in the nose, and visualization of the pilot lamp and the probe in the nose.

- One should aim for an ostium of about 10 mm vertically and at least 5 mm horizontally, taking care to see that it is in a position which is not likely to be obstructed by other structures in the nose like the septum or the turbinates.

- The Nasal endoscopic monitoring also ensures that there is no inadvertent injury to the nasal septum or other adjacent structures.

- In cases where space seems inadequate, infracture of the turbinate may generate more space and contribute to the continuing patency of the ostium.

- Irrigation with saline can be done if visualization is hampered by the appearance of smoke. Syringing with Mitomycin-C 2% solution is done and the appearance of the fluid in the nose can be seen through the nasal endoscope.

- If there is any evidence of haemorrhage into the nose, a pack can be placed and removed a few hours later.

A few studies have indicated that intubation of the ostium with silastic or other material is an useful adjunct to the procedure.

An alternative would be to use Mitomycin-C drops and steroid drops topically in the immediate postoperative period.

ECLAD thus comes across as a time saving, efficient procedure in which practically all morbidity associated with conventional external DCR is eliminated and is a more predictable and efficacious procedure than Endonasal DCR.

References


Jacksons Cross Cylinder (JCC)

Dr. R. Nirupama Balaji, Dr. K.S. Chandrakanth, Dr. Sheeja, Dr. Ramakrishnan, Dr. Tresa, Dr. Preetha

History
- L.V. Edward Jackson in 1887 discovered the JCC.
- He was also the first president of the American Academy of ophthalmology and otolaryngology.

Uses
1. To assess the strength and axis of the cylinder.
2. To check the accuracy of the distance spherical correction.

Quotes
In Edward Jackson’s words JCC is probably “far more useful and far more used” than any other lens in clinical refraction.
Every ophthalmologists should be familiar with the principles involved in its use.

Instruments
The cross cylinder is a combination of two cylinders of equal strength but with opposite signs placed with their axis at right angles to each other and mounted in a handle. The commonly used cross cylinders are of ± 0.25D and ± 0.5D.
Although the cross cylinder is usually used to refine the cylinder axis and power of refraction already obtained, it may also be used for the entire astigmatic refraction.

Method

1. Refinement of the Axis
This is always done first. This is because the correct axis can be found in the presence of an incorrect power but the full cylindrical power will not be found in the presence of an incorrect axis. To refine the axis, the cross cylinder is placed (+0.5D) before the eye with its axis at 45 degree to the axis of the cylinder in trial frame (first with -0.5 D cylinder and then +0.5D cylinder or vice versa) and the patient is asked to tell about any change in visual acuity. If the patient notices no difference between the two positions, the axis of the correcting cylinder in the trial frame is correct. However, if visual improvement is attained in one of the positions a “plus” correcting cylinder should be rotated in the direction of the plus cylinder component of the cross cylinder (and vice versa). The test is then repeated several times until the neutral point is reached.

2. Refinement of cylinder power
To check the power of cylinder, the cross cylinder of ± 0.25D is placed with its axis parallel to the axis of the cylinder in the trial frame. First with the same sign and then with the opposite sign. In the first position the cylindrical correction is enhanced by 0.25D and in the second it is diminished by the same amount. When the visual acuity does not improve in either of the position the power of cylinder in trial frame is correct. However if visual acuity improves in any of the positions a corresponding correction should be made and verified till final correction is attained.

3. Discovery of Astigmatism
If no cylindrical correction is present initially, the cross cylinder may still be used, placed arbitrarily at 90 degree
and 180 degree to check for astigmatism. If a preferred flip position is found, cylinder is added with axis parallel to the respective plus or minus axis of the cross cylinder until the two flip choices are equal. If no preference is found with cross cylinder axis at 90 degree and 180 degree the 45 degree and 135 degree should always be checked before assuring that no astigmatism is present.

**Points to Ponder – to Summarise**

- JCC is always a sphero cylindrical lens such that one meridian is plus power and the other meridian is of equal minus power.
- The red dots identify the axis of the minus power. The power in the meridian of the red dots is of plus power.
- The JCC in a plus cylinder phoropter is identical to that of minus cylinder phoropter.
- When you use a plus cylinder phoropter you “chase” the white dot instead of the red dots.
- When you perform the JCC power test the length of the astigmatic interval changes. Flipping the lens causes a change in the interval length (a change in image quality) between the two meridians if cylinder is not fully corrected.
- The end of JCC test is when both images appear equal or are equally blurred. This occurs because with the correct cylinder power in place and the JCC lens in place the astigmatic interval is the same length in each position.
- The end of JCC test occurs when the resultant cylinder caused by obliquely crossing two cylinders has been neutralized.
- Placed before an emmetropic eye the cross cylinder blurs the image. Placed before an ametropic eye the cross cylinder does not alter the spherical equivalent but it will enlarge or contract the interval of strum, blurring or clarifying the image as it increases or decreases the net astigmatic correction. The cross cylinder is used for subjective refinement of axis and power of cylinder after placing the best available estimate of refraction before the eye.

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Releasable and Adjustable Sutures for Safe and Predictable Outcome Following Glaucoma Filtration Surgery

Dr. Chockalingam, M. DO DNB FRCS (Glasgow) PGDHM, Dr. Anup Chirayath DOMS DNB FRCS (Glasgow)

Introduction

There has been a paradigm shift in our understanding of glaucoma which is currently defined as a progressive, multifactorial optic neuropathy characterized by specific morphological changes with acquired and accelerated loss of retinal ganglion cells resulting in a variety of functional changes including visual field loss. Thus, elevated intraocular pressure is now considered as one among the several but not the decisive risk factor for the development of glaucoma. Despite research on other ways of managing glaucoma all currently available therapeutic interventions – medical, lasers and surgery – have aimed at reducing the intraocular pressure, the benefit of which has been elucidated by several long term studies and trials.

Trabeculectomy, with or without cataract extraction and intraocular lens implantation and augmented with or without antimetabolites has been the mainstay of surgical treatment to reduce intraocular pressure in patients with glaucoma. While a lot of research and interest has been shown in the non – penetrating surgeries, laser surgeries, valve implants, trabeculectomy continues to be the most widely performed procedure. The surgical technique in trabeculectomy continues to be refined and reshaped to make it safer and predictable.

Trabeculectomy is fraught with complications related to shallow anterior chamber in the early post – operative period. Shallow anterior chamber leads to a host of complications in the anterior and posterior segment, like corneal decompensation, peripheral anterior synechiae, posterior synechiae, accelerated cataract formation, choroidal detachment, macular edema, suprachoroidal hemorrhage, all of which compromise the desired outcome of the filtration surgery in terms of lowering the intraocular pressure and decrease the vision. Although the incidence of these complications are less than what is encountered after a full – thickness filtration surgery, these complications may occur more frequently and with greater severity with intraoperative use of antimetabolites especially mitomycin – C (MMC). The guarded filtration trabeculectomy being performed now is not as successful as a full – thickness filtration procedure in terms of intraocular pressure control.

Releasable sutures permit a controlled achievement of the targeted intraocular pressure in the post – operative period after trabeculectomy. Placing relatively tight sutures at the time of surgery with subsequent selective suture lysis or removal enables reducing overfiltration in the immediate post operative period without sacrificing long term intraocular pressure control. Releasable sutures permit a controlled achievement of the targeted intraocular pressure in the post – operative period after trabeculectomy. Placing relatively tight sutures at the time of surgery with subsequent selective suture lysis or removal enables reducing overfiltration in the immediate post operative period without sacrificing long term intraocular pressure control. Releasable sutures permit a controlled achievement of the targeted intraocular pressure in the post – operative period after trabeculectomy.

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thickness filtration procedure. The combination of releasable sutures with intraoperative and postoperative application of antimetabolites have greatly enhanced the success of trabeculectomy in the recent past. This article discusses the principles, techniques, benefits, and practical issues of the widely used technique of laser suture lysis, releasable sutures and adjustable sutures and provides a brief insight into the intraoperative considerations while applying such sutures.

**Laser Suture Lysis**

**Principle**

Laser suture lysis allows the surgeon to place tight sutures and release them in the postoperative period as and when desired. The singular advantage of this technique is that the surgeon need not master any new intraoperative skill. The technique involves the transconjunctival application of laser energy to the scleral flap. The laser energy, by virtue of heat generated, dissolves the suture thus lysing them and effectively cutting them and permits an enhanced aqueous runoff.

Several lenses are available including the Hoskins, Ritch, Mandelkom and Zeiss four mirror. The Hoskins suture lysis lens (Ocular Instruments Inc. Bellevue, WA) is more popular in clinical settings due to its ease of usage. It is a 3 mm diameter biconvex glass button lens surrounded by a semi – circular lid retraction flange made of polymethylmethacrylate affixed to aluminum handle (Figure 1 and 2). The lens presses on the conjunctival bleb directly over the scleral flap whereas the superior rim pushes up the upper lid. Commonly the argon laser is used although a krypton (514 nm) or diode laser (800nm) may also be used. The target site for the delivery of laser is indicated by a red spot light and the suture is lysed taking care not to pass the laser on the overlying conjunctival or adjoining superficial scleral vessels. The suture is cut at the extreme end on the outer side of the scleral flap so that after cutting, one end retracts inside the sclera.

**Technique**

Following premedication with a topical anesthetic agent and with sterile precautions, the patient is asked to look down and the upper lid is gently lifted by an assistant to improve the surgeon’s access and visibility. Either one of the two lenses - the Hoskins nylon suture laser lens or the Zeiss four mirror lens is placed on the conjunctiva over the suture intended to be cut (Figure 3) and laser energy applied. Gentle pressure with the lens displaces the fluid from the overlying conjunctiva and blanches the overlying conjunctival vessels to enable better visualization of the scleral flap suture to be cut. With good visibility and in a cooperative patient, a suture can be cut in less than five applications. With the Zeiss four – mirror lens, the conjunctival vessels are blanched through the slightly rounded edge of the working end of the lens adjacent to its concave portion.

![Fig. 1. Hoskins Suture Lysis Lens](image1)

![Fig. 2. Hoskins Suture Lysis Lens](image2)

![Fig. 3. Hoskins suture lysis lens in position over the bleb, blanching the conjunctival vessels, thereby enabling better visualisation of the scleral flap suture to be cut.](image3)
The settings for the procedure are given in the following box.

**SETTINGS FOR ARGON LASER LYSIS OF TRABECULECTOMY FLAP SUTURES**

<table>
<thead>
<tr>
<th>Lens used</th>
<th>Hoskins (most commonly used), Ritch, Mandelkom and Zeiss four mirror</th>
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</thead>
<tbody>
<tr>
<td>Laser used</td>
<td>Argon, Krypton, diode</td>
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<tr>
<td>Spot size</td>
<td>50 – 100 microns</td>
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<tr>
<td>Power</td>
<td>250 to 1000 mW</td>
</tr>
<tr>
<td>Time</td>
<td>20 to 100 milliseconds</td>
</tr>
<tr>
<td>Applications</td>
<td>1 – 3</td>
</tr>
</tbody>
</table>

Following suture lysis, the patient is examined again on the slit lamp after a few minutes to notice if the bleb has formed spontaneously; if not, a careful digital massage is given to see the bleb gain in height and intraocular pressure is recorded. Figure 4 shows an eye undergoing argon laser suture lysis and Figure 5 the formation of a good bleb gaining in height after the procedure.

**Practical Considerations**

The most commonly encountered problem is poor visualization of the suture to be lysed due to presence of thick inflamed Tenon’s tissue or blood. Placing long sutures intraoperatively which are equidistant on both sides of the cut edge of the scleral flap may improve visualization of at least a small clear area of the suture in the postoperative period. Prolonged compression with the lens may blanch the conjunctiva permitting suture lysis. The **Zeiss four – mirror lens** is more effective than the Hoskins lens when the bleb is thickened because it can be applied to the eye with greater pressure than that of the Hoskins lens.

Laser assisted suture lysis may cause mild ocular pain since it is done in the early postoperative period. Perhaps the most serious complication is laser induced perforation of the conjunctival flap. This may occur if excessive power is used with several failed applications and may also occur in the presence of subconjunctival blood in the area. This may result in chronic bleb leaks especially when the procedure has been augmented with antimetabolites. Following lysis of the suture the cut end may stand vertically inside the bleb irritating the conjunctiva and thereby causing vascular congestion, fibrosis and eventual failure of the bleb or a non-healing button hole with eventual chronic bleb leak. This is possible if following suture lysis and added digital massage, the bleb fails to gain in height.

If the cut end of the suture stands vertically, it is wise to cut the base of the vertical segment to prevent it from puncturing the bleb.

The most important practical consideration though is the availability of the machine to the surgeon. Also, problems related to convenience and cost to the patients are pertinent since this method involves the cutting of multiple sutures over time. The patient has to make repeated visits for examination, sequential laser suture lysis and post laser intraocular pressure monitoring.

**Releasable Sutures**

**Principle**

To avoid the drawbacks of laser suture lysis, eliminate dependence on sophisticated machines and thus make the procedure feasible in the office room, surgeons have developed techniques whereby releasable sutures are applied intraoperatively and these are removed as and when necessary in the postoperative
period. Several methods of application and removal of releasable sutures including mattress, slip and loop have been adapted by different surgeons. The most widely used technique of interrupted externalized releasable sutures as developed by Cohen and Osher is described (Figure 6). The popularity of this particular technique is because it uses the traditional interrupted suture approach as used in conventional scleral flap closure and thus has a shorter learning curve.

**Technique**

**Placing the Suture**

Technique of interrupted externalized releasable sutures as developed by Cohen and Osher is described for a filtration procedure with a fornix based conjunctival flap (Figure 7). After the sclerostomy and peripheral iridotomy is completed, the scleral flap is closed with 10–0 nylon sutures. The flap may be closed with single releasable suture along with other fixed sutures or all sutures applied may be of the releasable type. The suture needle is passed through the cornea about 0.5 mm anterior and parallel to the limbus starting a little away from the area of the scleral flap (Fig 7 – A). After a short course through the corneal stroma it is externalized (Fig 7 – B). It is then passed through the base of the scleral flap near the limbus close to but not overlying the site of the sclerostomy (Fig 7 – C) and externalized again (Fig 7 – D). It is then passed through the apex of the scleral flap in the same fashion of a regular interrupted suture (Fig 7– E). The suture is tied with four throws made and the suture loop lying over the scleral flap is grasped completing the placement. The suture is trimmed leaving a very small residual loop over the scleral flap (Fig 7– F). The distal free end of the suture is cut flush with the cornea so that no suture end is exposed. When a limbal based conjunctival flap is fashioned, the suture passes beneath the intact insertion of the conjunctiva at the limbus.

**Releasing the Suture**

In the postoperative period following administration of topical anesthetic agents and with sterile precautions, the patient is asked to look down and the upper lid is gently lifted by an assistant to improve the surgeon’s access and visibility. A 26– gauge needle is used to lift the suture loop lying on the corneal surface thereby exposing the distal end of the suture which hangs freely on the surface of the cornea. The suture is then grasped and gently pulled with a fine forceps.

**Practical Considerations**

Access to laser is not required, minimum instrumentation is required and there is negligible pain or trauma to the tissue and no risk of conjunctival perforation. The releasable sutures can be removed more predictably than lysing the sutures in the presence of thick Tenon's tissue, subconjunctival pigmentation or hemorrhage. However, the slipknot can become enveloped and infiltrated with episcleral tissue or subconjunctival fibrous tissue which may preclude successful removal especially in the late post-operative period. Unlike the residual suture with cut ends left behind following laser suture lysis, the entire suture is removed and thus there is no cause of irritation or button holes developing in the conjunctiva.
The technique involves a learning curve to perfect it. The distal end if either not buried in the cornea or cut flush with the cornea may become loose and hang on the surface of the cornea resulting in a wind – shield wiper syndrome. This may also cause chronic mucus fishing or serve as a nidus for infection. Since the sutures have to be removed sequentially, problems related to convenience and cost to the patients are equally pertinent as the patient has to make repeated visits for examination, sequential suture removal and intraocular pressure monitoring.

**Trimming The Releasable Suture**

Even if the sutures are not required to be removed in the post – operative period for modulating the target intraocular pressure, they have to be trimmed six to eight weeks after surgery to avoid late slippage of the distal knot and thereby causing wind – shield wiper syndrome or chronic mucus fishing and to prevent any infection. This is important more so when we use antimetabolites for the surgery and 10 – 0 nylon suture is not biodegradable.

Following administration of topical anesthetic agents and with sterile precautions, the patient is asked to look down and the upper lid is gently lifted by an assistant to improve the surgeon’s access and visibility. A 26 – gauge needle is used to lift the suture loop lying on the corneal surface thereby exposing the distal end of the suture which hangs freely on the surface of the cornea. The suture is grasped and while being pulled with a fine forceps, it is cut flush with a fine blade near the proximal end of the exposed suture. It is important to gently pull the distal free end of the suture with a forceps while cutting it flush at the proximal end so that on being cut the suture retracts into the cornea and no free end is exposed.

**Transconjunctival Adjustment of Interrupted Sutures**

Various methods of suture application and removal have been developed to reduce the intraocular pressure in the postoperative period. However, till date there been no technique developed to tighten the scleral flap sutures to increase the intraocular pressure in the event of low postoperative pressures and its antecedent sequel occurring in the eye. Recently, a technique of transconjunctival adjustment of interrupted sutures has been described, which is promising, and adds a new dimension in the postoperative management following trabeculectomy.

In this technique, after a trabeculectomy with a 3 X 4 mm rectangular scleral flap with two 10 – 0 nylon sutures placed in the corners, the surgeon adjusts the sutures in the postoperative period by grasping the suture knot through the conjunctiva with a specially developedatraumatic forceps and pulls it either towards or away from the cornea 28. This specially designed forceps called the **Khaw Transconjunctival Adjustable Suture Control Forceps** (Figure 8) has round, smooth tips to avoid trauma or perforation of the conjunctiva during the sutures’ adjustment.

**Practical Considerations**

The problems encountered include poor visualization of the suture to be adjusted particularly due to thick inflamed Tenon’s tissue or blood. The procedure causes ocular pain and more discomfort to the patient than other methods. There is a risk of trauma to or disinsertion of conjunctival flap and possible perforation of the conjunctiva while doing the procedure. This may result in rebound inflammation leading to bleb fibrosis and ultimate failure or result in formation of chronic non – healing bleb leaks (especially when anti – metabolites are used) predisposing to hypotony and its sequel. While this procedure does not require sophisticated machinery support, it is always not able to be performed in the slit – lamp and may require mobilizing the patient to the operating room. This method of suture adjustment may be feasible and successful in the immediate post – operative period while later adjustment may be difficult when the suture becomes enveloped and infiltrated in the Tenon’s fascia.
or episcleral tissue. In the limited studies conducted so far the investigators have reported the capability to reduce the intraocular pressure by transconjunctival suture adjustments more predictably than the ability to raise the low intraocular pressure levels.

**Surgical Steps Revisited While Suturing the Scleral Flaps**

With the use of releasable or adjustable sutures becoming prevalent and its role in improving the success of outcome clearly documented by several studies, the surgical steps in the conventional trabeculectomy need to be revised and refined to enhance the outcome of surgery. A number of modifications have been suggested to improve the outcome of trabeculectomy even while making it safer sans the dreaded complications.

The scleral flap may be triangular or rectangular and should be one – third to one – half thick. Since the suture is passed full thickness through the flap very thin flaps pose a danger of button hole developing at the site of the entry in the flap when the releasable suture is removed subsequently. The sclerostomy made should be at the center of the bed of the designed flap and if it is close to either end of the base of the flap, it should be noted in the intraoperative notes and the corresponding suture in the flap edge should be tightened appropriately. While suturing, it is preferable to place sutures one – third length into the scleral flap from the cut edge of the flap and externalize them two – thirds length outside if fixed sutures are being placed. If releasable sutures are contemplated, the suture should be equidistantly placed on either side of the cut edge of the flap to facilitate easy removal. Generally, the sutures at the apices are most effective in closure than other sutures. However, the aqueous flow through the edges of the flap should be observed at the end of suturing the scleral flap before closing the conjunctiva and the sutures which are most and least effective in restricting the outflow of aqueous and whose manipulation in the postoperative period is planned should be noted in the intraoperative notes.

**Timing the Suture Removal**

The exact timing of lysing or releasing the suture depends on the technique used, initial level of intraocular pressure, target intraocular pressure desired, degree of subconjunctival filtration already present, presence of evidence of bleb fibrosis, anterior chamber depth etc. Suture lysis or removal has been performed as early as three days and as late as 21 weeks after trabeculectomy. The result of suture removal or lysis is most effective if done within 2 – 3 weeks of surgery. However it is better to delay suture lysis or removal after trabeculectomy augmented with mitomycin C to avoid the increased incidence of post – laser hypotony that results from the suppression of wound healing by mitomycin – C. Immediately after suture removal, it is ideal to make the patient wait in the office room for some time. The patient is examined again on the slit lamp to notice if the bleb has formed spontaneously; if not, a careful digital massage is given to see the bleb gain in height. The intraocular pressure is recorded one hour and one day after the suture removal / lysis. The time interval between suture removal or lysis can be from hours to days, depending on the response noted after the initial suture removal. However, it is preferred to remove one suture at a time and if further suture removal is desired it should be scheduled on some other day. The anterior chamber should be observed for depth and presence of any bleeding from the stoma after the suture removal / lysis. Studies place the risk of shallow anterior chamber and its sequel occurring in as many as 14 to 54% of eyes following suture removal after trabeculectomy. Thus shallow chambers remain a problem to be tackled with adjunctive measures like torpedo patching, large contact lenses, scleral shells and reformation of anterior chamber for which the surgeon must be equipped with in the event of such a need following suture removal / lysis.

**Conclusion**

Releasable sutures in trabeculectomy have to a large measure reduced the risk of postoperative complications associated with low intraocular pressure and shallow anterior chambers. The use of releasable / adjustable suture is of benefit in eyes at risk of complication of flat anterior chamber in the postoperative period (eyes with nanophthamos, elevated episcleral venous pressure, glaucoma in aphakia, glaucoma in young myopic individuals). Laser suture lysis and externalized releasable sutures are more popular and widely practiced. Newer techniques like externalized...
compression sutures over the scleral flap and transconjunctival adjustment of interrupted sutures throw up new options for improving surgeon’s ability to control intraocular pressure and anterior chamber depth in the postoperative period.

Reference


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Robert Marcus Gunn

(1850 -1909)

Dr. Padmaja Krishnan MS

Gunn's sign, Gunn's dots, the Marcus Gunn jaw- winking phenomenon and the Marcus Gunn pupil to name a few…..but who was Marcus Gunn?
Like Argyll Robertson, another ophthalmologist best remembered for the abnormal pupil he described, Marcus Gunn also was a Scot.
He was born in 1850 to a farmer in a remote Northwestern part of Scotland and studied Medicine at the University of St. Andrews and graduated with distinction from the University of Edinburgh in 1873.
In 1874, he spent six months teaching at Vienna, with the likes of Stellwag and Arlt. He returned to Moorfields in 1876 as a resident medical officer and improved the quality of cataract surgery by introducing the sterile principles of Joseph Lister. He also worked in comparative anatomy at University College, London. Later while working at the Perth district Asylum he learnt direct ophthalmoscopy and used this skill to see the retina of patients.
Marcus Gunn went to Australia in 1879 to collect specimens of eyes from native species of animals, especially marsupials. Returning to England, he studied these specimens as also those brought by Charles Darwin's Challenger expedition.
In 1882, at the age of 32, Marcus Gunn obtained a fellowship of the Royal College of Surgeons and the following year he became Assistant surgeon at Moorfields Eye Hospital. That same year, 1883, he described a synkinetic movement in a 15 year-old girl who had congenital ptosis: when she moved her jaw, the ptotic eye would open. This phenomenon now bears his name.
He was appointed Surgeon at Moorfields in 1888 and at the time of his death in 1909 was Senior surgeon at Moorfields.
Marcus Gunn was a good teacher and an excellent surgeon, who systemised the teaching of Ophthalmology. He loved the outdoors and with his interest in Botany, Zoology and Marine Biology spent his holidays collecting fossils. He had a large collection and these he donated to the British Museum.

Comtrust Hospital, Calicut
An Update on Eales’ Disease

Dr Jyotirmay Biswas MS¹, Dr Aditya Verma MS²

Introduction

Since the initial description by Henry Eales of Eales’ disease in his patients with recurrent retinal hemorrhages, history of headache, variation in peripheral circulation and chronic constipation in 1880 and 1882, this disease still remains a clinical enigma with an undetermined precise etiology. The disease is now considered a clinical syndrome which possesses a specific clinical picture and natural course.

History

It was first described by Dr. Henry Eales (Fig 1), a British ophthalmologist in 1880 and 1882. Elloits was the first to recognize the inflammation of retinal veins and described it as periphlebitis retinae. Subsequently many investigators documented both arteriolar and venular inflammation in Eales’ disease.

Definition

Eales’ disease is an idiopathic retinal periphlebitis characterized by capillary non-perfusion, neovascularization and recurrent vitreous hemorrhages, mainly involving the peripheral fundus, and occurring predominantly in young, healthy adult males (Fig 2).

Epidemiology

Initially having been reported in United Kingdom, it was subsequently reported in series from Canada, Germany, Greece, Korea, and Turkey. Presently it is more commonly seen in the Indian subcontinent with a frequency of 1 in 135-200 ophthalmic patients in a referral eye hospital in India. Male predominance of up to 97.6% has been found in most series. Mean age of onset is 26 years, although the disease has been seen to occur as early as 10 years of age.

Clinical features

Often Eales’ disease is asymptomatic in the initial stages of retinal perivasculitis. Some patients may develop symptoms such as floaters, blurring of vision or even
gross diminution of vision due to vitreous hemorrhage. In a series of Eales’ disease patients, 75 % had floaters and black spots, 60 % had painless dimness of vision. Bilaterality is common with the incidence being 50-90 %. Anterior uveitis is uncommon in Eales’ disease. However in severe active periphlebitis stage, spillover anterior uveitis may occur which is always non-granulomatous. The presence of granulomatous anterior uveitis points towards sarcoidosis. Eales’ disease shows patches of active and healed perivasculitis and vessel alteration in all quadrants unlike branch retinal vein occlusion, which generally remains confined to the affected quadrant.

Hypopyon is not seen in Eales’ disease and is a differentiating feature between it and Behcet’s disease. It also mimics sarcoidosis, and the presence of granulomatous anterior uveitis points towards sarcoidosis.

Healed perivasculitis is often seen as sheathing of veins. Other vascular changes include sclerosed cord of vessels, irregularity of vein caliber, and pigmentation along venules, kinky venules, abnormal vascular anastomosis and veins pulled into the vitreous cavity. Presence of active and healed choroiditis in Eales’ disease should make one suspect the presence of simulating disease such as sarcoidosis, tuberculosis or syphilis.

Central retinal periphlebitis involving the posterior pole, especially the macula, is markedly uncommon compared to peripheral periphlebitis. It is termed as central Eales’ disease which is a variant of classical Eales disease (Fig 3,4).

Macular changes are relatively uncommon. A recent series showed macular changes in 18 % of eyes, which included exudates over the macula, epimacular membrane and rarely subhyaloid hemorrhage. Peripheral retinal neovascularization was a frequent finding and was reported in 36-84 % cases. However optic disc neovascularization was a rare occurrence observed in only 9 % of cases. Dense vitritis is uncommon. Recurrent vitreous hemorrhage is the hallmark of this disease.

Surface neovascularization was seen in 50 % of eyes in one series. Fibrovascular proliferation also occurs in Eales’ disease (Fig 1, 2). Differential diagnosis of Eales’ disease is presented in Tables 1 and 2.

<table>
<thead>
<tr>
<th>Table 1. Proliferative Vascular Retinopathy Mimicking Eales’ Disease</th>
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<tr>
<td><strong>Systemic</strong></td>
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<td>Diabetes mellitus</td>
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<table>
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<th>Table 2. Retinal Vasculitis Mimicking Eales’ Disease</th>
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<tr>
<td><strong>Systemic</strong></td>
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</tr>
<tr>
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</tr>
<tr>
<td>Sarcoidosis</td>
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<tr>
<td>Syphilis</td>
</tr>
</tbody>
</table>

Fig. 3. Fundus picture (montage) left eye of a patient with Eales’ disease showing aggressive vasculitis, with sheathing of vessels in all the quadrants, hemorrhages in superior and temporal quadrants, and posterior pole involvement.

Fig. 4. Fundus photograph of right eye in a case of central Eales’ disease showing active retinal vasculitis and hard exudates in macular star configuration.
Investigations

Fundus Fluorescein Angiography

It is often helpful to distinguish Eales’ disease from other retinal vascular disease and is particularly beneficial in the ischemic stages to delineate areas of capillary non-perfusion, retinal and optic disc neovascularization and questionable macular edema.

In cases of active retinal vasculitis, staining of the veins can be seen in the early venous phase with extravasation of the dye in the late phase. This is highly characteristic of active local inflammation in the retinal vessels. Extent and location of the neovascularization can be precisely delineated by Fundus Fluorescein angiography.

Neovascularization if present can be characteristic with sea fan appearance (Fig 5) with intense hyperfluorescence in the early arteriovenous phase of FFA. These new vessels leak in the late venous phase. Venous obstruction and venous stasis are seen well on FFA. Engorged and tortuous capillaries and veno-venous shunts can also be seen in the ischemic stage.

FFA is used to delineate the location and extent of retinal ischemia (Fig 5) while performing laser photocoagulation. It helps to assess the adequacy of photocoagulation and the need for additional laser photocoagulation, when FFA is repeated in the follow up visits.

Ultrasonography

Ultrasound (combined A scan and B scan) is needed to rule out any associated retinal detachment, either tractional, rhegmatogenous or combined in an eye with opaque media. Ultrasound usually reveals echoes of variable density depending on the compaction of the vitreous hemorrhage.

Other findings in ultrasound are as follows
1. Subhyaloid echoes.
2. Posterior vitreous detachment complete or incomplete.
3. Membranes in the vitreous cavity.
4. Fibrovascular proliferation.

Natural Course

Clinical manifestations are due to 3 basic pathological changes:
1. Inflammation (peripheral retinal perivasculitis)
2. Ischemic changes; peripheral retinal capillary non-perfusion
3. Neovascularisation of the disc and retina which often leads to vitreous hemorrhage.

Loss of vision in Eales’ disease is due to the recurrent vitreous hemorrhage (Fig 6), macular changes (ischemia, edema, and hemorrhage), tractional and combined retinal detachment involving the macula. Vascular occlusions may also complicate the picture (Fig 7).
Blindness due to Eales’ disease is rare. In case series of 800 cases only 4 eyes had < 20/200 and 8% had visual acuity between 20/100 to 20/200.

Classification

Charmis in 1965, classified Eales’ disease into 4 stages
Stage 1: Very early in evolution and characterized by mild periphlebitis of small peripheral retinal capillaries arterioles and venules.
Stage 2: Perivasculitis of the venous capillary system is widespread. Vitreous haze is present.
Stage 3: New vessel formation with abundant hemorrhage in the retina and the vitreous is found.
Stage 4: End stage of massive and recurrent vitreous hemorrhages with retinitis proliferans and tractional retinal detachment.

Due to the overlap of the stages in the clinical setting the four stage classification is not very popular. Other investigators have proposed different system of grading depending on the extent of microangiopathy, proliferative retinopathy, and vitreous hemorrhage. These classification systems are useful for monitoring and assessment of the effect of the treatment.

More recently, Saxena and Kumar proposed a new classification system:

Stage 1: periphlebitis of small (1a) and large caliber vessels (1b) with superficial retinal hemorrhages.
Stage 2a: denotes capillary nonperfusion.

2b: neovascularization elsewhere /of the disc.
Stage 3a: fibrovascular proliferation.

3b: vitreous hemorrhage.
Stage 4a: tractional/ combined rhegmatogenous retinal detachment.

4b: rubeosis iridis, neovascular glaucoma, complicated cataract and optic atrophy.

The same authors also published a new classification system-based on visual outcomes in Eales’ disease, characterizing the visual outcomes based on the severity of the disease.

Yet to date there is no standard classification system which is accepted and practiced.

Etiopathogenesis

It still remains unclear what the cause of the disease is, in spite of the several clinical and basic studies; however the following have been noted to probably have an association with Eales’ disease.

Tuberculosis

Pathological demonstration of tubercle bacillus was reported by Gilbert in 1935 and Stock in 1937. However Finoff in 1924 injected tubercle bacilli in 46 experimental animals and demonstrate vasculitis in only 1 eye. It appears that Eales’ disease may not carry viable organisms, but harbor nonviable organism or DNA of mycobacterium tuberculosis in significant number of cases. In a study, 11 out of 23 epiretinal membranes removed from eyes with Eales’ disease showed mycobacterium tuberculosis genome by nested PCR technique. However, culture of vitreous specimen did not show any growth of mycobacterium tuberculosis. It appears that Eales’ disease patients may not carry viable organisms, but may probably harbor nonviable organisms or DNA of mycobacterium tuberculosis in a significant number of cases.

Hypersensitivity to tuberculoprotein

Mantoux positivity has been reported in 42-98 % of Eales’ disease. Moreover, Mantoux is commonly positive in healthy adults in India and Eales’ disease was found in Mantoux negative patients too.

Parasitic infestation

Wania et al proposed possible association of Ascaris lumbricoides with Eales’ disease. There was no difference between the levels of IgM and IgG antibodies to Toxocara canis and Ascaris between Eales’ disease patients and controls in a study done at Sankara Nethralaya.

Neurological involvement

Various neurological lesions, such as multiple sclerosis, acute myelopathy, multifocal white matter abnormality, cerebral stroke, internuclear ophthalmoplegia, spastic paraparesis and hemiplegia have been reported. Other systemic diseases may be associated and are listed in Table 3.
### Immunology and immunopathology

Class I and class II HLA have been associated with Eales’ disease. HLA DQ2, DR52 and Bw6 were found in higher frequency in Eales patients and thus strongly associated with it. Experimental evidence also suggests autoimmune mechanism in the etiopathogenesis of Eales’ disease.

Recently Saxena and coworkers studied lymphocyte proliferative response against retinal S antigen, its uveitopathogenic fragments, yeast histone H3 peptide, interphotoreceptor retinoid binding protein (IRBP).

### Biochemical studies

Prathap et al has found raised α-globulin and decreased albumin levels in the serum of patients with Eales’ disease. Rangarajan et al has identified a distinct protein with molecular weight around 23 kda in serum of Eales’ disease. Sen et al found a raised α-1 acid glycoprotein levels in serum of the Eales disease.

A close relationship between the prominent neovascular proliferation in Eales’ disease and the intense expression of VEGF has been studied. The increased expression of VEGF, when compared to other conditions inducing neovascularisation, might explain the severity of neovascular growth and the propensity of repeated vitreous hemorrhages in Eales’ disease.

Rao et al has reported that the damage inflicted on the ocular tissue is due to reactive oxygen in uveitis. Lowered levels of antioxidant vitamins E and C and consequent accumulation of oxygen and lipid free radicals have been studied and could be the cause of the inflammation, neovascularization and retinal pathology in patients with Eales’ disease.

One of the major effects of oxidative stress on cellular membranes in patients with Eales’ disease is a decrease in platelet membrane fluidity. The decreased membrane fluidity suggests alterations in the physiological events, which may result in alterations in the functioning of retinal photoreceptors.

### Investigation in Eales’ Disease

Investigations are done to differentiate Eales’ disease from sarcoidosis, syphilis, tuberculosis and sickle cell retinopathy. The list of investigations in a case of Eales’ disease are enumerated in Table 4.

<table>
<thead>
<tr>
<th>Table 3. Systemic Diseases Associated with Eales’ Disease</th>
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<tbody>
<tr>
<td>Tuberculosis</td>
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<tr>
<td>Hypersensitivity to tuberculoprotein</td>
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<tr>
<td>Thromboangitis obliterans</td>
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<tr>
<td>Neurological disease</td>
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<tr>
<td>Multiple sclerosis</td>
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<tr>
<td>Acute or subacute myelopathy</td>
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<tr>
<td>Multifocal white matter abnormality</td>
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<tr>
<td>Cerebral stroke</td>
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<tr>
<td>Others</td>
</tr>
<tr>
<td>Focal sepsis</td>
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<tr>
<td>Hematological abnormalities</td>
</tr>
<tr>
<td>Acanthocytosis</td>
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<tr>
<td>Increased plasma viscosity, erythrocyte rigidity and erythrocyte aggregation</td>
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<tr>
<td>Hypereosinophilia</td>
</tr>
<tr>
<td>Blood coagulation disorder</td>
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<tr>
<td>Impaired oxygen release from blood</td>
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<tr>
<td>Raised fibrinolytic activity</td>
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<tr>
<td>Vestibuloauditory dysfunction</td>
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<tr>
<td>Parasitic infection (Amoebiasis, Ascariasis)</td>
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<td>Others</td>
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<tr>
<th>Table 4. Investigations for Eales Disease</th>
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<tr>
<td>To rule out leukemia and hematological disease:</td>
</tr>
<tr>
<td>- Hemoglobin (Hb) and Hemacrit (PCV)</td>
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<tr>
<td>- Total RBC count</td>
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<tr>
<td>- Total WBC count and differential count</td>
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<tr>
<td>Other tests:</td>
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<tr>
<td>- Platelet count</td>
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<tr>
<td>- Erythrocyte sedimentation rate</td>
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<tr>
<td>- Reticulocyte count</td>
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<tr>
<td>- Postprandial blood sugar</td>
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<tr>
<td>- Lysozyme (sarcoidosis)</td>
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<tr>
<td>- Mantoux test</td>
</tr>
<tr>
<td>- Basic coagulation test</td>
</tr>
<tr>
<td>- Bleeding time</td>
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<tr>
<td>- Clotting time</td>
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<tr>
<td>- Clot retraction</td>
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<tr>
<td>- Plasma clotting time</td>
</tr>
<tr>
<td>- Sickle cell preparation</td>
</tr>
<tr>
<td>- Hemoglobin electrophoresis (sickle cell retinopathy)</td>
</tr>
<tr>
<td>- Immunoglobulin profile VDRL and Treponema</td>
</tr>
<tr>
<td>- Pallidum Hemagglutination Test (TPHA)</td>
</tr>
<tr>
<td>- Anti-nuclear antibody (SLE &amp; other collagen diseases)</td>
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<tr>
<td>- Serum angiotensin converting enzyme (sarcoidosis)</td>
</tr>
<tr>
<td>Radiological tests:</td>
</tr>
<tr>
<td>- X-ray chest (tuberculosis and sarcoidosis)</td>
</tr>
</tbody>
</table>
Management

It depends on the stage of the disease which includes:

1. No-treatment with periodic evaluation in the regressed stage of perivasculitis or fresh vitreous hemorrhage.
2. Treatment with oral or peri-ocular steroid in active peri-vasculitis stage.
3. Laser photocoagulation in case of the neovascularization of retina or optic disc, or gross capillary non-perfusion.
4. Vitreous surgery is indicated in non-resolving vitreous hemorrhage (usually > 3 months).
5. Any associated retinal detachment warrants a vitreoretinal surgery.
6. The roles of anticoagulant hyperbaric oxygen and anti-tubercular treatment remain controversial.

A particular patient may require one or more of the above modalities to treat Eales’ disease. Early and aggressive treatment of Eales’ disease have been shown to be beneficial in terms of anatomical and visual outcomes.

Observation

Patients with inactive vasculitis can be observed periodically at 6 months intervals. Patients with fresh vitreous hemorrhage are observed at an interval of 4-6 weeks, if the underlying retina is attached by ultrasound or indirect ophthalmoscopy. Such vitreous hemorrhage often clears by 6-8 weeks.

Medical therapy

Corticosteroids remain the mainstay of the treatment. There is no definitive dosage in active retinal perivasculitis stage. Dosage is tailored for each patient based on the severity of the inflammation. Some require a maintenance dose of 15-20 mg/day for 1-2 months. In case of associated macular edema one may add depot steroid injection. There are reports of intravitreal steroids being used for cases of Eales’ disease with favorable outcomes.

In general, the response to corticosteroid was found to be extremely good in several studies. Therefore the need for cyclosporine and other immunosuppressive agents is limited in Eales’ disease. As many investigators believe that hypersensitivity to tuberculoproteins play a role in the etiology of Eales’ disease, anti tubercular therapy has been given in Eales’ disease empirically which includes rifampicin 450 mg and isoniazid 300 mg once daily for a period of 9 months. In patients with positive Mantoux and active perivasculitis, oral corticosteroids and antitubercular therapy has been recommended. However the role of ATT in Eales’ disease treatment remains controversial.

Recently, low dose oral methotrexate pulse therapy (at a dose of 12.5 mg/week) has been described to be clinically effective in Eales’ disease, and was found to be associated with an acceptable safety profile.

Photocoagulation

It is the mainstay of therapy in proliferative stage of Eales’ disease. In case of gross capillary non-perfusion, photocoagulation is suggested. Currently laser photocoagulation is mostly used due to obvious advantage of reaching the periphery where retinal neovascularization and ischemia is mostly observed. Argon green laser is used usually. In case of significant cataract or mild vitreous hemorrhage red krypton laser can effectively be used. This is delivered by slit lamp or indirect ophthalmoscopy. Following vitrectomy, an endolaser probe or indirect ophthaloscope laser can be used for laser delivery on the operating table.

Aim of photocoagulation in Eales’ disease is to regulate the circulation by diverting blood from hypoxic areas to healthy retina, (thereby reducing the formation of vasoproliferative factors), to obliterate surface neovascularization and close leaking intraretinal microvascular abnormalities. In patients with retinal neovascularization direct treatment with moderately overlapping burns is suggested. In case of elevated neovascularization photocoagulation of the feeder vessels beneath the frond is done.

Aneurysms and arteriovenous shunts are also treated in a similar fashion. Pan retinal photocoagulation is necessary when there is optic disc neovascularization. There are few minor complications associated with laser photocoagulation. Retinal hemorrhages are possible in a few cases but major bleeding is uncommon with proper selection of intensity and other parameters of photocoagulation. Occasionally retinal gliosis laid down by the regressing new vessels undergoes further contraction and cause a variety of retinal complications.
such as macular distortion due to epiretinal membrane and retinal tear resulting in retinal detachment. Laser photocoagulation can be done once the inflammation has subsided reasonably with anti-inflammatory medications like steroids as it is not advised in the active inflammatory stage which can worsen the neovascularization due to the angiogenic factors liberated.

**Vitreoretinal Surgery**

Vitrectomy alone or combined with other vitreoretinal surgeries is often required in Eales’ disease (Please refer to the flowchart for treatment plan of vitrectomy). The aim of vitreous surgery is to clear vitreous opacities and evaluate the fundus for neovascularization. Along with vitrectomy, laser photocoagulation can also be performed by endophotocoagulation or indirect laser ophthalmoscopy. A standard 3 port pars plana vitrectomy is the method of choice. Patients with fewer episodes of vitreous hemorrhage and preoperative laser photocoagulation have a better visual prognosis. Early vitrectomy has been found to yield significantly better results as compared to deferred vitrectomy.

**Anterior retinal cryoablation (ARC)**

ARC is considered in small undilating pupil, hazy media due to cataract, after-cataract, or residual hazy vitreous, where it is usually reserved as adjunct to photocoagulation.

**Anti VEGF Agents**

Kumar A et al recently reported a successful and rapid regression of disc and retinal neovascularization in a case of Eales’ disease after intravitreal bevacizumab. These agents may prove as important adjunctive agents in the management of Eales’ disease in future, especially in patients who experience recurrent hemorrhages due to aggressive posterior segment neovascularization.

**Summary and Conclusions**

Eales’ disease, with its characteristic clinical features and fluorescein angiography findings is a specific vitreoretinal disease. The disease can mimic several other ocular and systemic diseases presenting as retinal vasculitis or proliferative vascular retinopathies. Since its original description, many investigators have considered an association with tuberculosis to be the prime cause of this disease. Recent immunological, molecular biological, and biochemical studies indicate a probable multifactorial etiology. Human leucocyte antigen, retinal autoimmunity, mycobacterial tuberculosis genome, and free radical mediated damage play their role in etiopathogenesis of the disease.

Although its etiopathogenesis remains unclear, the management options are quite well established. Systemic corticosteroids have been found to be beneficial in active perivasculitis stage. Photocoagulation
is indicated in cases with gross capillary nonperfusion or retinal neovascularisation. Results of vitrectomy in non resolving vitreous hemorrhage with or without retinal detachment are satisfactory.

References
A Rare Case Of Subfoveal Choroidal Neovascular Membrane In Radiation Retinopathy- Combination Therapy Works...

Dr Gopal S Pillai, MD DNB FICO FRCS, Dr Abhijith Khake MBBS (DNB), Dr Lakshmi Nisha Menon DO (DNB), Dr Meenakshi Dhar MS, Dr Anuradha Rao MS DO, Dr Lilan Bhat MS DNB

Radiation retinopathy is commonly seen 1-2 years after radiation and occurs due to delayed retinal microvascular changes in the endothelial layer leading to capillary occlusion and microaneurysm formation. The fundus changes are similar to diabetic retinopathy changes. Subfoveal choroidal neovascular membrane in radiation retinopathy has been reported only once before in the literature, but it had not been treated successfully. We report such a rare case of radiation retinopathy which lead to the formation of a subfoveal choroidal neovascular membrane (CNVM) and was treated successfully with intravitreal bevacizumab (avastin) followed by Photodynamic therapy (PDT).

A 39 year old male who had undergone excision of hemangiopericytoma of left orbit followed by chemotherapy and radiotherapy 18 months back presented to the vitreo retina services with diminution of vision associated with metamorphopsia in his left eye of 2 weeks duration. There was no history of flashes or floaters, any recent stressful event, or any steroid intake. He was having watering and redness of the left eye for the last year. BCVA in the left eye was 6/9, and apart from the conjunctival congestion and dry eye, the anterior segment examination of the left eye was within normal limits. There was no corneal edema, cells or flare in the anterior chamber, good anterior chamber depth, normal pupillary reaction and accurate projection of rays. Fundus examination revealed a few microaneurysms and two cotton wool spots near the arcades suggesting a diagnosis of radiation retinopathy. Surprisingly there was also a hemorrhage seen in the subfoveal area. On further investigation with OCT there was a shallow sensory neural detachment at the fovea. On account of a suspicion of choroidal neovascular membrane he was advised a fluorescein angiography.

But unfortunately the patient came back to us after 1 month and his vision at this point of time had decreased further. The visual acuity in the left eye had dropped to 6/36 and fundus examination showed enlargement of the hemorrhage in the subfoveal region in the left eye. A sensorineural detachment was noticed around the hemorrhage at this time. Fluorescein angiography was done which showed a late leakage of the dye around the site of the hemorrhage. Thus a diagnosis of choroidal neovascular membrane was made on fluorescein angiography. OCT showed thickening of the foveal area with subfoveal detachment and cystoid macular edema with increased hyperreflectivity of the pigment epithelial layer.

We had discussed the various treatment options with the patient. Since there was no documentation of any successfully treated choroidal neovascular membrane with radiation retinopathy in the literature, and due to obvious economic reasons, the patient preferred an intravitreal bevacizumab injection. Intravitreal avastin
Fig. 1 Initial picture showing macular hemorrhage

Fig. 2 Initial OCT showing sensorineural detachment

Fig. 3 Hemorrhage has increased in 1 month

Fig. 4 OCT at 1 month showing the CNVM

Fig. 5 Hemorrhage and sensorineural detachment has increased after avastin injection

Fig. 6 OCT shows worsening after avastin injection

Fig. 7 Hard exudates after PDT and avastin

Fig. 8 OCT shows resolution

Fig. 9 Hard exudates resolving at 6 months

Fig. 10 OCT becomes normal after 6 months
injection was given after 2 days and he was advised to follow after 2 weeks.

When he came for follow up after 2 weeks his visual acuity had further dropped to 5/60 and fundus examination showed enlargement of the hemorrhage with increase in surrounding macular edema. His OCT showed increase in the thickness and hyperreflectivity at the RPE layer suggesting an increase in activity. Since the visual acuity was reducing day by day, we had planned our next step, i.e. Photodynamic therapy with intravitreal bevacizumab. He underwent the procedures the same week.

Follow up visit showed a decrease in the hemorrhage size and an increase in the amount of hard exudates. There was significant decrease in the macular thickness, cystoid macular oedema and sensorineural detachment on OCT. His vision had improved to 6/36. Since the patient showed improvement in vision and OCT, despite the emergence of hard exudates, the patient was advised a review after 1 month.

At the time of his next review his visual acuity improved to 6/18, fundus examination showed complete disappearance of the hemorrhage as well as the hard exudates. His OCT showed that the retinal thickness had normalized with normal foveal depression.

So to summarize, this patient had radiation retinopathy and developed a choroidal neovascular membrane with significant macular thickening, cystoid macular oedema, sensorineural detachment, hard exudates and edema. After the avastin injection, the edema and macular thickening increased. With PDT and a second avastin treatment, the thickness decreased at a much faster rate and more completely with the resolution of the subfoveal hemorrhage as well.

Discussion

Choroidal neovascular membrane in radiation retinopathy is very rare. There are only 2 documented case reports of choroidal neovascular membrane after radiation therapy. Out of the 2, one was far from the fovea and the other was extensive and untreatable. The extrafoveal area of neovascularisation in the first case was lasered. But this approach is not feasible in cases of subfoveal choroidal neovascular membrane, which develop at the fovea. In such situations, combination treatment with photodynamic therapy and anti VEGF agents may prove to be of benefit.

In this case, the reason for the development of choroidal neovascular membrane is not known. It is known that a Bruchs membrane break is needed for the development of a choroidal neovascular membrane. Whether radiation can lead to a Bruchs membrane defect is debatable. The other possibility is a recurrence of the orbital tumor into the nearby structures. But after a follow-up of almost a year, the choroidal neovascular membrane has not increased further. Further follow-up is required to know the prognosis of this case as the radiation retinopathy and the choroidal neovascular membrane may progress with time.

References

Introduction

The incidence of dengue has increased dramatically in recent years and Kerala is at present in the throes of a viral fever epidemic comprising chiefly of chikungunya and dengue fever. Previously, ocular findings were considered rare in dengue fever, however, various types of intraocular haemorrhage, vasculitis, disc oedema and retinal pigment epithelitis have been described in small case series. Here we present a case of viral fever (serologically proven dengue fever) in which the patient developed a subhyaloid haemorrhage in one eye and presented with acute unilateral visual loss.

Case Report

A 14 year old female presented with history of sudden loss of vision right eye of 3 days duration. She gave a history of fever, anorexia, myalgia and vomiting for the past one week for which she was on treatment.

On examination, vision in the right eye was counting fingers at ½ metre, and 6/6 in the left eye. Anterior segment was within normal limits. Dilated fundus evaluation showed a large subhyaloid haemorrhage over the macula in the right eye (Fig 1). Left eye was normal. A baseline laboratory investigation showed reduced platelet count (1.12 lakh/mm$^3$), bleeding time of 1 minute 20 seconds and a clotting time of 3 minute 20 seconds.

A Nd Yag hyaloidotomy right eye was done and the subhyaloid blood drained (Fig 2a, b, c). She was referred to a physician to rule out dengue. Dengue antibody titre was found to be positive and she was started on treatment for the same.

On review 10 days later, her vision had improved to 6/9 in the right eye. Fundus evaluation showed adequate clearing of subhyaloid haemorrhage in the right eye with small vitreous haemorrhage in inferior part of vitreous cavity (Fig 3). Her platelet count had increased to 3.34 lakh/mm$^3$.

Discussion

Dengue fever may occur in two forms- the classic form and haemorrhagic form. Common symptoms such as fever, headache, prostration, myalgia, nausea and retro orbital pain occur in both whereas haemorrhagic signs and other signs of severe disease such as shock, gastrointestinal bleeding, petechiae, epistaxis, abdominal pain, effusion and death are strongly associated with haemorrhagic dengue fever.

The ocular manifestation include subconjunctival haemorrhage which is the commonest eye finding 1. Other studies have described macular haemorrhage as the principal finding 2. Fundus findings are dilation and tortuosity of vessels, superficial retinal haemorrhages both macular and retinal, cotton wool spots and hard exudates, maculopathy, diffuse retinal oedema, peripapillary haemorrhage, vitreous cells, optic disc oedema, retina vasculitis, exudative retinal detachment and anterior uveitis 3.

The fluorescein angiographic findings included poor choroidal flushing, delayed disc filling, disc extravasations, blocked fluorescein, capillary obliteration, non filling of macular network, capillary leakage and window defect 2, 4.

It has been found that dengue fever patients with significant thrombocytopenia (<50,000 cells/mm$^3$) are predisposed to spontaneous ocular haemorrhages.
described. The thrombocyte count in this case was only borderline and not significantly reduced. However, severe vomiting associated with the fever may have led to the haemorrhage, (Valsalva retinopathy).

Premacular subhyaloid haemorrhage may occur from proliferative diabetic retinopathy, ruptured retinal macroaneurysm, neovascularisation in branch retinal vein occlusion, Valsalva retinopathy and Terson Syndrome resulting in sudden profound visual loss. Valsalva retinopathy can also result in subconjunctival haemorrhage. The common causes are forceful coughing, sneezing, weight lifting, intercourse and other strenuous activities. Forced expiration against a closed glottis can lead to sudden increase in intrathoracic and intra-abdominal pressure thereby suddenly increasing pressure in the veins of head and neck leading to haemorrhage.

In young patients where there is no posterior vitreous detachment this blood collects in the premacular area resulting in a boat shaped haemorrhage with horizontal upper level.

Premacular haemorrhages can be managed conservatively, by vitrectomy or hyaloidotomy. Nd-YAG laser is used for creating a hole in the posterior hyaloid \(^7,8\). It is done where the blood is thickest and farthest away from the fovea. 1-4 shots of 3.8-4.2 mJ are used with Goldman 3 mirror contact lens for focusing after local anesthesia instillation. Spontaneous clearance may take many months. Long standing subhyaloid haemorrhage may result in poor visual outcome if a fibrotic epiretinal membrane develops. Altered blood products may result in pigment alteration in the macula too. However, with treatment, very good results are obtained.
Conclusion

In a patient with subhyaloid haemorrhage and no other cause, a platelet count and antibody titre for dengue is necessary especially if there is a history of fever preceeding the episode of bleeding.

References


“OF PUPILS..... AND TEACHERS”

Mind Your Language
RRV (Dr. Varma)

Debates go on among the medical fraternity as to how to tell and how much to tell the patient for the ‘informed consent’. Most of our patients are knowledgeable and hence easy to educate. The net and innumerable lay ‘medical’ periodicals have obviated the need for detailed explanations. Some twenty years back communicating technical details was like two people talking in two different languages. Sometimes there is the actual language barrier. Kochi, being a melting pot of cultures, has people who talk Hindi, Punjabi, Marathi, Gujarathi, Sindhi, Bengali, Konkani, Thulu, Kannada, Tamil and Telugu, not to mention the curious lingo of Kudumbis. And the tourist season brings in most of the European languages into our consultation rooms. [My neighborhood shop-keeper had been in the Merchant Navy and helped me out once with a Greek patient]. It is so frustrating not being able to explain. Of course, in the land of Kathakali, we can resort to mudras to a certain extent; yet.....

The year was 1980 and I was a P.G.student in the B.J.Medical College, Ahmedabad. A boy was brought with history of a goat butting him in the face. He was the son of migrant Tamil labourers and had extensive lacerations of the upper tarsus. To my Registrar, all space below the Vindhyas was Madras. And of course everyone spoke ‘Madrasi’. (Actually when I talked about Malayalam one of my classmates had asked me: “Malayalam is spoken in Malaysia, chhe na, Varma Bhai?”) I, as a fellow ‘madrasi’ was asked to explain the prognosis to the parents. My linguistic skills were still underdeveloped back then. So I thought in Malayalam, translated it to my version of Tamil, and explained to the by-standers quite lucidly, or so I thought. After I finished (and sighed in relief), the father of the boy said in suddh Hindi: “Doctor Saab, will you please repeat in Hindi what you have just said?”

I had once watched the late Dr. M.G.Krishnan deal with a North Indian patient quite efficiently. He wanted her to lie down and didn't know how to say so in Hindi. Nor did me, his helper. He thought for a while, pointed to the examination couch and with great aplomb, made a sound, “PDKO” (don’t ask me to pronounce it). The patient quietly went and lay down.
Acute Posterior Multifocal Placoid Pigment Epitheliopathy - A Case Report

Dr Bini S T MBBS, Dr Biju John MS DNB FRCS, Dr Pravada, N. MS DO

An 18 yr old male presented at the outpatient department with decreased vision in the right eye of seven days duration and blurring of vision in the left eye of three days duration. Visual loss in the right eye was rapidly progressing and was not associated with pain, redness or photophobia.

There was no history of floaters, flashes of light, scotomas, or coloured halos around the light or any associated micropsia, macropsia or metamorphopsia.

There was no relevant systemic or ocular illness in the past or any relevant ocular illness in the family. General examination and systemic examinations were normal. Best Corrected Visual acuity was 5/60 in the right eye and 6/18 in the left eye.

Slit lamp examination in the right eye showed few cells and strands in the anterior vitreous while the left eye showed only few cells, and no strands. Intraocular pressure was 18 and 16 in the right and left eye respectively.

Ophthalmoscopic examination revealed multiple small subretinal yellowish-white, round, discrete, flat lesions < 1/4th disc diameter in size scattered all over the posterior pole. More confluent lesions were present near the macula. Lesions were scattered temporal to the macula and nasal to the disc up to the midperiphery. The peripheral lesions were more oval and more ill defined. Associated retinal edema was present and the disc was normal.

Left eye also showed similar lesions in lesser numbers (Fig 1 & 2).

Fundus flourescein angiography showed hypofluorescence in the choroidal and arterial phase due to blocked fluorescence. In the early venous phase there was leakage, and in the late phases there was staining corresponding to the lesions (Fig 3).

Field examination with Humphrey field analyzer revealed a paracentral scotoma corresponding to the lesions in the right eye while the field charting of the left eye was normal. Laboratory evaluation revealed a normal blood and urine routine examination except ESR which was 40 mm/hr. ANA showed a positive result. Mantoux test...
and Rheumatoid factor were negative. Serology for syphilis was negative. Chest X ray was normal. He was diagnosed as having Acute Multifocal Posterior Placoid Pigment Epitheliopathy [AMPPE] and treated with tapering dose of systemic steroids. Clinical improvement was noted in the right eye after 3 days of treatment, but the LE developed more lesions with worsening of vision initially and started showing improvement after one week (Fig 4). At the end of 2 weeks BCVA in the right eye was 6/18 and left eye was 6/12 and by the 4th week it improved to 6/9 in both eyes.

is a disorder characterized by the sudden appearance of multiple, yellow white, flat inflammatory lesions at the level of the retinal pigment epithelium and choriocapillaris. A flu-like prodrome consisting of fever, malaise and headache precedes most cases of AMPPE. This is followed by a sudden, usually bilateral, painless loss of vision. In patients with a monocular onset of symptoms, involvement of the fellow eye may occur within the following days to weeks 4. Central or paracentral scotomas may occur in patients with retinal lesions involving the foveal or parafoveal areas. Fundus examination reveals the characteristic multiple round, circumscribed, flat, yellow-white subretinal lesions involving the retinal pigment epithelium 5. As these lesions resolve over several weeks, vision improves in most cases to slightly less than initial acuity, and in some patients acuity may return to pre-onset levels 6. With time, fundus lesions are replaced by areas of depigmentation and pigment epithelial clumping. Additional ocular findings may include episcleritis, anterior uveitis, vitritis, retinal vasculitis, and papillitis.3,7,9 Associations with cerebral vasculitis, and erythema nodosum along with a host of immune-mediated disorders have been reported 9-12. For this reason, it is suggested that all patients with AMPPE should undergo a systemic and neurologic evaluation. Fluorescein angiography reveals characteristic changes during the evolution of the disease. During the acute, active stage of the disease, early films disclose areas of hypofluorescence in inflamed areas, secondary to RPE cell edema, leukocyte infiltration, and capillary nonperfusion. However, hyperfluorescence occurs in late films, as leakage occurs from the choriocapillaris through damaged RPE cells 5. During the inactive stage, as AMPPE lesions resolve, areas of hyperfluorescence occur at these sites secondary to RPE atrophy.

Discussion

Originally described by Gass 1 in 1968, acute posterior multifocal placoid pigment epitheliopathy (APMPPE),

Two months later the patient had vision of 6/6 both eyes and the only residual finding was a mild granularity of the right fovea (Fig 5).
Indocyanine green angiography reveals areas of choroidal hypofluorescence during the acute stage of the disease, resulting from capillary non-perfusion, and these persist during the later stages of the disease.

Gass and colleagues suggest that inflammation begins at the level of the retinal pigment epithelium\textsuperscript{13}. Others, however, propose that the disorder primarily involves the choriocapillaris, and acute inflammation at this level occurs secondary to a hypersensitivity reaction to an external antigen, leading to occlusion of choroidal arterioles, ischemia, and secondary RPE changes\textsuperscript{14}.

Although the ocular disease has a self-limiting course, with approximately 80\% of untreated patients having a visual acuity of 20/40 or better, 20\% are left with impaired vision. Therefore all patients with APMPPE with macular involvement should be treated with systemic steroids. Use of systemic steroids rapidly resolves inflammation, and may result in a better final visual outcome.

References

A 9-year-old boy presented with complaints of defective vision in right eye of 6 months duration. On examination his visual acuity was 6/18 N6 in the right eye and 6/6 N6 in the left eye. Anterior segment examination was within normal limits. IOP was 12 mm Hg in both the eyes. Detailed fundus examination revealed a yellowish subretinal lesion with a tinge of haemorrhage nasal to the fovea (Figure 1A). Fundus examination of the left eye was within normal limits. OCT examination revealed a hyperreflective band in front of RPE Choriocapillary complex suggestive of choroidal neovascular membrane (CNVM) in the right eye. Fundus fluorescein angiography showed a well-delineated area of early hyperfluorescence, which was about $1/4^{th}$ disc diameter in size and located inferotemporal to the fovea surrounded by an area of hypofluorescence (Figure 1B). As the angiogram progressed, the hyperfluorescence increased in size and
intensity with fuzzy margins in the late phase suggestive of extrafoveal choroidal neovascular membrane in the right eye (Figure 1C). FFA of the left eye was within normal limits. There was no evidence of past inflammation or trauma. The child was diagnosed to have idiopathic choroidal neovascular membrane and was given thermal laser to the extrafoveal CNVM in the right eye. Figure 1D shows colour photo immediately after laser treatment. In the last follow up visit at 6 months, visual acuity in the right eye remained at 6/18 N6. Repeat OCT showed scarring of the CNVM with no retinal edema or subretinal fluid.

Discussion

CNVM is a rare entity in pediatric population. Inflammation, trauma, optic disc drusen and idiopathic causes have been described. Some of them undergo spontaneous involution. Treatment with thermal laser and submacular surgery are reported to be effective in a number of cases. Here we present a case of idiopathic CNVM in a 9 year old boy with no evidence of inflammation or trauma. Since the location of CNVM was extrafoveal it was amenable to thermal laser. He is not having any recurrence at 6 months. CNVM should be kept in mind as a rare cause of defective vision in pediatric age group.

References

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A 24 year old male patient was first seen here on 08/04/98 with complaints of defective vision of the right eye 6 years and recurrent episodes of redness and watering in the right eye. On examination - Vision was 2/60 is the right eye and 6/18 is the left eye with lid edema, corneal opacity and papillae on palpebral conjunctiva. He was diagnosed to have vernal conjunctivitis and treatment was instituted. On later visit, keratoconus in right eye was detected. He had recurrent attacks of vernal kerato conjunctivitis (VKC). In 2000, he developed steroid induced glaucoma which was treated with steroid withdrawal and antiglaucoma medications. He subsequently developed posterior subcapsular cataract both eyes for which cataract extraction with posterior chamber intraocular less implantation was done in the right eye (2000) and left eye (2000). Postoperative vision was 6/12 in the right eye and 6/9 in the left eye. By 2004, due to recurrent attacks of VKC, he had developed corneal opacity (right eye) with 360° vascularisation and V/A steadily declined. When seen last (21/09/06) visual acuity was 2/60 on the right eye and hand movements left eye. Applanation tonometry was 8 and 100 mm Hg in the right eye and left eye respectively. Pachymetry was 409 μm, 479 μm in the right and left eye respectively.

How would you proceed to manage this case?

Rasik B Vajpayee

The patient presented has bilateral pseudophakia with secondary keratoconus and poor ocular surface. A comprehensive ocular evaluation should include assessment of ocular surface and accurate estimation of potential visual acuity by Laser Retinometry. The vascularized corneal opacity in the central part may have been caused due to persistent low grade limbal inflammation resulting in a deficiency of limbal stem cells or it may be a result of occurrence of shield corneal ulcers typical of vernal catarrh. It is also possible, considering the history of recurrent pain and redness, a recurrent herpes simplex keratitis may have caused this. Any giant papillae present in the upper palpebral
conjunctiva should be checked for and if present, should be treated optimally. Giant papillae of vernal catarrh rub against the cornea and cause chronic ulcers called as Shield Ulcers. Additionally ocular surface should be improved with tear substitutes. Excision of corneal scar tissue and an ‘On Lay’ Transplantation of Amniotic membrane with cultured stem cells would help in improving the ocular surface and increasing the corneal clarity and vision. If required and if the results of laser retinometry test are normal, a subsequent DALK or penetrating keratoplasty may be performed to further improve the visual acuity.

The reduction in visual acuity of the left eye may have been caused due to any single or multiple factors. These may be steroid induced glaucoma, after cataract, and high astigmatism due to a possible presence of keratoconus. The complete ophthalmic evaluation should also include specular microscopy to rule out presence of corneal decompensation. The photograph of the left eye reveals presence of some haziness of the cornea in the upper nasal half which may represent corneal edema due to corneal endothelial decompensation. However, looking at the corneal pachymetry findings, the most probable cause of low vision may be the presence of keratoconus which would require appropriate management starting from spectacles and contact lenses.

**Virender S Sangwan**

To me it seems it is a chronic advanced case of VKC with blinding complications of glaucoma and severe ocular surface disease. From the history it is not clear whether the patient has itching or not. This part of the history should normally be volunteered by the patients because the itching is so intense. He also has keratoconus. Now we have this young gentleman with pseudophakia with severe ocular surface disease and keratoconus. First thing we have to establish is the contribution from each factor in the decrease in vision. The factors responsible for decrease in vision are keratoconus, severe ocular surface disease and glaucomatous optic damage. Another issue we need to address is whether the VKC is active or not? If it is active then we need to use appropriate medications to control the ongoing allergic eye disease and plan for visual rehabilitation. From the slit lamp photographs provided it looks like there is an ongoing disease and I am not sure if there is an ulcer in the right eye. If there is an ulcer that needs to be treated appropriately, if it is a shield ulcer with no infection then it should be treated with intense topical steroids. If infection is suspected then treatment should be guided by corneal scrapings and culture sensitivity. Presence of superficial vascularisation and or pannus indicates the presence of stem cell deficiency. I do not have the disc changes or disc photographs so it is not possible to comment on what stage is the glaucomatous damage and whether this component needs active intervention or management.

Based on the response to the questions that I have raised my plan is as follows: 1. Control the ongoing inflammation with topical and or systemic medications including the management of ulcer in the right eye. 2. Address the ocular surface management medically, essentially using preservative free lubricating drops and gel. 3. For visual rehabilitation first I would think of non surgical options such as use of Boston contact lens which we have at LV Prasad Eye Institute and that will also help us to assess the visual potential of the eye. 4. If the vision does not improve with the Boston contact lens and we think that corneal condition is responsible for poor vision, in that situation I would think of cultivated limbal allograft with or without LK/DLK to be followed by penetrating keratoplasty if required. Of course these management steps will be sequential and will take its own time and the success of one step would determine the next intervention.

**Dr Rajesh Fogla**

At present this patient has active recalcitrant vernal conjunctivitis, with keratoconus, and limbal stem cell deficiency. I think we need to treat him in a stepwise manner. There is significant amount of surface inflammation in both eyes. As he has been noted to be a steroid responder, topical steroids cannot be used frequently. I would start him on a short course of systemic steroids after clearance from the physician. Topically he can be started on fluorometholone 6 times daily, cyclosporine 1% 4 times daily, genteval gel 4 times daily and tetracycline ointment at bedtime. Intraocular pressure needs to be monitored at periodic intervals. One can also consider supratarsal injection of
corticosteroids to manage the giant papillae on the tarsal surface of upper lids. If his surface is not intact, one can also consider performing an amniotic membrane transplantation surgery. Once the surface inflammation and vernal activity is under good control, this patient would require a deep lamellar keratoplasty, live related limbal allograft, with amniotic membrane transplantation to restore his eyesight. In case he has some stem cell function still active, then limbal transplantation can be avoided.

Dr. Quresh B. Maskati

The left eye seems to be doing fine. The eye is quiet, there does not seem to be any active allergic conjunctivitis; cornea is fairly clear and the IOL appears in situ. Nothing much needs to be done except to monitor the glaucoma status in the left eye.

The right eye on slit lamp does not seem to have keratoconus. However, there is a severe stem cell deficiency, with vascularisation of cornea and corneal opacity. This eye needs to be treated with topical patanolol hydrochloride drops (Patanol - Alcon or Winolap or Olopat eye drops). The glaucoma also needs medical therapy. As the vision is poor, one can try a deep anterior lamellar keratoplasty with guarded prognosis. A penetrating keratoplasty can be tried if one is not well versed with DALK. The limbal vessels can be cauterised using Dr. Harminder Dua’s technique of a fine needle cautery at the time of surgery. However, postoperatively the patient will need aggressive management in view of expected surface problems due to his stem cell deficiency, with lubricants and frequent steroids, keeping a close watch on his IOP. If the graft fails, we may either leave the eye alone or do a living related or cadaveric stem cell transplant with a new corneal graft and put the patient on immunosuppressive medication for a long long time. However, 5 year survival of such grafts is less than 50% currently.

Dr. C.V. Anthrayose

This is the unfortunate case history of an young patient who had been suffering from vernal keratoconjunctivitis (VKC) and all its complications; with a probable clinical diagnosis of Ocular Surface Disorder (OSD) in his right eye and fairly clear cornea with probably optic atrophy in the left eye. VKC is one of the known causative factor producing OSD because of the Limbal Stem Cell Deficiency (LSCD). LSCD is produced by the loss of goblets cells and damage to the conjunctival epithelium at mucocutaneous junction producing dry eye. Now the patient’s right eye has all the hallmark signs of LSCD like conjunctivalization and neovascularisation, due to chronic inflammation with visual loss.

Suggested Management

The management of this case involves the management of dry eye and adnexal condition. Since this is a case of right eye total LSCD with left eye fairly healthy, limbal stem cell with optic atrophy we can think of Limbal Stem Cell Transplantation (LSCT) with penetrating keratoplasty (PKP) in one sitting or in 2 stages. We can try one of the following alternatives for LSCT like direct conjunctival limbal auto graft or cultivated limbal auto graft on amniotic membrane from left eye or conjunctival limbal allograft from donor eye with simultaneous PKP or PKP at a second stage after 3 months.

Surgical steps

The surgical steps include preparation of recipient bed with peritomy, peeling of neovascular membrane, keratectomy with conjunctival limbal auto graft (CLAG) with amniotic membrane (AM) transplant or AM cultured CLAG and PKP as second stage, preferably after 3 months with a young donor cornea with good endothelial count. Left eye we can try only conservative line of management. We have to give a guarded surgical prognosis because of the dry eye with OSD in a pseudophakic secondary glaucoma patient.

Dr. Freddy T. Simon

In this case I would first try if the vision in the left eye could be improved. A topography of the left eye should be done and if there is keratoconus then it needs appropriate management. But the anterior segment photograh of the left eye does not show any gross keratoconus and the vision of HM only could not be due to keratoconus alone.

If the vision in the left eye cannot be improved then alone the right eye is to be tackled. The two options are
1. First an amniotic membrane with a limbal cell transplant and once the ocular surface is tackled a keratoplasty will be required. These procedures will have a lot of difficulties like
- Is there limbal deficiency in the left eye also since he has vernal disease in both eyes? If so he will need limbal cells either from a relative or an allograft.
- In both the above situations he will need long term immunosuppressives.
- Post keratoplasty he will need steroids and will this increase the IOP since he is a steroid responder?
2. The second option for the right eye will be an odontokeratoprosthesis once the eye is quiet.
For him to get any vision with either of these procedures he will need a fairly healthy optic nerve.

**Dr. Noel Moniz**

Subsequent to this case going to the consultants, a lot of change have taken place over the past 5 or 6 months. Almost all the consultants have the same management plans. This patient underwent an amniotic membrane transplant at a major referral hospital in his RE after which he was put on heavy topical steroids and antibiotics. Unfortunately he developed an infection under the Amniotic membrane and the cornea and the membrane melted. The IOP was controlled medically and he was put on aggressive topical antibiotic therapy to control his infection. Subsequently the infection was controlled and now he has got a totally vascularised cornea. Ultrasound showed a normal posterior segment.

Around this time the left eye developed an ulcer and this showed a good response to treatment. No cause for poor visual acuity in the left eye could be found. Though the refraction did show some change there were no signs of keratoconus in the left eye. An RGP lens did not increase his vision in the left eye.

The only hope, if at all, is in the right eye but before undertaking any further procedures it would be worthwhile knowing the status of his retina and optic nerve. Some idea could be got through electrophysiological studies of the optic nerve and retina. Also if a keratoplasty is being planned it would be worth doing an ultrasound biomicroscopy so that the surgeon will know what to expect.

All in all a very difficult case to manage and more disheartening is the fact that you see a young man going blind in front of you.

Compiled by Dr. Noel Moniz, L.F.Hospital, Angamaly
What is Computer Vision Syndrome?

Dr. Meena Chakrabarti MS DO DNB

Since computer use is such a visually demanding task, vision problems and symptoms have become very common in today’s workplace. Most studies indicate that computer operators who view their video display terminals (VDT) report more eye related problems than non VDT office workers. NIOSH Survey (National Institute of Occupational Safety and Health) has reported that visual symptoms occur in 75-90% of VDT workers as opposed to 22% musculoskeletal disorders (carpel tunnel syndrome) in computer users.

A series of visual symptoms which are a by-product of excessive viewing of VDT screens without proper attention to practical visual hygiene is called Computer Vision Syndrome (CVS).

This new found entity, frequently described in the lay press and world wide web has now been accepted in medical literature.

The cause of these visual complaints are a combination of individual visual problems, poor work place conditions and improper work habits. Prolonged work on the computers has been associated with diminished power of accommodation, removal of near point of convergence, and deviation or phorias for near which are transient.

The symptom of CVS have been divided into 4 categories 1) Asthenopic, 2) Ocular surface related, 3) Visual and 4) Extraocular.

Eyestrain (Asthenopia): A subjective complaint of uncomfortable, painful and irritable vision. In the VDT environment, eyestrain in all its manifestations can be caused by a number of different environmental and visual conditions. The symptoms of eyestrain manifests as headaches, focusing difficulties, burning, tired and aching eyes, dry eye symptoms, double vision or blurred vision, light sensitivity, neck or shoulder pain, changes in colour perception and pain in and around the eyes.

Headaches

Headaches are discomfort symptoms for which most computer users seek medical advice.

Characteristics of headache in computer users are its frontal location or one sided headache occurring in the middle or end of day. The patients are fine in the morning and the headache exhibits a different pattern during weekdays and weekends. Tension and stress, numerous eye conditions, improper work place conditions etc (glare, poor lighting, improper workstation setup) are responsible for this symptom.

Blurred Vision

Blurred Vision in a computer user could be due to a variety of factors such as refractive error, improper prescription lenses, age related focusing problem (presbyopia), dirty screen, poor quality monitors, poor viewing angle and reflected glare from the screen.

Dry and Irritated Eyes

Tear secretion covers the eye surface and maintains moisture for normal eye function. Tears also help to maintain oxygen balance of the external eye structures and to maintain the optical properties of visual system. Blinking facilitates resurfacing of precorneal tear film. Blink reflex rate varies depending on the activity that you are engaged in. The blink reflex rate becomes faster.
when you are active and slower when you are sleepy or concentrating. While you are at work on the computer, the blink rate reduces considerably because of your concentration on the task and relatively limited range of ocular movements.

In addition, the size of palpebral aperture widens on up gaze to view the VDT. This results in greater evaporative dryness and greater number of incomplete blinks both of which predispose to dry eye symptoms.

**Neck Ache and Back Ache**

“The eye leads the body” and hence nature has designed our visual system to be so dominant that we are forced to alter our body posture to accommodate for any deficiency in the way we see. In many office situations, if the vision of a worker is compromised he must adapt his posture to ease the strain on the visual system.

If an older worker is using single vision glasses, which are designed for a 16" viewing distance, they must lean towards the screen, which is 20"-25" away in order to clear the image.

If the older worker is using bifocals, which are designed to see near objects in the lower visual field, they must tilt their heads backwards and lean forwards to put the viewing section of the lenses into proper position to see the screen.

These situations will cause obvious physical problems.

(Tables 1 and 2)

**Light Sensitivity**

Discomfort due to glare is largely caused by disparities in brightness in the field of view. Overhead light fixtures, bright open windows, dark background display screen, white paper on the desk, light colored desk surfaces, desk lamps directed towards the eyes, all cause reflected light (or glare) to fall on the computer users’ eye.

**Double Vision**

When viewing a near point object, the eye muscles “converge” the eyes inward towards the nose. Convergence allows the eyes to maintain the alignment of the image on the same place on both retinas. When we lose the ability to maintain the “lock” between the 2 eyes, they misalign and aim at different points in space. If both eyes continue to transmit the image back to the brain, we will experience double vision.

Individuals with COMPUTER VISION SYNDROME may complain of any or all of the following symptom after a day’s work (Table 2).

<table>
<thead>
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<th>TABLE 2</th>
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<tbody>
<tr>
<td>List of common complaints for which a computer user seeks medical aid</td>
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<tr>
<td>1. Headache during or after working at the computer</td>
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<tr>
<td>2. Overall bodily fatigue or tiredness.</td>
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<td>3. Burning Eyes</td>
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<td>4. Distance vision is blurry on looking up from the computer.</td>
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<tr>
<td>5. Dry, tired or sore eyes</td>
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<td>6. Squinting helps while looking at computer</td>
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<td>7. Neck, Shoulder or Back pain</td>
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<td>8. Double Vision</td>
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<td>9. Letters on the screen run together</td>
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<tr>
<td>10. Driving / Night Vision is worse after computer use</td>
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<tr>
<td>11. ‘Haloes’ appear around objects on the screen</td>
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<tr>
<td>12. Forced to interrupt work frequently to rest the eyes</td>
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**Why Do Eye Strain Occur While Viewing The Characters on VDT (Videodisplay Terminal) or Computer Screen?**

The human focusing system responds very well to images that have well defined edges with good contrast between the background and letters.

Characters displayed on the computer screen are made up of many dots or pixels. Pixels are the result of electron beam striking the phosphor coated rear surface of the screen. The resolution of the images made of pixels is measured in dots/inch. Each pixel is brightest...
at the centre with the brightness decreasing towards the outer edges.

When a light meter with a small aperture is passed across a pixel, with the light amplitude being charted against luminance we get a light amplitude graph as shown in Fig 1 which compares the difference between a sharp edged print and fuzzy edged pixel. The characters in the LCD screen have sharper edges than those on the VDT monitors and are hence more comfortable to view.

* a pixel shows a bell shaped gaussian curve
* a printed character: a perfect square wave

Fig1: LIGHT AMPLITUDE GRAPH drawn using a lightmeter showing the difference between the appearance of a pixelated image and printed character.

The eyes have a hard time focusing continuously on the pixel characters on the computer screen. An effort is made to focus on the plane of the computer screen but usually they cannot sustain that focus.

Hence they relax to a point behind the screen termed as RPA (Resting point of Accommodation) or dark focus. The RPA is different for different individuals but for almost every one it is further away than the working distance to the computer. (Fig: 2)

Thus the eyes are constantly relaxing to the RPA and then straining to refocus on the screen. When the eyes have to do that for several times a day they naturally get tired.6-12

Many people suffering from blurred vision at the computer are in their 40’s or older.13 You probably only notice this problem at the computer because your monitor falls into the intermediate zone of your vision (as opposed to the near and the far), which you ordinarily don’t use much.

Middle aged and older people also have a little trouble with the ciliary muscles that control accommodation: the eyes ability to switch focus quickly (from the key board to the monitor and back). This “lag of accommodation” can lead to eyestrain and blurred vision.14

Computer monitor are often too close to the user because of the space constraints or lack of understanding of how the eye functions while working on a computer. Some young people whose near point is around 16 inches compensate for the closeness of the monitor with out significant eyestrain. Other who have binocular problems, uncorrected hyperopia and astigmatism have significant eyestrain and symptoms.

Constant effort is needed to focus near objects and to sustain accommodative spasm in which the focusing muscles lock into position and the eyes will not relax easily. This will lead to myopia (near sightedness) or pseudo myopia (When these eyes are examined they will accept minus powered concave lenses but when the eyes are dilated and the focusing muscles relaxed, the true prescription is reached). If this over focusing is not corrected, the pseudo myopia can become structuralized.15, 18

Examination for CVS:- There is a definite examination sequence for patients with visual and related complaints following prolonged computer use.

1. The most important finding for writing a “Computer Specific” prescription is the Manifest Refraction at distance.
2. Check for correction at 40 cm near point and also take care to assess whether the patient is comfortably and consistently binocular. If not assess the need for prisms to achieve binocularity.
3. Assess the patient’s computer working distance.
4. You can then make the patient sit at his actual working distance in front of a computer screen and based on your 40 cm near point evaluation, adjust the power by trial of loose lenses on the trial frame till he’s comfortable.
5. There are reliable methods (PRIOR VISION testers) developed to duplicate the light characteristics (pixelated image) of the computer screen, allowing the patients eyes to react in exactly the same way as when he sits in front of the computer. Trial of
glasses carried out at the patients working distance also help in arriving at the correction he needs to wear to avoid eyestrain.

**COMPUTER LENS DESIGN**

After obtaining the basic computer-distance–related sphero-cylinder prescription for your patient you are left with several choices to choose from after discussing the various options with your patient.

The computer monitor falls in the intermediate zone of vision. Normally regular glasses with out near vision addition are used for viewing objects at a distance, while the reading glasses help correct near vision. Bifocals correct both near and far vision while trifocals and progressive only have a small portion or the intermediate viewing, not large enough for comfortable computer work. Thus computer work requires the use of glasses which provides a larger intermediate zone viewing area.

One choice might be to select a single vision lens for the computer glass. This serves well for a person who works in a small cubicle all day with a distance visual demand of no greater than 4 feet. If the person needs to look around or across the room the simple vision lenses will not be adequate.

If your patient has previously worn a flat top segmental bifocal lens, you could prescribe an occupational trifocal design. The newer trifocals have a 14 mm x 35 mm ribbon intermediate segment with an add power set at 2/3 of the distance power. These lenses provide the width of field which is desirable in a computer environment.

Patients who are not comfortable with trifocals can be prescribed bifocals with either intermediate and near correction or intermediate and distance correction.

Ideally an occupational progressive lens that corrects near, intermediate and up to a point for distance (allowing the wearer to see a distance of about a room’s length) can be prescribed (Fig. 3 and Fig. 4). This lens tends to be poorly suited for regular wear. Commercially available occupational progressives are

1) TECHNICIA: has a wide intermediate field to facilitate computer viewing. The near zone is 25 mm wide and the add progression is such that 75 % of the full add power is achieved at a distance of only 9 mm below the major reference point (MRP). This lens also

has a small distance viewing area at the very top of the lens, so that the patient can see the office environment without a blur. Peripheral distortion is minimized by placing most of the unwanted astigmatism in the nasal portion of the lens.

2) SOLA “ACCESS” lens: This is a double progressive lens which offers clear viewing for both computer and distance with minimal peripheral distortion.

3) SHAMIR OPTICALS “OFFICE” Lens / (DESKTOP Lens):

4) VARILUX (INTERVIEW Lens)
"TO COAT" or "NOT TO COAT"? To answer those questions, you must know something about the patients’ working environment. If work is done at home, without fluorescent lighting and with windows where the amount of light that is let in can be controlled, the only coating you might consider is the anti reflection treatment of the lens (AR) \(^{(19)}\) (Fig. 5).

AR coating increases the efficiency of the lenses as a refracting medium. However the AR coating is still more difficult to keep clean even with the newest technology applications.

However, if work is done in a typical office setting with too bright fluorescent lighting you might want to consider two additional coatings along with the AR mentioned above.

400 nm Coating (UV Coating) : Most daylight or cool white fluorescent tubes have output that is rich in harsh, short wavelength light. This blue light is difficult for the human eye to focus due to its scattering characteristics. The UV coating eliminates blue component light to atleast some extent \(^{(18)}\).

Tint coating : In most offices the fluorescent lighting is too bright. A 10 % absorbing tint can reduce the eyestrain associated with such high light levels. In addition to the degree of tint, the hue of the tint can also help improve the visual performance. Amber coloured tint selectively reduces the illuminance for rods and cones.

Thus if your office has fluorescent lights you might want to consider both these options as well. UV coating can cut down on the amount of blue light that reaches the eye as can amber tint and hence makes focusing a lot easier.

9 steps to reduce computer eyestrain have been suggested by the National Institute of Health and Occupational Safety. These include

1. Regular, yearly complete eye examination.
2. Use proper lighting: Avoid excessive bright light coming from outside and excessive bright light inside. When you are using the computer your ambient lighting should be around 50 % of that in a regular office.
3. Minimize glare: Glare from the wall, reflective surfaces and computer screen can cause eyestrain. If possible paint the walls of the room a darker shade with a matte finish. Install an anti reflective screen for your computer. Reduce exterior lighting by use of blinds. Using anti glare-coated goggles will also help reduce the eyestrain (Fig. 6).
4. Adjust brightness of computer screen: Adjust also the contrast, text size and colour for optimum comfort. Match the computer screen to the brightness of the environment. The contrast between the background and on screen characters should be light.
5. Take frequent breaks: Full time computer users should take a 10 minutes break every hour to reduce eye strain.
6. Refocus your eyes: Look away from the computer screen every 10-15 min and focus on a distant object for 5-10 seconds. It also lets you blink, which wets your eye.
7. Blink more often: When staring at the computer, people blink less frequently- (about 5 times less than normal according to studies). Tears coating the eyes evaporate rapidly during the long non-blinking phases and cause dry eyes. Office buildings may have excessively dry atmosphere, which reduces...
tearing. For significant dry eye symptoms prescription of artificial tears may be helpful. It’s a good idea to blink every time you hit the “ENTER” key.

8. Modify workstation: Use a copy stand placed adjacent to the monitor if you need to look back and forth between the print on the written page and the computer. Purchase ergonomic furniture to assume proper screen location and posture.

9. Exercise even while sitting:

Sitting, stretching and joint rotating exercises have been advised for computer users

The results of a study on knowledge, awareness and practices in Indian ophthalmologists with reference to Computer Vision Syndrome showed that all doctors who responded to the questionnaire were aware of CVS. The main mode of treatment was by using tear substitutes in 50.7%. Ophthalmologists in this study were not prescribing any special spectacle nor did they have any preference for a specific type of glass or special filters. Computer users were likely to be prescribed sedatives/anxiolytics and advised frequent conscious blinking than non computer users.

Computer Vision Syndrome is a diagnosis of exclusion as almost everyone uses computers. Hence an universally acceptable diagnostic and grading system needs to be established and the tendency to label any vague collection of symptoms as CVS needs to be discouraged.

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“Instant Vision” Compared With Postoperative Patching: Clinical Evaluation and Patient Satisfaction After Bilateral Cataract Surgery

Eva Stifter, MD, Rupert Menance, MD.

The application of an eye patch after cataract surgery has been a long established and generally accepted routine, until it was abandoned recently. This has been replaced by the ‘no patch’ surgery which offers ‘instant vision’ to the patient. In this prospective randomized clinical trial the authors compare two methods of post operative dressing regimen: patching vs “instant vision” with out patch. Sixty consecutive hospitalized, nonambulatory patients with cataract surgery under topical anesthesia on both eyes on different days were enrolled prospectively. In randomized order, one eye was patched for the first 24 hours postoperatively: the other eye was left open without patch to obtain “instant vision”. Both eyes received the same anti-inflammatory and antibiotic drop therapy. Twenty four hours postoperatively, no significant differences between patching and “instant vision” could be found for corrected and uncorrected visual acuity, corneal epithelial defects, conjunctival inflammation, anterior chamber flare, and intraocular pressure. During the first 24 hours postoperatively, all tear film parameters were significantly worse in the “instant vision” eyes (P< .001), indicating a transient tear film instability. During the first four hours after cataract surgery, pain scores in the “instant vision” eye were significantly higher than the patched eyes (P< .001). Eight hours postoperatively and later, there were no significant differences in any pain scores. After experiencing both methods, 27% of the patients subjectively rated the two methods as equivalent; 65% of the tested patients preferred patching to “instant vision” because of lower pain and foreign body sensations and psychologic arguments. The authors conclude the study by saying that both methods were equally safe for postoperative therapy. However, further efforts have to be made to increase the patient’s comfort with “instant vision” in the first few hours after cataract surgery.

Presumed Infectious Endophthalmitis Following Cataract Surgery In The UK: A Case-Control Study Of Risk Factors

S Kamalarajah, R Ling, G Silvestri, NK Sharma, MD Cole, G Cran and RM Best.

Endophthalmitis still remains one of the most dreaded complications of modern cataract surgery despite refinements in surgical technique and use of prophylactic antibiotics. In this article, the authors
report variables affecting presumed infectious Endophthalmitis after cataract surgery with intraocular lens implantation.

Two hundred and fourteen clinically diagnosed patients with presumed infectious Endophthalmitis were compared with 445 control patients throughout the United Kingdom in a prospective case control study. The cases were identified through the British Ophthalmological Surveillance Unit reporting card system. Control patients undergoing cataract surgery from 13 ‘control centre’ throughout United Kingdom were selected randomly. Risk factors were identified by univariate and multivariate logistic regression analyses. Statistically significant risk factors in multivariate models included inpatient cataract surgery (P=0.001), surgery in dedicated eye theatres (P<0.001), consultant grade surgeon (compared to registrar) (P=0.001), posterior capsule tear during cataract surgery (P=0.001). The use of face masks by the scrub nurse and surgeon during cataract surgery (P<0.001) and the administration of subconjunctival antibiotics at the end of surgery (P<0.001) were protective against postoperative infection. Here surprisingly, dedicated eye theatre and consultant grade surgeons had higher rates of Endophthalmitis than multi specialty theatre and registrar grade surgeons. The authors pointed out that more in - patients (older and sicker) were operated in dedicated eye theatres. Also, complex cataract surgeries were more likely to be operated by consultants.

In order to minimize the risk of postoperative Endophthalmitis authors recommend the wearing of face masks by the surgeon and scrub nurse during cataract surgery and subconjunctival antibiotics at the end of surgery.

The Effect Of Aspirin and Warfarin Therapy In Trabeculectomy

CJ Cobb, S Chakrabarti, V Chadha and R Sanders

Currently there are no agreed guidelines on the pre- and postoperative management of patients on antiplatelet and anticoagulation therapy (APACT) in glaucoma surgery. Cataract surgery does not seem to have an adverse outcome in patients on APACT. Despite this a proportion of ophthalmic surgeons discontinue such therapy before cataract surgery. The authors aimed to establish the risk of surgical out come in patients on APACT in glaucoma therapy.

In this authors retrospectively examined 367 consecutive trabeculectomies performed between 1994 and 1998. Preoperatively 60 (16.4%) patients were on APACT (55 on aspirin and five on warfarin). The incidence of hyphema and hemorrhagic complications between patients with and without APACT was documented. Surgical success was defined in two categories as an intraocular pressure (IOP) <21mmHg and IOP <16 mmHg 2 years following trabeculectomy with and without antiglaucoma medication.

This study showed that none of the patients on aspirin suffered significant intra or postoperative hemorrhage. Aspirin was associated with a significantly higher risk of hyphema (P=0.0015) but this was not found to significantly affect IOP control at 2 years. Patients on warfarin suffered hemorrhagic complications and trabeculectomy failure.

The conclusion of the study is that aspirin appears to be safe to continue during trabeculectomy. Warfarinised patients are at risk of serious bleeding complications and trabeculectomy failure.

Reviewed by Dr. Praveen Suresh Talwar DO. Little Flower Hospital and Research Centre, Angamaly.
Optometry A-Z

Edited by Nathan Efron
Price: £ 99.00

This work is designed to supplement conventional books by providing an easily accessible ready reference source for information about all aspects of optometric practice. Entries are set out alphabetically in what is more of an encyclopedic than a dictionary approach. Thus, in comparison to a dictionary of the same size and covering the same material, Optometry A-Z contains fewer entries, but each entry is of greater length. Therefore, there are few narrow ‘technical definitions’ in this book; rather, the entries tend to cover more general themes.

Thus, this is a true reference text in that it is not really designed to be read from cover to cover.

The author has tried to give appropriate weight to entries to reflect their current importance in the field, and to adopt the terminology that is most widely used. Thus, while this book covers all aspects of optometry, it can not cover every topic within the various aspects. Extensive cross-referencing and the incorporation of alternative terminology (cited alphabetically with cross references to the primary entry) will hopefully allow readers to quickly find the information they are seeking.

The layout of the book is straightforward. Each term is set in blue type, and the descriptive text follows in plain black type. Useful cross-references are sometimes given at the conclusion of an entry, and occasionally key synonyms and antonyms are provided.

A comprehensive encyclopedia of all aspects of Optometry, including colour vision, eye disease, low vision, binocular vision, orthoptics, contact lenses, spectacle lenses, ocular anatomy and physiology, geometrical and physical optics, optometric techniques, visual fields, occupational optometry, therapeutics and optometric law. Arranged in a convenient A-Z format, it will be an indispensable reference source for the busy practitioner as well as a useful study aid for students new to the field.

A book that will assist every eye care practitioner, this text will be invaluable to optometrists, ophthalmologists and dispensing opticians. It will also serve as a handy reference for clinical support staff and those working in industries and profession allied to eye care. Indeed anyone working or studying in this field will find Optometry A-Z an essential purchase.

A Manual of Systematic Eyelid Surgery

Edited by J.R.O. Collin
Price: £ 39.99

This Manual is written for all surgeons and ophthalmologists who operate on eyelids. Its purpose is to promote a systematic approach to eyelid and lacrimal surgery and to simplify the choice of operation.
Flow charts are used, which allow the reader to see at a glance which is the most appropriate procedure for any given set of circumstances. Each operation is described under the headings of ‘Principle’, ‘Indications’, ‘Method’ and ‘Complications’. A large number of simple diagrams have been included to make the text as clear as possible.

In this third edition complications have been added as a separate heading for each operation, reflecting the recognized need to inform patients about these prior to surgery. The chapter on ectropion has been rewritten. There are new chapters on important topics such as facial palsy and thyroid eye disease. The chapters on socket and cosmetic surgery have been considerably expanded and updated, reflecting the advances in these subjects.

A Manual of Systematic Eyelid Surgery, Third Edition delivers clear, step-by-step descriptions and detailed line diagrams depicting many of the most commonly performed eyelid surgery procedures which the author finds most useful, including the newest aesthetic techniques.

Fully revised and updated, the 3rd edition of this best-selling resource keeps you up to date with new coverage of lower lid blepharoplasty, adjustable sutures thyroid eye disease, management of corneal exposure, lacrimal surgery and more.

‘Collin has managed to include those extremely pertinent points which are important to the operating surgeon - this book is full of a wealth of information on eyelid surgical procedures.’

Nutrition and the Eye- A Practical Approach

Edited by Frank Eperjesi, Stephen Beatty
Price: £ 39.99

Recent research has shown that the course of the most common blinding eye disease in the developed world, age-related macular degeneration, can be modified through the use of specific nutritional supplements.

In this book the authors discuss nutrients and micronutrients with respect to the eye and with respect to vision. This is followed by a section on the ophthalmic manifestations of nutritional deficiencies and then a section on the physiological and pathological effects of ocular senescence, with particular emphasis on the role that nutrition plays in the ageing process; here the authors discuss in some detail the relationship between nutrition and common ocular disease such as dry-eye syndrome, glaucoma, cataract and age-related macular disease. This is then followed by evidence in support of dietary modification and / or supplementation in the prevention and/or management of ocular disease. The penultimate section reviews contraindications and offers guidance on avoiding potential adverse reactions associated with dietary modification, and offers some broad conclusions in the final section.

Contemporary, current and clinical, Nutrition and the Eye: a Practical Approach provides essential, up-to-date information on the effects of nutrition on vision. Separating myths from realities, it will assist ‘front-line’ eye care practitioners in their clinical procedures and help them in providing information, all of which is backed by evidence-based research, to the patients in their care. From basic sciences to supplements, this book provides a concise, practical yet complete overview of our current understanding.

- Down-to-earth, practical yet evidence-based information means we will be able to answer patients’ questions concerning nutrition and the eye.
- This book builds upon basic sciences, which will improve our understanding of the topic and enable informed clinical decision making.
- An essential practice manual with easy-to-retrieve information.
Some of the newer diagnostic instruments described in this book rely on complex technology, produce a vast array of data and often beautiful and sometimes beguiling pictures.

This book describes both old and new examination and investigative techniques that those involved in eye care will use or may encounter in their work. The authors of each chapter not only describe how to undertake each test or examination but also outline the problems and pitfalls that will be encountered in their performance and interpretation.

No book can hope to replace a skilled teacher well versed in the examination of the eye. Practical teaching must remain the cornerstone of learning how to examine the ophthalmic patient. Similarly, time spent discussing the most appropriate test to perform with colleagues working in associated areas, for example optometrists, orthoptists, ophthalmologists, neurophysiologists, neurologists or radiologists, is never wasted. This book will provide a sound framework for this practical teaching and discussion.

Clear, concise, and clinical, OPHTHALMOLOGY guides you through the many techniques used for ocular examination and diagnosis today.

- It discusses the surgical principles underlying each technique, the selection criteria, performance, indications and contraindications as well as pitfalls of the procedure.
- Offers a full chapter covering new imaging techniques for the eye, including digital imaging, image analysis, and OCT.
- Explores standard assessment procedures as well as microbiological examination and investigation, ultrasound and radiological evaluation, clinical visual electrophysiology, and fluorescein angiography.
- Features abundantly illustrated coverage in a clear format for quick reference.

(Dr. C. V. Andrews Kakkanatt, JMMC Thrissur)
Welcome to KSOS

All new members to this community are welcome to use the KSOS website that is feature rich and informational. The website address is www.ksos.in

How to register as in the website?

All members are requested to provide the webmaster of KSOS, their details such as
1. First Name     2. Last Name     3. email id

What the webmaster will do:

The webmaster will add these details into the member list. The system will generate the User Id and Password, which will then be emailed to you.

Once the members receive their user ids and passwords, they are requested to visit the website and log in using the Member Login area (top right of the website). Then use the “View My Account” link to see your account details. Please update your details in that section. You are also allowed to change your User Id and Password, to your liking. Please use names that you can always remember, for your user id. If you change your password, do memorise the new password.

Online facilities for the Members

KSOS members have access to very good online resources. All members can then take advantage of the advanced facilities provided such as

1. KSOS journals: Members alone can access the KSOS journals online. You have to login to access the journals.
2. Message Board Members alone can feed messages into the message board for the rest of the KSOS members.
3. Discussion Forum for members All members are also requested to take part in the discussion forum (a new feature) in the website. For this, you are requested to register separately. The instructions for this have been given in the “News Update” section of the website.
4. Video Streaming has been added to the website
This feature has been added to the KSOS website, making the website even more vibrant. It features streaming video content of medical surgeries and procedures.
5. Useful links The KSOS Website provides links to useful and important websites across the world.
6. Member search KSOS members can access the contact details of any other member, using the website. All members are requested to visit the website and see the features in the website and get accustomed to the online system.
CME Programmes

STATE

34th Annual Conference of Kerala Society of Ophthalmic Surgeons
Date : 23, 24, 25th Nov. 2007
Venue : Palghat
Contact : Dr. Anup Chirayath
Email: dranup@afeh.org

Annual Conference of Uttaranchal State Ophthalmological Society
Date : 27, 28th October 2007
Venue : Ram Nagar
Contact : Dr. Satanshu Mathur
Email: satanshu@vsnl.com

Mid-Term Conference of Delhi Ophthalmological Society
Date : 17th – 18th Nov 2007
Venue : Delhi
Contact: Dr. Namrata Sharma
Email: namrata.sharma@gmail.com

STATE

20th-22nd September ‘07
XLV Annual Conference of Vitreoretinal Society of India
Contact Person : Dr. Saurabh Luthra
Drishti Eye Centre for Retina & Lasers, 9B Astley Hall, Dehradun 248001.
E-mail: vrsi2007@gmail.com, drsaurabhluthra@yahoo.com;
Website : www.vrsi2007.com

National

20th-22nd September ‘07
XLV Annual Conference of Vitreoretinal Society of India
Contact Person : Dr. Saurabh Luthra
Drishti Eye Centre for Retina & Lasers, 9B Astley Hall, Dehradun 248001.
E-mail: vrsi2007@gmail.com, drsaurabhluthra@yahoo.com;
Website : www.vrsi2007.com

NATIONAL

28th-30th September ‘07
Bombay Ophthalmological Society
Contact Person: Dr.T.P.Lahane
Doctor's Quarters, Bldg No.1, Flat No.5, Sir J J Hospital Campus, Byculla, Mumbai 400 008.
E-mail: drtplahane@rediffmail.com

28th-30th September ‘07
Bombay Ophthalmological Society
Contact Person: Dr.T.P.Lahane
Doctor's Quarters, Bldg No.1, Flat No.5, Sir J J Hospital Campus, Byculla, Mumbai 400 008.
E-mail: drtplahane@rediffmail.com

26-28th October ‘07
31st Annual Conference of M.P.State Ophthalmic Society “Chitranayan” 2007
Contact Person: Dr. Kuldeep Srivastava
Sadguru Netra Chikitsalaya, Jankikund, Chitrakoot- 210204
Tel: 07670 – 265320
Mobile: 09424617200

1-2nd December ‘07
VII All India Uveitis Conference
Contact Person: Dr. Dipankar, Dr. Kalyan Das
Sri Sankara Nethralaya Guwahati, Assam, Pin 781 028, India. Tel : 91-0361-2228879/80, 2305516,2228921/22; Fax: 91-0361-2228878,
E-mail: Usissn2007@sify.com;
Website:http://www.ssnguwahati.org

1-2nd December ,07
Contact Person : Dr.K.Ramesh
L.V.Prasad Eye Institute, L.V.Prasad Marg, Banjara Hills, Hyderabad 500 034, Andhra Pradesh.
Email: rameshak@lvpei.org, drrk123@rediffmail.com;
Web:www.lvpei.org
7-9th December ‘07
17th Annual Conference of the Glaucoma Society of India.
Cont. Person: Dr Chandrima Paul (Mob: 00919830079189)
B B Eye Foundation, 2/5 Sarat Bose Road, Sukhsagar, 1st & 2nd floors, Kolkata 700 020.
Ph : 91 33 24746608/ 8816 Fax: 91 33 248662720.
E-mail: bbeyefoundation@yahoo.co.in

14th-16th December ‘07
Bihar Ophthalmological Annual Conference (EYECON-2007)
Contact Person: Dr C.S Shah
Masakchak, Bhagalpur-812001, Bihar.
Ph: 0641-2401234, 09431295959
E-mail: drcssshahbgp@yahoo.co.in

INTERNATIONAL

November 10-13 2007
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New Orleans
American Academy of Ophthalmology,
P.O.Box 7424 San Francisco, CA 94120-7424
E-Mail: meetings@aao.org
www.aao.org/annual_meeting
Management of Posterior Segment
Intraocular Foreign Bodies

DR. Meena Chakrabarti, MS,DO, DNB

Foreign Body Injuries
- Chisel & Hammer Injuries
- Grinding Marbles
- Blast Injuries In Quarries
- Fire Cracker Injuries
  - Young Male; 20-40 Yrs.

Important of Detailed History
- Medicolegal Purpose
- Raise Suspicion of IOFB
- Identify Preexisting Ocular Disease
- Asso Systemic Life Threatening Injuries
- Time/Mode/Circumstance/ Magnetic Properties Of FB.
- Previous Records.
  - Treatment Received (Medical/Surgical)
  - Antibiotic Prophylaxis
  - Tetanus Prophylaxis

Fb Location
- Vitreous : 61 %
- Intraretinal : 14 %
- Subretinal : 5 %
- AC : 15 %
- Lens : 8 %

Entrance Wound
- Corneal Entrance Wound:65 %
- Scleral:25 %
- Corneo Scleral:10 %
  - 92 % Single Site Entry
  - 8 % Multiple Sites

Ocular Damage Due To IOFB Injuries
- Direct Damage
  - At Site Of Entry
  - Along FB Track
- Concussive Damage
- Endophthalmitis
- RD
- Metallosis: Siderosis Chalacosis
- Other Sequulae (SRNVM/ERM)

Types of IOFB
- Metallic - 90 % (80 % Magnetic)
  - Iron & Lead
  - Cu, Ag, Au, Pt, Ni
- Non Metallic
  - Cilia, Caterpillar Hair
  - Pencil Lead, PVC, Gunpowder
  - Iatrogenic: Suture, Lint, Cotton Fibre

Ocular Examination
- Gentle Examination
- No Contact Examination In Presence of Open Globe
- Slit Lamp Microscopy
  - Wound Of Entry
  - FB Track
  - Cataract /Lens Rupture
  - Infection
  - Localised Corneal Oedema
    (Goiniscopy To Rule Out IOFB At Angle)
- Indirect Ophthalmoscopy
  - (Initial Examination May Provide View of IOFB, RD, CD, VH etc.)
Timing Of IOFB Removal

- Size of IOFB
- Amt of Ocular Reaction
  - Nonreactive: Observe
  - Reactive: Remove
- Material of FB
- Duration Between Injury And time Patient Is First Seen
- Immediate Sx:
  - Primary Repair Required
  - Reactive / Vegetative FB
  - Frank Infection
- Elective
  - Primary Repair Done Elsewhere
  - Relatively Inert FB
  - No signs of Infection
- Conservative
  - Long Standing, Nonreactive, Inert, Encapsulated FB.

FB: Foreign body; IOFB: Intraocular Foreign body; AC: Anterior Chamber; RD: Retinal Detachment; SRNVM: Subretinal neovascular membrane; ERM: Epiretinal Membrane; Cu: Copper; Ag: Silver; Au: Gold; Pt: Platinum; Ni: Nickel; CD: Choroidal detachment; VH: Vitreous Haemorrhage.
AP: Anteroposterior; Lat: Lateral; IO: Intraocular; EO: Extraocular; RT: Retinal Tear; Vit Tr: Vitreous Traction; PPV: Pars Plana Vitrectomy; REM: Rare Earth Magnet; E/L: Endalasers; I/VIT AB: Intravitreal Antibiotics; PFCL: Perfluorocarbon liquid; PPL: Pars plana lensectomy.
GENERAL INSTRUCTIONS TO AUTHORS

The Kerala Journal of Ophthalmology (KJO) is a quarterly; peer reviewed one, devoted to dissemination of the latest in ophthalmology to the general ophthalmologists as well as to specialists in the various subspecialties of this discipline. It invites submission of original work dealing with clinical and laboratory materials.

Authors submitting materials to this journal are requested to adhere STRICTLY to the norms laid down below. The matter must be typed on one side of the paper. A margin of 1” must be left all around and the material must be double-spaced. A page should contain not more than 25 lines. Two copies of the text in paper and one copy in a CD must be submitted to the Editor and the corresponding author is advised to keep another copy with him. The corresponding author must give it in writing in his covering letter that the same matter will not be submitted elsewhere if accepted. He must also enclose the copyright transfer of his work to this journal. The papers sent will be subjected to peer review. The accepted manuscripts become the permanent property of this Journal. The author is informed that, if his work is returned to him for correction / clarification after peer review, he should effect the same and send the manuscript back to the Editor within one month. Each manuscript component mentioned here under must begin with a new page and the pages are to be numbered at the right tip corner starting from the Title page.

1. **TITLE:** The title of the work must be brief and precise. It should not exceed two lines and 40 characters (including comma, period) Author (s) full name (s) must be given along with his (their) degree and the affiliations. Corresponding author’s name, correct address (including e-mail and Fax, if available) and phone number must be mentioned at the bottom left hand corner of the first page.

2. **ABSTRACT:** The abstract is to be given in the beginning itself. It should not exceed 200 words. It must contain the aim, methodology, results and conclusion. For case report, summary / conclusion alone is to be given.

   **KEY WORDS** (maximum five) in capitals are to be included at the end of Abstract.

3. **INTRODUCTION:** Describe the aim of the study, along with the hypotheses that were tested. Only necessary references are to be given

4. **METHOD:** Give in detail the materials used and the methods employed. Describe the type of study. Pharmacological names only must be mentioned for the drugs used and, if proprietary name is used, then the manufacturers name must be given in parentheses. Except for standard, well-accepted abbreviations (Including SI Units), all others must be introduced in parentheses when the full term is used for the first time in the article.

5. **RESULTS:** Give only the results obtained by the study under discussion. State the statistics in the correct scientific form (P value, mean etc). Results based on assumptions must not be given. Indicate in the text the place where the tables have to be inserted

6. **DISCUSSION:** The discussion should be to the point and relevant to the subject under discussion. This section can be combined with the previous one if the author desires. Avoid speculations. Use only standard abbreviations or the abbreviations already introduced.

7. **ACKNOWLEDGEMENT:** This is to be made only to those who were directly and scientifically involved with the preparation of the paper. Permitting authorities, technicians, photographers who assisted in the work need not be mentioned.
8. REFERENCES: The references should be given in numerical order in which they first appear in text and not in alphabetical order (Citation Order System). It should be numbered consecutively in the text. The references will not be checked by the Editor or by the Peer reviewer and hence the author is solely responsible for its completeness and the accuracy. Period should not be employed anywhere in the references. Personal communication, unpublished data and poster references, if mentioned, should be in the text itself and the source mentioned in parentheses. References should be in the following form:

Journal reference: Author(s) full title, Journal name (as abbreviated in Index Medicus), volume number, pages and year. If there are more than three authors, then mention the first three authors and then 'et al'.

Book reference: Authors(s) (& Editor, if any), title of book (and chapter), publisher, place of publication, page number(s) of the cited portion and year.

9. THE LEGEND: The legend for the illustrations (and tables, if necessary) must be given in a separate sheet of paper and should be typed double-spaced.

Illustrations: The photos and figures should be prepared in glossy prints with good contrast and of the size 6” x 4”. Only salient details should be included. On the back of the illustration, the figure number in text, title of the paper, the first author’s name and the top side (marked with an arrow) must be specified. Except for arrows, no text is to be on the photos. It is the duty of the author(s) to get the patient’s written permission when the subject is identifiable in the photo. Submit two sets of illustrations. Illustrations from other Journals and books are usually not accepted. If used, it rests with the author(s) to get the copy right permission from the original author / publisher and this permission letter must be sent to the Editor at the time of submitting the manuscript. For Histological figures the stain and magnification used should be noted e.g.: - H & E Stain x 70.

10. TABLE: It should be in double space. Each table must have an Arabic numeral (except for single table) and a title both in a single line. Each column in the table must have a short heading. If a table is large, then it must be continued in a second page, which also must have the table number and the title. Avoid vertical lines in the tables. Two sets must be submitted.

The manuscripts are to be sent to The Editor by Courier Mail or by Registered post. The corresponding author will receive communication from the Editor within two weeks of receiving the manuscript.

11. All manuscripts are subjected to editorial board review.

12. Other Categories of Manuscript

a) Original Articles should generally not exceed 3,000 words or 12 double – spaced pages.

b) Review Articles: can be on topics of relevance to clinical practice, research methodology, community ophthalmology or investigative work, of relevance to visual science. These articles should include up to date review of existing literature, and summarize the current status / preferred practice for that particular topic.

Brief reports are short communication of new instruments, new laboratory techniques or surgical techniques as well as interesting case reports with unique findings. These should not exceed 1000 words with a maximum of 2 illustrations. They should follow the format - introduction, case, and discussion. No more than 8 references should be cited. Each brief report must begin with a 75-100 word summary that highlights the significance of the articles.
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