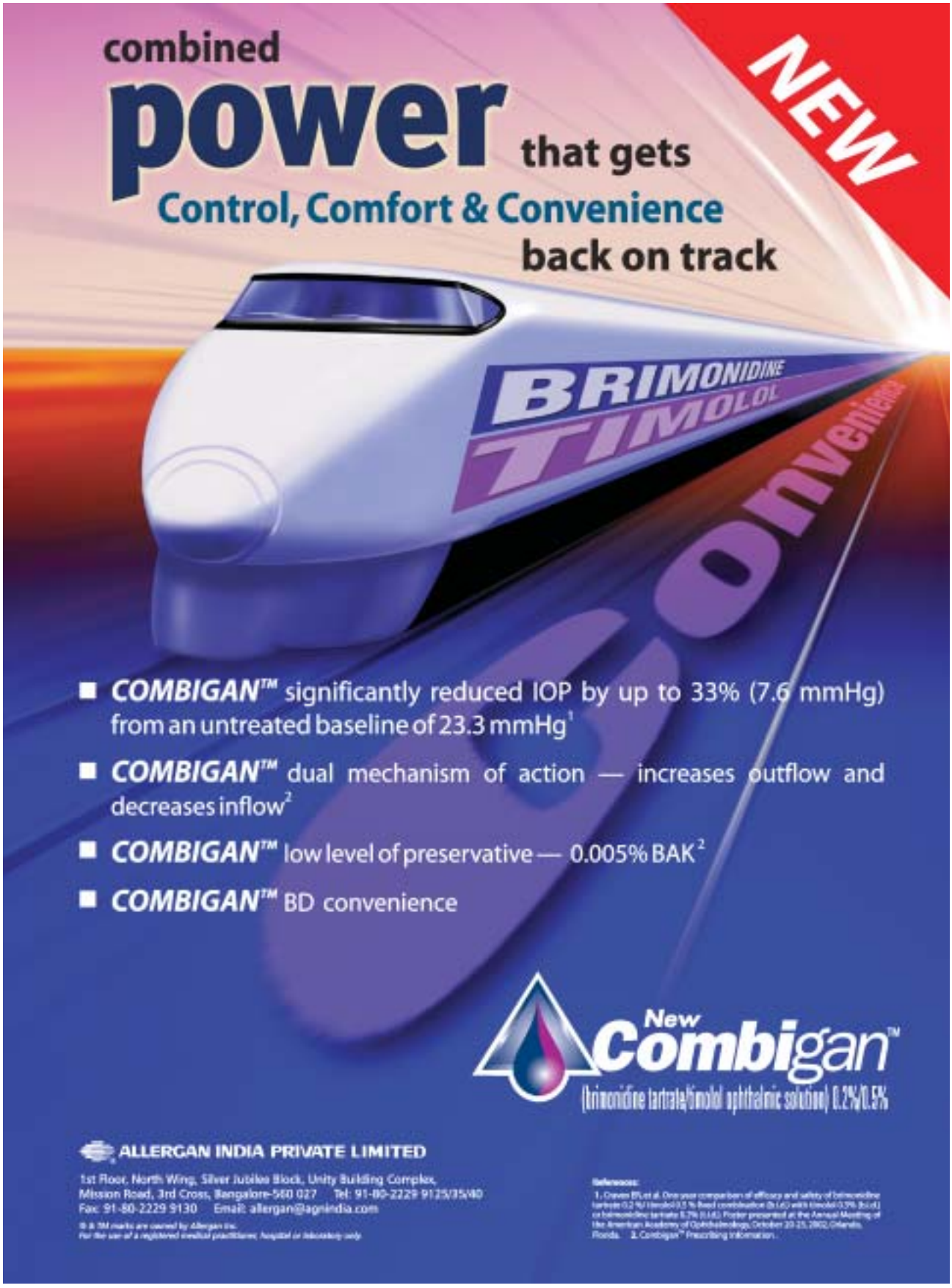

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Table 1 Composition of Hartmann's solution, BSS® and aqueous fluid*

	Aqueous	BSS®	Hartmann's Solution (Lactated Ringer's)
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Calcium	1.5	3.4	2
Chloride	108	130	111
Magnesium	1	1.5	-
	lactate	acetate	lactate
	ascorbate	citrate	
	protein		
Osmolality:	300	304	256

*Electrolytic concentration, mmol/l: osmolality, mosmol/l

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Can the Risks of a Malpractice Law Suits be Reduced?

As per the recent statistics on consumer litigation, ophthalmology takes a prestigious 5th position accounting for 3.75% of all consumer litigations in medical practice. This list is topped by Obstetrics and Gynaecology (33%) followed in a descending order by Orthopaedics (18%), General Medicine (15.15%), General Surgery (6.75%) and other surgical super specialities (<10%).

Analysis of available data on malpractice law suits in ophthalmology shows that endophthalmitis, postcataract corneal decompensation, retained intra ocular foreign body, retained sutures, delay in referral, decentered intra ocular lenses, incorrect glass prescription and under or over correction by Lasik and dropped nucleus account for the majority of cases.

Risks of facing a malpractice lawsuit can be minimized by utilizing risk management strategies which involves (a) adopting preventive measures to prevent the occurrence of complications, b)recognizing complications when they occur, c) appropriate management of complications, d) timely referral and e) effective communication with the patient and his relatives.

Attention to preoperative and postoperative counseling and timely complication management are key steps that has to be emphasized and underscored.

Documentation is another key step and most surgeons realize its value only when faced with a medical malpractice litigation. It is absolutely essential to record a) the date and time of each hospital visit, b)a brief history of the present illness with relevant past history, c)clinical findings and diagnosis, d)record of the treatment strategy advised and counseling given and e)a note on the patients' compliance to therapy. As far as the courts are concerned, whatever is not documented in the case record has not occured. If you claim to have performed a procedure, but have not documented it, your claim will not hold water in the court room.

All ophthalmologists who perform surgical procedures will definitely encounter complications at some time or the other. These complications need not all be a result of medical negligence. IOL related problems which hold the major proportion of cataract surgery malpractices suits include incorrect power, size and type. Make sure that the right type of IOL is implanted in the patients' eye and have safeguards in the operating theatre to avoid mistakes.

When a complication arises, the support and time that the surgeon gives to the patient is usually greatly appreciated. Ignoring the situations is a sure sign of "abandonment" of the trust placed by the patient on you and is usually the main impetus for the patient to seek redressal.

Meticulous documentation, updating on the most current technology, adopting treatment strategies that are accepted by Medical Speciality Societies, acknowledging one's own surgical limitations are absolutely vital in reducing the incidence of potentially compensable complications.

Informed consent is a must before surgery and has to be taken in advance rather than on the day of the surgery. Doing so allows the patient ample time to have a satisfactory discussion on the risk, benefits and alternative to the surgical procedure that is planned. While discussing the surgical procedure, an honest appraisal of the patients' condition should be presented rather than trivializing the surgery and minimizing the complication in an attempt to ease the patients' mind of worry. The patient has his own high and unrealistic expectations regarding the outcome of the surgical procedure. Unless effective communication and appropriate counseling is carried out and the risks and benefits of the procedure explained clearly, any adverse outcome can cause confusion, disappointment, anger and a sense of abandonment, all together may snowball into a malpractice claim.

Before performing any surgical procedure be reasonably sure that the benefit will outweigh the risks. Be also doubly sure that the surgical pathology that you are going to handle is the main cause for the patients' visual loss. Unrealistic patient expectation should be managed preoperatively itself, rather than having to struggle to explain a bad prognosis after a poor surgical outcome.

It rests in our own hands to reduce the risks of a malpractice law suit. Let us adopt strategies to minimise these risks and also remember that any patient that we deal with can be a potential trouble maker. Law suits can be agonising, extremely demoralising and can undermine and destroy our professional and personal lives.

Let good sense prevail and let us adopt strategies that will make our profession litigation free!

The editorial board joins me in wishing you a very Happy, Prosperous and Litigation-free New Year!

Dr. Meena Chakrabarti
Editor, KJO

Idiopathic Central Serous Chorioretinopathy

Dr. Chandran Abraham

The fundus features of idiopathic central serous chorioretinopathy (ICSC) were first described by von Graefe nearly 140 years ago, in 1866.[1] A wide variety of terms were then used to describe the condition, depending on what different investigators felt about its cause or the mechanism behind it.[1] The most common terms used currently, are central serous retinopathy, central serous chorioretinopathy, and idiopathic central serous chorioretinopathy. The tendency for spontaneous remission and recurrences was recognised quite early. Though much progress has been made in understanding its clinical features, natural course, and response to treatment, the precise mechanism that causes it is not fully known.

Age, sex and symptoms

ICSC has been classically described to occur in young males between 20 and 40 years of age, but is being increasingly reported in older age groups[2-4] and in women.[4,5] Though rare in patients below 20 years of age, and without a familial predilection, it has been reported in a 7-year old girl,[6] and in members of the same family.[7-9] The presenting symptoms are central or paracentral scotomas, metamorphopsia, reduced vision, micropsia or macropsia. The symptoms are of acute onset and very worrisome to the patient. They may be troublesome only when the normal fellow eye is closed, but some patients experience considerable difficulty in binocular visual functions. Some may report decreased sensitivity to contrast or colour. ICSC may be unilateral or bilateral.

Consultant Ophthalmologist, Apollo Medical Centre, Chennai

Fundus features

The fundus appearance is quite characteristic, and a diagnosis of ICSC can usually be made after an examination with the direct or indirect ophthalmoscope, or with a slitlamp biomicroscope, using contact or noncontact lenses. Classically, there is a round or oval serous detachment of the retina at the macula that is localised and well delineated from the surrounding normal retina. The detachment invariably, though not always, involves the fovea, and may rarely be outside the macula.[10] The fluid under the sensory retina may be clear or turbid, and may sometimes contain protein deposits, or fibrinous exudates (Figs 1 & 2).[11,12] More than one such localised detachment may be occasionally found in different areas of the same fundus, and could be located peripherally as well.[13,14] The sensory detachment may sometimes be extensive and bullous,[13,15-24] with shifting subretinal fluid that may extend to the fundus periphery (Fig 3). Eyes with fibrin in the subretinal space or eyes with extensive detachments may develop subretinal fibrosis.[12,21] Serous detachments of the retinal pigment epithelium (RPE)[25-28] may often be found in eyes with localised or extensive retinal detachments. These appear as smooth, round or oval, dome-like elevations of the RPE with clear fluid and distinct margins. RPE detachments are usually around ¼ disc diameter in size, but can be much larger. They may be found within the area of serous retinal elevation, at its border, or outside it. RPE tears[12,20,29] and choroidal neovascularisation[30] are rare in ICSC, but have been described. Care must be taken to exclude polypoidal choroidal vasculopathy,[31] and other conditions that may cause a secondary retinal detachment, before a diagnosis of ICSC is made. It is also important not to

overlook a congenital pit of the optic nerve head that may be associated with a serous detachment of the macula. When ICSC resolves or becomes chronic, there may be alterations in the fundus such as RPE atrophy (Fig 4) in different patterns, RPE hyperpigmentation, subretinal yellow deposits, or retinal neovascularisation.[18,32-38] An atrophic tract in the RPE might be noticed in eyes after absorption of fluid that had gravitated inferiorly from the macula (Fig 5).[39,40] While acute ICSC may not pose a problem in diagnosis, the chronic stages can be easily misdiagnosed unless one is familiar with the fundus changes associated with it.[41] ICSC may sometimes be bilateral with symptoms developing days or weeks apart in each eye. Fellow eyes may be normal, may show localised serous detachments of the retina away from the fovea, RPE detachments, or evidence of previous ICSC.

Visual function

Visual acuity in ICSC can range from 6/6 to 6/36, typically worsening with pinhole, and improving with a small hyperopic correction. Vision can be worse if the detachment is bullous and involves the macula. Near vision may be more significantly affected. The Amsler chart easily documents any metamorphopsia, scotoma, micropsia or macropsia. Several tests of macular function could be abnormal.[42-49] These include delayed recovery time after dazzling the macula,[42] reduction of responses on local and multifocal electroretinograms,[43,44] increased latencies[45,46] or reduced amplitudes[47] on visually evoked responses, and depression of retinal function using psychophysical and photochemical tests.[48] Electrophysiological tests designed to test RPE function could not detect any abnormality.[49]

Fluorescein angiography

Fluorescein angiography in ICSC clearly documented that intravenously injected fluorescein entered the subretinal space from the choroid, through the RPE.[26,50] The characteristic features on the angiogram is the appearance of one or more spots of hyperfluorescence in the early phase that increase in size and intensity during the later phases of the dye transit, with fuzzy borders in the late phase that characterise leakage of the dye (Figs 6 A,B,C). The leaked dye may pool in a dependent area of the serous retinal detachment, but almost never fills the entire subretinal space. The patterns of dye leakage may resemble an inkblot or a smokestack. The number of spots leaking fluorescein could vary from 1 to 22, and may be observed at different distances and locations from the fovea.[21,51,52] The leaks are confined to the macula or posterior pole when the serous detachment is localised, and may be found more peripherally when the detachment is extensive. Associated serous RPE detachments show early hyperfluorescence of the entire lesion, with increasing hyperfluorescence during the dye transit and in the late phase, but without any increase in size. Typical leaks of ICSC may develop from existing RPE detachments.[53] Areas of RPE atrophy that may be seen in chronic cases are also readily appreciated as window defects on the angiogram. Some of these angiographic findings may be present in asymptomatic fellow eyes.[54]

Indocyanine green angiography

Indocyanine green, by virtue of its properties, allows the choroidal circulation to be more easily visualised and studied in detail by digital imaging and video techniques. Apart from an initial observation that there was choroidal circulatory insufficiency in ICSC,[55] all subsequent studies found the choroid to be hyperpermeable, seen as areas of hyperfluorescence on the ICG angiogram.[56-63] Increased choroidal permeability has been found not only around areas of fluorescein leakage, but in areas devoid of fluorescein leakage as well.[56] Such permeability could persist in chronic ICSC, and in ICSC that had resolved as determined by other clinical signs.[58,63] ICG angiography may also be helpful in detecting serous pigment epithelial detachments not obvious on fluorescein angiography.[56,62]

Optical coherence tomography

The introduction of optical coherence tomography (OCT), and its use in ICSC provides information that is unique and also complements other methods of evaluatuion.[64-70] OCT can detect shallow sensory retinal detachments that cannot be made out with slitlamp biomicroscopy, allows quantification of the detachments, and is useful in follow-up.[65,66] Some of the unique features observed on OCT include thickening

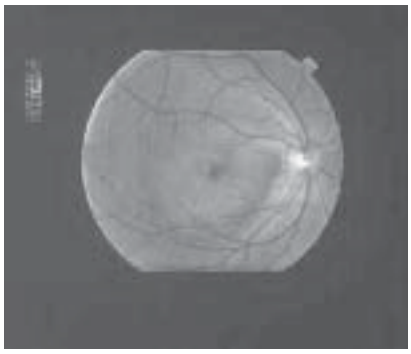


Fig. 1. Fundus photograph of ICSC with clear fluid under sensory retina

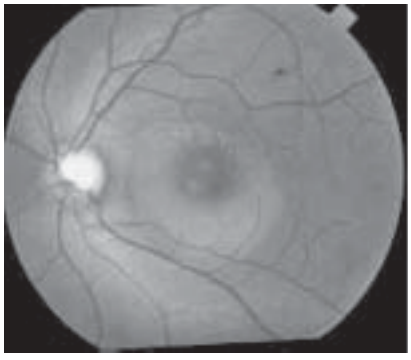


Fig. 2. Fundus photograph of ICSC with turbid fluid and protein deposits under sensory retina

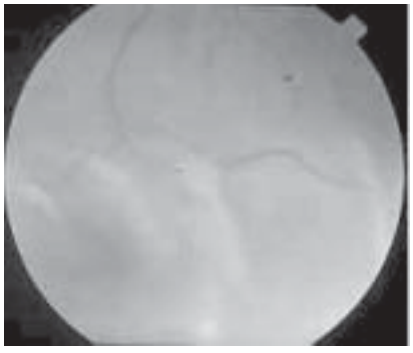


Fig. 3. Fundus photograph of inferior quadrant with ICSC and bullous sensory retinal detachment



Fig. 4. Fundus photograph of retinal pigment epithelial atrophy in spontaneously resolving ICSC

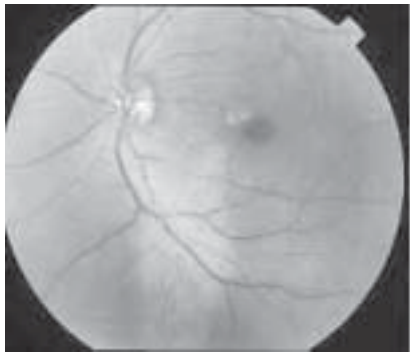


Fig. 5. Fundus photograph with retinal pigment epithelial tract from macula to inferior quadrant

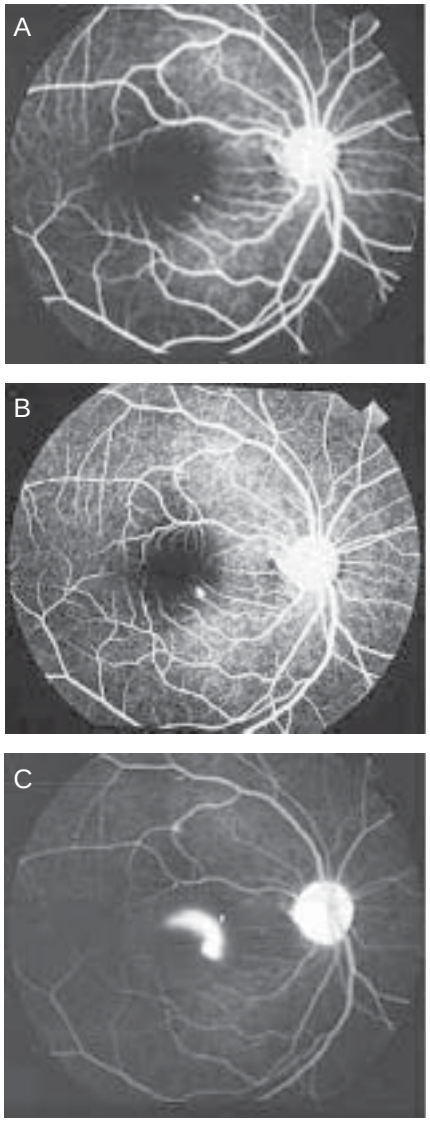


Fig. 6. (A) Early arteriovenous phase fluorescein angiogram with a point of hyperfluorescence inferonasal to fovea (B) Mid-arteriovenous phase fluorescein angiogram showing enlarging fluorescence (C) Late phase fluorescein angiogram showing a smokestack leak

of the detached sensory retina in the acute phase, decreasing thickness of the retina as the detachment subsides,[67] optically empty vaulted areas under the sensory retina that are related to fluorescein-filled areas, protrusions of the RPE corresponding to leaking fluorescein spots, and semicircular spaces under the RPE with thinning of the overlying retina.[68] Foveal thinning, absence of a discrete signal from the internal limiting membrane, and an indistinct boundary between the photoreceptor and outer segment layers in resolved ICSC were features that correlated with visual acuity.[69]

Other imaging techniques

Digital imaging processors, scanning laser ophthalmoscopes, and high-resolution ultrasound are other methods that provide precise localisation and quantification of the lesions in ICSC, and enable correlations with other phenomena such as autofluorescence, fluorescein and indocyanine green angiographic patterns.[71-79] One study found sites of fluorescein leakage to cluster around the geometric centres of the serous detachments, and smokestack leaks to be associated with larger detachments.[71] Another study found no such correlation.[72] Observations on fundus autofluorescence have been varied, with increased or decreased autofluorescence over areas of sensory retinal detachment, and over sites of fluorescein leakage at the RPE.[74-78] A nonechogenic linear band has been described on high-resolution ultrasonography, although a possible shadowing of the RPE could not be ruled out.[79]

Associated conditions

ICSC has been reported in patients with a wide variety of medical disorders. These include infections and infestations,[80-82] inflammatory bowel disease,[83,84] disorders of the blood and its components,[85-87] renal and adrenal disorders,[88,89] ocular disorders and syndromes,[90-92] and other conditions.[93-98] Patients with ICSC have also been found to have a Type A behavioural pattern,[99] severe psychological stress,[100] and increased sympathetic activity of the autonomous nervous system.[101,102]

ICSC in pregnancy can occur in healthy women, [103-107] and is usually associated with subretinal exudation.[104-106] Spontaneous resolution occurs around the third trimester of pregnancy or soon after delivery,[103-105] and there has been no recurrence of ICSC during a subsequent pregnancy.[105]

Corticosteroids are now being increasingly implicated as an important factor associated with the development or worsening of ICSC.[97,108-132] Patients receiving steroids for their systemic[110,115,116,121,124,131] or ocular conditions[109,112,123,128,129] can develop ICSC that may be unilateral or bilateral. This includes patients who are not only on oral steroids, but those who receive steroids through several other routes as well.[115,117,120,123,125,127,130] Though rare, ICSC has been recently reported in patients who have received periocular steroid for ocular inflammation,[128] and intravitreal steroid during vitrectomy.[129] The occurrence of ICSC in Cushing's syndrome[111] and elevated levels of endogenous cortisol[118,119] in patients with ICSC offers some support to this association. Though the dose and route of steroid intake is very varied in the different studies, ICSC could occur in patients who are on oral doses of over 20 mg a day, for a period of about two months.[113] Resolution of ICSC has been reported after reducing the dose or withdrawing the steroid.[109,114-116,121,133]

Mechanism

Though not completely understood, there is evidence that normal apposition between the sensory retina and the RPE, and between the RPE and choroid are maintained by a higher hydrostatic pressure in the vitreous, higher osmotic pressure in the choroid, structural connections between RPE and Bruch's membrane, the metabolic activity of the RPE, and perhaps a glue-like function of the interphotoreceptor matrix.[134] The RPE also constantly moves ions and water unidirectionally, from the vitreous towards the choroid.[134] There is general agreement that the fluid under the RPE and serous retina in ICSC are derived from the choroid. This is based on the fluorescein angiographic features described above as well as from experimental studies.[135] The question as to whether the RPE or the choroid is primarily involved remains unsettled. While simple loss of functional integrity of the RPE alone cannot account for fluid in the subretinal space,[134,136,137] derangement of the metabolic transport system in the RPE,[136] or reversed polarity in one or more

of the RPE cells[138] could account for such fluid accumulation. Earlier clinical observations, and more recent observations from indocyanine green angiograms described above have documented choroidal hyperpermeability, suggesting that the choroid may be primarily affected in ICSC. Damage to choriocapillaries, in addition to damage to the RPE was seen in an animal model where central serous chorioretinopathy was experimentally induced.[139] However, persistence of ICG changes after spontaneous or laser-induced resolution of ICSC[58] are not fully explained, if the primary defect were to be entirely in the choroid. Clinical observations suggest that corticosteroid intake and increased sympathetic activity of the autonomous nervous system could be a factor in the development of ICSC. Repeated intravenous injections of adrenaline in an animal model resulted in features similar to ICSC seen in humans,[140] though a concomitant rise in blood pressure could have been responsible.[134] Pathological material from patients with ICSC remains sparse. No abnormality was detected in the RPE or choroid during a routine autopsy in a patient with serous detachment of the retina and RPE, who died of a cardiac problem.[141] The presence of fibrin in the grey-white subretinal exudation in yet another autopsy specimen suggests that the origin of fibrin was from the choroid, though the choroid itself was reported to be normal.[141]

Natural Course

The natural course of ICSC is favourable in most patients, with resolution of the serous detachment and return of visual activity,[142,143] with sustained long-term visual gain.[144] About 5% will be left with a visual acuity of less than 6/9, 20 to 50% will have one or more recurrences, with most recurrences occurring within one year, and about 20% can develop ICSC in the other eye.[141-143,145] Recurrences were most often seen within one disc diameter of the original site of fluorescein leakage.[146] Eyes with serous bullous detachments can also reattach spontaneously with recovery of vision.[22,23] In spite of clinical resolution and improvement in visual acuity, other tests of macular function determined by Amsler's grid, visual field charting, colour vision testing, contrast sensitivity determination, multifocal electroretinograms, and electro-oculograms could be abnormal.[34,147-153] Morphological changes in the retinal pigment epithelium such as pigment epithelial atrophy, and hyperpigmentation may be seen in eyes after resolution of the serous detachment.[33,35,154] These areas of atrophy may be located within, or even outside the area of serous detachments that had resolved.[154] Serous pigment epithelial detachments associated with ICSC in younger patients can spontaneously flatten and show some pigmentation, without the development of geographic atrophy or choroidal neovascularisation.[155] Hyperfluorescence on the ICG,[57,63] and foveal thinning on the OCT[69,156] may be evident long after the sensory detachment subsides.

Management

Patients with ICSC can be reassured and observed because of the favourable natural outcome that has been consistently documented. In patients taking steroid for an ocular problem, the drug may be tapered off and stopped if there are clear indications that it is not necessary. In patients taking steroids for other medical conditions, a careful consideration of the reason for steroid intake must be made, and any plans to reduce the dosage or withdraw the steroid must be made in consultation with the patient's physician. There may be patients with systemic disorders who need the drug for prolonged periods. There is no proved medication for ICSC at present. Resolution of the serous detachment has been reported after discontinuation of steroids and institution of anti-tubercular drugs,[108] and beta blockers have been found to be useful in a small number of patients.[157-159] Psychosomatic assessment of patients with ICSC did not reveal any conspicuous abnormality that warranted psychotherapy.[160]

Photocoagulation using different wavelengths, and different techniques can bring about a quicker resolution of fluid, amelioration of symptoms, and improvement in vision.[17,19,21,22,39,141,143,145,161-194] Such a response to laser treatment is seen in eyes with bullous serous detachments as well.[17,21,22,193] However, some residual defects in macular function, and chronic changes in the RPE may remain, and recurrences are possible. The most important indication for laser treatment is the patients' intolerance to symptoms, inability to perform daily tasks because of symptoms, or the need for normal vision in each eye that is mandatory in certain professions. Other considerations include the duration

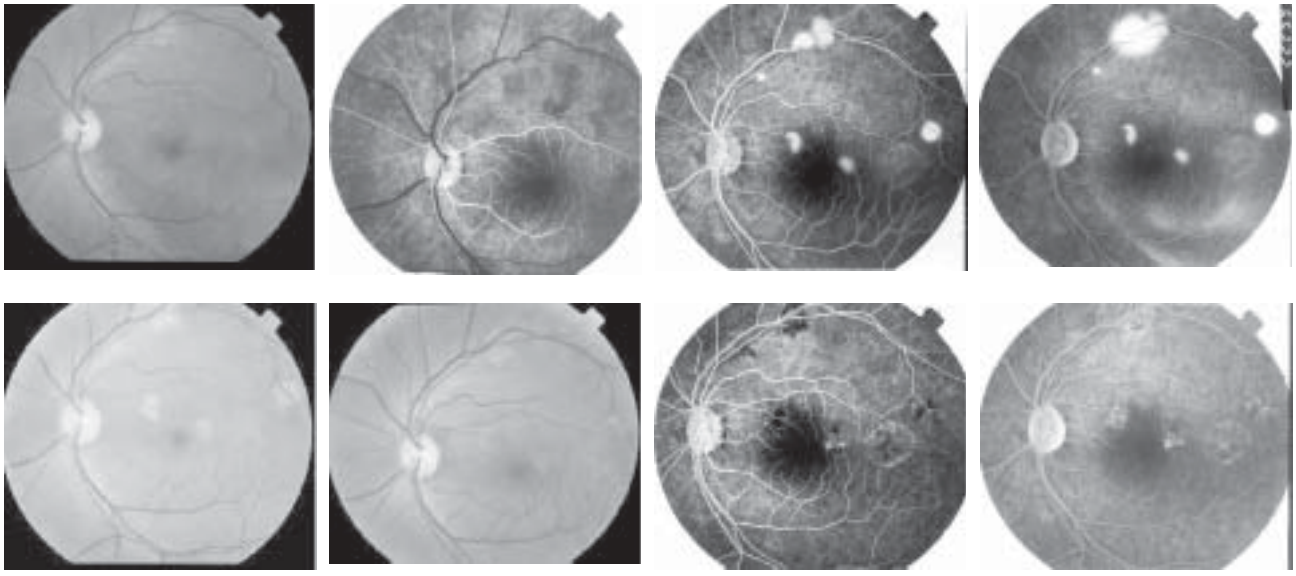


Fig. 7 (A) Pre-treatment fundus photograph with serous fluid at macula, and pigment epithelial detachments temporal to macula and superotemporal to the optic disc (B) Arterial phase fluorescein angiogram showing some hyperfluorescence superotemporal to optic disc (C) Late arteriovenous phase angiogram showing hyperfluorescence from three ICSC leaks and from two retinal pigment epithelial detachments (D) Late phase angiogram with some pooling of fluorescein within the serous detachment (E) Fundus photograph immediately after laser photocoagulation to the ICSC leaks and pigment epithelial detachments (F) Fundus photograph two months after laser treatment with resolution of serous detachment (G) Late arteriovenous phase fluorescein angiogram showing hypofluorescence in treated areas (H) Late phase angiogram showing hypofluorescence in treated areas, with obliteration of all leaks

of the serous detachment, location of leakage on the fluorescein angiogram in relation to the fovea, and recurrences.[143] Laser photocoagulation consists of identifying the origin of dye leakage at the RPE from a recent fluorescein angiogram, and treating these sites directly with burns that are just visible (Figs 7 A-H) . Associated RPE detachments may be treated entirely in the same manner. Such direct treatment was found to be superior to indirect treatment,[168,169] and is the most common method adopted. However, disappearance of a foveal leak after treatment of other leaks,[182] and resolution of serous detachment after a grid pattern of treatment in eyes with diffuse RPE changes have been reported.[179,183] Apart from the argon green laser, effective treatment of ICSC has been reported with other wavelengths,[173,175,178,181,186,192] and with the subthreshold technique as well.[189,191] The krypton red has been successfully used to treat leaks much closer to the fovea.[173,175,178,181] An accidental foveal burn is a potential complication with laser treatment of ICSC, and laser burns may enlarge over a period. Choroidal neovascularisation is rare, but may occur at the site of laser treatment, and care must be taken not to overlook a small choroidal neovascularisation prior to treatment.[168,184,188]

More recent approaches in the treatment of CSCR include the use of photodynamic therapy, sometimes guided by indocyanine green angiography,[195-198] and transpupillary thermotherapy.[199] These procedures, performed on patients who had chronic CSCR,[196,197,199] or fluorescein leaks at or close to the fovea,[196,198,199] resulted in resolution of serous detachment, decrease or cessation of dye (fluorescein and indocyanine green) leakage, and stabilisation of vision in most patients,[195-199] though one developed choroidal neovascularisation.[196] Vitrectomy using perfluorocarbon liquid and endolaser was successful when a bullous serous detachment did not allow adequate laser treatment.[200]

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ORIGINAL ARTICLE

23 Gauge Sutureless Vitrectomy – An Outcome Analysis

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Since the introduction of pars plana vitrectomy in the early 1970s by Dr. Robert Machemer, advances in the field of vitreoretinal surgery have been dramatic. The initial pars plana vitrectomy was performed by Dr. Robert Machemer using 17 gauge (1.5 mm diameter) multifunction probes capable of cutting and aspirating the vitreous. This instrument utilized a fibreoptic sleeve and needed a sclerotomy of 2.3 mm size for safe introduction and removal of instruments.

In 1974, O' Malley designed a smaller vitreous cutter with a diameter of 0.9 mm (20 gauge). This less invasive three port 20 gauge cannula entry system is still used today. The use of divided function instruments through three separate sclerotomies made bimanual vitreous surgery possible.

In 1990 Dr. de Juan and colleagues designed a variety of 25 gauge (0.5 mm diameter) vitreo retinal instruments for more delicate and precise surgical maneouvres.

Recently Fuji et al designed a 25 gauge microcannular system and an array of 25 gauge instruments referred to as transconjunctional sutureless vitrectomy system (TVS). One of the disadvantages of 25 gauge TVS is the extreme flexibility of instruments making the complicated tasks performed in the vitreous cavity difficult.

The TVS is considered as one of the most innovative vitreoretinal surgical technique developed by Fuji et al. In this technique three polyamide microcannulas are inserted transconjunctivally through the sclera in the area of parsplana. The VR instruments and infusion lines are then introduced through these cannulas into the vitreous cavity. Because a thin 25 gauge instrumentarium is used, the incision left in the sclera after removal of the cannulas are so small that they self seal without suturing. This procedure has no surgical trauma to the conjunctiva, requires no scleral sutures and this leaves no postoperative suture related astigmatism and entails a distinctly reduced rehabilitation time.

Sutureless self sealing sclerotomies for parsplana vitrectomy were first described by Chen in 1996. Instead of using the usual right angled incision through the sclera, a tunnel like tangential incision was made at a 30^{O}- 45^{O} angle through the sclera. Suture closure was not required as the wound borders are pressed together by the intraocular pressure ensuing from the oblique course of incision.

Although the conjunctiva is always opened more or less wide in most sutureless self sealing sclerotomy technique, transconjunctival vitrectomy merely requires a point incision for the microcannulas. Sclerotomies in 25 gauge vitrectomy require no suturing because they are only 0.5 mm in diameter compared with 1.15 mm width of the sclerotomies for 20 G conventional vitrectomy. When 23 gauge microcannulas are introduced through the sclerotomy, they are too large and will definitely require suture closure.

23 gauge cannulas can be placed in tunnel incisions 0.72 mm wide running tangential to the scleral surface. In few cases the infusion fluid drained out from under the conjunctiva when the cannula was withdrawn at the conclusion of surgery. Absence of postoperative hypotony speaks for the tight self sealing nature of the

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tunnel incision. In contrast Fuji et al observed transient postoperative hypotony after vitrectomy with 25 gauge system. Hence it appears that the angle of the incision rather than its size is of utmost importance for achieving self sealing nature.

The aim of the study is to analyse the complications and outcome of 23 gauge transconjunctival sutureless vitrectomy using 23 gauge instrumentarium.

Material and Method

In a retrospective study, sutureless 23 gauge vitrectomy was performed on 37 consecutive eyes. The indications for vitrectomy are given in table 1.

Table 1. Indications for Vitrectomy

1	Giant Retinal Tear Repair	4
2	Silicone Oil Removal	4
3	Dislocated IOL Removal	1
4	Epimacular Membrane Surgery	2
5	Diabetic Vitrectomy	26
	• Vitreous Hemorrhage	14
	• TRD	12

The surgical technique adopted was as follows. The procedure was started by pushing the conjunctiva 1mm to 2 mm laterally (ie, parallel to corneoscleral limbus) in the inferotemporal, superotemporal and superonasal quadrants using a special pressure plate (DORC; Holland) to hold it firmly to the sclera. A 23 gauge stilletto blade, 45° angled is inserted at 30°-40° angle through the conjunctiva, sclera and parsplana 3.5 mm from the corneoscleral limbus. To obtain scleral tunnels parallel to the limbus, the scleral incisions are made radial to the limbus. The incision with the 23 gauge stilletto blade is 0.72 mm wide. Constant pressure is applied to the pressure plate during introduction and withdrawal of the stilletto blade to prevent slippage of conjunctiva against the sclera (Fig. 1).

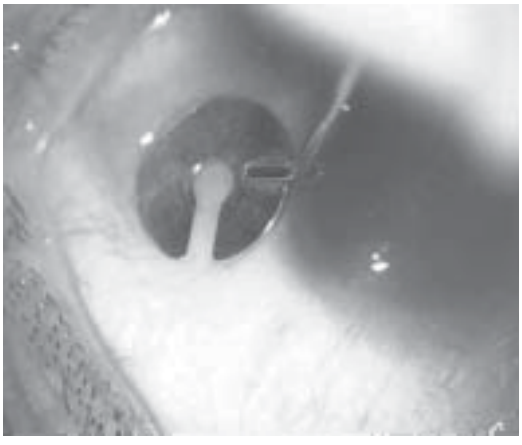


Fig. 1. A specially designed pressure plate is used to fix the conjunctiva and hold it firmly against the sclera.

The microcannula was then inserted through the (Fig. 2-Fig. 5) conjunctival incision into the scleral tunnel using a specially designed blunt inserter (DORC). The length of microcannula is 4 mm, internal diameter 0.65 mm and external diameter 0.75 mm.

The instruments used for transconjunctival 23 gauge sutureless vitrectomy are given in TABLE-2 and Fig. 6 and 7

Table 2. Instruments For 23 Guage Sutureless Vitrectomy

1	23 Gauge Vitrectomy Cutter
2	Wide field Endoilluminator
3	Eckardt's Twin Endoilluminator (chandlier)
4	Flute needle with back flush
5	End gripping forceps, scissors
6	Endodiathermy probe

Bimanual vitrectomy was performed in 6 patients with posterior pole tractional RD. In these patients a self retaining 25 gauge Eckardts twin endoilluminator (DORC) was used in addition to the standard three vitrectomy ports. The membranes on the retinal surface were gently grasped with forceps and cut with scissors. Perfluorocarbon liquid was injected intraoperatively in 12 eyes to achieve anatomic retinal reattachment. During injection of PFCL with a blunt cannula, the infusion line was opened to let out superfluous irrigating fluid and maintain the IOP within normal limit. Silicone oil injection in 8 eyes and fluid air exchange (3 eyes) could be carried out with ease.

At the conclusion of the surgeries the cannulas were simply withdrawn from the scleral tunnel. The conjunctiva was pushed laterally using a cotton wool applicator to separate its incision from the scleral incision. Topical antibiotics and steroid drops were given postoperatively.

The patients were duly examined on a daily basis for the first 4 days after the procedure and at 4 weekly intervals thereafter. The duration of follow up lasted for 4-12 weeks.

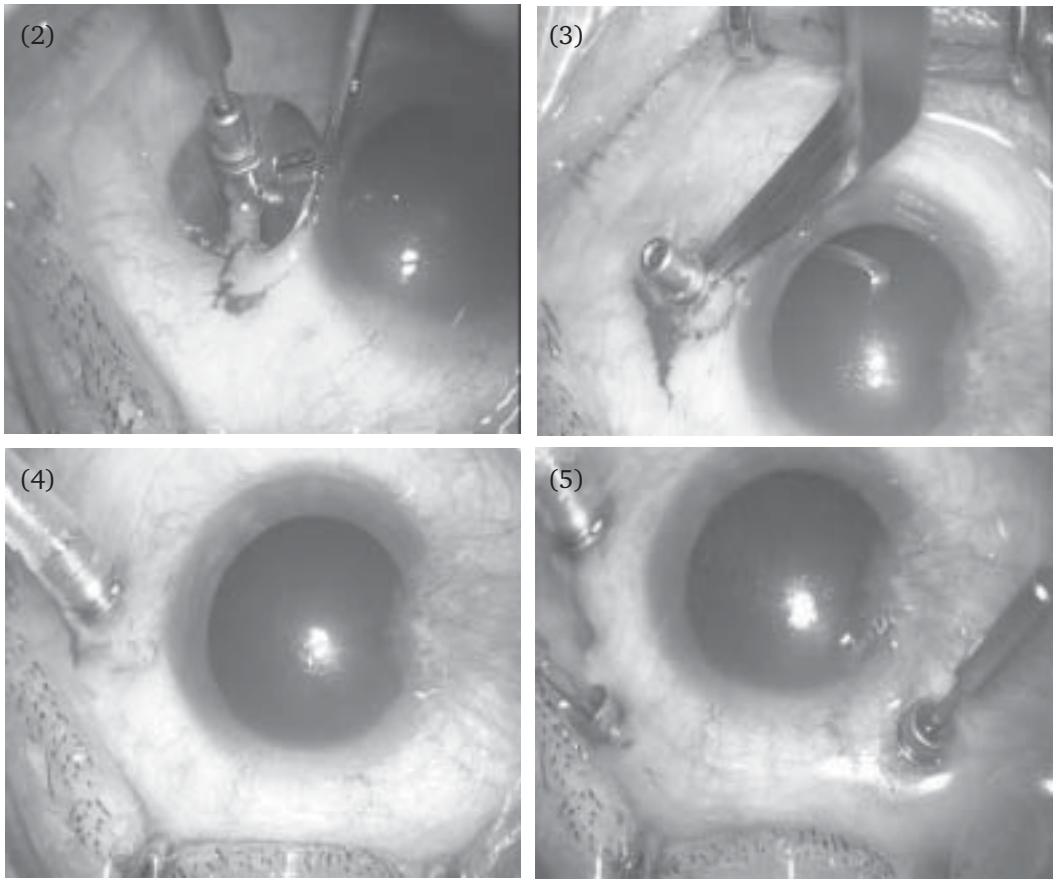


Fig. 2-5. The microcannulas are introduced through the conjunctival incision into the scleral tunnel using a specially designed inserter (DORC)

Results

In about 30 % of cases, scleral incision with the stilletto blade led to microbleeding from the episcleral vessels. Usually this was only a small bleed for which diathermy application was not warranted. The presence of pressure plate enabled successful insertion of the cannulas into the conjunctival and scleral incision in 90 % of eyes. In 10% of patients conjunctival slippage occurred making identification and successful insertion of the cannula impossible.

Placement of the cannula within the tunnel lead to spontaneous stoppage of bleeding. Use of microcannulas was free of complication in all 37 eyes. Not even in cases requiring scleral indentation to perform vitreous base excision did slippage or loss of cannula occur. There was also no cannula induced lesion of the posterior capsule in phakic or pseudophakic eyes.

The vitreoretinal instruments used in the present study were of similar design and utility as their 20 gauge counterparts. The 23 gauge cutter was found to be less efficient and slower at performing extensive vitrectomy. The infusion bottle height was much higher than that for a standard vitrectomy. The suction used was also higher since the efficiency of the cutter was not comparable to a 20 gauge cutter. The intraocular instruments used during vitrectomy were more flexible than 20 gauge instruments. However this increased flexibility did not hamper their function or their ability to guide the globe. Superior rotation of the globe for vitrectomising the superior and peripheral vitreous was found to be difficult in 30 % of patients. However with scleral indentation sufficient vitrectomy could be performed.

Withdrawal of the cannula resulted in subconjunctival bleeding in 30% of patients. Accumulation of small amount of fluid or silicone oil after withdrawal of the cannula was noticed. This ooze stopped spontaneously when pressure was applied to the conjunctival incision pushing it laterally to separate the conjunctival incision



Fig. 6. Instrumentarium for transconjunctival sutureless 23 g vitrectomy

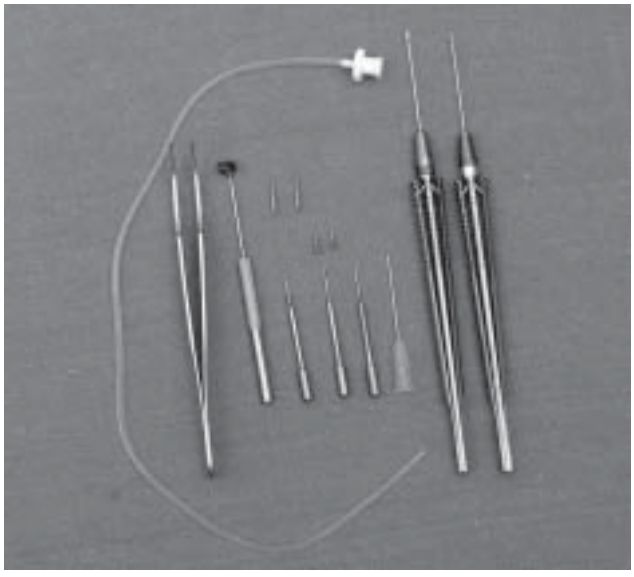


Fig. 7. 23 Gauge Intraocular forceps and scissors used for 23 Gauge transconjunctival sutureless vitrectomy.

from the scleral incision. Subconjunctival haemorrhage was observed in 2 eyes which cleared spontaneously on 3rd postoperative day. Sutures were not used in any of the cases to close the conjunctival or scleral incisions. During postoperative follow up applanation tonometry was consistently within the normal range in 36/37 cases. In one patient postoperative IOP recording was 8 mm Hg and the IOP increased to normal by the third postoperative day. No postoperative subconjunctival gas or silicone oil was observed on follow up.

This procedure provides certain definite advantages over the standard 20 gauge pars plana vitrectomy. There is minimal trauma of the conjunctiva and sclera. Postoperative scleral thinning over areas of sclerotomies is not seen. The incidence of postoperative suture related astigmatism is reduced and the postoperative period is comfortable with earlier and easier rehabilitation.

The advantages of 23 gauge vitrectomy over 25 gauge TVS are

1) less flexibility of instruments, 2) shorter operating time, 3) longer durability of instruments and 4) lesser learning curve

Conclusion

In summary, the 23 gauge sutureless vitrectomy procedure appears to be a viable alternative to 25 gauge vitrectomy. It offers all the advantages of minimally invasive tranconjunctival vitrectomy system with benefits of a sturdier and larger instrumentarium.

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ORIGINAL ARTICLE

ND Yag Hyaloidotomy for Premacular Haemorrhage in Valsalva Retinopathy

Dr. Mahesh G., Dr. A. Giridhar, Dr. S.J. Saikumar, Dr. Savita Bhat, Dr. Sachin Fegde, Dr. Sandhya N., Dr. Anna Elias

Abstract: Premacular subhyaloid haemorrhage can result in sudden profound visual loss after Valsalva maneuver. Nd Yag Hyaloidotomy is a simple and effective method of treatment in these cases. We present a series of subhyaloid haemorrhage in Valsalva retinopathy, which were successfully treated with Nd Yag Hyaloidotomy.

Key words: Nd Yag Hyaloidotomy, Valsalva retinopathy, subhyaloid haemorrhage.

Introduction

Premacular subhyaloid haemorrhage may occur from proliferative diabetic retinopathy, ruptured retinal macro aneurysm, neovascularization in branch retinal vein occlusion, Valsalva retinopathy and Terson Syndrome. This can result in sudden profound visual loss. A fibrotic epiretinal membrane may develop in longstanding subhyaloid haemorrhage resulting in relatively poor visual outcome. Altered blood products may result in pigment alteration in the macula in persistent subhyaloid haemorrhage. Valsalva retinopathy is a rather uncommon condition and this can result in dense premacular haemorrhage. Valsalva maneuver is forced expiration against a closed glottis, which can lead to sudden increase in intrathoracic and intra abdominal pressure. This results in sudden increase in pressure in the veins of head and neck. Different ocular manifestations can occur which includes subconjunctival or subhyaloid haemorrhage. The source of blood in the subhyaloid haemorrhage is the capillaries. In young patients where there is no posterior vitreous detachment this blood collect in the premacular area resulting in a boat shaped haemorrhage with a horizontal upper level. The common causes of Valsalva Retinopathy include forceful coughing, sneezing, weight lifting, intercourse and other strenuous activities. The result may be sudden and alarming loss of vision. There are reports on the natural course of this blood and it takes months for clearing. Observation or vitrectomy is the current way of managing a premacular haemorrhage. Hyaloidotomy or membranotomy using different lasers are reported in a variety of premacular haemorrhages due to causes other than Valsalva retinopathy. This study was undertaken to evaluate the role of photo disruptive Nd: Yag Hyaloidotomy to displace the premacular haemorrhage in cases of Valsalva related premacular haemorrhage.

Methods

This was a prospective consecutive single center interventional case series. The study period was from January 2002 to December 2003. Six eyes of six patients with dense premacular haemorrhage following Valsalva Retinopathy were included in the analysis. All the patients reported with sudden loss of vision noticed after Valsalva maneuver. There were 3 males and 3 females. Age group of the patients ranged from 17 to 44 with an average age of 29.16 years. Average time duration between the onset of the disease and

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No	Age	Sex	Duration	Vision at presentation	Baseline LogMAR vision	Cause	Final log MAR vision	Final Vision
1	28	Female	2 days	CF 1m	1.8	Vomiting, pregnant	0	6-Jun
2	18	Male	7 days	CF 3m	1.3	Weight lifting	0	6-Jun
3	39	Female	3 days	CF 1/2m	2.1	Vomiting, pregnant	0.48	18-Jun
4	29	Male	1 day	CF 1m	1.8	Weight lifting	0	6-Jun
5	44	Male	15 days	CF 3/4m	1.89	Weight lifting	0	6-Jun
6	17	Female	7 days	CF 1/2m	2.1	Vomiting, appendicitis	0	6-Jun

presentation was 5.8 days, which ranged from 24 hours to 15 days. The cause of the bleeding was lifting of heavy weights in three patients, vomiting associated with pregnancy in two patients and vomiting following acute appendicitis in one patient. Visual acuity on presentation ranged from counting fingers close to face to counting fingers at three meters. Colour fundus photograph was taken at base line. Detailed peripheral retinal examination with indirect ophthalmoscopy was carried out to rule out any other peripheral retinal lesions or retinal vascular diseases.

Nd-Yag laser was used for creating a hole in the posterior hyaloid. The area for Hyaloidotomy was selected as the area, where the blood was thickest and which was farthest from the fovea. The same settings of 3.8 – 4.2 mJ were used in all cases. 1-4 shots were applied to create a hole in the posterior hyaloid. Goldman 3 mirror contact lens was used for focusing after local anaesthetic (proparacaine) instillation. Immediate post laser colour fundus photographs were taken. (Figure 1 & 2) Patient was discharged the same day and reviewed after 1 week, 1 month, 3 months, 6 months and 1 year after the procedure. In all follow up visits Snellen visual acuity was recorded. A detailed peripheral retinal examination was done and colour photos were taken. Fluorescein angiography was not done as the cause of bleeding was obvious.

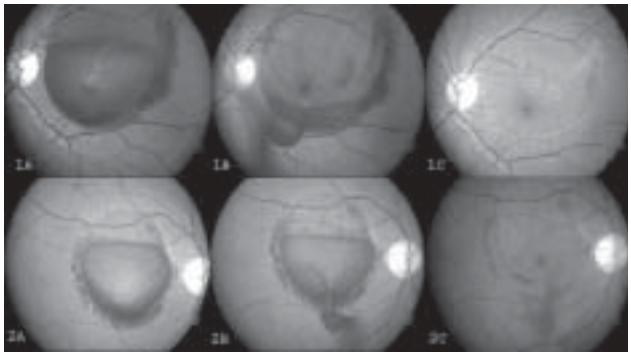


Fig. 1A. Pretreatment photograph showing dense subhyaloid haemorrhage. 1B: Immediate post treatment photograph showing displacement of blood 1C: After 1 month Fig. 2A. Premacular haemorrhage after Valsalva maneuver 2B: After Nd Yag Hyalodotomy 2C: 6 weeks later]

Results

Successful Hyaloidotomy using photo disruptive Nd-Yag laser was possible in all the cases. Immediate displacement was seen in all cases. Blood drained into the vitreous cavity clearing the visual axis. In all cases except one the lower dependent area of the boat shaped subhyaloid haemorrhage was chosen for laser application. In one case the lower end of the blood was at the fovea. So laser photo disruption was done at the center of the haemorrhage and patient was asked to lie in prone position. All the others were advised head elevated position till the blood cleared from the visual axis. Primary outcome measure was the visual acuity. At one week 5 out of 6 patients had regained 6/6 vision. One patient had 6/18 vision due to persistent vitreous haemorrhage. This visual acuity was maintained in the subsequent follow-ups. Average follow up duration was 12.5 months (Range 3 months to 22 months). No peripheral retinal lesion was noticed in any cases. Wrinkling of posterior hyaloid was seen in two cases, which reduced in the subsequent follow up visits. This was unrelated to the visual acuity. No other complications were noticed in any of the cases. All the patients complained of floaters in the first visit, which reduced in the follow up visits.

Statistical analysis

For statistical purposes the Snellen visual acuity was converted to logMAR. The mean logMAR visual acuity at presentation was –1.829 with a standard deviation of 0.292. Final logMAR vision had a mean of

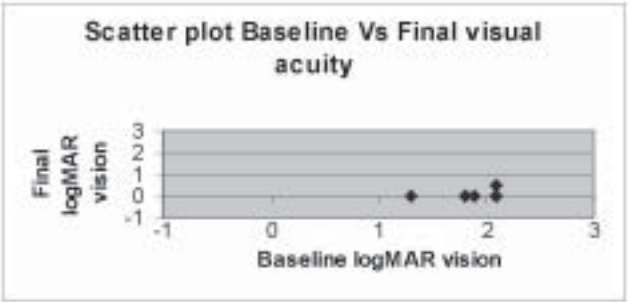


Fig. 2. Scatter plot showing baseline and final visual acuities

0.080 with a standard deviation of 0.197. This definitely shows improvement. The change was statistically significant (Wilcoxon signed rank test p = 0.027). The scatter plot with baseline and final visual acuity is given in Fig. 2.

Discussion

Laser Hyaloidotomy has been described by many authors in the past for the treatment of dense premacular haemorrhage. [1]Raymond et al has used the same parameters in premacular subhyaloid haemorrhage due to proliferative diabetic retinopathy and ruptured retinal artery macroaneurysm. The visual acuity was not satisfactory in that series due to the co-existing retinal changes. Similar treatment was given by [2]Ulbig et al for a series of premacular subhyaloid haemorrhage due to different causes and a subject with Valsalva retinopathy fared the best. Our findings support this good visual prognosis in Valsalva related subhyaloid haemorrhage. [3]Frequency double Nd-Yag also had been used to treat different causes of subhyaloid haemorrhage. [4] Kwok AK et al had reported formation of epiretinal membrane following membranotomy in Valsalva retinopathy.

This interventional case series clearly underlines the safety and efficacy of Nd-Yag Hyaloidotomy in the treatment of Valsalva retinopathy. No complications were reported on the last follow up. Detailed retinal examination is suggested because of the photo disruptive action of Nd-Yag laser, which can theoretically create tractional or even rhegmatogenous retinal detachment secondary to the induction of posterior vitreous detachment. Though all the patients complained of floaters, this cleared within a few weeks. The use of Goldmann 3 mirror contact lens enabled accurate focusing of aiming beam, which was absolutely essential considering the photo disruptive action of Yag laser which can even create retinal breaks. The good visual prognosis in Valsalva retinopathy is related to the fact that the retinal vessels and macula were healthy unlike in other causes of subhyaloid haemorrhage like proliferative diabetic retinopathy.

Not many natural history studies are available on the outcome of Valsalva retinopathy. It is described by many authors that spontaneous clearance will take many months. Considering the safe outcome we think that Nd-Yag Hyaloidotomy is an excellent tool in the early rehabilitation of the patients. Within one week all the patients recovered nearly complete vision.

We conclude that Nd-Yag Hyaloidotomy is a safe and effective treatment for profound visual loss due to premacular subhyaloid haemorrhage in Valsalva retinopathy. Prospective randomized controlled trials are necessary to give the final answer to this problem.

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ORIGINAL ARTICLE

Core Vitrectomy (CV) Versus Near Total Vitrectomy (NTV) and Posterior Vitreous Detachment Induction with Retinal Surface Cleaning in Visual Outcome of Post Operative Endophthalmitis

Dr Gopal S Pillai, MD, DNB, FICO, FRCS

Endophthalmitis is one of the most devastating eye complications that can occur following intraocular surgery. The most common cause of endophthalmitis is a bacterial infection after cataract surgery. This complication develops in fewer than one out of every 1000 patients who have had cataracts removed. It is a serious problem that can lead to permanent loss of vision. Symptoms vary slightly, depending on whether the infection occurs early (six weeks or less) or late (months or years) after surgery. If the outcome is to be successful, it is essential that the diagnosis is recognized and not denied. Once the diagnosis is made, treatment should begin without delay to avoid further deterioration of visual acuity.

To date, there has only been one large prospective randomised study on the management of endophthalmitis. The Endophthalmitis Vitrectomy Study (EVS), recently completed in the USA, has greatly simplified our approach to treatment, which should begin immediately after the diagnosis is made.

In all cases, where the acuity is better than light perception, a single-port vitreous biopsy via the pars plana should be performed using a vitreous cutting-suction device. (Disposable devices are now available which allow the procedure to be done outside the operating theatre if necessary with even less delay. This can be useful in outreach clinics). The specimens are directly smeared, for Gram stain etc, and plated for culture.

The space created by the biopsy is sufficient for direct intravitreal injection of antibiotics. In the EVS, amikacin and vancomycin were used. Now many people have switched over to vancomycin and ceftazidime. The study showed that there was no advantage in the concurrent administration of intravenous antibiotics.

Only when the visual acuity is Hand movements is there an advantage in performing a formal three port vitrectomy, from the point of view of both final acuity and media clarity.

What EVS described for the vitrectomy was a core vitrectomy which removed part of the central vitreous involved by the infection. They did not proceed to see the retina, assess it or to induce a posterior vitreous detachment.

Now with technology advancing at a rapid pace, our instrumentation is far better than what it was and vitreo

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retinal surgeons are far more comfortable and brave to go nearer the retina to induce a PVD, even in endophthalmitis. In this study we compare and contrast core vitrectomy with near total vitrectomy with PVD induction and retinal surface cleansing in the visual outcome of post operative endophthalmitis.

Lacuna in knowledge

Paucity of studies in which near total vitrectomy has been attempted in the management of endophthalmits.

Aims

To compare core vitrectomy (CV) and near total vitrectomy with PVD induction and retinal surface cleaning (NTV) in the visual outcome of postoperative endophthalmitis.

Type of study

Randomized clinical trial

Randomization

14 cases of endopthalmitis managed surgically were randomly allocated to the core vitrectomy group or the near total vitrectomy group. Random allocation to either group was done after the 6 mm infusion cannula was placed in position.

Inclusion criteria

1 Endophthalmitis following cataract surgery
2 Visual acuity less than hand movements

Exclusion criteria

1 Any other intraocular surgery done in the past
2 Any ocular disease like age related macular degeneration and glaucoma which worsens visual prognosis.
3 Corneal involvement

Methods

All cases were done with Bausch and Lomb Millenium vitrectomy machine with 750 cuts per minute. Random allocation to either group was done after the 6 mm infusion cannula was placed in position. In the core vitrectomy group, after a central vitrectomy, without going into the periphery of the vitreous or near the retinal surface, ports were closed and cryopexy performed.

In the near total vitrectomy group, after a core vitrectomy, a wide angle system was put in place and a near total vitrectomy was performed. PVD was induced with aspiration or forceps near the optic disc. After PVD induction, the rest of the vitreous was also eaten up. Retinal surface was studied and was cleansed with a silicone tipped backflush (Dorc)

All results were analyzed by statistical methods.

Outcomes studied were

1 Post operative media clarity as graded by the EVS classification
2 Post operative visual acuity at 1 week, 6 weeks and 12 weeks.
3 Number of surgeries
4 Complications

Results

There were 14 eyes of 14 patients in the study. The two groups had 7 eyes of 7 patients each. 10 males and 4 females were enrolled in the study. The average age of the patients was 68.2 years and was comparable between the two groups.

Pre operative visual acuity was hand movements or less in all the cases. The duration of endophthalmitis was from 1 day to 1 week. There was no statistically significant difference between the two groups.

Post operative media clarity was assessed on day 1, 1st week and 2nd week. It was classified according to the EVS study where grade 5 was normal and grade 0 was no visibility of the fundus.

Media clarity was statistically better in the near total vitrectomy group when compared to the core vitrectomy group. ($p<0.05$) (Table 1)

Visual outcomes were studied in both the groups at day1, 1st week, 6 weeks and 12 weeks. Snellen visual acuity was taken and was reciprocated to form decimal visual acuity for statistical analysis.

Visual acuity was statistically better in the NTV group than the CV group ($p<0.03$) at all the follow ups (Table 2).

Table 1. Grade of media haziness as per EVS in core vitrectomy group (CV)) and near total vitrectomy group(NTV)

CV	Day 1	0	0	0	0	0	0	0
CV	1st week	0	1	0	1	0	0	1
CV	2nd week	1	1	2	2	2	1	2
NTV	Day1	2	2	1	3	1	2	2
NTV	1st week	3	3	1	4	2	2	3
NTV	2nd week	4	3	2	4	3	2	3

Table 2. Snellens visual acuity in core vitrectomy group (CV)) and near total vitrectomy group(NTV)

Duration	Case 1	2	3	4	5	6	7	CV
Day 1	HM	PL	PL	HM	HM	HM	PL	
CV	1st week	FCCF	FCCF	HM	FCCF	HM	HM	FCCF
CV	6 weeks	1/60	2/60	4/60	HM	FCCF	HM	FCCF
CV	12weeks	2/60	PL	6/60	HM	2/60	HM	FCCF
NTV	Day1	HM	FFCF	HM	1/60	FFCF	PL	FCCF
NTV	1st week	3/60	2/60	1/60	4/60	2/60	PL	1/60
NTV	6 weeks	6/60	4/60	2/60	6/36	6/60	PL	3/60
NTV	12weeks	6/18	4/60	2/60	6/18	6/60	PL	6/36

3 cases underwent a secondary vitrectomy in the core vitrectomy group. None of the near total vitrectomy group needed a second surgery. Thus the need for resurgery was more in the core vitrectomy group.

No surgical complications were faced in both the groups.

Conclusions

To conclude, near total vitrectomy seems to be a more effective, but more aggressive approach to post operative endophthalmitis.

Media clarity during early as well as late post operative phase is uniformally better in the near total vitrectomy group.

Post operative visual acuity was better in the near total vitrectomy group when compared to the core vitrectomy group.

Secondary vitrectomy to take out vitreous opacities were necessary in the core vitrectomy group. No resurgery was required in the near total vitrectomy group.

No complications were seen even after PVD induction in endophthalmitis. There was no case of retinal detachment.

We could see, assess and prognosticate the retina after visualizing it in the near total vitrectomy group. It was not possible in the core vitrectomy group.

Though larger multicentric randomized studies are required in asserting the benefit of near total vitrectomy with PVD induction and retinal surface cleansing over core vitrectomy, the results of this pilot trial is encouraging to vitreous surgeons who have split hair in the management of this most dreaded complication of cataract surgery.

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ORIGINAL ARTICLE

An Ode To The Cupped Disc

Dr Chandrima Paul, Dr Ajoy Paul, Dr Partha Biswas, Dr P. K. Bakshi

Introduction

The presence and the course of glaucoma are generally assessed by means of optic disc evaluation and visual field tests. There is a close relationship between the morphology of the optic disc and the visual field. Changes in the optic disc usually preceed detectable visual field loss in early glaucoma.

Expressions of optic nerve damage typically rely on the cup/disc ratio(c/d ratio) which has been shown to correlate with the level of visual field loss. This staging however, has some shortcomings.When emphasis is placed on increasing cup size as a sign of worsening of glaucomatous damage,there is no direct description of the actual change occurring in glaucoma – the loss of neuroretinal rim tissue.It also does not consider the effects of optic disc size, which may affect the ability to detect glaucomatous damage.The neuroretinal rim area is an optic disc variable that has been shown to be superior to the c/d ratio in its correlation with visual function and its ability to differentiate eyes with suspected glaucoma and those with glaucomatous progression.

Aim

To evaluate which morphological features of the optic disc are predictive factors for development or progression of visual field loss in POAG.

Materials and Methods

The study included 582 eyes of 342 Indian individuals who attended the glaucoma service of B B Eye Foundation over last 3 years.

274 eyes were labelled as ocular hypertensives on the basis of 1)BCVA of atleast 20/20 with =+5 or -5 Dsph and =+2 or -2 Dcyl, 2) IOP = 22mmHg 3) Central Corneal Thickness-within normal limits, 4) Asymmetrical Cupping>0.2 difference in two eyes or >0.6 in either eye 5) Open angles on gonioscopy 6)Transparent ocular media 7) Humphrey Visual Field Analysis – within normal limits(24-2 Full Threshold) 308 had POAG with1)IOP = 22mmHg 2)Central Corneal Thickness-within normal limits 3) Asymmetrical Cupping>0.2 difference in two eyes or >0.6 in either eye 4) Open angles on gonioscopy 5) Transparent ocular media 6) Humphrey Visual Field Analysis-24-2 full threshold protocol was used

For baseline diagnosis, fields were documented as abnormal when there were

1) 4 points depressed at p<5% or 2)a cluster of 3 points depressed at p<1% Field progression was defined as reproducible decline of atleast 2dB in Mean Deviation and Pattern Standard Deviation from baseline.

Optical Coherence Tomography was performed and 3-Optic Nerve Head analyses using fast optical disc scan was used.

For baseline diagnosis cup area, cup disc ratio, rim area and rim disc ratios were correlated with standard visual field parameters of mean deviation and pattern standard deviation.The unit of measurement was the rim area, rim/disc ratio,cup area and cup/disc ratio as depicted by the optic nerve head changes in the optical scan of the OCT3. When there is no rim, the rim/disc ratio was taken as 0.0.When there is no cup, the rim/disc ratio is taken 0.5.

Glaucomatous progression was recognized by documentation of loss of rim area and reduced rim disc ratio and increased cup area and cup disc ratio.

Visual Field tests and optical disc scans were performed every 3 months and reviewed.

BB Eye Foundation, Kolkatta

Optic discs OH

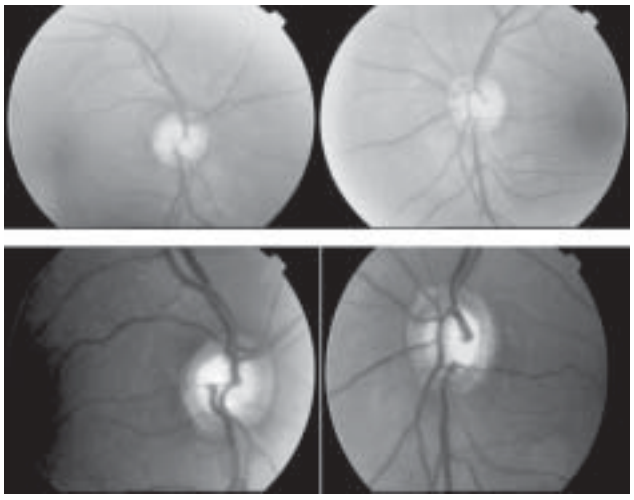
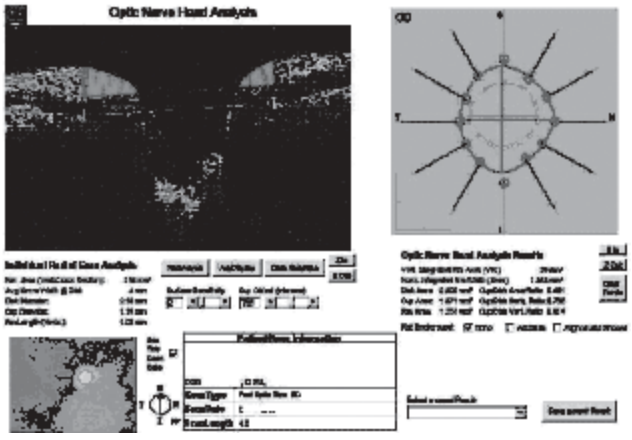


Fig 1a. Optic nerve imaging in an ocular hypertensive Note the asymmetric cupping and a CD ratio of 0.6 (RE) 0.4 (LE)

Ocular Hypertensive



OD: Disc area - 3.529, Cup area - 1.955, CD area ratio - 0.6, Rim area - 1.574, RD area ratio - 0.5

Fig 1b. Optic nerve head analysis by OCT 3 in a patient with ocular hypertension

POAG

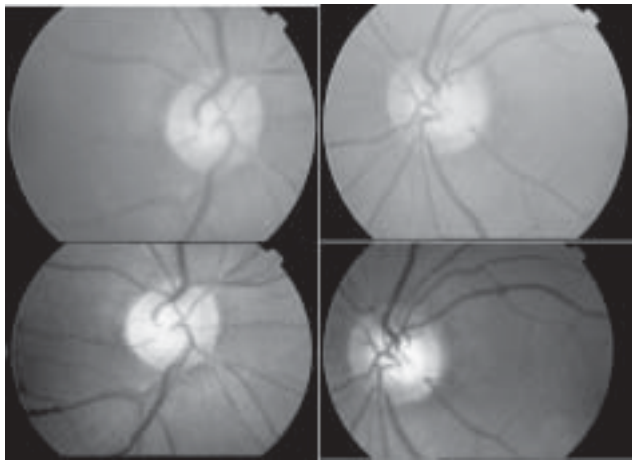
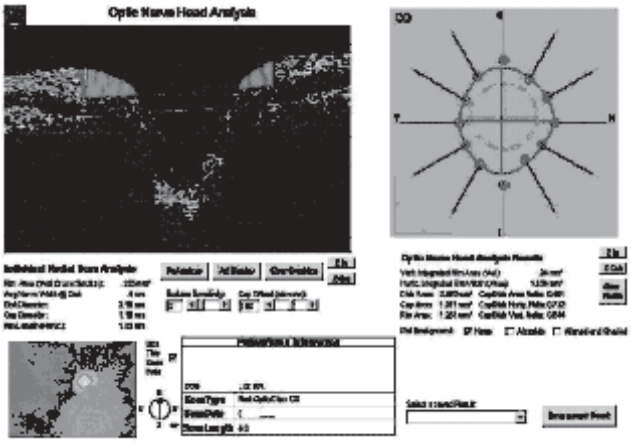


Fig 2a. Optic nerve head imaging in a known glaucoma patient. Note the asymmetric cupping with CD ratio of RE 0.4 and LE 0.6

POAG



OD: Disc area - 2.322, Cup area - 1.071, CD area ratio - 0.5, Rim area - 1.251, RD area ratio - 0.5

Fig 2b. Optic nerve head analysis by OCT gives a CD area ratio of 0.5 (OU) in a patient with Primary Open Angle Glaucoma

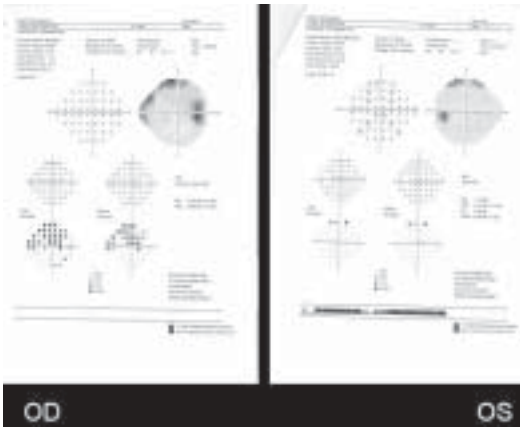
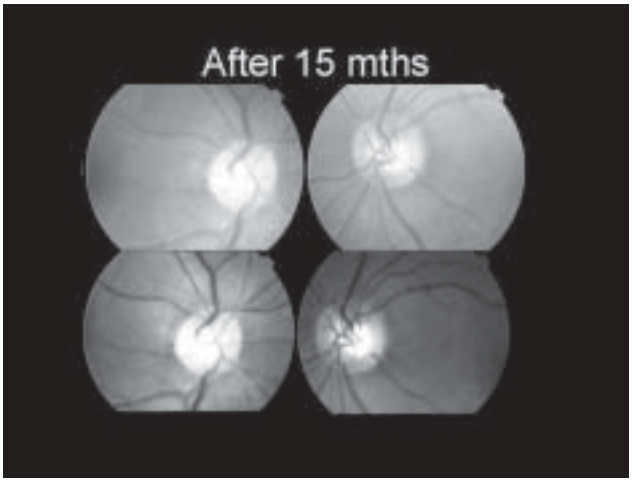


Fig 2c. C 30-2 Analysis in a POAG patient

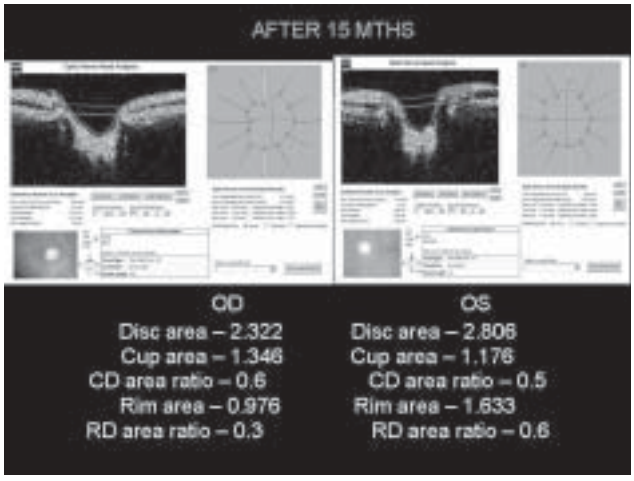
Results

Baseline glaucoma diagnosis and progression by ONH analysis was detected in 68 eyes. When correlated with the VF, statistically significant correlation was noted with decreased rim area ($p<0.001$) and reduced rim/disc ratio($p<0.01$) in all disc sizes (Table 1). But the correlation of VF with increased cup area and cup/disc ratio was($p>0.05$) insignificant in small (<1.5 mm)and average (1.5-2.0 mm) size disc and ($p>0.07$) in large disc size. (Table 2 & Fig 4)

(a)



(b)



(c)

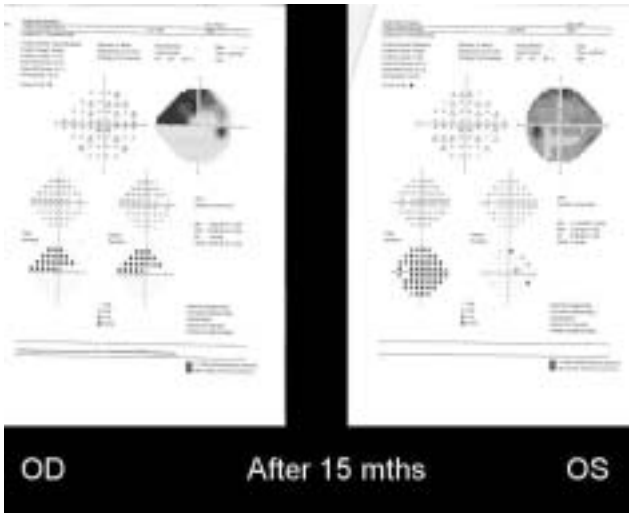


Fig. 3. (a-c) Progression of disc changes, optic nerve head analysis parameters, and visual field documented on follow up after 15 months in an ocular hypertension

Table 1. Visual field correlation with Optic Nerve Head parameters

RESULTS		
OH	Converters	P value
Increased cup area **n=28**	12	>0.05
Increased cup/disc area ratio **n=30**	12	>0.05
Decreased rim area **n=34**	28	<0.001
Decreased rim disc area ratio **n=22**	16	<0.01

Table 2. Visual field correlation with Optic Nerve Head parameters in the POAG group

RESULTS		
POAG	Progression of visual field	P value
Increased cup area **n=76**	20	>0.05
Increased cup/disc area ratio **n=76**	20	>0.05
Decreased rim area **n=42**	36	<0.001
Decreased rim disc area ratio **n=42**	24	<0.01

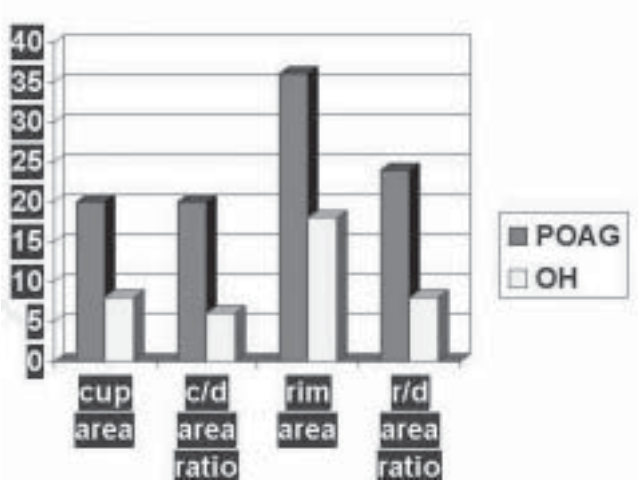


Fig 4. Bar diagram showing correlation between the visual field progression and optic disc parameters in POAG and Ocular Hypertension Group.

Discussion

Documentation of loss of rim tissue was the surest sign of progression when correlated with reduction of sensitivity in the pattern deviation plot of the VF. In correlation with the VF, neuroretinal rim area and rim/disc ratio are superior to cup area and cup/disc ratio in their ability to recognize ocular hypertensives and POAG patients.

Conclusion

The morphological features of the optic disc which are the predictive factors for development or progression of Visual Field loss are loss of rim area and reduced rim/disc area ratio.

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Humor In Ophthalmology

Dr. Verma R.R.

Malapropos Ophthalmicus

Each specialty has its own language, vocabulary and grammar. Any outsider may be pardoned for mispronunciations, choice of wrong words, or spellings. But some of them are too hilarious to be forgotten.

I am sure a lot of you have had patients talking about 'diluting' the eyes. Of course, if you put some liquid in anything, it should get diluted, shouldn't it? One patient even asked me if I will ' concentrate' his eyes afterwards, as he had no driver to drive him home. (He was not far off the mark, you see; dictionary defines concentrate as bringing to the centre).

Confusions between short sight, long sight etc. are too numerous. One person claimed that he had 'axes' on his right sided glass. May be they gave him 'sharp' vision. Yet another wanted a mirror for reading purposes. Of course he meant glasses but got confused as he thought in vernacular and translated literally. Then there was a patient who did not have 'clearance' for the last line but would 'take risk' and read. The number of first time patients who try to read lines on Snellen's Chart as words are many.

Names of medicines too lend themselves to hilarious malapropisms. One elderly lady with constricted pupils solemnly assured me that she was regularly putting 'Pilex 'in her eyes. During the early eighties another of my patients said that she was prescribed 'Janatha-mycin' by doctor. Considering the very low cost of Gentamicin Eye Drops (Rs. 2.50, I think) at that time, I thought the name very apt. The number of patients putting 'Allergan' eye drops is not small. (One even said she was putting 'Allergy' eye drops).

Haven't we all had patients who said they had 'B.P.' in their eyes? Or wanted to know if they should 'ferment' the hordeolum? Talking of hordeolum, one of my younger patients told me that he had a 'tie' in his eye. When an elderly man asked if 'senses' could be put in his eyes, I did not dismiss it as nonsense. I explained about the rigid and foldable ones.

About ten years ago one of my patients visited her son in Mumbai and had a 'fake emulsification' done there. As she had a 165-degree limbal would stitched with 10/0, the name was apt indeed.

If only one could remember all such trivia……

* Malapropism- Use of a word with similar sound or spelling instead of the right one. This word is derived from the character "Mrs. Malaprop" in the play "The Rivals" (1775) by Richard Brinsly Sheridan

Preservatives and Ocular Surface Diseases

Dr. Samar K. Basak, MD, DNB

No ophthalmologist wants to prescribe an ophthalmic preparation, which, though effective, would cause adverse effects on ocular surface over a period of time due to preservative. However, this effect can occur, if he or she does not pay close attention to a medication's ingredients and possible side effects. This is especially important if a patient may need to use ophthalmic solutions indefinitely.

An ocular medication is much more than the active drug. Its other component may cause problem for some patients. This is especially true for those who overuse their artificial tears products, use multiple topical medications; suffer chronic eye diseases like dry eye or glaucoma or require long term post-surgery medication.

Inside a multi-dose ophthalmic vial

Ocular medications are composed of unique mixtures of the active drug, a preservative, the drug delivery system, viscosity-increasing agents, and an aqueous buffered vehicle (Table 1).

Of these, it is the preservative, that has most often been considered the culprit in damaging the corneal epithelium leading to disruption of the apical glycocalyx. This usually happens when the drops are used beyond the recommended dosage. This sequence leaves the epithelium unable to keep the tear-film in place and can lead to ocular surface diseases (OSD).

Advantages of preservatives

All multi-dose topical ophthalmic preparations use preservatives. In fact, it is mandatory by drug regulation.

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Preservatives play a key role in maintaining the sterility of tears substitutes, antibiotics or other ocular medications through multiple uses. They protect against bacterial, fungal or viral contamination of ophthalmic solutions (drop) that can occur when the dropper tip touches the skin, eyelids, fingers or other non-sterile surfaces. Once the tip is contaminated, the offending agent may be aspirated back into the bottle; or it may blend into the solution with the next use as the medication collects on the tip before dropping into the eye. But the ophthalmic ointments are often supplied non-preserved, presumably because they are not as susceptible to contamination from retrograde flow back into the tube.

Preservatives also prolong the shelf life of the formulation by preventing bio-degradation and maintaining drug potency. The primary concern with many preservatives is not their value or efficacy, but rather their recognized cytotoxic side-effects. Although intermittent use of preserved multi-dose eye drops in normal individuals is probably not harmful, high concentrations of some preservatives can cause damage and irritation to the ocular tissue, particularly in patient with dry eye or glaucoma.

Preservative surface toxicity may be difficult to recognize (Fig. 1 to 3). The differential diagnosis includes chronic inflammation from dry eye, episcleritis, conjunctivitis or blepharitis. That means preservative-induced toxicity does not have distinct signs or symptomatology. *So, damage due to ophthalmic preservatives often goes undiagnosed because it is difficult to differentiate toxicity of a medication from the damage caused by a preservative.*

Awareness of the potential effects of preservatives on overall ocular surface health is relatively low.

Table 1. Composition and Preservative of commonly used artificial tears available in India*

No	Trade Name	Composition	Preservative	Company
1	**Refresh Tears**	CMC 0.5%	Purite	*Allergan*
2	**Refresh Liquigel**	CMC 1.0%	Purite	*Allergan*
3	**Celluvisc Gel**	CMC 1.0%	Nil	*Allergan*
4	**Genteal**	HPMC 0.3%	Perborate	*Novartis*
5	**Eye Mist**	HPMC	Purite	*Milmet*
6	**I-Lube (Minim)**	1.4% PVA 0.6% Povn	Nil	*FDC*
7	**Lacrisol**	1.4% PVA 0.6% Povn	BAK 0.02%	*Microvi*
8	**Polyvin**	1.4% PVA 0.6% Povn	Chlorbu 0.5%	*Optho*
9	**Irisol Plus**	1.4% PVA 0.6% Povn	BAK 0.02%	*Warren*
10	**Ocuwet**	1.4% PVA 0.6% Povn	BAK 0.01%	*Centaur*
11	**Dudrop**	1.4% PVA 0.6% Povn	BAK 0.01%	*Milmet*
12	**Moss**	1.4% PVA 0.6% Povn	BAK 0.01%	*Syntho*
13	**I-Lube**	1.4% PVA 0.6% Povn	BAK 0.01%	*FDC*
14	**Moisol**	HPMC 0.07% + Salts	BAK 0.01%	*FDC*
15	**HypoTears plus**	5% Polyvidone + Elec.	BAK 0.005%	*Novartis*
16	**Tears Plus**	1.4% PVA 0.6% Povn	Chlorbutanol 0.05%	*Allergan*
17	**Tears Naturale**	HPMC 0.3%	Polyquad 0.001%	Alcon
18	**GenTeal Gel**	HPMC 0.3%	Perborate	Novartis

Nearly every type of ophthalmic product uses preservatives from artificial tears and contact lens solution to medications such as antihistaminics, antimicrobials, NSAIDS, antiglaucoma and corticosteroids.

Types and Mechanism of Action of Preservatives

Ocular preservatives can be classified into *two main categories*: **detergents and oxidative.**

Detergent preservatives

They act upon micro-organisms by altering cell membrane permeability and lysing the cytoplasmic content *e.g.,* Benzalkonium chloride (BAK), Chlorobutanol, Thiomersol and Polyquad. Ocular toxicity can occur because some detergent preservatives can affect eukaryotic cells and the preservative is incorporated into the cell and cause ocular damage.

Oxidative preservatives

They are usually smaller molecules that penetrate the cell membranes and interfere with cellular function. They can destabilize cell membranes, but to a lesser degree than detergent preservatives. Stabilized oxychloro complex (SOC) and Sodium perborate are two examples of oxidative preservatives.

At low levels, oxidative preservatives have an advantage over detergent preservatives because they can provide

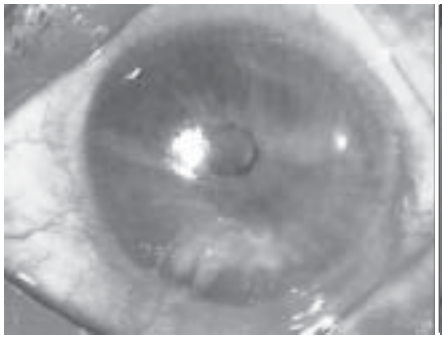


Fig. 1. Medicomentosa in normal cornea

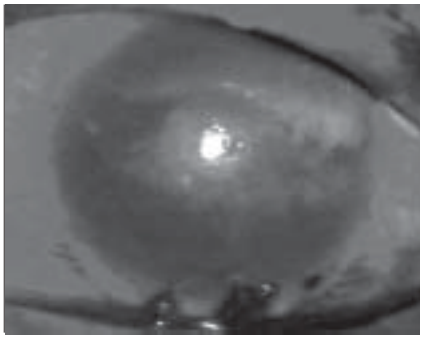


Fig. 2. Medicomentosa after Fluorescein staining

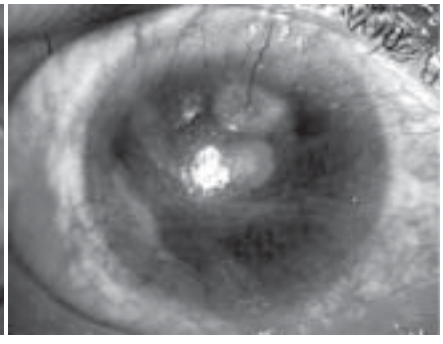


Fig. 3. Medicomentosa in severe dry eye patient

enough activity against micro-organisms while having negligible toxicity on eukaryotic cells. This is because many micro-organisms do not have the ability to cope with oxidative stress. Mammalian cells are equipped with antioxidant, oxidase and catalase to neutralize the effect of a low-level oxidants (Fig 4a to 4d).

Common ocular preservatives

Benzalkonium chloride (BAK)

BAK is a quaternary ammonium compound, and is the most common preservative used in topical multi-dose vials. It is often used in conjunction with disodium EDTA.

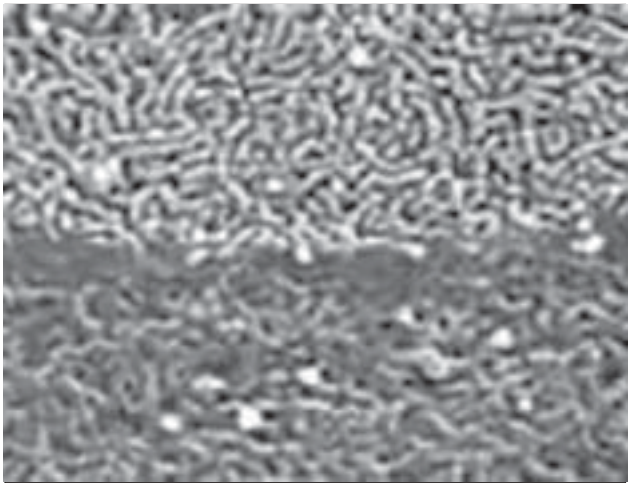


Fig. 4a. SEM of an untreated rabbit corneal epithelium with extensive microvilli and tight intercellular junctions (14,000X).

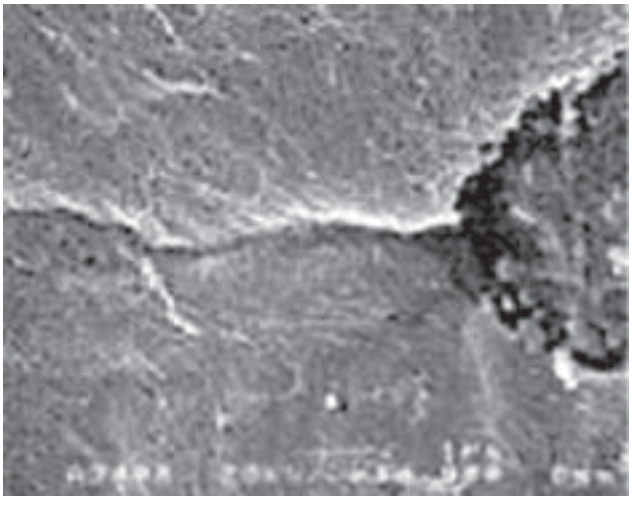


Fig 4b. Same corneal epithelium treated with a product preserved with 0.001% Polyquad 4 times for seven days (14,000X).

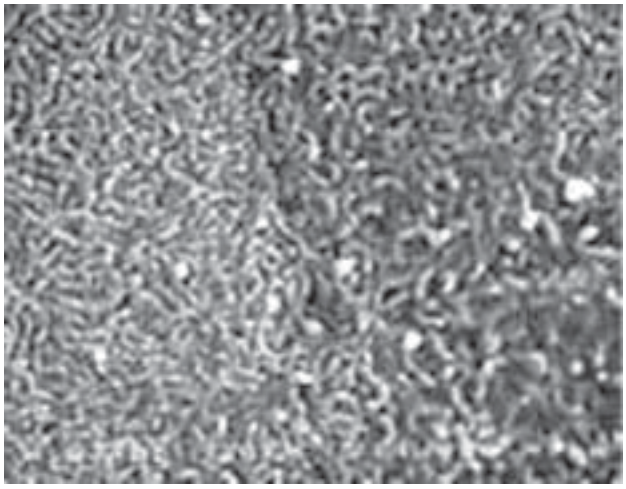


Fig 4c. Same corneal epithelium after Sodium perborate 4 times for seven days.

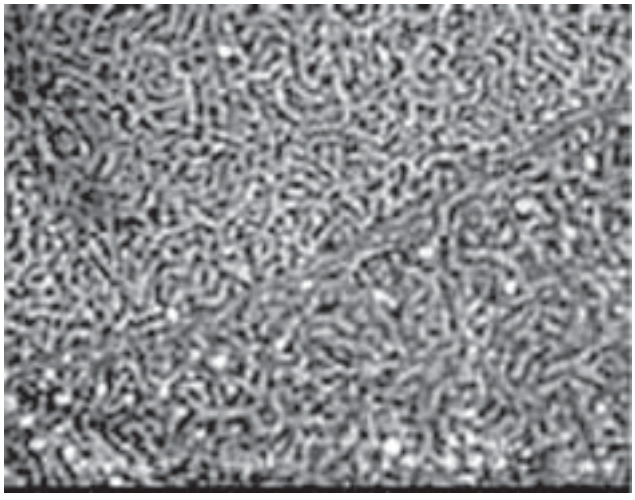


Fig 4d. Same corneal epithelium after treated with Purite 4 times for seven days.

(Fig 4a to 4d: Courtesy Dr. Robert Noecker, MD)

Edetate sodium is an additive which augments the preservative activity of BAK and other preservatives. However, it is not a true preservative by itself. This is just to lower the concentration of primary preservatives. Although EDTA may not be toxic to the normal rabbit epithelium, patients with severe dry eye often complains of irritation with preservatives that contain disodium edetate.

BAK has been the gold standard of preservatives for many years. It is chemically stable, does not degrade easily, even at a higher temperature. It is usually used at a concentration of 0.01% to 0.02%. BAK acts upon microorganisms by altering cell membrane permeability and lysing cytoplasmic contents. It is also shown to

increase the corneal penetration of some drug by causing a separation of the epithelium.

BAK can induce different types of cell destructions in a dose-dependent manner. It causes growth arrest at a very low concentration (0.0001%), apoptosis at 0.01% and cell necrosis at higher concentrations (0.05-0.1%) (Fig 5a and 5b).

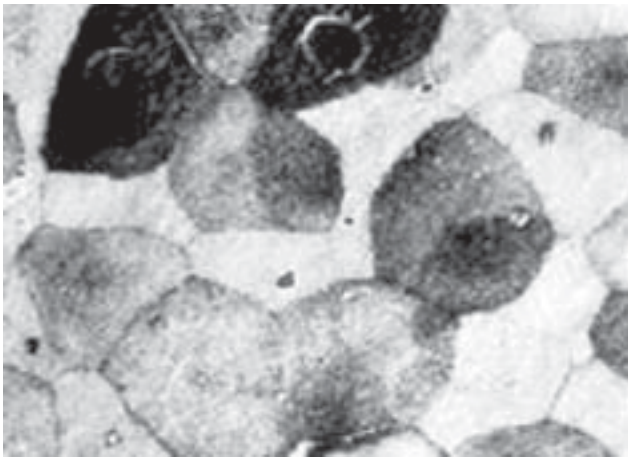


Fig 5a. SEM of rabbit corneal epithelium after 0.02% BAK 4 times for 7 days.

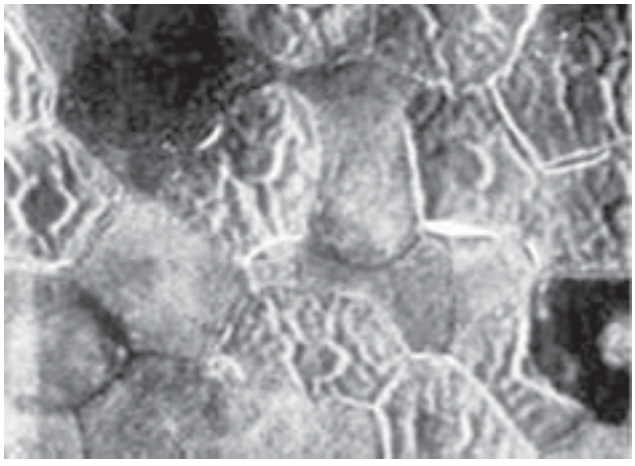


Fig 5b. Same cornea with 0.02% BAK 8 times for 7 days. An increase in epithelial holes, a loss of microvilli and wrinkling of surface cells.

(Fig 5a and 5b: Courtesy Dr. Debbasch C et al)

It is thought that patients at greatest risk of BAK-induced toxicity are those suffering from dry-eye syndrome. Because of lack of natural tears in these patients, the BAK in each eye drop is not as diluted as it would be in a patient with normal tear production.

At concentration and dosing used clinically, however, BAK does not appear to have significant adverse effects unless its frequency exceeds four to six times / day. This becomes a concern when patients use other drops on top of chronic medications, such as glaucoma drops. Additionally, patients with soft contact lenses must remove their contact lens for 5 minutes to prevent the absorption of BAK into the lens.

Clearly BAK is a wonderful, safe, potent preservative. We need to be careful with its use in patients when they are using several medications; are over-dosed or have a history of compromised corneal epithelium.

Chlorobutanol

It is an alcohol-based preservative; so it does not have surfactant action. It works by disorganizing the lipid structure of the cell membrane which increases cell permeability and leads to cell lysis.

Chlorobutanol has broad-spectrum antimicrobial activity. It is used as 0.5% in ophthalmic preparation and 0.05% concentration of chlorobutanol does not affect the lipid layer of tear film. In human corneal epithelial cells, the cytotoxic effects of chlorobutanol occur less rapidly than those of BAK and toxicity changes are less severe.

Polyquad (Polyquaternium-1)

It is a new polymeric quaternary ammonium antimicrobial preservative. It has been proven to have less toxic effect on corneal epithelial cells than BAK. It does not go into deep level and only cause superficial epithelial damage compared to BAK. It is used as 0.001% in ophthalmic solution.

Thiomersol

It is used as 0.001-0.004% in ophthalmic solutions. It causes cellular retraction, cessation of mitotic activity, and superficial corneal epithelial cell loss. *Phenyl mercuric nitrate, Methyl paraben and Propyl paraben* have similar antimicrobial activities as chlorobutanol, but they are relatively more epitheliotoxic than chlorobutanol or Thiomersol.

Sodium perborate

It is one of the first oxidative preservatives developed; and works by oxidizing cell walls or membranes and

thereby disrupting the cellular functions. It destroys most bacteria and can destroy even *Aspegillus niger.*

When Sodium perborate is combined with water it is converted into hydrogen peroxide, an effective antimicrobial agent. Once sodium perborate enters the eye, it is decomposed to water and oxygen by catalase and other enzymes present in the conjunctival surface (Fig 6). While gentler than other preservatives, sodium perborate may still cause ocular toxicity. Hydrogen peroxide levels above 100 ppm can cause ocular stinging

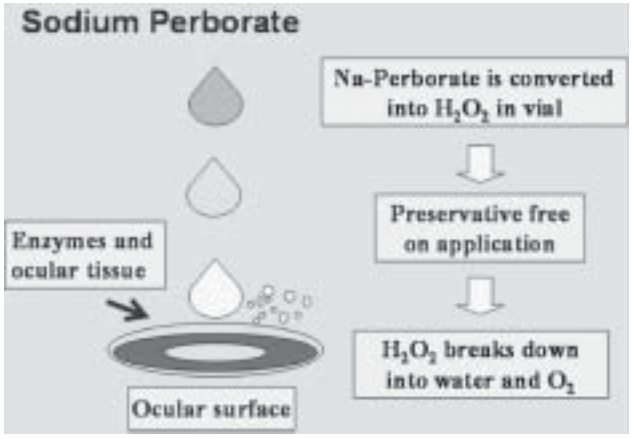


Fig 6. Mechanism of action of Sodium perborate

Stabilized oxychloro complex (SOC) or Purite

SOC is a relatively new preservative, first introduced in ophthalmic solution in 1996 and consists of an equilibrium mixture of oxychloro species: 99.5% chlorite (ClO_2^-), 0.5% chlorate (ClO_3) and trace amount of chlorine dioxide (ClO_2), which has bactericidal, fungicidal and viricidal activity.

SOC dissipates by converting into component normally found in tears, such as sodium-ion (Na^+), chloride-ion (Cl^-), oxygen and water. Due to oxidation potential of chlorite and possibly from the generation of chlorine dioxide in presence of acidic environments of the microbes, it leads to disrupt the protein synthesis and thereby kill them.

Sodium chlorite, the key component of SOC, has been used in water purification plant since 1944. It is also used in toothpaste, mouthwash and some antacids. SOC has wide spectrum of antimicrobial activities at a low concentration of 0.005% w/v. It has mild cytotoxic effect and has been given Environment Protection Agency Category II rating.

Both Sodium perborate and purite are noble preservatives and offer attractive options as multi-dose drugs for patients who either require more than six doses /day or for those who use more than one type of drop to treat concomitant diseases (*e.g.,* glaucoma and dry eye).

Conclusion

Today ophthalmologists have numerous topical preparations to choose from. When choosing a treatment regimen, both systemic side effects and ocular tolerability should be considered. Patients with KCS, corneal transplants, scarred conjunctiva from multiple surgeries and post-LASIK patients are at a higher risk for iatrogenic ocular surface problems. Prescribing eye drops with less harmful preservatives or oxidative preservatives is helpful in maintaining good ocular surface health. It will reduce the irritation and discomfort and thereby improve patient's compliance. One should choose the medicines with lower concentration of BAK for short term therapy or if the doses are four times or less. Thus, the efficient treatment strategies are required to minimize preservative-induced toxicity.

For the patient who uses an artificial tears product intermittently or as indicated, his concern may be few. But for individuals with some combination of glaucoma, dry eye, ocular infection or allergy and decreased corneal sensitivity, the choice of product is more important in relation to preservatives. In future, the manufacturers may reformulate the existing product with less toxic preservatives, offer less concentrated form of current preservatives or develop a new one.

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Specular Microscopy

Dr. Reena Rasheed, MS. DO

Specular Microscopy is a method of evaluation that allows the direct observation of corneal endothelium in clinical or eye bank set up. Other tissues that may be seen with specular microscope include corneal epithelium, the cells of crystalline lens, and various types of ocular debris, inflammatory cells and other optically reflecting structures. Changes in cell morphological characteristics of the corneal endothelium as seen in specular microscopy, reflects the stresses and strains that have been placed on that cornea and its functional reserve to respond favorably to unexpected stress.

History

Early work by Vogt and Goldmann visualized endothelial cell pattern in low magnification. Vogt coined the term *'Speigelmikroskopic'* to describe the process which is then translated to English as specular microscopy. In 1968,David Maurice developed the first high powered specular microscope to photograph endothelial cells in vivo at 500X.

Principles & Technique

Specular microscope is an optical reflection microscope that reflects a slit of light from the cornea and allows observation of this specularly reflected beam. Image that is seen is attributable solely to specularly reflected light rays. Depending on the objective lens used, the specular microsope may be either a contact microscope in which the front of the objective lens touches the cornea or a non contact microscope in which there is an airspace between the front of objective lens and cornea.

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Eye bank contact specular microscopes are used for observation of the corneal endothelium in the whole globe. However modern eye banks prefer to observe the cornea after it has been excised and placed in storage media. Observation in storage chambers using a non contact objective lens is the popular method of endothelial observation in eye banks. For most corneas endothelium is not seen easily at 4 degree C and must be warmed to room temperature for proper assessment. Corneas having a shorter warming time may be expected to have more active endothelial pump and thus better functional reserve.

For clinical use, the specular microscopy technique depends on the particular instrument that is used. Basic technique is to align the instrument to patient's eye so that the conditions for specular imaging are obtained. This essentially requires that the region of endothelium being observed be perpendicular to the optic axis of objective lens.

Most specular microscopes sold today capture video images that can be seen instantly on a video monitor screen. The image can be stored digitally and printed out on a laser printer and the data can be analyzed later. These machines are also capable of evaluating the following parameters like cell density, degree of pleomorphism and polymegathism.

Factors affecting image quality

Larger the numerical aperture of the objective lens greater the resolution of image. In addition it is very important that the front surface of the lens be clear. For eye bank specular microscopes the condition of the plastic or glass between the corneal endothelium

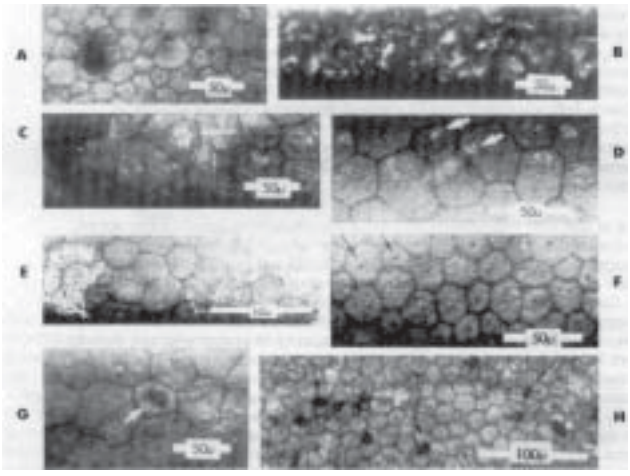


Fig. 1. Miscellaneous endothelial structures. A, Isolated smooth excrescences (cornea guttaga). B. Multiple coalesced excrescenes. C and D, Intracellular bright structures, E, Pigmented endothelial deposits. F and G, intracellular dark structures. H, Intercellular dark structures, believed to be invading inflammatory cells. (D, E and F courtesy of L. Neubauer, M.D.)

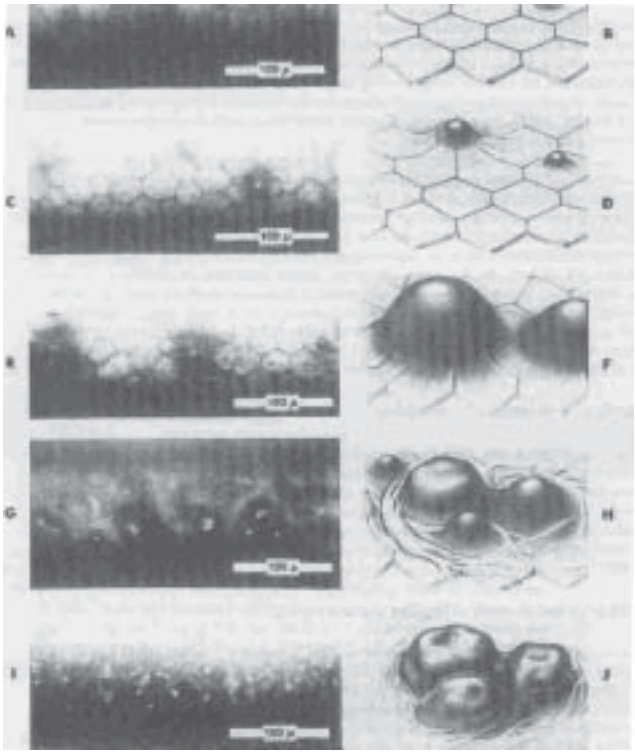


Fig. 2. Specular photomicrographs and drawings of various stages of cornea guttata in Fuchs' dystrophy (x 100) A and B, Stage 1: Early form of cornea guttata. Excrescences are smaller than individual endothelial cells. Dark areas are the sides of the excescence. Bright spots are each a reflex from the apex of the excrescence, C and D, Stage 2: Cornea guttata approximate the average endothelial cell in size, E and F, Stage 3: Cornea guttata are considerably larger than the average endothelial cell. Adjacent endothelial cells are abnormal in appearance. G and H, Stage 4: Individual cornea guttata have coalesced and contain more than one apical bright spot. Boundaries of adjacent endothelial cells are absent or difficult to identify 1 and 3, Stage 5: Coalesced excrescences and nearly complete disorganization of adjacent endothelial mosaic, Numerous bright structures presumably are collagenous material deposited at the level of Descemet's membrane or on the posterior corneal surface

and objective lens is very important and optical distortions in these materials can adversely affect the image quality.

Interpretation of image

Specular microscopic image of a young normal endothelium is shown in the picture. The cells are similar in size and shape with no abnormal dark or bright structures, cell density varies between 2000 to 3500 cells per sq mm. Normal endothelial cell has got dark cell borders and bright cell surface. Percentage of cells with six apices should approach 100%.Lower percentage indicate a diminishing state of health of the endothelium. Surfaces that are smooth, rough, wavy and with excrescences are demonstrated. Curved surfaces such as cell borders, the sides of rough areas and the sides of an excrescence appear dark to the observer.

The normal endothelial cell having a smooth surface appears as a bright circle surrounded by a dark border. If instead of a smooth surface, the cell has a rough surface the inside of the cell have a granular appearance with the spacing between the granular dark regions being indicative of the degree of roughness of the surface. Isolated corneal guttae appear as dark structures with central bright spot. Corneas that are in the process of healing or recovering from a stress are characterized by coalescing cells (cell fusion) showing disappearance of the common cell border between two cells.

Some of the characteristics of an **abnormal endothelium** which are functionally deficient and compromised are:

a. An endothelial cell density less than 1500 cells/sq mm.



Fig. 3. Eye bank specular microscope of modern design. Various magnifications and modes of operation are possible when objective or oculars are changed. As well as high-resolution direct viewing, this instrument allows viewing on a television monitor, provides hard copy documentation on 35 mm Polaroid, or other film, and allows video recording of the image and direct input of the image into a computer for detailed image analysis

b. Severe polymegathism or pleomorphism of endothelial cell pattern
c. Presence of corneal guttatta
d. Presence of abnormal shaped cells such as those seen in coalescence
e. Abnormal single cell defects
f. Extensive areas of severe oedema
g. Presence of inflammatory cells or bacteria
h. Presence of ghost vessels in stroma

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Fine Needle Aspiration Cytology (FNAC) In Ophthalmology

Dr. Sankar

FNAC/FNAB (fine needle aspiration cytology/biopsy) has evolved to be a rapid, reliable and cheap outpatient procedure for the diagnosis of palpable and superficial lesions in the human body. This technique involves the use of a fine bore needle to obtain diagnostic material from the lesion. It has superseded core needle biopsy in many situations as a valuable diagnostic tool without any major complications. This procedure can have accuracy in experienced hands parallel to histopathology in arriving at an equivocal diagnosis. However FNAC is not a substitute to surgical pathology but only an extremely valuable compliment to it.

The advantages of FNAC include the following.

1. it is a relatively painless procedure and minimally invasive only.
2. it is repeatable
3. it is useful in multiple lesions or lesions at multiple sites
4. avoids costly stay in the hospital
5. Saves money for the patients, doctors and the tax-payer.
6. Even negative result can provide information for further line of investigation and/or management
7. Could be a basis for reference to a higher centre.
8. Provides an early result,within a few hours.
9. Obviates the need for unnecessary surgery in patients who need radiotherapy, such as in patients with Non Hodgkins Lymphoma.
10. Avoidance of surgery prevents tumour spillage.

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The two fundamental requirements for FNAC reporting are good representativeness of the sample and high quality of preparation of the slide. Both these can be improved if

1. FNAC is performed with the help of CT or ultrasound images
2. A pathologist does the FNAC or is consulted about the procedure. FNA material can also be subjected to investigations like immunohistochemistry,flow cytometry, morphometry and microbiological methods like culture and sensitivity. Thus an accurate diagnosis is possible within hours with FNAC and hence the demand for this procedure is increasing.

Technique of FNAC

Any localized mass is an indication for FNAC. The feasibility and informative value of FNAC in a case should be discussed with the pathologist beforehand. Highly malignant tumors like malignant melanoma or germ cell tumors are better diagnosed with biopsy as there are a few reports which warn of needle track dissemination. However with the use of small bore needles and by avoiding multiple passes, and, if normal tissue covering the neoplasm is present, we can avoid dissemination.

The patient is informed and explained about the procedure. Informed written consent is obtained and cooperation is ensured. It is better to do the procedure in the theatre to ensure sterility. For FNA of orbital masses the patient may be put in sitting or lying

position. Local anesthesia is mandatory as orbital tissue and eyelids are highly sensitive.

Equipments needed

1. 22-25 gauge 1-1½ inch needles are used. Thicker needles cause bleeding, may become plugged and induce pain. Thus "fine "needles are preferred.
2. 2.5 Or 10 ml syringes of rigid material with good negative pressure are used.
3. Microslides-should be clean, dry and labeled.
4. Coplin Jar (with lid) with the fixative-ethanol or isopropyl alcohol and ether –this acts as a slide carrier as well.
5. Skin disinfectant
6. Sterile pad-for pressure bandage after FNAC
7. Test tubes for collecting fluids.
8. Culture plates-optional
9. Local anesthetic
10. An assistant-While doing FNAC both hands are engaged in holding the barrel and piston of the syringe. Hence an assistant is needed to help in fixing the mass during aspiration.

Steps of FNAC

1. Sterilization of the skin with disinfectant.
2. Local anesthesia
3. The needle is inserted supporting the barrel of the syringe. A vertical approach is less painful. With experience fingertip sensitivity while entering the lesion can be felt.
4. The needle is moved back and forth 3-4 times slowly with slight negative aspiration(Pulling the piston)
5. Piston is released and the syringe and needle are withdrawn.

   Note: Whenever material is noted in the hub of the needle, the negative pressure is stopped because material aspirated into the syringe cannot be transferred to the slide without drying.
6. The needle and syringe are detached and the material in the needle is expressed onto the slide immediately
7. The material is spread with the tip of the needle and the slides are dropped into the fixative in the Coplin jar. Steps 6 & 7 should be completed in 15 seconds as drying is instantaneous.
8. The used syringe, needle etc. is disposed of.
9. Pressure bandage is applied if there is bleeding.

Ideal aspirate

An ideal aspirate has a high cell content and small amount of fluid. It is identified by its creamy nature. The cell yield is high when the material remains in the needle without entering the syringe.

Staining air dried smears (not put in fixative) are stained with MGG (May-Gruenweld-Giemsa) stain. Wet fixed smears are stained with Pap stain. While nuclear details are seen better with Pap stain, the cytoplasmic features are better appreciated in the MGG stain. The staining procedure takes 1 ½ hours. Thus the FNA and its interpretation is possible within 2 hours and hence is considered as an outpatient procedure.

FNAC in Ophthalmology

Although the concept of FNA was proposed in 1927, and became popular in 1950,it is only since 1975 that FNA has been in use in ophthalmology, especially for evaluating orbital lesions. Orbital masses can be congenital, inflammatory or neoplastic.FNA can be done on orbital,periorbital and eyelid masses and the FNAC diagnosis can alter diagnostic evaluation and therapeutic regimen significantly. It may obviate the need for open biopsy. However retroorbital and intraconal masses are difficult to approach for doing FNAC. In this situation FNAC is better avoided because of likely injury to the eyeball.FNA can also be performed on eyelid masses,conjunctival masses,cysts etc.In selective cases intraocular FNA is also done. Non-neoplastic lesions of orbit which can be diagnosed by FNAC include

1. Suppurative and granulomatous lesions.
2. Mucocele and retention cysts.
3. Epidermal cysts
4. Inflammatory pseudotumors.

Neoplastic lesions of orbit which can be diagnosed by FNAC include

1. Lymphoproliferative disorders (lymphoma and plasmacytoma)

2. Mesenchymal (Haemangioma, schwannoma, fibrous histiocytoma, rhabdomyosarcoma etc.)
3. Lacrimal gland tumours(eg: pleomorphic adenoma)
4. Secondaries (from sinonasal carcinoma, neuroblastoma, leukemia, carcinoma breast etc.)
5. Adnexal tumours of skin (eccrine tumours, sebaceous carcinoma etc.).

In orbital FNAC, the pathologist should have all the clinical data to be used as a safeguard and not as a bias. The FNA report should be clear and indicate whether it is a definite diagnosis or an indeterminate one requiring further evaluation. Since the varied conditions seen in the eye and orbit are seen more commonly in other parts of the body, a well experienced general pathologist/cytologist would be better equipped to report accurately on orbital lesions.

Table Lesions diagnosed by FNAC on ocular and orbital lesions in RIO (sep 2003-may 2006)

No	Lesion
1.	Non Hodgkin's Lymphoma
2.	Acute/Chronic suppurative lesion
3.	Inflammatory Pseudotumour
4.	Retention cysts
5.	Haemangioma
6.	Sebaceous carcinoma
7.	Pleomorphic adenoma
8.	Mucocele
9.	Schwannoma
10.	Nodular faciitis
11.	Sinonasal carcinoma infiltration
12.	Cutaneous adnexal neoplasm(eccrine)
13.	Malignant round cell neoplasm
14.	Epidermal cyst
15.	Parasitic granuloma
16.	Meibomian carcinoma lid
17.	Intra ocular-a/c leukemia infiltration and malignant melanoma and suppurative/fungal endophthalmitis

Limitations of FNA in ophthalmology

1. difficulty in reaching the lesions due to the complexity in the anatomy of eye and orbit.
2. paucity of material
3. rare complications like,retroorbital haematoma, damage to globe,optic nerve and ptosis.

FNAC - The RIO experience.

During the past 3 years 77 patients admitted in the RIO, Trivandrum were subjected to FNAC.Each case was discussed in detail before arriving at a diagnosis. There was no complication in any patient during or after the FNA.The most common lesion encountered was Non-Hodgkin's Lymphoma of orbit. The incidence of lymphomas was found to be higher compared to other FNA series in the world. All the NHL were of low or intermediate grade. No high grade NHL was encountered. The diagnostic accuracy of FNAC in lymphomas is 100% keeping histopathology as the gold standard. In 4 patients the mass could not be accessed for FNA.Only differential diagnosis was possible in 6 cases.

Conclusion

Fine needle aspiration cytology has become an important diagnostic tool in evaluating lesions in ophthalmology. If the help of a pathologist is taken, the accuracy of this procedure in diagnosis can parallel that of histopathology. FNAC can save money and time for patients and doctors in the evaluation and management of orbital and ocular masses. The risk and complications are minimal. Preoperative evaluation and diagnosis with FNAC helps in planning oculoplastic and other surgeries.

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Endonasal Endoscopic Dacryocystorhinostomy

Dr. Biji Varghese MS ENT, **Dr. John Panicker** MS ENT

Introduction

Conventional dacryocystorhinostomy (DCR) requires an external skin incision, opening the periostium, removal of the bone at the lacrimal fossa and making a fistula through the medial wall of the lacrimal sac to the nasal cavity. Development of rigid nasal endoscope and endoscopic sinus surgery made it possible to do this surgery from the nasal side, there by avoiding the external scar and dissection through orbicularis oculi muscle and medial canthal ligaments,retaining the lacrimal pump function. Also it has the advantage of avoiding excessive bleeding, which sometimes occurs due to injury to angular vessels.The success rate of conventional DCR varies from 80 to 99%, whereas in case of endoscopic DCR, it is 70 to 95%; probably a little less than the conventional DCR.Introduction of laser in nasal surgery has further improved the results of endoscopic DCR.The choice of external or endoscopic DCR is better left to the patient, after explaining the relative advantages and disadvantages. The endoscopic DCR technique we use in our hospital is described below.

Anaesthesia

Most of the cases can be easily done under local anaesthesia.In children and some apprehensive patients, general anesthesia may be required. For cases under local anesthesia the nose is first anasthetised by 4%xylocaine mixed with decongestant nasal drops. For this we use a nasal pack to be kept for a minimum period of 10 minutes. After removing the pack,the mucosa in front of uncinate process is injected with 1%xylocaine with adrenaline. Two or three drops of 4% xylocaine is put in the conjunctival sac, as most of the time we may need to probe the lacrimal canaliculus.

Surgical Technique

The operation is done using 4mm zero degree rigid telescopes. Using an eleven size knife, a rectangular area of mucosa is incised just in front of the upper half of uncinate, below the attachment of the middle turbinate. Any bleeding point at the edges of mucosal incision is cauterised by bipolar cautery.After removing the mucosal flap, the exposed bone is thinned out by a drill.The bone medial to the lower part of the sac and naso lacrimal duct is relatively thin and we may expose the sac and duct at this point.In case the bone is thick,we can use a gouge to remove the thick bone. Once part of the sac wall is exposed,a Kerrison punch is used to nibble away the relatively thick bone above and in front. By applying pressure externally, we can appreciate the lacrimal sac wall. In case of doubt a probe can be introduced through the lower canaliculus and press on the medial wall. Once confirmed,using a small sickle knife,the medial wall of the sac is opened. Most of the time we may be able to appreciate pus or lacrimal fluid gushing out from the opening. After sufficient medial wall is removed, a gel foam socked with antibiotics solution is introduced into the sac through the new fistula and kept in place using another gel foam. This helps to epithelialize the new opening without granulation formation.

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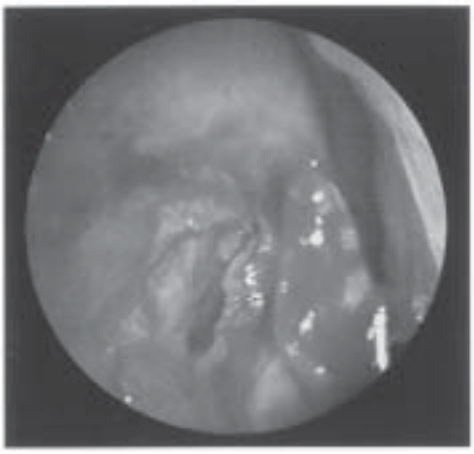


Fig. 1. Exposure of the right upper duct the and lower lacrimal sac

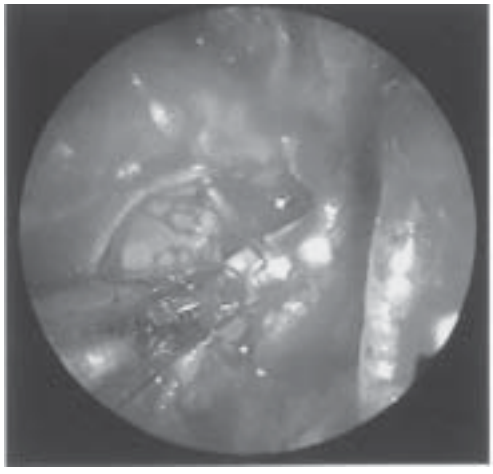


Fig. 2. A lacrimal window is created at the and lower lacrimal sac right lateral nasal wall, showing the lacrimal probe through the common canaliculus.

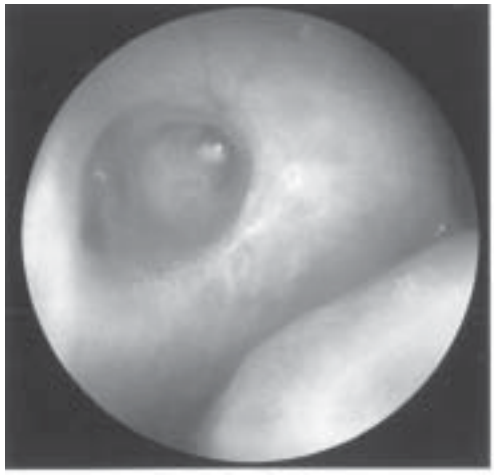


Fig. 3. The 12-month postoperative appearance of the lacrimal window on the right side,showing entry of fluorescein dye into the sac through the common canaliculus

Post operative care

Antibiotics and Steroid eye drops are used for 10 to 14 days and short course of systemic antibiotics given along with antihistamines to avoid sneezing. The patient is discharged the next day.

Conclusion

Endocopic DCR, resultwise is almost as equal as external DCR.Relative advantages like absence of scar and less postoperative morbidity makes it increasingly popular.

Corneal Endothelial Replacement – Current State-of-the-Art

Dr. Srinivas K Rao, DO, DNB, FRCSEd

Until very recently, surgical management of the dysfunctional corneal endothelium required a penetrating corneal transplant. This procedure has stood the test of time for the past century, since Zirm first performed it in 1905. However, despite numerous improvements, there are many issues that still need to be addressed. Of these, the important ones are the loss of corneal and ocular structural integrity due to the keratoplasty wound, the alterations in corneal shape due to the wound and its apposition by sutures, immunological considerations caused by exposure of donor antigens to the ocular surface immune defense, and the possibility of long-term complications due to the presence of sutures – loosening, vascularization, infection and rejection. Any procedure that would obviate these concerns would definitely improve outcomes of corneal endothelial transplant surgery and in recent times, such a procedure has gradually evolved. This article traces the evolution and present status of these exciting developments in corneal surgery in the past decade.

A procedure that addresses these issues would avoid the use of sutures to keep the donor graft in position – thus, reducing corneal shape distortion which can result in compromised visual function. The lack of sutures and a full thickness corneal penetrating wound would also help avoid the long-term complications of corneal sutures and enhance corneal and ocular structural integrity. Since anterior chamber associated immune deviation (ACAID) helps reduce the chance of host immune system sensitization, the ability to transplant lesser amounts of donor tissue with presumed reduced antigenicity is attractive, especially if this tissue is also introduced into the anterior chamber, with no exposure to the elements of the ocular surface defense. And finally, if all of these can be achieved through a small wound in the ocular coats, the results are likely to be better.

A review of corneal embryology and anatomy will facilitate a better understanding of the mechanics of these newer techniques. During development, once the surface ectoderm separates from the optic vesicle, it differentiates to form a two-layered epithelium – the primitive cornea. In week 7, mesenchymal cells from the neural crest migrate forward from around the lens vesicle and this occurs as three waves. The first wave migrates between the surface ectoderm and the lens to form the corneal and trabecular endothelium. The second wave migrates between the corneal epithelium and the endothelium to form the stroma. The third wave migrates between the corneal endothelium and the lens to form the iris stroma.[1] By week 8, the corneal endothelial cells produce a basement membrane – the Descemet's membrane. In adults, this membrane has an anterior banded layer and a posterior non-banded layer. This embryological development of the endothelium and the corneal stroma as separate entities results in a potential cleavage plane between the posterior stroma and the Descemet's membrane, which is the basis for the Descemet's stripping procedure, resulting in smooth interface in the recipient eye.

Anatomically, in the adult cornea, keratocyte cell density is highest in the anterior stroma of the cornea

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immediately posterior to Bowman's membrane (24320 cells/mm^3 +/- 6740 [SD]), lowest in the central area (11,610 +/- 4290 cells/mm^3), and has an intermediate density in the posterior stroma immediately adjacent to Descemet's membrane (18,850 +/- 4610 cells/mm^3).[2] The vast majority of the corneal stroma consists of 200 to 500 layers of flattened collagenous lamellae extending from limbus to limbus, with some crossing the apex (center) of the corneal dome. In the anterior one third of the stroma, collagen lamellae are thin (about 0.2 to 1.2 microns thick and 0.5 to 30 microns wide), run obliquely to the corneal surface, and sometimes split into two to three sub layers that become interwoven. In the posterior stroma, collagen lamellae tend to be arranged parallel to the surface and are thicker (1.0 to 2.5 microns thick and 100 to 250 microns wide). (http://www.grendahl.com/eyeworks/eyeanatomy.html). The more regular collagen lamellar arrangement in the posterior cornea and the reduced keratocyte density in the central and posterior stromal layers, aid posterior corneal lamellar replacement by reducing the healing response and providing better visual outcomes.

Such a procedure was first described by Gerrit Melles from Rotterdam. In its initial development, a large 9 to 10 mm superior limbal incision was used.[3] Dissection of the limbal tissue and peripheral cornea is initiated with a crescent knife at a depth of about 300 to 350 microns **(figure A3)**. Subsequent dissection of the posterior corneal stromal lamellae is performed with a set of specially designed semi-sharp dissectors that are variably curved to accommodate the corneal shape **(figure A4)**. The goal of the dissection is to split the corneal stroma into two layers – an anterior 80% and a posterior 20%, extending all the way to the limbus. A specially designed trephine with a very low height profile is then introduced into the stromal dissection plane. This is connected to a larger knurled handle which rests above the cornea. The trephine is centered on the pupil and by rotating the knurled external handle clockwise and counter-clockwise, the trephine is used to cut through the posterior lamellar tissue. The anterior chamber (AC) is pressurized with a cohesive viscoelastic like hyaluronate to provide the counter force for the trephination. Trephination is continued until the anterior chamber is entered. The cut is completed with specially designed scissors and the disc of posterior corneal stroma is removed.

A whole donor globe is utilized and a similar dissection of the corneal stroma is performed. After this is completed, the entire cornea with a scleral rim of at least 3 mm is excised. If the eye bank only provides a donor corneo-scleral rim, an artificial anterior chamber is used to create a pressurized corneal dome **(figure A8,9)** – after which the dissection is completed **(figure A10)**. The dissected corneo-scleral rim is placed endothelial surface upwards in a donor corneal punch **(figure A11)**, and a matched trephine is used to excise a central corneal button of the same diameter as the recipient corneal bed.

A coaxial irrigation aspiration instrument (preferably automated) is used to remove the viscoelastic from the anterior chamber of the recipient eye **(figure A12)**. It is important that the viscoelastic be completely removed from the AC and the recipient stromal bed, since retained viscoelastic can interfere with the adherence of the donor stromal tissue. It is therefore important to use a cohesive viscoelastic such as hyaluronate and not a dispersive variety as the latter are hard to remove completely. After the viscoelastic is completely removed, the anterior chamber is filled by injecting balanced salt solution through a paracentesis.

The dissected posterior corneal stromal disc is carefully separated and placed endothelial side down, on a specially designed spatula which is coated with hyaluronate to protect the endothelium. The spatula is used to carry the dissected donor disc into the anterior chamber of the recipient eye and is elevated to seat the donor disc into the recipient bed. The spatula is gently withdrawn and an air bubble is injected into the AC to support and keep the donor disc in place. The superior limbal wound is closed with 10-0 nylon sutures. The position of the disc is inspected and adjusted, if required. It is especially important to ensure that the edges of the recipient stromal bed are not interposed between the donor graft and the recipient stromal bed. The air bubble is then partially replaced with BSS to avoid a pupillary block and the procedure is completed.

Although Melles reported good outcomes with this approach, which he termed posterior endothelial lamellar keratoplasty (PELK), the large limbal wound was a source of astigmatism, as well as a zone of potential weakness in the ocular coats. In order to overcome these disadvantages, he described the small incision variant of this technique.[4]

In this approach, the corneal epithelial surface is marked with a suitably sized trephine – usually 8 to 8.5 mm, centered on the pupil, and the outline is inked with a series of closely spaced dots, using a marking pen **(figure A1)**. This outline serves to provide a visual cue to the location and size of the disc of posterior lamellar tissue that must be removed to create a recipient corneal stromal bed. Hyaluronate is instilled in the anterior chamber through a paracentesis, and the corneal stromal dissection is performed as described previously, using a 5 mm temporal limbal incision **(figure A2)**. The anterior chamber is entered by placing a sharp keratome in the superior tunnel and engaging the posterior stromal layer in line with the surface markings at 12 o'clock **(figure A5)**. This will result in a 3 mm cut in the posterior corneal layers superiorly. Specially designed curved scissors are then used to complete this cut in a circular fashion, following the external marking **(figure A6,7)**. This results in the separation of a disc of posterior stromal tissue, which conforms in size and shape to the trephine mark on the corneal surface. This is removed from the anterior chamber and inspected for size, shape, thickness, and regularity of dissection. The outlines of the circular bed thus created in the posterior corneal stroma are also inspected and if any tags are noted, protruding into the central bed, they are trimmed and removed. The anterior chamber viscoelastic is removed completely and the AC reformed with BSS. The donor cornea is dissected as described previously. The posterior corneal stromal disc is then folded like a flexible intraocular lens in a 60:40 fold, over a thin tube of hyaluronate (to protect the endothelium). It is grasped with a specially designed forceps which does not crush the tissue **(figure A13)** and is quickly introduced into the AC **(figure A14)**. In the AC, the folded 60% surface is placed in apposition with the recipient stromal bed, and the forceps is carefully withdrawn **(figure A15)**. Although the fluid filled AC tends to shallow at this stage, the fold in the graft protects the endothelium. BSS is then injected onto the iris surface through the paracentesis. The fluid helps reform the anterior chamber and the flow from the iris towards the graft helps unfold the 40% fold of the graft which then fits itself into the remaining portion of the recipient stromal bed. Air is injected and the position of the graft adjusted, as described previously. The 5 mm limbal wound is closed using 3 interrupted 10-0 nylon sutures **(figure A16)**.

This procedure was further modified and popularized by Mark Terry, who designed his own set of instruments and termed the procedure – deep lamellar endothelial keratoplasty (DLEK).[5]

The mechanism by which the donor disc maintains its position is not clear. It is likely that there is a physical adhesion between the dissected stromal surfaces of the recipient and the donor – a sort of 'velcro' effect. Subsequently, the functioning of the endothelial pump may create a directional gradient of fluid movement in the stroma and the resultant negative pressure may serve to seal the interface and keep the graft in position, as in the laser in situ keratomileusis flap. In time, sealing of the edges of the graft-host junction by endothelial cell migration, and stromal healing in the interface will serve to complete the process.

This procedure resulted in further improvements due to the reduced size of the limbal wound. However, the need to use scissors in the AC to excise the posterior stromal disc in the recipient is technically challenging and the natural evolution of the procedure attempted to address this issue and avoid the need for extensive AC manipulations. This was again pioneered by Melles and he described the procedure of simply removing the endothelium in the recipient eye to create the bed.[6] After the chamber is stabilized with hyaluronate, a reverse Sinskey hook (with the tip facing upwards) is introduced into the AC **(figure B1)** and the blunt tip is used to gently score the endothelium in line with the surface trephine marking. This results in a circular tear in the endothelium **(figure B2)**, and using specially designed endothelial scrapers or even the reverse Sinskey hook itself, this central disc of Descemet's membrane and endothelium can be stripped from the posterior corneal stroma **(figure B3)**. It is placed on the surface of the cornea and inspected to ensure that the entire central disc has been removed **(figure B4).** The folded donor disc is introduced into the AC as described for DLEK, after removing the viscoelastic. However, in the absence of exposed collagen lamellae in the host corneal surface, the adhesion of the donor disc tends to be weaker and in order to reduce the chance of donor dislocation the following day, a complete air fill of the AC should be maintained for 10 minutes. After this period, the air is partially removed

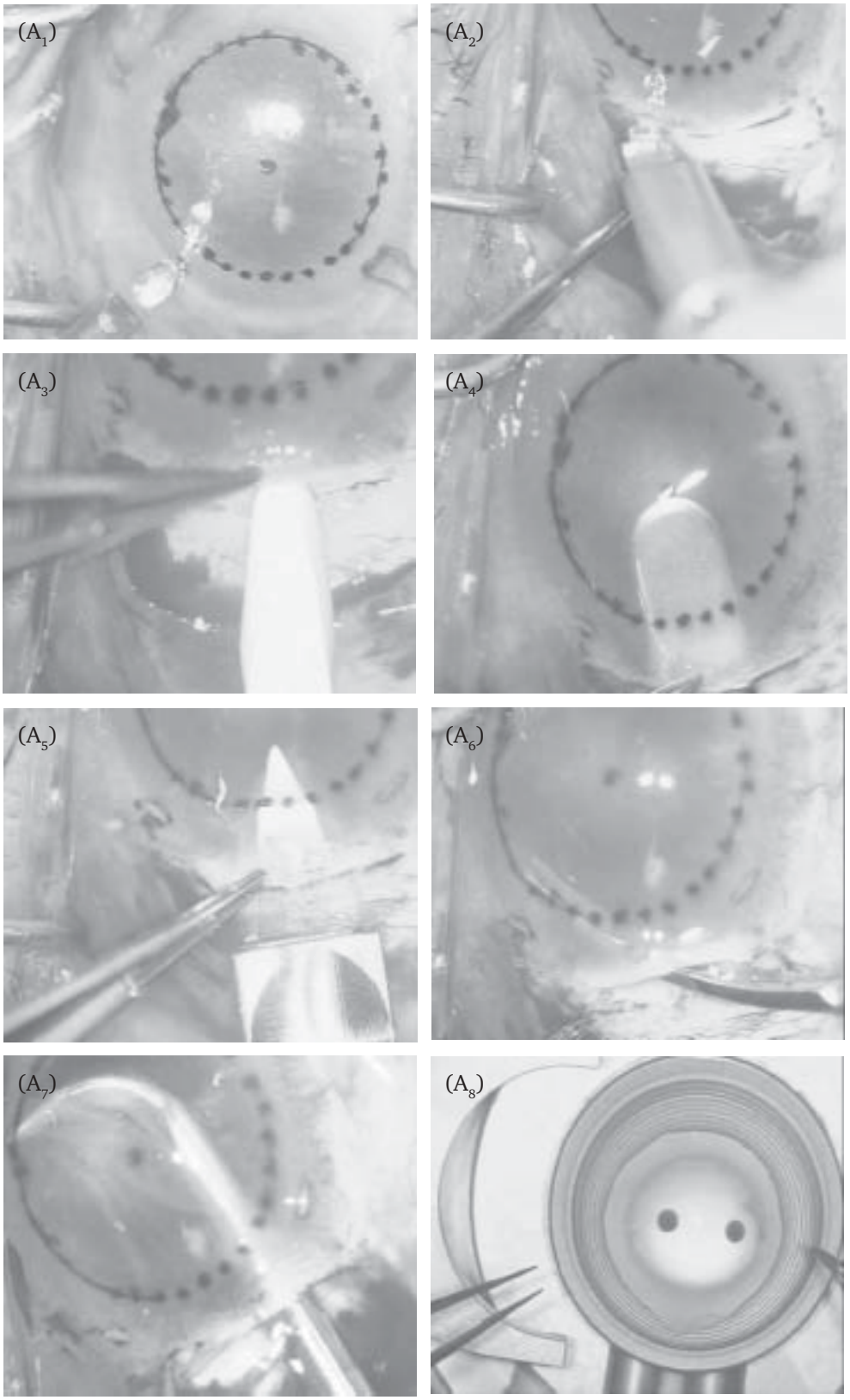


Fig. A. Steps of Deep Lamellar Endothelial Keratoplasty (DLEK) A1–Marking of corneal surface, A2–5 mm, 350 micron temporal limbal/scleral incision A3–Dissection initiated with crescent into corneal periphery A4–Corneal stromal dissectors used to complete dissection A5–Anterior chamber entry with keratome A6–Mildly curved scissors for proximal wound A7–Sharply curved scissors for distal wound A8–Donor corneo-scleral rim placed on artificial anterior chamber

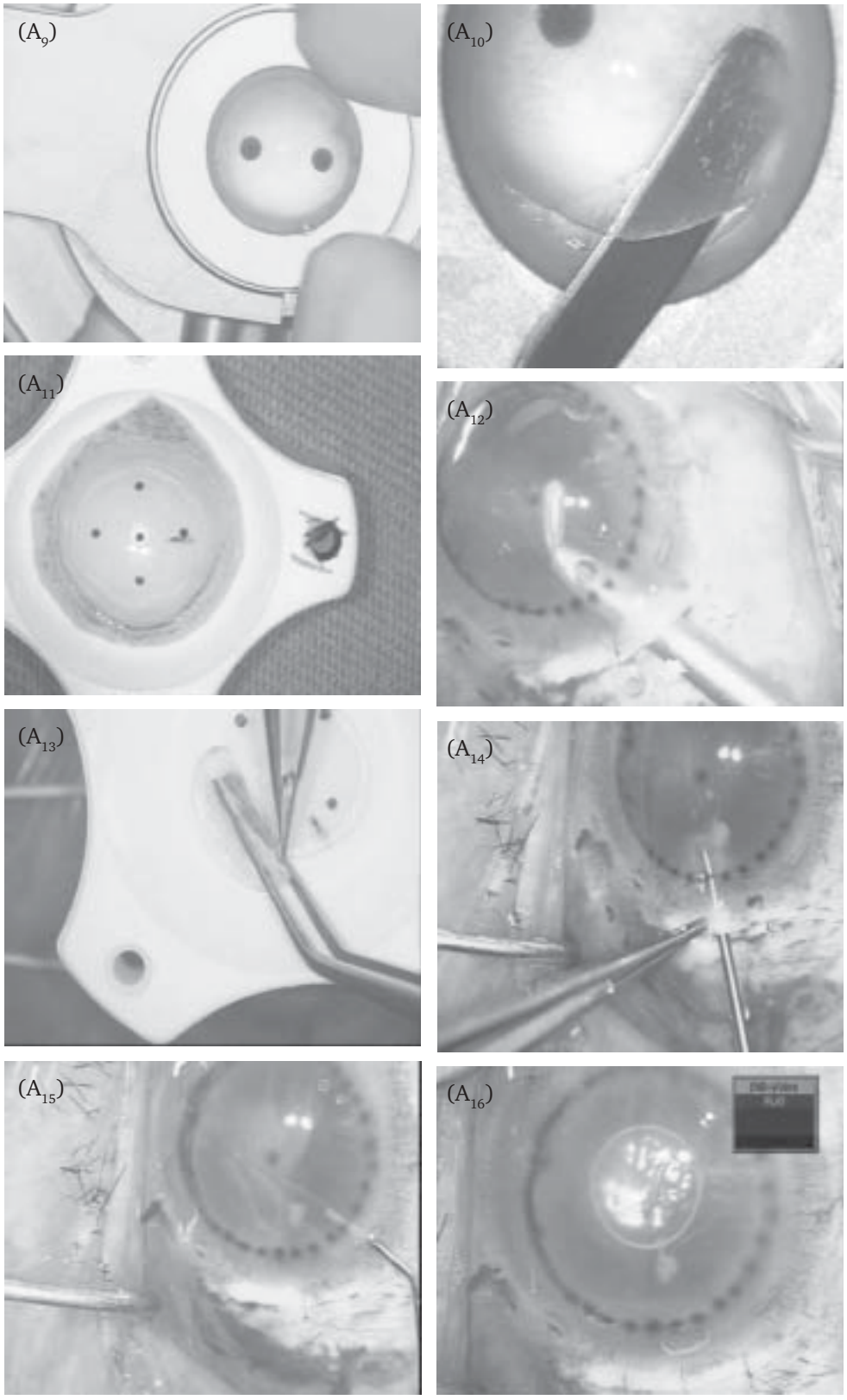
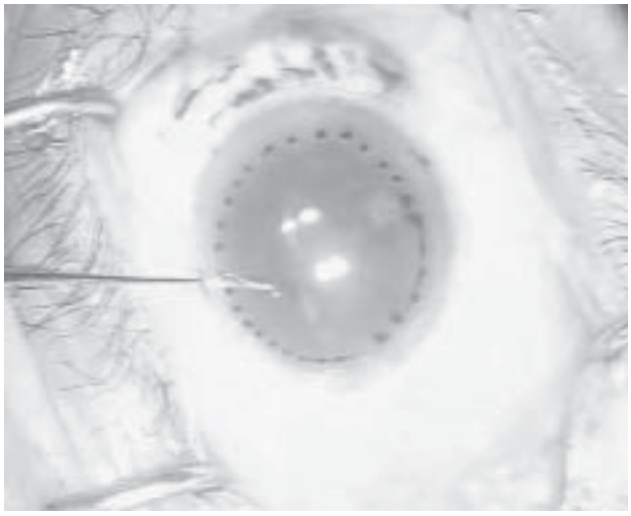
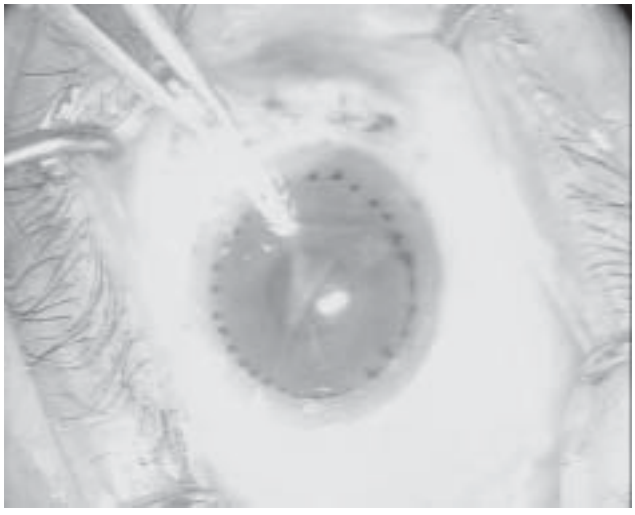


Fig. A. Steps of Deep Lamellar Endothelial Keratoplasty (DLEK) A9–Rim locked into position and pressurized A10–Dissection of donor cornea A11–Dissected cornea placed in teflon block for punching disc A12–Viscoelastic removed from anterior chamber A13–Folded graft held with forceps A14–Folded graft introduced into anterior chamber A15–60% fold placed in apposition with recipient bed A16–Unfolded graft supported by air bubble and wound closed





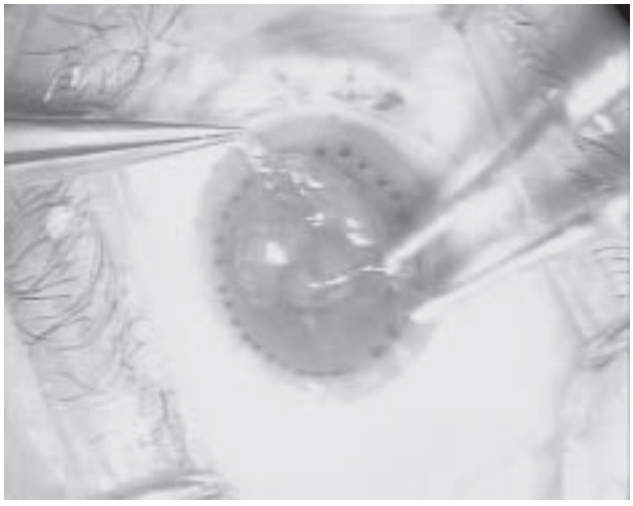
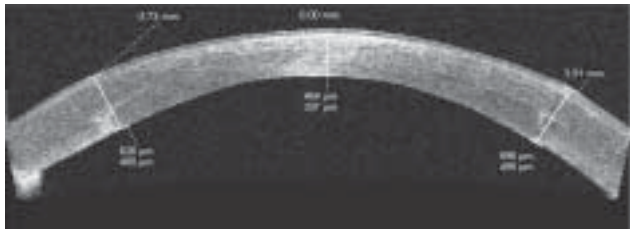


Fig. B. Descemet's Stripping Endothelial Keratoplasty (DSEK) B1–Reverse Sinskey in anterior chamber to cut Descemet's membrane B2 – Cut Descemet's removed from anterior chamber B3 – Descemet's membrane inspected to ensure complete removal



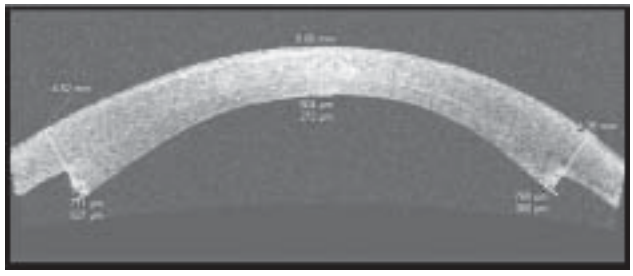


Fig. C. Anterior segment OCT imaging after DLEK and DSEK C1–DLEK–Interface and measurements of donor and recipient thickness C2–DSEK–Increased central corneal thickness with well apposed donor graft protruding into anterior chamber.

to avoid pupillary block, the wound is closed with sutures and the patient is asked to lie supine without pillows for the next few hours. This procedure has been termed Descemet's stripping endothelial keratoplasty (DSEK). The first large series with this approach was reported by Francis Price.[7]

Several methods have been described to increase the adhesion of the donor disc — scoring the peripheral part of the recipient stromal bed with a sharp needle tip to expose corneal stromal lamellae to promote adhesion of the donor disc. Stab incisions in the recipient cornea – one in each quadrant, reaching the donor-recipient interface, have been suggested, to help remove fluid trapped in the interface. Another method suggested to promote adherence of the donor is the use of an air fill for 1 hour. In my experience, a 10 minute complete air fill of the AC is sufficient for graft adherence. After the air fills the AC, a smooth spatula is used to stroke the corneal surface from the center to the periphery, to aid removal of fluid trapped in the interface and iron out folds in the donor disc.

In a further modification, Gorovoy described the use of an automated lamellar microkeratome to cut a flap of about 300 to 350 microns in the donor cornea. A disc of the desired diameter is then punched from the posterior corneal tissue. This increases the predictability

of the procedure and makes it safer. Proponents also claim that the greater smoothness of the microkeratome cut, compared to manual dissection, can improve the speed and extent of visual recovery in these eyes. The microkeratome however, is expensive, and is not freely available in all centers.

With these advances, visual rehabilitation for patients with bullous keratopathy has certainly improved. Astigmatism and delayed visual recovery are less common with these approaches. On the other hand, there is a learning curve for this procedure, it has its own complications, there is possibly greater handling of the endothelium (although published series of DLEK indicate postoperative endothelial counts comparable to those after penetrating keratoplasty). It will also require longer follow-up of larger series of patients to determine if these approaches do confer immunological advantages with a lesser incidence of corneal rejection. With these procedures, it is likely that the final visual acuity of the patient may be one Snellen line or so less than with penetrating keratoplasty – possibly due to the presence of the interface in the central cornea. With the DSEK procedures, there is also the issue of the non-physiological increase in central corneal thickness, due to the addition of a donor stromal disc to a recipient cornea that has had only its Descemet's membrane and endothelium removed.

Hence, although we are currently at an exciting phase in the development and refinement of endothelial replacement procedures, it is clear that despite the incremental advances in the last decade, there is enormous scope for further improvement. Chief among these is the possibility of using only the Descemet's membrane and endothelium from the donor to replace similar tissues in the recipient. Although the procedure does have its problems, it is possible, although reproducibility is as yet lacking (Melles, personal communication). And finally, the holy grail of corneal surgeons – the possibility transplanting a cultured sheet of endothelium into a recipient eye, is still in the laboratory and with current limitations in culturing endothelium, may take a while to translate into a clinical technique.

Developments in diagnostic instruments have kept pace with these changes and with the availability of the anterior segment OCT, and it is now possible to image *in vivo*, the changes in corneal structure that is achieved with these techniques. The image of a cornea that has undergone DLEK is shown in **figure C1**, and the corneal interface between the donor and recipient corneal stroma is clearly seen, and measurements of the two layers are possible. Note also the precise fit of the posterior donor disc in the recipient bed and the normal central corneal thickness. In contrast, **figure C2**, is of a cornea after DSEK, and in this image, the increased central corneal thickness with the addition of the donor disc, is clearly seen. Note however, the well apposed donor-host interface and normal reflectivity of the corneal stroma. With these ongoing developments - the present level of refinement of corneal endothelial replacement procedures, and our ability to image and understand these newer procedures, it is an exciting time to be a corneal surgeon.

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Current Concepts in Consumer Litigation

Dr. B.V. Bhat

"An ounce of PREVENTION in worth more than a tonne of CURE" – Panchathanthra

Fighting the litigation in the consumer forum is becoming an exhaustive and tiresome process, and the judgment against doctors and hospitals is alarmingly on the increase.

The amendments in consumer law have made the process of appeal in state forum still more complex. There was no necessity to make any cash deposit for filing appeal during earlier years.

A deposit of Rs. 25000 is becoming mandatory in all cases of appeal at state forum. A further deposit of an amount up to 50% of the sum awarded is also being made as a prerequisite to get a stay at the state forum against the judgment of district forums. However, this is at the sole discretion of the chairman, which leaves us totally at the mercy of the whims and fancies of the chairman of the state forum. However, the awareness about the possible litigation by the patient, seems to be still inadequate among doctors as well as hospitals.

An effort is being made through these columns to update your knowledge on consumer litigation.

Ever since the consumer law was made applicable to healthcare services, about 2250 cases have come up against doctors and hospitals in Kerala. Though consumer courts were established to give speedy relief to consumers, majority of the cases are at various stages of hearing and the final verdict from the state forum has come in only less than 60 cases. The only consolation is that as much as 2/3 of these verdicts were in our favour.

The parody of the drama lies in the fact that, cases as old as the year 1992-93 are still pending final hearing at the state forum; and in some cases the litigants or the defendant doctors have already left this world.

Bhat's eye clinic, Asoka Hospital, Kannur

Ophthalmology ranks number 5, in the number of cases with its share at 3.75 % of the total cases while OBG 33 %, Ortho 18 %, Medicine 15.75 % and General surgery 6.75 % and other surgical super specialist above 10 % as per the recent statistics.

Thanks to the Hi-tech advances in our field - only five new cases were reported during the year 2005-06 pertaining to ophthalmology in Kerala.

The cases that came up were due to sepsis, corneal decompensation, retained foreign bodies, retained sutures, delay in referring cases to higher center, decentered IOL, wrong prescription of glasses, under/over correction by lasik etc.

This shows that with a proper counseling of the patients and relatives prior to and after treatment many of the litigations can be prevented.

Documentation of Records

Improper documentation was the biggest hurdle in defending many cases. Hence a few lines on documentation and record keeping. Majority of the consultants do "restaurant type of practice.' – Patients enter, service is provided, payment collected and there ends the matter ! However there has been a good change in many institutions due to computerization of OP records.

Patients keeping on changing doctors have posed a big problem in maintaining continuity of records. Hence it will be extremely helpful to note down the telephone number and address of all the patients in your records or diary for further follow up in deserving cases.

Medical curriculum should give due importance to inculcate habit of record keeping while moulding up young medical graduates. At present doctors realise its importance only when they face a medico legal or consumer cases.

Guidelines for Record keeping

1. Try to get the records of the past treatment if any, while taking the history itself. This will give you lot of tips in deciding the medication or assessing the possible cause of complication.
2. Record the date and time of examination on each visit. This is very important as some of the patients may turn up later on with the claim form for insurance or for a certificate.
3. Maintain an accident register to record all the details of the injuries in all case of trauma and report to the police in case the same happens to be a medico legal case.
4. Keep a copy of all investigation reports that are handed over to patients.
5. Make a record of whatever advice you give to the patients orally so that tomorrow he doesn't blame you about not advising properly.
6. Prescription and dosage should be legibly written or typed. There have been cases of over dosage and even change of the drug dispensed / administered in the hands of chemists or nursing staff.
7. Always keep a copy of the discharge card, this being the most powerful weapon in the hands of the patients. Never take things for granted by entrusting the job of writing the card to junior doctors without a final supervision by you.

Inpatient case sheet record must be maintained in chronological order and must have entry of doctors orders and nurses report of compliance of the same. Progress of the condition of the patients must be regularly indicated in the case sheet.

Do not tamper with entries or alter them. If you must, do it properly by rewriting and initialing the same.

Abbreviations are better to be avoided. Since most of them do not have an universal usage.

Remember – as far as courts are concerned whatever is not documented in your notes, "never happened" regardless of whatever you may have actually done.

Informed consent - can be taken in a printed form in the language familiar to the patient or close relative. It is desirable to get in writing from them that they have read / heard and understood the contents of the consent letter. Special mention must be made in cases of Eviseration / Enucleation, Oculoplastic and Cosmetic surgeries.

Reference letter: Always keep a copy of the reference letter when the patient is referred to another center. In case a patient is getting discharged against your advice, get the same endorsed in the case sheet and discharge card. Obtain a receipt from the patient for having received the discharge card, investigation reports, x-ray film etc. Keep a photocopy with you.

As regards out patient records, computer or hard copy can be maintained in the clinic if possible. If not keep the sheets properly filed in a small file and hand it over to the patient so that he will keep them safely. Medical records should be kept atleast for 7 years.

Consider that every patient is a potential litigant. The initiation of legal action against a doctor can be done by just sending a lawyers notice before the expiry of two calendar years from the date of occurrence of the cause of action. This notice has to be properly replied to by denying the allegations. Every word in the reply has legal implications. If you are protected by any of the schemes, they will fight for you in the court. However you have to keep a constant vigil and give timely advice to the advocate concerned. Remember - you have to teach your advocate about your case.

IMA and MAPS are in the forefront in defending the doctors and hospitals effectively. However it is surprising to note that only less than 10,000 doctors are protected by any insurance or schemes. As for the private hospitals – only about 25% of them have any sort of protection by GIC / IMA / MAPS. This clearly reflects the lack of awareneness of the facts by the hospital management, that, the hospitals are fully liable for damages to the patient irrespective of the fact that the concerned doctor is employed full time or part time or is a freelancer.

Consumer litigation might go on for decades. Hence be prepared for a long fight However do not lose your heart because, while in the sea if you have to survive you should know how to swim against the waves.

Every consumer litigation is a medical accident. Let us try to prevent such accidents and face them with courage and wisdom when they really happen.

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Treatment of Retinal Pigment Epithelial Detachment with Transpupillary Thermotherapy and Intravitreal Triamcinolone Acetonide

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Summary

Retinal pigment epithelial detachment (PED) is a common feature of wet age related macular degeneration. Symptoms develop with the involvement of macula and patients report blurred vision, metamorphopsia, micropsia or positive scotoma. Several reports on the natural course of PED associated with age related macular degeneration has revealed a high predisposition to choroidal neovascular membranes. We report a case of complete flattening of PED following Transpupillary thermotherapy and intravitreal triamcinolone acetonide.

Case Report

A 72-year old man presented with history of defective vision for the past one and a half years. He was a known diabetic, hypertensive and suffered from coronary artery disease for the past 5-10 years. His best- corrected visual acuity in the right eye was 6/24 N6 and in the left eye was 6/18 N6. Amsler grid examination was normal. Extra ocular movements and anterior segment were normal. Fundus in the right macula revealed a large oval notched PED measuring 8 disc areas with few drusen at its inferonasal margin (Fig. 1). Left eye fundus showed multiple soft drusen at the macula. Early phase fluorescein angiography revealed hyperfluorescence of the dye (Fig. 2), which increased in intensity with the course of the angiogram towards the late phase but remained the same in size. The areas corresponding to the drusen in both the eyes revealed hyperfluorescence due to late staining. OCT of the right eye showed a large dome shaped PED measuring 4417 μm in width and 1090 μm in height with relative attenuation of the signal from the deep choroid. Located at the dome of the PED and under the fovea was a localized area of serous sensory macular detachment measuring 149 μm in height (Fig. 3). OCT of the left eye showed multiple irregular elevations in the macular region and also below the fovea. There was no evidence of intra retinal fluid or activity in the left eye. Based on this a diagnosis of serous pigment epithelial detachment in the right eye due to age related macular degeneration was made. He was treated with Transpupillary thermo therapy in the right eye with two applications of 500 mW each with 3 mm spot size for 2 minutes using 810 diode laser followed by 4mg intravitreal triamcinolone injection in the right eye on the same day. A month following treatment vision had improved to 6/18 N6 and the fundus examination showed a reduction in size of the PED (Fig. 4). Repeat angiogram performed two months after treatment revealed early hyperfluorescence in the area around the macula which

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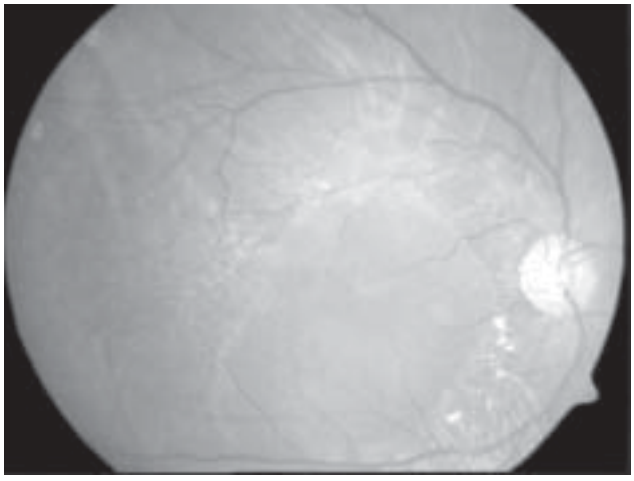


Fig. 1. Reveals a large PED at the right macula

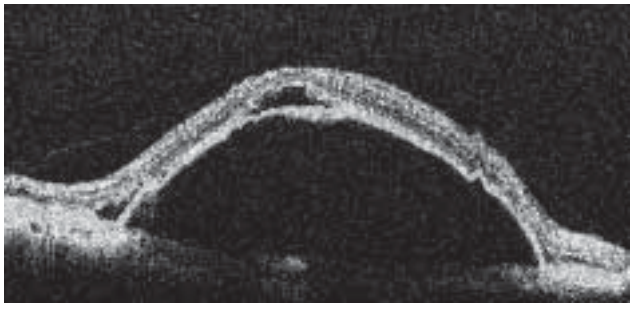


Fig. 2. Shows early phase angiogram with uniform pooling in the PED

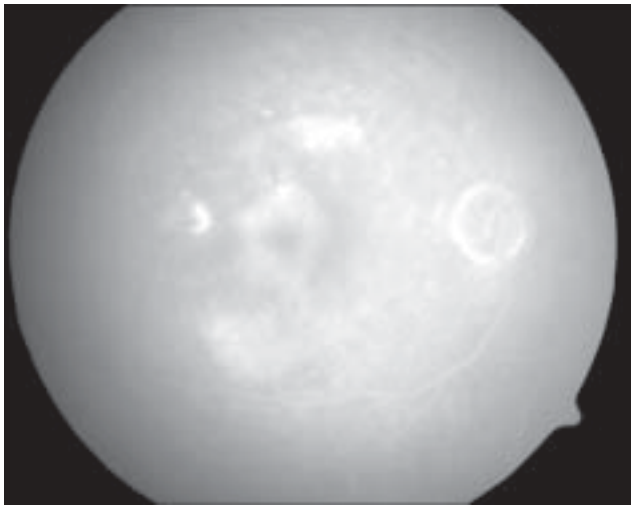


Fig. 3. OCT showing dome shaped elevation of macula with serous macular detachment

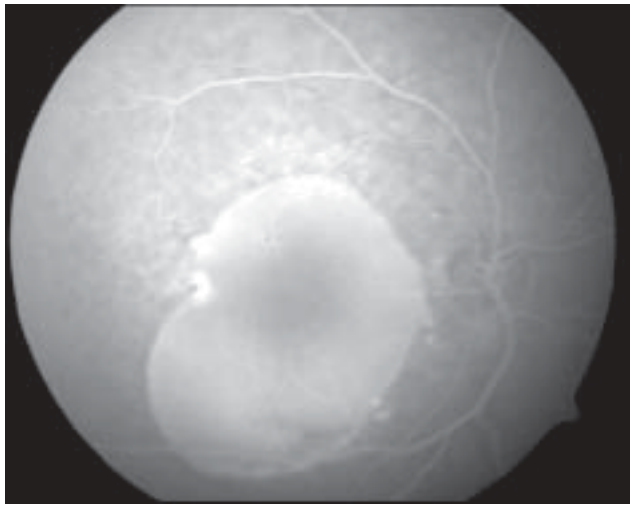


Fig. 4. Fundus photograph shows collapse of the PED

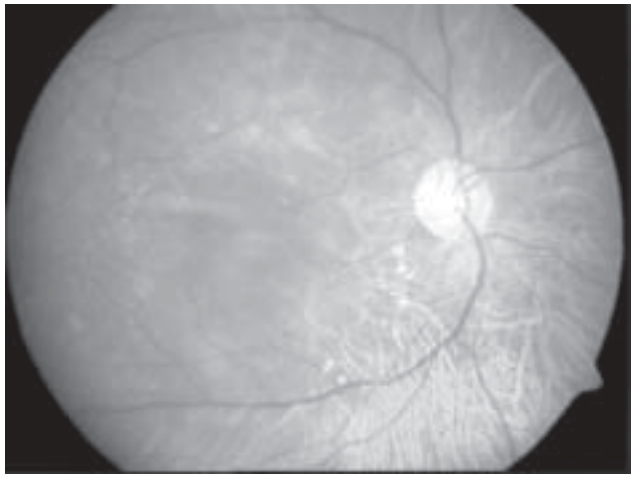


Fig. 5. Shows late phase of the angiogram following treatment of PED

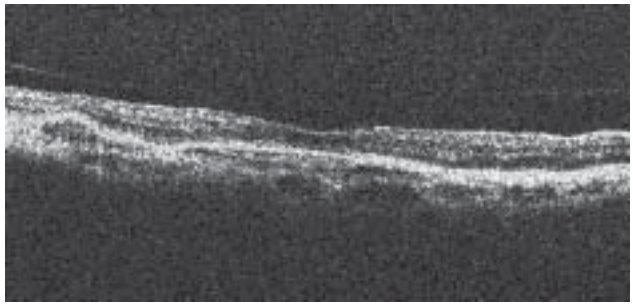


Fig. 6. OCT of the RE six months following TTT with normal foveal contour

did not intensify or expand in the late phase suggesting a scar (Fig. 5). The corresponding OCT showed complete resolution of serous subfoveal macular detachment and residual PED measuring 1496 μm wide and 182 μm in height. At 6-month follow up the right eye vision remained 6/18 N6 and left eye was 6/18 N10. Fundus examination showed a collapsed PED and repeat OCT showed a near normal foveal contour with an area of drusenoid PED temporal to the fovea (Fig.6)

Discussion

Retinal pigment epithelial detachments result from any choroidal disorder that may disrupt the normal junction between basement membrane of the retinal pigment epithelium and natural collagenous layer of the Bruch's membrane. Different types of retinal pigment epithelial detachments have been described viz. serous, turbid, haemorrhagic associated with or without neovascular membrane.[1] Casswell et al studied the natural history of pigment epithelial detachments with the aim to identify prognostic factors. He classified pigment epithelial detachments into early fluorescence, late fluorescence, shallow detachment with limited fluorescence (drusen type) and irregular fluorescence. They reported spontaneous flattening as a feature of the drusen type and early fluorescence group. Spontaneous flattening of the Pigment epithelial detachments was associated with pigment epithelial atrophy and invariable loss of vision. Visual acuity was maintained only in those with persistent detachment. They also concluded that 30% of Pigment epithelial detachments developed choroidal neovascular membranes (CNVM) and 10% developed RPE tears.[2] Patients above the age of 55 years have an increased risk of development of haemorrhagic Pigment epithelial detachments or CNVM. Spontaneous resolution of Pigment epithelial detachements has been reported for avascular serous pigment epithelial detachment with loss of vision.[3]

We report a single case with a large serous avascular Pigment epithelial detachments, which was treated with the intention to reduce the risk of developing choroidal neovascular membrane. At 6 months after treatment, the vision was maintained in this case. However, longer period of follow up and a larger case series is necessary to know the beneficial effect of therapeutic flattening of PED.

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Trilateral Retinoblastoma: A Case Report

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Trilateral retinoblastoma is a rare, but well recognized syndrome. These tumors usually occur in the pineal, parasellar, or suprasellar regions several years after successful management of ocular retinoblastomas without evidence of direct extension or distant metastasis. Here we report a case of trilateral retinoblastoma presenting 2 years after successful treatment of bilateral retinoblastoma. The patient was a 1 year old baby girl showing white reflex in the right eye. She had no family history of retinoblastoma. Detailed Indirect Ophthalmoscopic examination showed a dense tumour extending into the midvitreous. Left eye examination revealed two small lesions, less than a disc diameter, nasal to the disc. A clinical diagnosis of bilateral retinolastoma was made and CT scan was planned. It showed no involvement of the optic nerve and the brain was normal. Right eye was enucleated and retinoblastoma was confirmed by histopathological examination. Laser photocoagulation was done for the left eye lesions. The child was followed up regularly. Two years later she presented with drowsiness and on examination there was papilloedema in the left eye. She was subjected to CT again, which revealed a midline suprasellar tumor without evidence of cerebrospinal fluid seeding or extracranial metastasis. The child was referred to Regional Cancer Institute for further management.

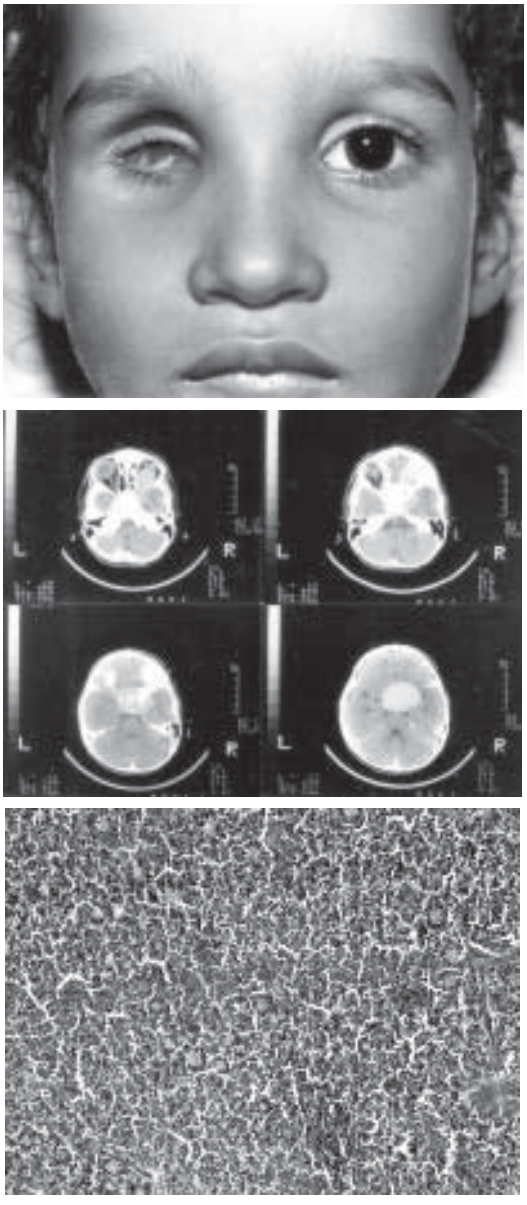


Fig. 1. a) 5year old child with enucleated socket (RE) b) CT Scan showing the midline suprasellar lesion c) Histopathology showing retinoblastoma rossettes

Discussion

Retinoblastoma (RB) is the most common intraocular malignancy found in children. It is caused by the inactivation of both copies of a tumor suppressor gene (*Rb1*), which participates in the control of cell cycling.

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Approximately one-third of these tumors are bilateral and is associated with germinal mutations. All bilateral tumors and one tenth of unilateral tumors are caused by a germline mutation inherited as an autosomal dominant trait. This hereditary predisposition to retinoblastoma is caused by mutant alleles occurring at the q14 band of chromosome 13.

The association of bilateral RB with ectopic midline intracranial tumors, termed trilateral retinoblastoma (TRB), is a well recognized but uncommon syndrome. The association of ocular RB with brain tumors was first reported in 1971. The intracranial tumor arises most often in the pineal region but can also be a suprasellar or parasellar tumor, and is considered to be an isolated independent primary focus without evidence of retinal disease.

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Sturge Weber Syndrome – A Variant

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Introduction

Sturge Weber Syndrome is the most commonly diagnosed phakomatosis. It is a dermato oculoneural syndrome which affects skin, eye and central nervous system, glaucoma being the most common ocular complication. Usually Sturge Weber syndrome occurs unilaterally. Rarely bilateral findings can occur in unilateral cases. Here is a case report of a boy who presented with bilateral choroidal haemangioma and features of unilateral Sturge Weber syndrome.

Case Report

A 11 year old boy attended our clinic with complaints of diminution of vision in the left eye of about 1 month duration. He was diagnosed to have Sturge Weber syndrome at the age of two years. CT scan done at that time was normal. But no ophthalmic work up had been done so far.

Examination of the face revealed a diffuse reddish lesion of the skin involving both upper lid and lower lid on the left side (Fig. 1). Anterior segment examination showed multiple, dilated and tortuous vessels over the conjunctiva in the left side (Fig. 2a-d) more so in the superior and nasal aspects.

Gonioscopy in the right eye was normal, while the left eye revealed vascularisation of the periphery of the cornea. The pachymetry readings were 557 and 560 microns in the right eye and left eye respectively.

On dilatation, there was diffuse dark red appearance of the fundus which is characteristically described as 'tomato ketchup fundus' (Fig. 3). There was an asymmetrical cupping which was more in the left eye (Fig. 4). Digital fluorescein angiogram showed diffuse "leopard spot" pattern of hyperfluorescence with areas of blocked fluorescence in between (Fig. 5). C 30-2 field evaluation was within normal limits in both the eyes



Fig. 1. Hemangioma of the skin of upper and lower lid of left eye

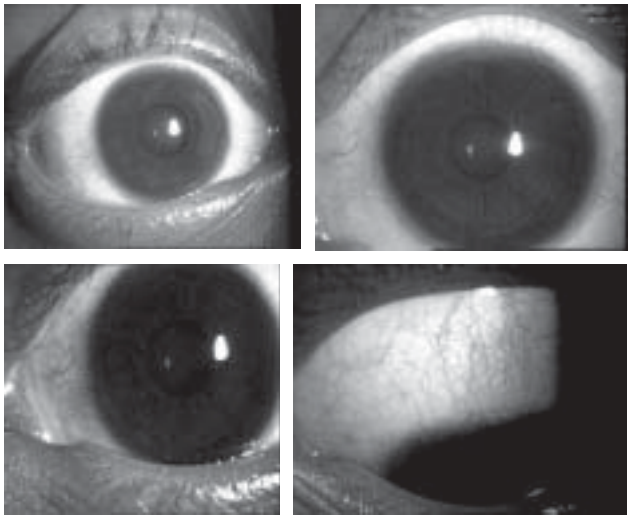


Fig. 2 a-d (a) Normal anterior segment of right eye, (b & c) Multiple dilated tortuous episcleral vessels in the nasal and superior part of conjunctiva left eye (d) vascularisation of peripheral cornea left eye

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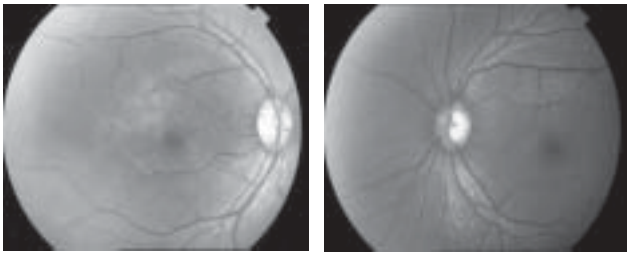


Fig. 3. Tomato ketchup fundus appearance bilaterally suggestive of diffuse choroidal haemangioma

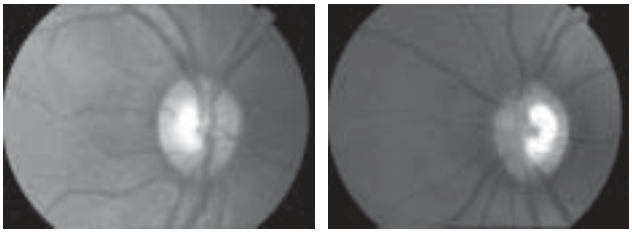


Fig. 4. Disc imaging demonstrating asymmetric cupping LE>RE

(Fig. 6). OCT scan findings were as follows:- CD ratio – Horizontal/Vertical = 0.69/0.538 RE and 0.896/0.777 LE. No retinal nerve fibre layer thinning was made out. Ultrasound B Scan (Fig. 8) revealed thickening of the choroid in both eyes. CT scan of head and orbit was

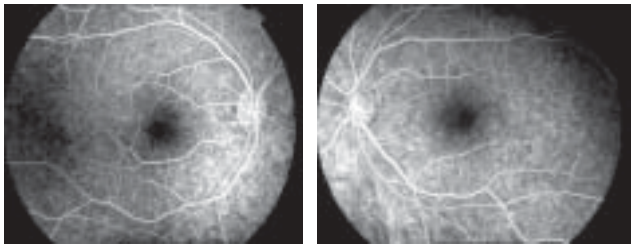


Fig. 5. Diffuse "Leopard Spot" pattern of hyperfluorescence with areas of blocked fluoroscence

normal. An MRI with contrast has been advised to demonstrate choroidal involvement.

The patient was started on beta blockers in his left eye and now the IOP has come down to 18 in a period of 4 weeks.

Discussion

Sturge Weber syndrome comes under the group of phakomatosis – (first described by Van der Hoeve ('Phakoma' means mother spot). The other members of this group are:- Neurofibromatosis (1/4000),

Vou Hippel – Lindau disease, Tuberous Sclerosis (1/30,000), Wyburn Mason Syndrome, Ataxia Telengiectasia, Klippel – Trenuanay – Weber

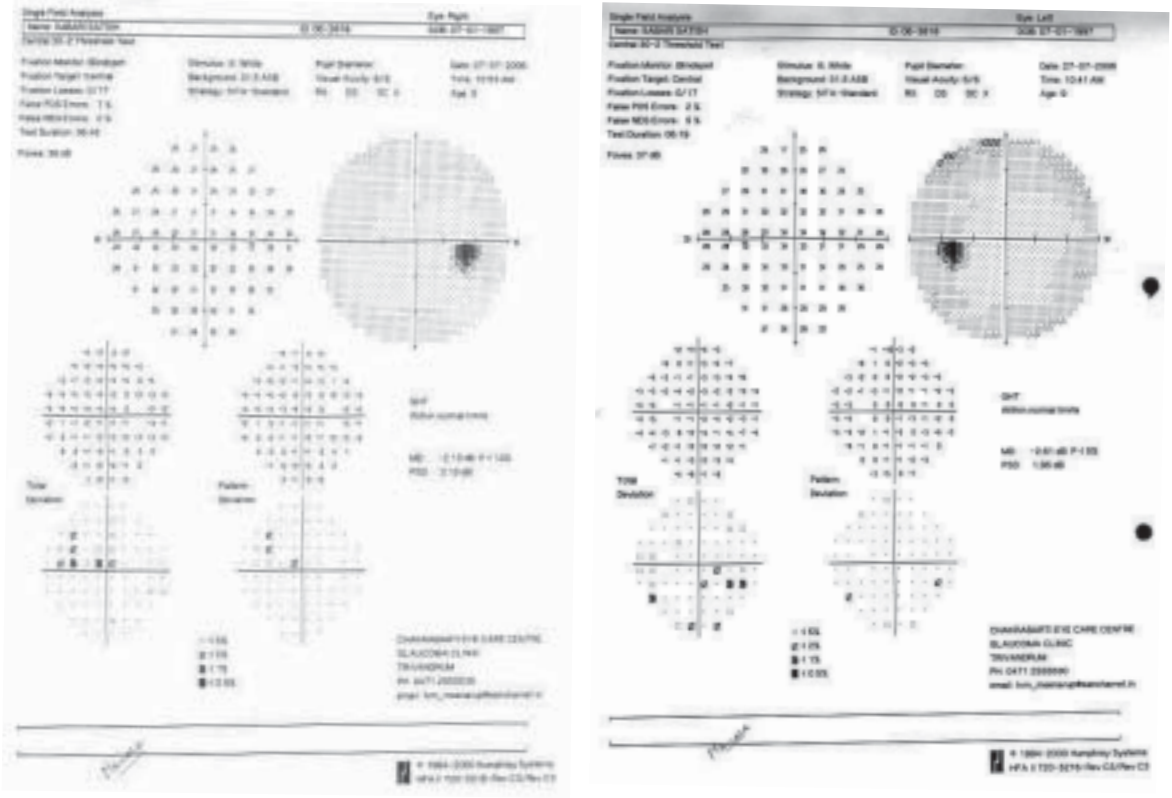
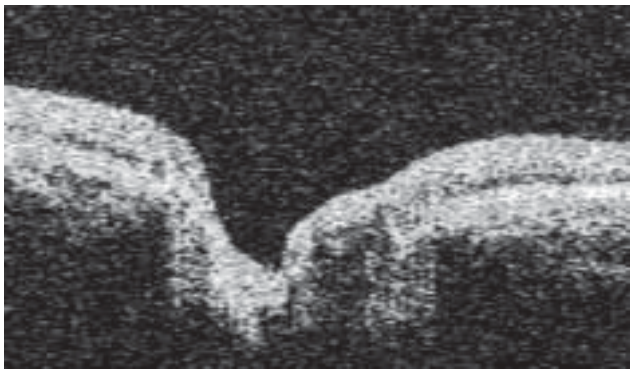
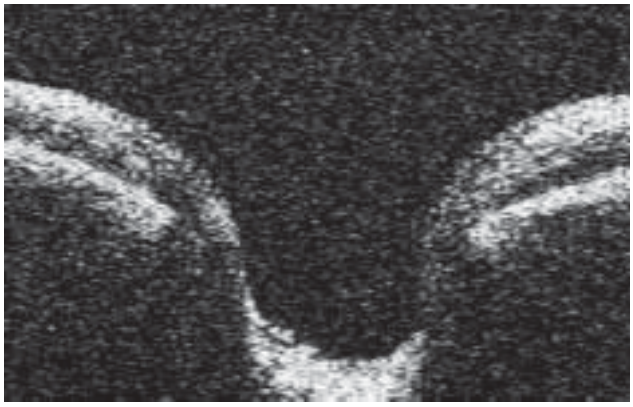


Fig. 6. C 30-2 evaluation demonstrating normal fields in both eyes





C D Ratio	OD	OS
Horizontal	0.69	0.896
Vertical	0.538	0.777

Fig. 7. OCT of right and left eye with optic nerve head analysis

Syndrome, Oculodermal Melanocytosis and Phakomatosis Pigmentovascularis

Sturge Weber syndrome:- (1/50,000) is otherwise called Encephalotrigeminal angiomatosis. It is a dermatooculoneural syndrome with an unclear mechanism of genetic transmission.

The triad of Sturge Weber syndrome consists of (1) Naevus flammeus – which is a hamartomatous haemangioma of the facial skin, otherwise called portwine stain. (2) Ipsilateral leptomeningeal angiomatosis These patients usually present with history of seizures, hemispheric motor or sensory deficits and mental retardation. A characteristic radiographic finding is cortical calcifications that develop after several years and appear as double densities or "railroad tracks". (3) Ipsilateral ocular involvement. This includes haemangiomas of the episclera, iris, cilary body and choroid.

The incidence of glaucoma in patients with Sturge Weber syndrome is 50% (30-71%). Those patients with both upper lid and lower lid involvement are at higher risk of developing glaucoma. There are 3 mechanisms by which glaucoma develops in patients with Sturge Weber syndrome (1) Angle anomaly (60%). These patients normally present during infancy. (2) Raised episcleral venous pressure (60%), secondary to the development of episcleral haemangiomas (3) A rare mechanism of glaucoma is neovascularisation of angle which occurs as a sequelae of chronic retinal ischemia secondary to exudative retinal detachment.

The incidence of choroidal haemangioma in Sturge Weber syndrome is 40%. Bilateral choroidal haemangioma is rare which occurs in 6.12%. The characteristic diffuse dark red appearance of the fundus is described as "Tomato ketchup fundus". Complications of choroidal haemangioma include cystoid degeneration of the overlying retina and exudative retinal detachment.

Management

Since the IOP is now well controlled with beta blockers, this patient can be continued with the same. If the IOP

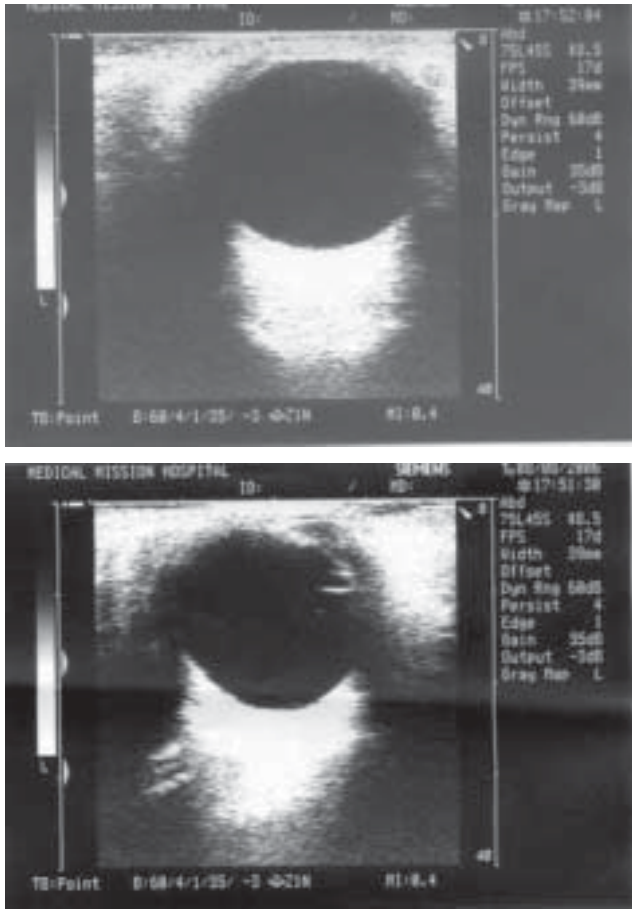


Fig. 8. USG B - Scan: thickened choroid in both eyes

goes out of the target pressure range, prostaglandins can be added. Even though the exact mechanism by which prostaglandins lower the IOP in cases with raised episcleral venous pressure remains unclear, literature says that prostaglandins do lower the IOP in such cases. The final resort will be a filtering procedure which, of course is associated with many potential complications.

Considerations in filtering surgery

The most common complication following filtering procedure in patients with Sturge Weber syndrome is intraoperative or postoperative choroidal effusion. This occurs in about 25% of the patients.

Another dreaded complication which can occur intraoperatively is expulsive haemorrhage (6%) or suprachoroidal haemorrhage. Intraoperative flat anterior chamber can occur in 20% of the patients undergoing filtering surgery.

Precautions

Regarding mode of anaesthesia , topical anaesthesia is the preferred choice in adults. This avoids the danger of bleeding during peribulbar injection. A judicious use of cautery is also recommended in these patients.

Preoperatively, ocular hypotony should be achieved. Sudden lowering of intraocular pressure intraoperatively can lead to choroidal effusion. Controlled decompression of the eye during surgery is important to prevent the above complication. Some surgeons perform prophylactic posterior sclerotomy in conjunction with trabeculectomy surgery in patients with Sturge Weber syndrome.

Conclusion

This is a case of bilateral diffuse choroidal haemangioma in a case of unilateral Sturge Weber syndrome with previously undiagnosed glaucoma.

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A case series of DUSN (Diffuse Unilateral Subacute Neuroretinitis)

Dr. Sarath Ravi, Dr. Nimi R, Dr Biju John, Dr K. Mahadevan, Dr E.V. Suguna Devi

DUSN or Diffuse Unilateral Subacute Neuroretinitis is a progressive parasitic disease of the outer retina and retinal pigment epithelium where nematodes are seen deep in the retina or in the subretinal space.The nematodes primarily causing DUSN are Ankylostoma Canium, Toxocara Canis, and Baylisascaris procyonis. DUSN usually presents in children with the insidious onset of unilateral paracentral scotoma and transient visual obscurations. Here we report 2 cases of DUSN which presented in rather unusual age group and also were different from each other in the way they presented.

The first case is that of a 38 year old otherwise healthy female who presented with defective vision in her left eye of 2 weeks duration. On examination her best corrected visual acuity in left eye was 6/36. There was a grade two relative afferent pupillary defect and fundus examination showed a hazy media due to fine vitreous floaters, hyperemic disc, and on careful examination showed a live nematode in the subretinal space temporal to the macula, seen moving actively from one location to another. (Fig 1a & b)

Laser photocoagulation was done using diode laser (power of 600 mW, spot size 150 microns and duration 100 ms). End point was death of worm with no movement noted. (Fig. 2a)

Following laser ablation of the nematode, patient was started on systemic steroids resulting in a resolution of the retinitis and a final visual outcome of 6/12. (Fig. 2b)

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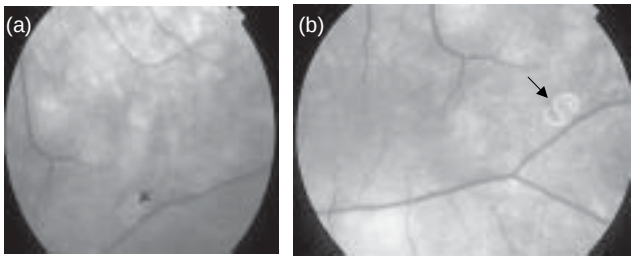


Fig. 1. (a) shows the worm in the subretinal space inferior to the macula (b) shows the worm just temporal to macula taken after a period of one minute after the first

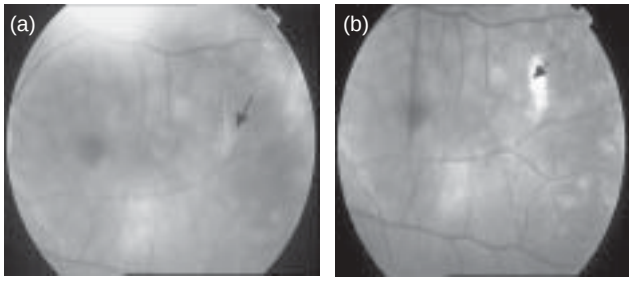


Fig. 2. (a) shows the laser application done on the worm. (b) shows the tissue reaction 24hrs post laser)

The second case is that of a 55 year old male patient who presented with acute onset of defective vision of 3 days duration. On examination the vision in the right eye was CFCF, with circumcorneal congestion, cells in anterior chamber and in anterior vitreous face.The fundus picture (Fig. 3a & b)showed multiple grey white lesions in the outer retina. So we had the differential diagnosis of a multifocal choroiditis, neuroretintitis or white dot syndrome and DUSN. The left eye examination was normal.

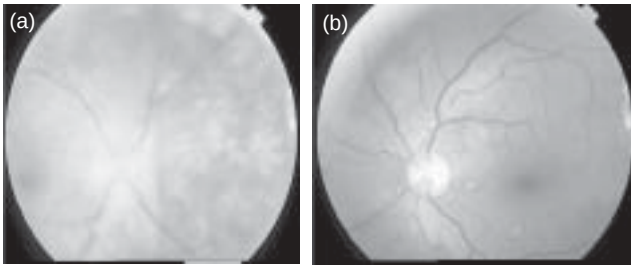


Fig 3 (a & b) on the left shows the multiple grey white lesions in the outer retina Fig. 3b fundus picture was normal in the left eye

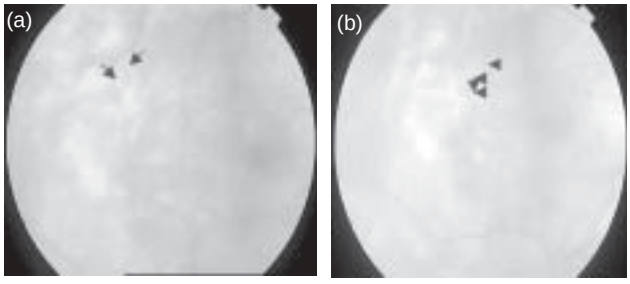


Fig 4. (a & b) the worm is seen about ½ DD above the superior edge of disc.

So we proceeded with routine and specific investigations and the patient was reviewed after 24 hours. The investigation results were negative. The repeat fundus examination revealed an actively motile nematode with one end of the worm close to the centre of fovea due to which laser treatment was deferred.

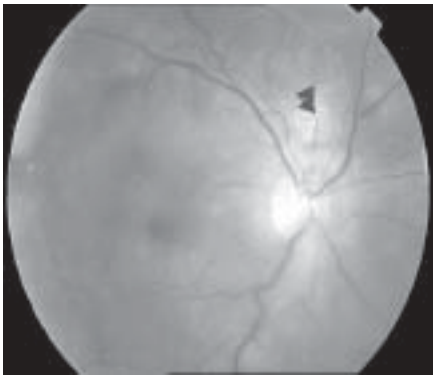


Fig. 4. (c) The worm is seen about 2 DD above the superior edge of disc)

So we thought of waiting till the worm moved sufficiently away from the fovea to a 'safe' location where it could be lasered.In the mean time the patient was started on systemic steroids.

After 48 hours the retinal inflammation had subsided and the nematode was then seen very clearly but still in an unsafe location close to the disc. (Fig. 4 a,b & c)

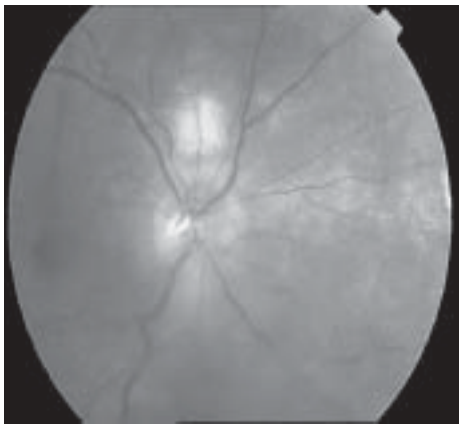


Fig. 5. Shows the worm lasered above the disc.

We waited for another 48 hours for the worm to move away from disc but the worm seemed stuck and not moving out.So we went ahead with the laser. Laser settings used(Diode Laser) were 500 mW power, 150 microns spot size and duration 100 ms.15 shots were applied. (Fig. 5)

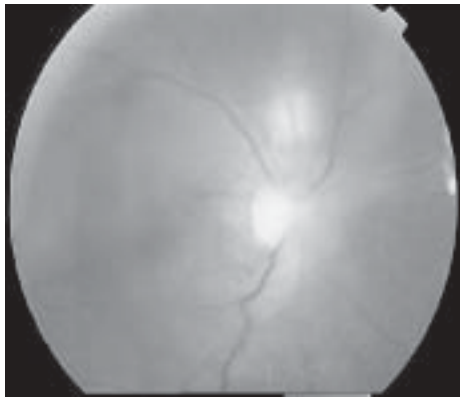


Fig. 6 (a) Demonstrating increase in retinal inflammation and vitritis after laser

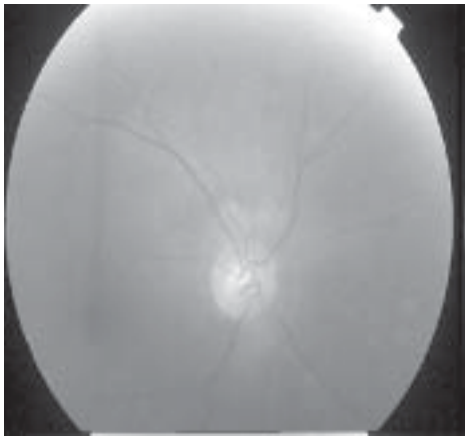


Fig. 6 (b) Fundus photo of right eye after a single posterior subtenon's injection of Triamcinolone acetonide

Following the laser there was a marginal increase in the retinal inflammation and vitritis (Fig. 6a)

The patient was given a posterior subtenon's injection of 20 mg Triamcinalone acetate (Fig. 6b). Systemic steroids was also started.Vitritis and retinal inflammation subsided.



Fig. 7. Demonstrates remarkable clearing of ocular media after a course of systemic steroids

Vision in the RE had then improved to 6/12.

Two weeks post laser the vision in the right eye improved to 6/6.

DUSN is thought to be a rare clinical entity and so we thought of reporting these two cases which presented in our hospital in the last one year, 4 months apart.

DUSN or diffuse unilateral subacute neuroretinitis is a progressive disease of the outer retina and RPE where nematodes are seen deep in the retina or in the subretinal space. The nematodes primarily causing DUSN are Ankylostoma Canium, Toxocara Canis, and Baylisascaris procyonis.Human infection is thought to result from ingestion of soil contaminated with larvae or eggs. Presumably the worm hatches in the intestine and migrates via the bloodstream to the subretinal space. The life cycle of these worms in humans is not well-characterized Retinal pathology is thought to be caused by an inflammatory reaction to the presence of the worm in the subretinal space. Fundoscopic findings in early stages of the disease include vitritis, papillitis, and clusters of gray-white outer retinal lesions. Later in the course of the disease, there is optic nerve atrophy, retinal arterial attenuation, and diffuse pigment epithelial disturbance. If the worm is killed early in the course of disease, severe vision loss may be prevented. In both our cases we could restore the patient's vision adequately thanks to early detection of the worm and timely treatment. However only in 25% of cases, a worm is visualized on fundoscopic exam. In those cases where the history is not clear or the worm is not seen, diagnosis can be quite difficult. Early retinal changes may be confused with multifocal evanescent white dot syndrome, acute posterior placoid pigment eiptheliopathy, optic neuritis, or papillitis.A careful and repeated fundus examination in such cases keeping in mind the possibility of DUSN may prove to be rewarding.

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Fulminant Tribacterial Traumatic Endophthalmitis - All Is Not Lost!

Dr Gopal S Pillai MD, DNB, FRCS, **Dr Abhijeet Khake** MBBS, **Dr Meenakshi Dhar** MS, **Dr Anuradha Rao** MS, **Dr Rajasree Nambyar** DO

Introduction

Trauma to the eye can be complicated by retained intraocular foreign body (RIOFB) and it can be further complicated by post traumatic endophthalmitis. Problems are amplified when the infection is caused by more than one organism. The prognosis of such a case is extremely poor. We present this case of tribacterial endophthalmitis as it is a very rare entity and combinations of Pseudomonas and Acinetobacter has never been reported to be successfully treated before. We also want to highlight the role of an ophthalmologist in managing polybacterial traumatic endophthalmitis including gram positive and gram negative organisms to optimize the results of such treatment.

Case report

A 40-year-old male, electrical technician in the Middle East was referred to our center with a history of injury in the right eye. He was watching some hammering work when he suddenly felt some thing flying into his eye. He started having redness, pain and watering immediately. There was bleeding from his right upper lid and the vision had become cloudy. He was seen at the local hospital and was diagnosed to have a retained intraocular foreign body. He was immediately referred to our center.

On examination at our center, his visual acuity in the right eye was perception of light with inaccurate projection in two quadrants. The upper lid in the right eye was edematous. There was a wound in the upper lid about 3mm superior to the lid margin. Conjunctival and ciliary congestion were seen along with a wound of entry on the sclera at 12 O' clock position, 2 mm above the limbus. Cornea was hazy and edematous. Anterior chamber was filled with fibrin and exudate.

The pupil was faintly visible and was 4 mm in size. It was not dilating. Lens and fundus details were not visible. The intraocular pressure of that eye was 47 mm Hg measured by Applanation tonometry. Examination of the left eye was within normal limits. A CT scan was done immediately which revealed a retained intraocular foreign body, 2-3 mm in size, lying in the inferonasal quadrant very close to the retina.

He was immediately admitted and started on empirical treatment with systemic and topical vancomycin and ceftazidime and systemic steroids. He was taken up for a pars plana lensectomy with total vitrectomy, vitreous base excision, with removal of Intraocular foreign body and silicone oil injection. The procedure was completed without any complications.

During surgery, vitreous and aqueous tap was sent for culture and sensitivity. There were 3 organisms identified on culture: Coagulase –ve staphylococcus epidermidis, Pseudomonas, and Acinetobacter.

Post operatively, the patient was managed with systemic and topical vancomycin, ceftazidime and gentamicin. He was also given intravenous steroids. He steadily improved with good corneal clarity and progressively increasing red glow. At one month follow up, the patient

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had an aphakic visual acuity of 2/60 and a best corrected visual acuity of 6/60. The intraocular pressure was controlled and there was a pupillary membrane, which was resolving well. The retina was well seen and was without exudates.

Discussion

Incidence of endophthalmitis in trauma is 2-7%. Upto 40% of open globe injuries contain IOFB and out of these 90% are metallic. Common composition of these IOFB's are iron, copper, glass and wood. Retained IOFB is an emergency as it may lead to severe vision loss due to endophthalmitis, retinal detachment & metallosis bulbi. About 8-13% of IOFB's develop endopthalmitis

CT scan is the method of choice for investigation of such cases. It is highly sensitive and can accurately localise the IOFB. It can also suggest the composition of IOFB. But negative CT does not rule out the presence of metallic IOFB. Ultrasound examination is very important in all cases of traumatic endophthalmitis to find out whether there is a foreign body, the location of the foreign body and the presence of retinal detachment, choroidal detachment or optic nerve damage. MRI scan is used in selected patients in whom we do not doubt a magnetic foreign body.

Common organisms isolated are Gram-positive cocci, Gram-positive bacilli and Gram-negative bacilli. *Staphylococcus epidermidis* and *Pseudomonas aeuruginosa* were the most common amongst single isolates[1]. Bacillus cereus & Streptococcus species are also commonly identified organisms. Polymicrobial infections are rare. In a latest report from India polymicrobial infection was seen in 3.26%[1] Trimicrobial infections are still rarer. The combination of acinetobacter and pseudomonas has never been reported before in literature.

Posttraumatic endophthalmitis caused by acinetobacter is rare as is polymicrobial infection with acinetobacter. It is an ubiquitous saprophytic-gram-negative bacillus of low virulence. Up to 25% of healthy adults exhibit cutaneous colonization[2] *Acinetobacter* has been known to cause infection in every organ of the body. Crawford et al reported this organism as a cause of recurrent posttraumatic endophthalmitis. [3]

In considering antibiotic treatment of polybacterial endophthalmitis, it is important to recognize that no single antibiotic provides coverage for all of the microbes isolated from eyes with endophthalmitis. Combination therapy is recommended as the initial empiric treatment of suspected bacterial endophthalmitis[4]. After removal of IOFB it is important to give broad spectrum intravitreal antibiotics with gram negative and gram positive coverage. The commonly used antibiotics are Vancomycin(1.0 mg) + ceftazidime(2.25 mg)/ amikacin(200-400 micrograms). The doubts that intravitreal amikacin can cause macular infarction has prompted the use of ceftazidime more frequently. Intravitreal antibiotics are given even following vitrectomy.

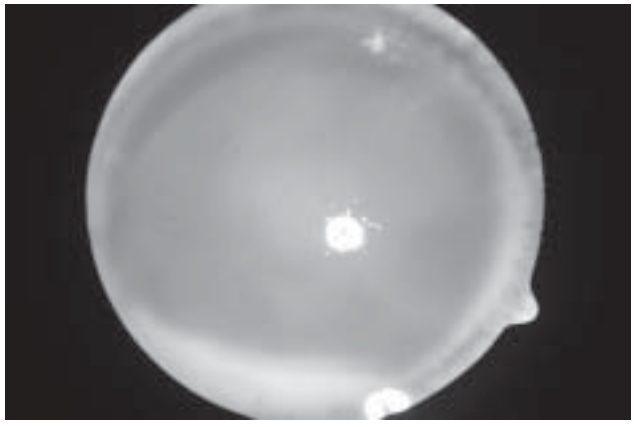


Fig. 1. Anterior segment of the right eye showing corneal edema and severe anterior chamber reaction with fibrinous membrane. Pupil is very hazily seen through the cornea. Iris details are not seen

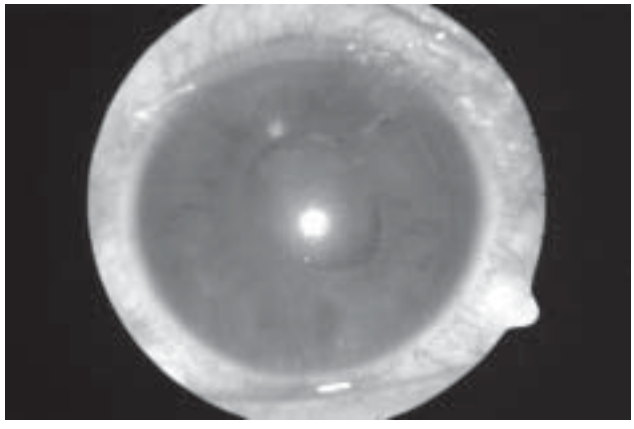


Fig. 2. Anterior segment of the right eye after surgery showing the clarity of anterior chamber. Iris and pupil are well seen. A pupillary membrane and inferior peripheral iridotomy is also seen.

The next important step is to start the patient on systemic antibiotics. Cefazolin, ceftazidime, vancomycin and ciprofloxacin are commonly used intravenous antibiotics. There is a definite role of intravenous antibiotics for post traumatic endophthalmitis as against postoperative endophthalmitis.

Vitrectomy is indicated in cases with retained intraocular foreign bodies with or without endophthalmitis. In cases of retained IOFB, visual prognosis is best if IOFB is removed as soon as possible during the primary wound repair. Delay in repair and IOFB removal for 24 hours produces a four fold increase in endophthalmitis risk. But if vitreo retinal support is not available, one can suture the primary wound and then send the case for expert vitreo retinal management.

This case also hilights that even in the presence of such virulent endophthalmitis with an IOP of 47 and vision of inaccurate projection of rays, the treatment option has shifted from evisceration to pars plana vitrectomy. Evisceration as a mode of management of severely damaged eyes with endophthalmitis is replaced by more aggressive and eye salvaging managements like pars plana vitrectomy and silicone oil injection.

Earlier, the surgical management of traumatic endophthalmitis was with core vitrectomy. But with the advent of more superior vitreo retinal instruments and machines, it is now possible to remove cyclitic membranes and prevent permanent hypotony and phthisis bulbi. After doing a complete vitrectomy with vitreous base dissection and cyclitic membrane removal semipermanent tamponade with silicone oil is usually performed. In most cases managed this way, the results have been excellent.

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Combined Hamartoma of the Retina and Retinal Pigment Epithelium

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

Combined Hamartoma of the retina and retinal pigment epithelium are benign tumors that cause significant visual loss. Accurate diagnosis is essential as these lesions can simulate choroidal melanomas and other intraocular tumors. This presentation demonstrates the clinical features, angiographic findings and OCT imaging characteristics of combined hamartoma of the retina and retinal pigment epithelium presenting unilaterally in a five year old female child.

Case History

A five year old girl was referred with a diagnosis of regressed retinopathy of prematurity in her left eye. Her mother gave the history of a normal antenatal period, normal full term vacuum extraction; and an uneventful neonatal period. They had noticed squinting of a variable degree in the child's left eye by the age of 2 years. They considered taking her for ophthalmic consultation at the insistence of her class teacher at school, who felt that the child had poor visual acuity.

Ocular examination revealed an uncorrected visual acuity of 6/6 in the right eye and 5/60 in the left eye not improving further with either trial of glasses or pin hole and 15° of left divergent squint. Anterior segment examination revealed a normal anterior segment, clear lens and normal intraocular pressures (Tn_A=18 0U). Fundus examination revealed a normal retina and optic disc in the right eye(Fig:1). The left eye showed a large whitish elevated lesion in the peripapillary area and posterior pole with dilated tortuous vessels and area of schisis centrally. (Fig.2). The optic disc appeared hyperemic and vertically oval in shape. Fluorescein fundus angiography showed early hyperfluoresence progressively increasing in intensity, with multiple dilated tortuous blood vessels on its surface (Fig.3). The blood vessels peripheral to the lesion appeared stretched and straightened. B scan Ultrasonography showed a moderately elevated lesion over the posterior pole (Fig.4). Macular scan of optical coherence tomography showed a normal macular contour in the right eye (Fig 5a). The left eye scan revealed a grossly thickened inner retina with multiple large and small schitic cavities (Fig.5b)

The findings of this case were suggestive of a Combined Hamartoma. The parents were counselled about the cause of visual loss and about amblyopia in the child's left eye. The possible complications on long term follow up such as vitreous hemorrhage, choroidal neovascular membranes, retinoschisis, retinal tear, posterior pole retinal detachments and epiretinal membrane formation were discussed and they were counselled on the necessity for follow up examinations.

Discussion

Combined hamartomas are solitary unilateral lesions located at the optic disc or posterior pole. They typically appear slightly elevated and have varying amounts of pigmentation, vascular tortiosity and epiretinal membrance formation. The mean age at the time of diagnosis is 15 years with a range of 10 months to 66 years.

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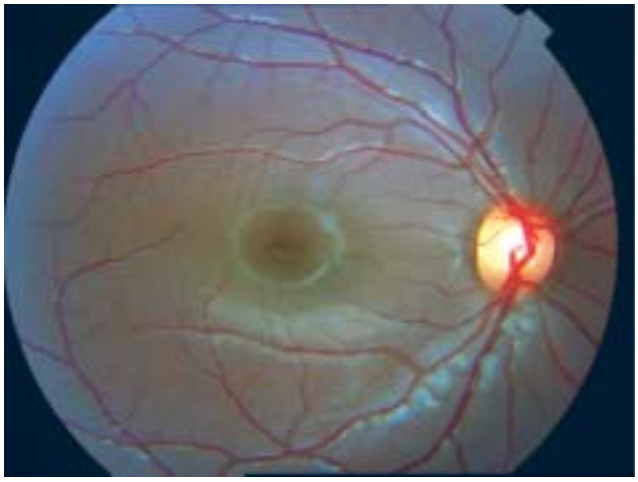


Fig. 1. Fundus Photography of the right eye



Fig. 2. Fundus Photography of LE showing an elevated whitish lesion with vascular tortuosity and central dark area representing a schisis cavity

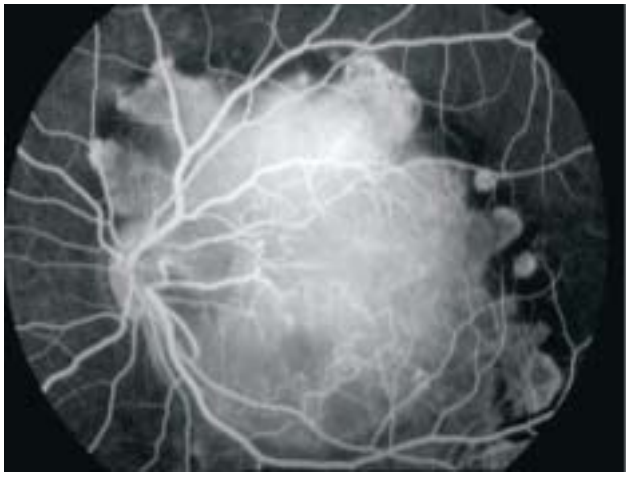


Fig. 3. Late phase angiogram showing hyperfluorescence over the entire lesion , and tortuous blood vessels

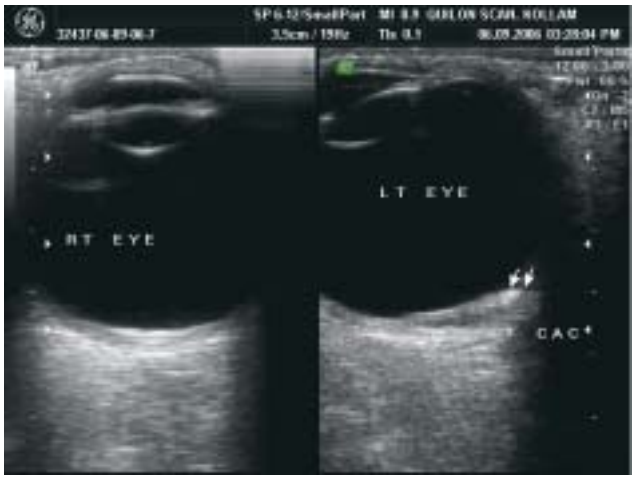


Fig. 4. B-Scan USG demonstrating the lesion with high reflectivity surface echoes and central dark schisis cavity

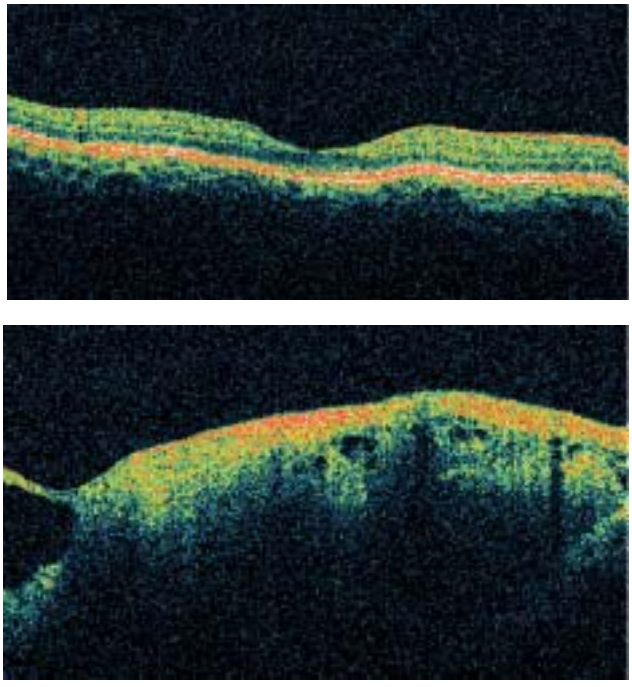


Fig. 5. (a) Normal OCT Macular scan right eye (b) Macular OCT Scan of left eye showing grossly thickened inner retinal layers and multiple intraretinal schitic cavities

Painless visual loss is the chief complaint of 60% of patients. Other presenting symptoms were strabismus (18%), floaters (5%), leucocoria (3%) and pain (3%). Visual function varies with the location of the lesion. Direct involvement of the optic nerve, papillomacular bundle or fovea will result in gross visual defect. If these structures are not involved, visual loss may result from macular distortion by retinal striae or by epiretinal membranes.

The lesions may be located on the optic disc(18%), juxta papillary region (10%), in the macula (38%), or in the mid periphery (5%). The lesions are usually solitary and unilateral. Bilateral lesions have been reported in association with neurofibromatosis.

Associated ocular findings include relative afferent pupillary defect in patients with extensive disc or macular lesions, strabismus, vitreous hemorrhage, chorodial neovascularisation at the margin of the lesion, secondary epiretinal membrane formation, macular hole and detachment. Combined hamartomas have been reported in association with X linked juvenile retinoschisis, optic nerve head pits, optic nerve head colobomas and optic nerve head drusens. Systemic associations include neurofibromatosis, facial haemangiomas, incontinentia pigmenti and tuberous sclerosis.

The diagnosis of combined hamartoma is a clinical one. The ophthalmoscopic appearance is sufficient to make a diagnosis, however, fluorescein angiography serves as an useful adjunct. The mid phase angiogram shows the striking vascular anomalies. Pseudovascularity and straightening of the vessels may be present. Leakage from these tortuous blood vessels cause late hyperfluoresence.

The management is usually conservative. In combined hamartomas associated with tractional distorsion of macula and epiretinal membrane proliferation, parsplana vitrectomy and epimacular membrane surgery has been tried, however, improvement in visual acuity have been rarely reported.

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Lasik – Why Not ?

Dr. Anil Radhakrishnan MS[1], **Dr. Ashish K. Bansal**[2], **Dr. K. Narayankutty**[3], **Dr. Pravin Krishna**[4], **Dr. Sitalekshmi**[5]

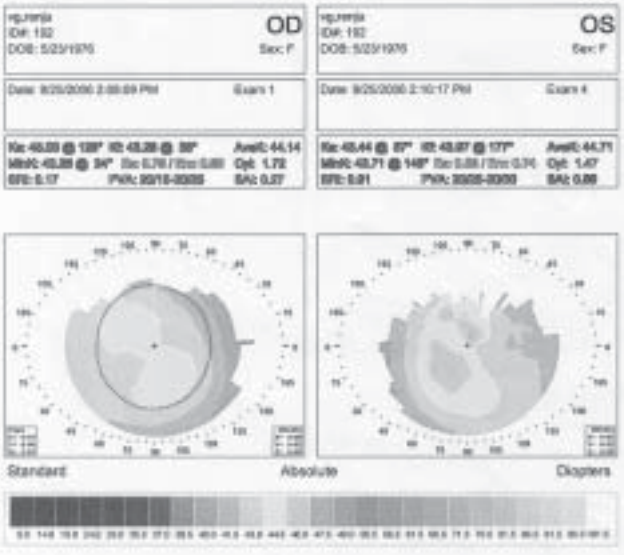
A thirty year old lady presented to our institute to explore the possibility of laser refractive correction. She was a soft contact lens wearer using them on a daily basis, more than ten hours a day, for the past four years and her present pair was less than a year old. Though her vision was not as crisp as with glasses she continued using contact lenses for improved cosmesis. For the past one and a half years her eyes felt dry often for which she was using 0.5% carboxymethyl cellulose eye drops 4 times a day. She had never changed her glasses in the last five years. She had no known systemic illnesses or allergies and was not on any medications. She came for evaluation five days after discontinuing her lenses.

On examination, her BCVA was 6/9 OU with -8.5 D sphere/-2.0 cyl @ 40 degrees in the right eye and -8.5 D sphere / -1.5 cyl @ 150 degrees in the left eye. Her spectacle prescription was -9.0 D sphere/ -1.25 cyl @ 30 degrees in the right eye and -9.0 DS / -1.0 cyl @ 180 degrees in the left eye. Her IOP was 12 mm Hg in both the eyes. She had 15 prism diopters of esophoria for distance and near and had normal ocular movements. Her lids were normal, and clear meibomian secretions were easily expressible on pressure over the tarsus. The tear meniscus measured about 0.5mm with no debris on indirect illumination. Her conjunctiva was quiet and cornea clear in both the eyes. The anterior chamber was deep & quiet with normal pupils and crystalline lens was clear. Dilated fundus examination was within normal limits.

Dry eye work-up showed T-BUT of 11 seconds in both the eyes. Schirmer's-1 values were 13 & 35 and Schirmer's-2 values were 3 & 7 in the right and left eye respectively. Rose Bengal staining was negative but corneal fluorescein score was 3 in the right eye and 2.5 in the left eye.

Her minimum pachymetry values were 551 microns in the right eye and 535 microns in the left eye. Her topography picture – absolute scale is shown in Fig no:1. The picture showed an asymmetric bow tie with high SAI. Klyce / Smolek software for keratoconus screening interpreted 'keratoconus suspect' though Klyce/ Maeda software interpreted 'normal pattern'. Assessment of posterior corneal contour was not done. She was prescribed preservative free lubricants and was requested to review after a week.

In the second visit, T-BUT was 12mm in both the eyes. Schirmer's-1 values were 4 and 35 and Schirmer's-2 values were 6 and 15 in the right eye and left eye respectively. The corneal fluorescin score had reduced by 50%. Repeat topography was done and is shown in



[1]*Chaithanya Eye Hospital and Research Institute, Kesavadasapuram, Trivandrum,* [2 & 4] *L.V. Prasad Eye Institute, Hyderabad,* [3] *Medea Lasik Centre, Kochi,* [5] *Shankara Nethralaya, Chennai*

Figure 2. Her aberrometry values were -7.74 D sphere/ -1.54 cyl@ 52 degrees and -7.32 D sphere / -1.45 cyl@ 160 degrees in right and left eye respectively.

Can she undergo laser refractive surgery? If so when?

Dr. K. Narayanan Kutty

You can confidently go ahead with LASIK for the right eye without any danger of keratectasia. Regarding the left eye repeat the topography immediately after instilling lubricant eye drops. If the picture is same still you can do LASIK with 90 micron head or LASEK.

Dr. G. Sitalakshmi

I will not offer LASIK at this juncture

1. There is an improvement in her topography with time as reflected by pictures and the indices and topography should be repeated after a couple of weeks
2. Go ahead with LASIK when two topography pictures are stable and normal looking. The diagnosis as of now is in favor of contact lens induced corneal warpage
3. As the fluorescein staining is still positive continue lubricants (preservative free) to improve her ocular surface
4. If there is any blepharitis /meibomitis treat it
5. Pachymetry also should be repeated prior to LASIK

Dr. Ashish. K. Bansal

I think we have to be cautious in this case and need not take a decision immediately. The topography has shown some change, even if a small one, after removing the lenses. It will be good to wait further for a week and repeat topography and see if it is still changing. Once it stops changing, then make a final opinion. If similar picture persists it is highly unlikely to be a pathological cornea.

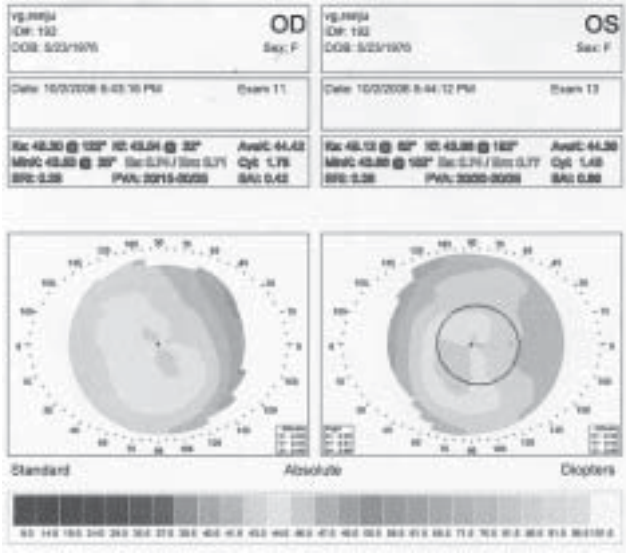
Secondly, it will good to follow up on her corneal staining status. Does it disappear with lubricants? If not, then further investigations are warranted.

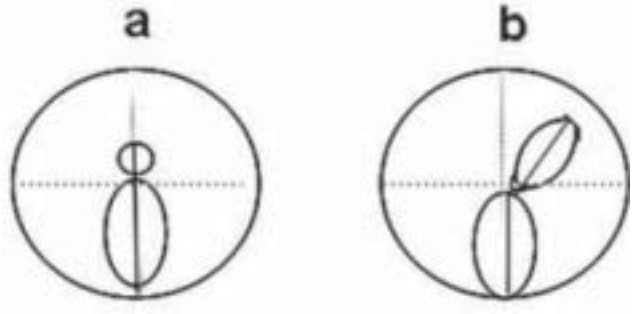
Thirdly, some of these eyes having phorias, can convert into manifest squint after undergoing LASIK. The patient should be made aware of this fact.

So, if the topography remains more or less same, as it is now, the corneal staining disappears and the patient has been informed about the possibility of developing squint, one may go ahead with LASIK.

Dr. Pravin Krishna

I feel the decision for LASIK has to be taken only after re-evaluation. I would like to treat blepharitis or meibomian gland disease if present. Corneal fluorescein staining is more likely due to mechanical effect of contact lens than due to dry eye as it is asymmetrical. Liberal tear substitutes with contact lens abstinence for two weeks should bring out the actual topographic pattern. If the topography pattern is changing, repeat readings are taken till there is no change. Most likely,





How to interpet SRAX[skewed radial axes] An imaginary line is drawn to bisect the upper and lower lobes of asymmetric bowtie. If there is no deviation, there is no skewing [**a**]. If the lines bisecting the two lobes appear skewed by more than 30 degrees from the vertical meridian it is called skewed [**b**].

the topography will become normal in a few weeks when LASIK can be performed.

Though improbable, it is not impossible that a 'suspicious pattern' is unmasked. In such a scenario I would like to perform an Orbscan and take a decision.

Discussion

Keratectasia is one of the dreaded complications of laser refractive surgery. The possible risk factors are high myopia, thin corneas, steep corneas, reduced stromal bed after laser ablation and asymmetrical corneal steepening [forme fruste keratoconus] [1]. Forme fruste keratoconus is not a variant of keratoconus but a topographic diagnosis. It implies a subclinical disease with the potential of progression to clinically evident keratoconus. An asymmetric bow tie with inferior steepening or asymmetric bow tie with skewed steep radial axes above and below the horizontal meridian suggest forme fruste keratoconus [fig 3] and it is recommended not to perform LASIK in such eyes [3].

Contact lens induced corneal warpage with rigid lenses can mimick a pattern similar to corneal ectasia. A sudden bump in keratometry, typical location at 6'o clock position and a 'smile pattern' of localized steepening are suggestive of superior riding rigid lens [2,4]. CL induced warpage, though much less common can be produced by soft lenses as well, commonly manifesting as central irregular astigmatism and loss of radial symmetry of the image [2,5] .

Keratoconjunctivitis sicca can cause significant alterations in topographic indices and can result in abnormal topographic patterns similar to corneal ectasia [6,7,8] . It is well known that LASIK can induce or worsen dry eye status [10,11].

In this patient there are mainly two issues. [1] Abnormal topography pattern and [2] Ocular surface damage.

Ocular surface damage could be due to ill-fitting contact lenses or due to CL-induced dry eye. Badly fitted lenses especially when worn for long hours can produce superficial punctate keratopathy. Reduced corneal sensitivity seen in long-term CL wearers can interfere with efferent limb of lacrimal reflex arc resulting in tear-deficient KCS, in addition to evaporative loss. Preservative free lubricants with discontinuation of CL wear can address both the issues.

But if Schirmer's test results are not reaching normal values and if epitheliopathy persists after a couple of weeks, the etiology of aqueous deficiency need to be looked into, more so in this age group. In such a scenario any laser ablative surgery can exacerbate the situation.

Regarding the topography, right eye shows a nearly symmetric bow-tie with normal topographic indices. In the left eye there is an asymmetric bow tie pattern, but no skewing of radial axes. The Surface Asymmetry Index is slightly high [normal value is 0.5], indicating that two halves of the cornea in the central part are not symmetrical. Abnormal topographic pattern in this case could be due to [1] CL induced corneal warpage or [2] a variant of normal pattern modified by dry eye or [3] 'forme fruste keratoconus', the possibility of which should be ruled out before considering LASIK.

In soft CL induced corneal warpage topography will revert to normalcy on discontinuing lenses for a few weeks, though it can take longer in a minority [2,5]. Once the topography picture is stable, LASIK can be safely performed.

If the picture is exactly similar after few weeks, the topography picture should be considered a normal variant as there is no skewing of steep radial axes or steepening. LASIK can then be performed quite safely.

If the picture fits into 'forme fruste keratoconus' after stabilisation laser ablative surgery is better avoided. Orbscan or pentacam analysis can give information about the posterior corneal contour.

Also, rarely phorias can break into tropias after refractive surgery. This usually happens in situations where distance vision in one eye is compromised for 'monovision' treatment [12].

To conclude, this case elucidates the fact that LASIK, though a relatively simple surgical procedure demands a detailed preoperative work-up, proper patient counseling and adherence to scientific principles to ensure a successful outcome.

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Compiled & Edited by Dr. Anil Radhakrishnan, Chaithanya Eye Institute, Trivandrum

Results of Deep Lamellar Keratoplasty Using Big- Bubble Technique in Patients With Keratoconus

Rajesh Fogla, Prema Padmnabhan, Am. J. Ophthalmol FEB 2006;141:254-259

This is an interventional case series including 13 eyes of 12 patients with moderate to advanced keratoconus and intolerance to contact lens wear in order to evaluate the efficacy of deep lamellar keratoplasty using the big bubble technique. Patients with scarring of Descemet's membrane were excluded.

Mean age of the patients was 24.5 +/- 8.0(14-41 years). Deep lamellar keratoplasty was performed by intrastromal air injection technique after partial trephination. Full thickness donor tissue oversized by 0.5 mm, devoid of Descemets membrane and endothelium was sutured into place using 10-0 nylon with 8 interrupted and 16 continuous sutures. The entire cone was included in the area of trephination.

Big bubble was successfully achieved in 9 eyes (69.2%). Average BCVA improved from 20/200 – 20/25. Mean keratometry postop was 44.5+/- 1.6 and astigmatism 2.57+/- 1.3 as compared to preop K value of 49.8+/- 5.98 and astigmatism of 2.57+/- 1.3. Mean CCT postop improved to 531+/- 35.9 as compared to the preop value of 365+/- 68.4. Avg endothelial cell count 2899+/- 499 cells/ mm^2. Mean follow-up was 5 months. Intraoperative micro perforation of descemets membrane was seen in 2 eyes (15.3%), 3 eyes developed a steroid induced increase in IOP, which responded to medical treatment.

Deep lamellar keratoplasty with big bubble is safe and effective in moderate to advanced keratoconus and provides satisfactory visual outcome comparable to standard penetrating keratoplasty without risk of endothelial rejection.

Subconjunctival Placement of Human Amniotic Membrane During High Risk Glaucoma Filtration Surgery

Christina A. Bruno, Jonathan A. Eisengart, Pauline A. Radenbaugh, Sayoko E. Moroi. Ophthalmic surgery, Lasers and Imaging, May/June 2006; 37:190-197

This is a retrospective review of 17 eyes of 15 patients who had amniotic membrane applied during glaucoma surgery with or without mitomycin C/ 5 flourouracil in order to determine if subconjunctival placement of amniotic membrane improves filtration results in patients with glaucoma at high risk for surgical failure.

High risk factors included age<40 years, black race, previous conjunctival surgeries, long-term use of anti glaucoma medications and uveitis. Outcome measures were postop IOP, need for further surgery and complications.

Subconjunctival placement of amniotic membrane was carried in any of these 3 ways –1. amniotic membrane sutured epithelial side up in a broad fashion over the sutured scleral flap. 2.Folded in half and placed underneath the scleral flap and then scleral flap closed with sutures. 3. Folded into half twice and placed under flap and completed as in 2.

Mean IOP reduced from 27.0+/- 9.1 preop to 18.1+/- 11.0 postop. Mean follow up was 179 days. 9 out of 17 achieved IOP <15 mm HG, and of these 6 didn't need antiglaucoma medications. Complications included self-limited conjunctival wound dehiscence (n=5), recurrence of uveitis (n=2) and cataract (n=1)

These results suggest that surgical placement of amniotic membrane may improve filtration outcome in high risk eyes which may be due to its anti-inflammatory, anti- angiogenic and anti-fibrotic properties. But it is not ideal in bleb leaks, as wound-healing processes are desired in this clinical situation.

Surgical Embolus Removal in Retinal Artery Occlusion

J.Garcia-Arumi et al, Br. J. Ophthalmol 2006;90:1252-1255

This is a prospective interventional study done to assess anatomical outcome, safety and functional effectiveness of surgical embolus removal in 7 consecutive patients with Retinal Artery Occlusion (RAO) (5- temporal branch RAO, 1 -Hemispheric A occlusion, 1- central RAO) of <36 hrs duration with sudden decrease in visual acuity or visual field defect in area of obstructed vessel, absence of perfusion or marked perfusion delay in affected areas of FFA with visible embolus.

Mean age of patients was 65 years with median preop vision 20/400 and preop macular thickness 358 ìm. Complete scotoma was seen in all patients. Standard 3 port, 20 G pars plana vitrectomy was done and posterior hyaloid dissected from the retina under active aspiration. Longitudinal incision made adjacent to embolus on the anterior wall of the arteriole with 25 G MVR blade. Tano asymmetrical vitreoretinal forceps was used to express the emboli from the vessel if it didn't pass thro the wall incision spontaneously. The arteriole was left unsutured and vasospasm closed the incision.

Surgical embolus removal was achieved in 6/7 patients (87.5%). Median VA postop was 20/40 and macular thickness decreased to 225µm. Reperfusion was achieved within 48 hours in 4 out of the 6 patients who had their emboli removed and this was confirmed angiographically. Mean follow up was 12 months.

Complications like rubeosis iridis and neovascular glaucoma were not seen. 1 patient developed cataract. Vitreous hemorrhage occurred in 2 patients, which spontaneously cleared within 2 weeks.

80% of eyes improved slowly after a week or a month to VA of 20/40, had residual visual field defect. This short time interval in restoration of VA or visual fields suggests that surgical embolus removal may be beneficial.

This is a pilot study, limitation being a small sample size, but showed good reperfusion of occluded vessels with 71% of patients having improved VA or visual fields.

Compiled by Dr. Radha J. DO, Little Flower Hospital, Angamaly

Hospital Waste Management and Its Monitoring

Madhuri Sharma MBBS MBA (HCA) FMS, Pushlisher - Jaypee Brothers, New Delhi, First Edition: 2002 Price Rs.100/-

Management of waste generated by health care institutions and hospitals has emerged as a prime area of concern in recent times. Safe and effective management of biomedical waste is not only a legal necessity but also a social responsibility.

Hospital waste management has been brought into focus recently, particularly with the ruling by Hon'ble Supreme Court of India and the notification of the Biomedical Waste (Management and Handling) Rules, 1998, which makes it mandatory for the health care establishments to segregate, disinfect and dispose their waste in an eco-friendly manner.

No effort needs to be soared to ensure implementing strategies for safe and sound management of hospital waste. Rising to the need, National Guidelines for Management of Hospital Waste, based on the Biomedical Waste (Management and Handling) Rules, 1998 were prepared by the Directorate General of Health Services/Ministry of Health and Family Welfare.

Hospital Waste Management and its Monitoring is a unique book giving adequate knowledge about health hazards of hospital waste, proper technique and methods of handling the waste and practice of safety measures while handling the waste and its safe disposal. It intends to give knowledge, awareness, and methodology including the latest techniques for disposal of biomedical waste. It will be of immense practical help to personnels at different levels in the health care establishments and will serve as an educational aid focusing on varied aspects of handling and disposal of hospital waste.

This book intends to give knowledge, awareness and methodology including the latest techniques for disposal of biomedical waste. It will be of practical help to the grass root level workers, nurses, paramedical and medical professionals (in the health care establishments), who are at an enhanced risk to the hazards of improper handling of biomedical waste. It will serve as an educational aid focusing on varied aspects of handling and disposal of hospital waste.

Berry & Kohn's Operating Room Technique

NancyMarie Fortunato Philips, Publisher: Mosby- Elsevier Science, St.Louis USA, Tenth Edition – 2004, Price $=45/-

Berry and Kohn's Operating Room Technique covers the foundations of surgical technique in a step-in-step format that enables you to effectively apply perioperative nursing and surgical principles to practice. Focusing on the physiologic and psychologic needs of patients it provides guidelines for planning and implementing comprehensive, individualized care. It also reviews the most common surgical procedures and emphasizes teamwork among perioperative caregivers to encourage cooperation in attaining positive patient care outcomes.

This book has 12 sections and 44 chapters.

Section One is dedicated to the learner and the educator. Section Two delineates the roles of the

members of the health care team as both direct and indirect caregivers. Section Three provides in- depth information on patient assessment and the development of an individualized plan of care, with the patient viewed as a unique individual. Section Four examines the physical plan of the perioperative environment-both hospital-based and free-standing ambulatory facilities. Section Five provides an updated chapter on microbiology and the importance of microbiologic control in the perioperative environment, with an emphasis on standard precautions. It delineates aseptic and sterile techniques in fundamental to intermediate aspects such as attire, scrubbing, gowning and gloving. Separate chapters are provided regarding the sterilization and disinfection of surgical instrumentation and patient care supplies. Section Six details the surgical instrumentation and equipment used during surgical procedures. The safe use of specialized surgical equipment is presented.

Section Seven discusses preoperative patient care and includes the family/significant others in the plan of care. Section Eight covers methods of anaesthetic administration and the role of caregivers during this process. Section Nine describes intraoperative patient care, including positioning, prepping and draping. Section Ten focuses on the surgical site. Hemostasis and wound closure are discussed in detail. Section Eleven presents an expanded view of postoperative patient care. The postanesthesia care unit is explained. Section Twelve covers the surgical specialties.

In this new edition (Tenth) the author added new chapters on the Surgical First Assistant (Chapter 5); on Surgical Pharmacology (Chapter 23) and an expanded, up-to-date content covering topics that affect perioperative practice, such as the latest technology for brushless skin cleansing. A dedicated website (evolve) providing links to perioperative resources as well as online learning tools designed to test content knowledge and critical thinking skills.

This Practical, authoritative resource is an ideal text for surgical technology or perioperative nursing courses and an invaluable reference or in-service manual for practicing surgical technologists, perioperative nurses, and other caregivers!

This book is a text of choice for basic surgical principles and techniques.

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Many of us have been chilled and thrilled by Robin Cook's novel "Coma" and by Michael Crichton's highly successful film version of the same. But did we know that both Cook and Crichton were doctors and that Cook was a trained and practising Ophthalmologist who describes himself as "on leave" from the Massachusetts Eye and Ear Infirmary?



Robin Cook (1940-)

Robin Cook, who is described as "the Master of the Medical Thriller", was born on 4th May 1940 in New York City to Audrey Cook and Edgar Lee. His first love was Archaeology but he became a doctor instead, graduating from Colombia Medical School in 1966. After his residency in General Surgery at Queen's Hospital, Honolulu, he became a doctor in the US Navy and reached the rank of Lieutenant Commander. He then went on to do his residency in Ophthalmology from 1971 to 1975 at the Massachusetts Eye and Ear Infirmary, affiliated to the Harvard Medical School, Boston. He then set up Private Practice north of Boston.

His very first book, published in 1972, was "Year of the Intern" and dealt with his experiences as a resident in Honolulu.

He was always interested in fiction and read a good many books before evolving his own distinctive style. "Coma", written in just 6 weeks, was published in 1977. It was an instant success. The film version, directed by Michael Crichton, was released in 1978.

He has written several books since. These have dealt with issues at the forefront of current Medical Practice including organ donation and transplantation, genetic engineering, treatment for infertility and *in vitro* fertilization, drug research and funding, managed care and bioterrorism. He continued to practise while he wrote and this kept him abreast of medical knowledge. He thus felt that while he was quite capable of writing a medical article, the issues would be more widely read and appreciated by far more people if he dramatized it as fiction. Over the years, his storyline has become predictable. But all his books are not only eminently readable, Cook's knowledge of Medicine and Surgery helps to define the subject clearly and accurately.

Besides writing, Cook is also a painter and enjoys diving, surfing and skiing. He married twice but both marriages were unsuccessful; he has no children.

Cook divides his time between his two homes in Boston and Florida, doing most of his writing in the New Hampshire area of Boston. As the list of his books shows, he continues to write and educate his readers while entertaining them.

His books are- Year of the Intern(1972), Coma (1977), Sphinx (1979), Brain (1981), Fever (1982), Godplayer (1983), Mindbend (1985), Outbreak (1987), Mortal Fear (1988), Mutation (1989), Harmful Intent (1990), Vital signs (1991), Blind Sight (1992). Terminal (1993), Fatal Cure (1993), Contagion (1995), Acceptable Risk (1996), Chromosome 6 (1997), Invasion (1997), Toxin (1998), Vector (1999), Abduction (2000), Shock (2001), Seizure (2003), Marker (2005), Crisis (2006).

Up Coming CME Programmes

NATIONAL

1. SEAGIG
Chennai 2006
International Glaucoma Convention
Dec 1-3 2006.
Venue: Chennai Convention Centre
CTC Complex, Nandanbakkam, Chennai 600 089.
Organizing Chair Person: Dr. L Vijaya
Shankara Nethralaya, Chennai
Ph:-2827 1616, 2823 3556
Fax:- 91-44-2525 4180
e-mail: seagig_2006@yahoo.com
Website:- www. seagig Chennai.org

2. Sixth All India Uveitis Conference
Aravind Eye Hospital, Madurai
December 2 and 3, 2006
Dr. Rathinam Sivakumar
Ph:- 0452 4356100, 0452 2530984.

3. XV Annual Conference of Vitreo Retinal Society of India
Dec 7-9, 2006
Conference Venue:- Hotel Riviera Suites, Kochi, Kerala
Conference Secretariat : Giridhar Eye Institute, Kochi
Ph:- 91-484-2316791, 2312303.
e-mail: conference@Nrsi2006.com
web:- www.vrsi 2006.com

4. 3rd International Symposium on "Diabetic Retinopathy and Age Related Macular Degeneration"
5th,6th, 7th, January 2007.
At ARAVIND TIFAC Core Centre for Relevance & Excellence
Detailed Programe Information
www.aravind.org / dr2007/index.htm

5. Sunayana 2007
65th All India Ophthalmological Conference
Feb: 1-4 2007
Hyderabad
Contact:- Dr. Pradeep Swarup
Organizing Secretary: Swarup Eye Centre
145, Dwarakapuri Colony, Panjagutta
Hyderabad 500 082
Ph:- 040-23354558, Fax:- 040 23352617
e-mail:-pswarup@sunayana2007.org

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SYSTEMIC THERAPEUTIC AGENTS AND RETINAL TOXICITY

Sl. No.	Medication	Clinical Symptoms	Clinical Findings	Dosage	Mechanism of action	Test for early detection	Treatment & Course	Prevention/ Early detection
1	Thioridazine (Mellaril)	*Blurring, *Brownish discolouration of vision	*Salt & Pepper Pigment Disturbance *Plaque like pigment clumping *Nummular loss of RPE & choriocapillaries * RP like picture	1000-800 mg/day 100% of cases over 10 years of therapy	Piperidyl side chain leads to toxic inhibition of oxidative phosphorylation & derangement of Rhodopsin	*Perimetry *Colour vision	Pigment degeneration progresses despite stoppage of medication	Annual Exam
2	Chloroquine	* Central Scotoma *Loss of central fixation *Constricted fields	*Perifoveal pigment granularity *Bulls eye maculopathy *Whitening of eyelashes *Subepi corneal deposits *↓ Corneal sensitivity *EOM Palsies	Total dose > 300 gm/day Rare at dosage <250gm/ day		Amslers Grid Kinetic & Static perimetry Ishiharas colour vision chart Testing with red objects, FFA, ERG	Toxic effects continues after stopping Drug *Baseline evaluation * Serial FFA (6-12m) * Fundus photo (monthly) *Central Field (Annual)	
3	Hydroxy Chloroquine (Plaquenil)	"	" less severe macular lesions; well tolerated by patients * Keratopathy	Dosages up to 400 mg/day		"	Toxic effects continue after stopping Drug Daily dose >3 mg/Kg-yearly check up Daily dose 6.5 mg/kg-6 monthly check up, Higher dosages, & > 6 years duration for therapy- frequent examination.	
4	Clofazimine	Asymptomatic	Corneal Whorl shaped lesions Macular pigmentary mottling (Bulls Eye) rarely			Slit lamp examination; Amslers grid, Fundus photography	Reversible	
5	Quinine Sulphate	*Blurring, * Loss of visual field, * Nyctalopia *Photophobia	Disc pallor, vascular alteration, Fine pigmentary mottling	4 gm/day	*Direct toxicity to ganglion cells, Retinal Pigment Epithelium & photoreceptors *Secondary Ischaemic changes from vaso constriction	Fundus Photography FFA	Toxic effects persists after stopping the drug	Haemodialysis Vasodilators
6	Ergot Alkaloids	*Acute visual loss	Vasoconstriction of retinal vessels, CME, CRVO Papillitis, optic disc pallor	Single dose	Vasoconstructive effect	Secondary ischaemic changes from Vaso constriction	Predisposition for toxicity in patients with pre existing vascular disease	
7	Oral Contra-ceptives	*Transient disturbance of colour vision: * Blurring	CME, retinal hemorrhages, CRVO, CRAO	If Oestrogen content higher	Thromboembolic phenomena	Avoid in patients with pre existing systemic vascular disease	Discontinue drug	Take OC with lesser oestrogen
8	Niacin (Nicotinic acid) (Rx of hypercholes terolemia	*Blurring worse on waking up *Metamorphopsia *Spokes around light	CME sparing foveola; miniature 'sun burst' wrinkling of inner retina	>3.0-5.0 gm / day	Direct toxic effect on muller cells → intracellular oedema	FP & FFA: (no leak) Amsler Grid	Discontinue, V_A returns to (normal) in 4-8 wks	

SYSTEMIC THERAPEUTIC AGENTS AND RETINAL TOXICITY (Contd.)

Sl. No.	Medication	Clinical Symptoms	Clinical Findings	Dosage	Mechanism of action	Test for early detection	Treatment	Prevention
9	Sulpha derivatives (anti biotics, diuretics)	*Blurring	Transient myopia 1-5 D	Accommodative Spasm * RR Lenticular oedema * Amsler CB oedema Peripheral serous CD			Reversible on discontinuing the drug	
10	Tetracyclines Promethazine Corticosteroids Ampicillin Acetaminophen	*Blurring of vision	Transient myopia 1-5 D				*Discontinue drug *Reversible	
11	Methoxyflurane (penthrane)	*Asymptomatic	Flecked retina syndrome	Prolonged anaesthetic administration	Secondary Hyper oxaluria	FP	Spontaneously reversible	
12	Tamoxifen	*Asymptomatic *Blurring	White refractive deposits at RPE level in perifoveal area	60-100 mg/day	Effect on lysosomal enzymes of RPE	Onset with in 12-18 months FP, FFA Amsler	Discontinue drug; changes reversible Baseline evaluation Rpt after starting therapy every 6 months	Dose 10-20 mg /day
14	Talc (in tablets crushed and used as intravenous Preparation)	D/V	Macular Ischaemia, Choroidal Infarction FB granuloma of retina, peripheral proliferative retinopathy	>1000 inj of any crushed tablet prep as intraveneous injection especially in drug abusers	Embolism	FP FFA	Discontinue drug Retinal changes will persist	? Role of laser photo coagulation for proliferative change
15	Chemotherapeutic Agents Chlorambucil Tilorone Intra Carotid Cisplatin	Visual *Blurring	Hemorrhage Retinopathy RPE Mottling Arteriolar narrowing Retinal and ON Ischaemia		Affects cells of retina with high Metabolic rate. ↓ Platelet count, vascular damage and Ischaemia ↓ DNA production	Monthly Fundus examination	Discontinue	
16	Cardiac Glycosides (CCF, Arrythmias)	*Blurred Vision *Scintillating Scotomas *Altered Colour Perception	Normal		Direct PR toxicity Altered cone mediated ERG response	Reversible	Discontinue Drug	
17	Rifabutin (Anti TB Chemotherapeutic agent) effective against Mycobacterium Avium Complex	Pain, D/V	Uveitis with Hypopyon	300-600 mg/day used along with Antifungal or Clarithromycin	S/L examination	Discontinue Reversible	Discontinue drug	
18	Methanol	Blindness	ON hyperemia Retinal Oedema	Minute dose	Formic Acid – direct effect on inner retina	Irreversible	Severe metabolic Acidosis-necessitating Haemodialysis	
19	Intranasal Coccaine And Amphetamine Abuse	D/V	CRAO Intraretinal hemorrhages	Single use	Severe Systemic Hypertension	Irreversible		
20	I/V Sympathomimmetics	D/V Paracentral Scotomas	Acute macular Neuro retinopathy	Single inj.	Direct interaction between adrenergic agent and photo receptor	Reversible	Reversible	

Author: Dr. Meena Chakrabarti
Editor, KJO

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 I" 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:-

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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 2 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.

c) 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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The Editorial Board of the Kerala Journal of Ophthalmology offers its token of gratitude to the following Ophthalmologists for the Peer Reviewing the Journal articles

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MICROSURGICAL VITRECTOMY 2000 P



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GALAXY COLD PHACO SYSTEM

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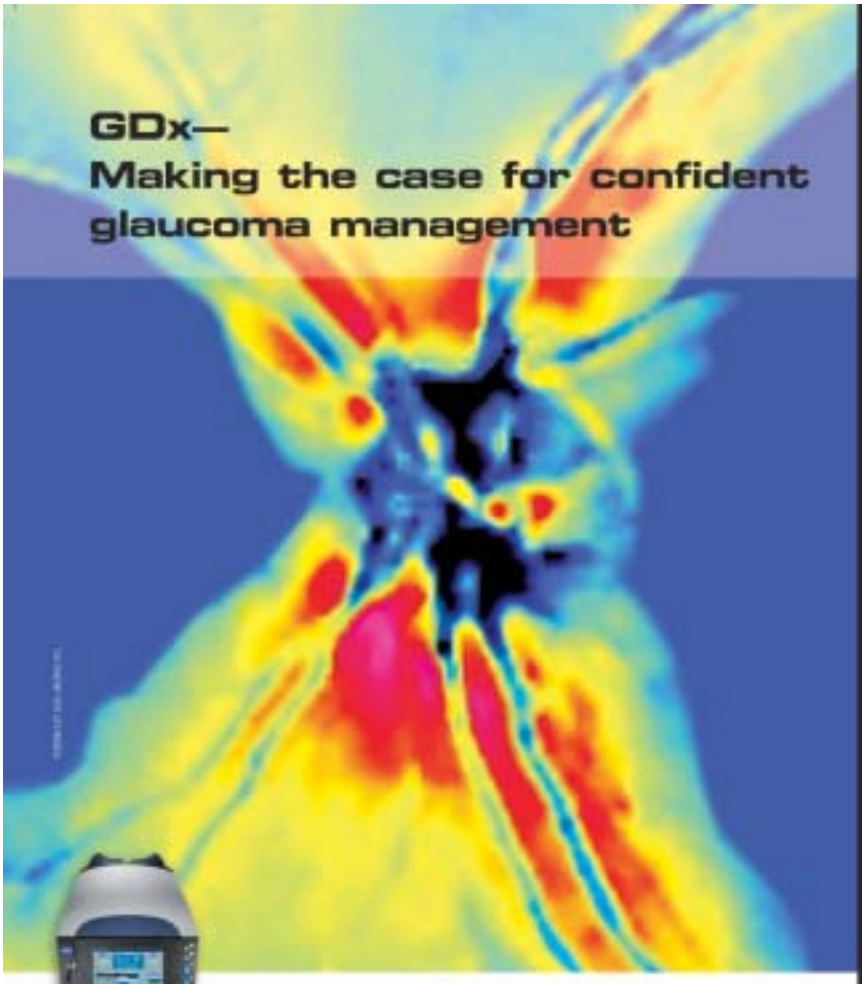
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To get the specifics on GDx and see compelling case studies, just visit **www.meditec.zeiss.com/GDx**. Or, contact your Carl Zeiss Meditec representative for more information. Because the answers are there.

Ocular hypertension or glaucoma: How can you be sure?



White male, age 56 IOP 30 mm Hg
Positive family history Thick cornea
Borderline visual fields



GDx deviation map compares RNFL to normative data; inferior defects are consistent with glaucomatous damage.

For further details, please contact :

Carl Zeiss India Private Limited

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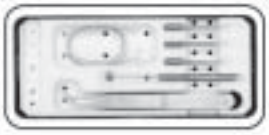
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VITRECTOMY SYSTEM
The High lights

ECKARDT 23 GAUGE VITRECTOMY SYSTEM & ACCESSORIES

23 GAUGE VITRECTOMY SYSTEM

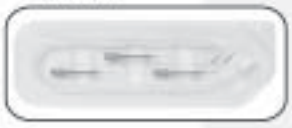


ECKARDT 23 GAUGE CANNULA SYSTEM CONSISTING OF:
- 3 X INSTRUMENT CANNULAS
- 2 X INFUSION CANNULAS
- 3 X BLUNT INSERTORS
- 3 X CLOSURE PLUGS
- STERILIZATION BOX

1272-23

ECKARDT 23 GAUGE CANNULA SYSTEM CONSISTING OF
• 2 X INSTRUMENT CANNULAS • 1 X INFUSION CANNULAS
• 3 X BLUNT INSERTORS • 2 X CLOSURE PLUGS • 1278 ECKARDT CANNULA FORCEPS
• 2115 PRESSURE PLATE STERILIZATION BOX

1272-ED06



DISPOSABLE ECKARDT 23 GAUGE CANNULA SYSTEM CONSISTING OF:
- 2 X INSTRUMENT CANNULAS
- 1 X INFUSION CANNULAS
- 3 X BLUNT INSERTORS
- 1 X INFUSION LINE
- 2 X CLOSURE PLUGS
- BOX/1 STERILE

1267-NMDO6



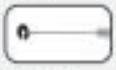
PNEUMATIC VITRECTOME, 23 GAUGE / 0.6 mm. FOR MOST VITRECTOMY UNITS WORKING WITH 30 PSI MAX 1500 CUTS/MIN (BOX/1,STERILE)

1278



ECKARDT 23 GAUGE / 0.6 mm. CANNULA FORCEPS

2115



ECKARDT PRESSURE PLATE

1272-E-DIS



DISPOSABLE ECKARDT 23 GAUGE VITRECTOMY KIT CONSISTING OF:
• 1272-ED-06 • 1267-NMD-06 • 3269-D-06 • BOX/1 STERILE

1281-A5D06



REPLACEMANT BACKFLUSH NEEDLE 23 GAUGE / 0.6 mm. (BOX/10,STERILE)

1281-C1



REPLACEMANT BACKFLUSH RESERVOIR (BOX/10)

1281-A506



REPLACEMANT BACKFLUSH NEEDLE STRAIGHT 23 GAUGE / 0.6 mm.

1281-B1D06



REPLACEMANT BACKFLUSH NEEDLE 23 GAUGE / 0.6 mm. (BOX/10,STERILE)

23 GAUGE REUSABLE MICRO-FORCEPS



1286-B06



END-GRIPPING FORCEPS, 23 GAUGE / 0.6 mm.

1286-C06



SERRATED JAWS FORCEPS, 23 GAUGE / 0.6 mm.

1286-WO6



ECKARDT END GRIPPING FORCEPS, 23 GAUGE / 0.6 mm.

2286-K06



ASYMMETRICAL ENDGRIPPING FORCEPS, 23 GAUGE / 0.6 mm.

23 GAUGE DISPOSABLE MICRO FORCEPS



1286-CD06



23 GAUGE / 0.6 mm. (BOX/1,STERILE)

1286-WD06



ECKARDT END-GRIPPING FORCEPS 23 GAUGE / 0.6 mm. (BOX/1,STERILE)

23 GAUGE REUSABLE MICRO-SCISSORS



1286-E06



VERTICAL SCISSORS, 23 GAUGE / 0.6 mm.

1286-J06



STRAIGHT SCISSORS, 23 GAUGE / 0.6 mm.

1286-M06



CUVERED HORIZENTAL SCISSORS, 23 GAUGE / 0.6 mm.

23 GAUGE DISPOSABLE MICRO-SCISSORS



1286-MD06

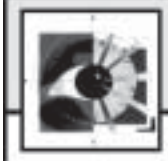


1281-C



ECKARDT 23 GAUGE / 0.6 mm. PASSIVE BACKFLUSH INSTRUMENT WITH EXTRA LARGE RESERVOIR FOR INCREASED BACKFLUSH CAPACITY.

CURVED SCISSIORS, HORIZENTAL 23 GAUGE / 0.6 mm. (BOX/5,STERILE)



VITRECTOMY SYSTEM
The High lights

DORC

1281-AD06



1281ADO6



BACKFLUSH INSTRUMENT WITH 23 GAUGE / 0.6 mm.BLUNT NEEDLE W/O ACTIVE ASPIRATION(BOX/1,STERILE)

1281-BT06



1281-BTD06



BRUSH BACKFLUSH INSTRUMENT WITH 23 GAUGE / 0.6 mm. BRUSH NEEDLE(BOX/1,STERILE)

23 GAUGE MEMBRANE INSTRUMENT

1292-E06





ECKARDT MEMBRANE PIC,23 GAUGE / 0.6 mm.

23 GAUGE DIAMOND DUSTED SWEEPER

1290-DSS06





DIAMOND DUSTED SWEEPER, 23 GAUGE / 0.6 mm.

1120-06 23 GAUGE DIATHERMY



ENDO DIATHERMY HANDLE WITH STRAIGHT TIP, 23 GAUGE / 0.6 mm.

1120-A

DIATHERMY CABLE FOR 1120-06

23 GAUGE SYSTEM ACCESSORIES FOR INTRAOCULAR ILLUMINATION

3269-E06



TOTAL VIEW ENDOILLUMINATION PROBE WITH ADJUSTABLE FIBER/ LIGHT INTENSITY 23 GAUGE / 0.6 mm.,INCLUDES COMBINED STANDARD AND TOTAL VIEW ENDOILLUMINATION TIP ILLUMINATED SPATULA PIC TIP (BOX/6, STERILE)



ALL FIBERS WITH TRIPLE-PORT CONNECTOR

3269-SB06



SPADE TOTAL VIEW ENDOILLUMINATION PROBE,23 GAUGE / 0.6mm.(BOX/6, STERILE)

3269-D06



ENDOILLUMINATION PROBE23 GAUGE / 0.6mm.(BOX/6, STERILE)

23 GAUGE MISCELLANEOUS

1279-P

DISPOSABLE REPLACEMENT TUBINGS (BOX/ 6,STERILE)

1279-R
DISPOSABLE REPLACEMENT TUBINGS VFI(BOX/ 6,STERILE)

EFD-100-06



EFTIAR DUAL BORE CANNULA, 23 GAUGE / 0.6 mm. (BOX/5,STERILE)

1272-E-RPP1

REUSABLE INSTRUMENT CANNULAS (SET/2)

1272-E-RPP2
REUSABLE INFUSION CANNULAS (SET/2)

1272-E-RPP3
REUSABLE CLOSURE PLUGS (SET/2)

1272-E-RPP4
REUSABLE BLUNT INSERTOR (SET/2)

1282-D06

45 BENT MVR BLADE, 23 GAUGE / 0.6 mm.(BOX/5,STERILE)

1282-E06
ILM MEMBRANCE PIC PRE-BENT 23 GAUGE / 0.6 mm.(BOX/5,STERILE)

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KERALA SOCIETY OF OPHTHALMIC SURGEONS
APPLICATION FOR LIFE MEMBERSHIP

1. Full name (in capitals).....
2. Father's/ Husband's name.....
3. Sex..... 4. Date of birth.....
5. Permanent address.....
.....
6. Mailing address.....
.....
7. Phone Nos. Hospitals.....Residence.....
8. Mobile No.....Fax No.....
9. E-mail Address.....
10. Qualifications

.....

Degree/Diploma Institution University Year passing

.....

Non-medical

.....

Medical

.....

Honorary

.....

11. Whether registered for basic qualification / speciality training Yes/No

if yes Number Date State Council

.....

12. Are you a member of All India Ophthalmological Society? Yes/No

if yes, quote AIOS number.....

13. Introduced by: Name and address of life member.....

.....Signature.....

I hereby apply for life membership of Kerala Society of Ophthalmic Surgeons and agree to abide by the rules and regulations of the Society.

Place : Signature of applicant

Date :

INSTRUCTIONS FOR APPLICANTS FOR KSOS MEMBERSHIP

1. Please enclose a Photostat copy of your Degree/Diploma certificate and Medical Registration Certificate.

2. Please send two stamp size photographs for issuing your photo identity card.

3. Life Membership Fee : Rs. 1000

   Reg.Fee : Rs. 50

   Bank commission for outstation cheques : Rs.50

4. Cheque/Demand draft is to be made in favour of Kerala Society of Ophthalmic Surgeons payable at Cochin.

5. The completed application form along with relevant certificates and Cheque/DD is to be sent to:

Dr. V. SAHASRANAMAM M.S, D.O
EYE SPECIALIST
No.30, VINAYAKA NAGAR
PAPPANAMCODE PO
TRIVANDRUM - 695 018
PH:- 0471-2490421

Receipt No..... Membership No.....

Date.....

ALL INDIA OPHTHALMOLOGICAL SOCIETY

Membership Application Form

NAME …….. ……..

(in Block Letters, please furnish first name under which you wish to be registered and then the other name)

(Enter in the box the alphabet under which your name should be indexed) ☐

DATE OF BIRTH:……..……AGE: ……..

ADDRESS: ……..……

(in block letters)……..……

STATE: ……..……PIN CODE: ……..

PRESENT STATUS: ……..……

	Qualifications	University	Year
1.			
2.			
3.			

Registration No. & State in which registered ……..……..

Have you been a member of this Society before ? Yes / No

If Yes (Furnish details) ……..……

Proposed by ……..

(Name)

Signature.....

Membership No. ……..…… (Life Member / Annual Member)

Seconded by ……..……

(Name)

Signature.....

Membership No. ……..…… (Life Member / Annual Member)

I wish to be a Life Member/Life Member on instalment basis.

Declaration by applicant: I declare that the above details are correct. I have read the instructions overleaf. I shall abide by the Rules & Regulations of the Society in force and any subsequent amendments made from time to time.

I am enclosing Cash/Bank Draft No…..……..……on ……..Bank

Dated……..……for Rs. ……..……..

Dated …….. Signature……..……

FOR OFFICE USE:

The Above application is in order. His/her application is to be put before the next Meeting of Managing Committee/General Body.

Date: *Hony. Gen. Secretary*

ALL INDIA OPHTHALMOLOGICAL SOCIETY

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All India Institute of Medical Sciences
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Date of Birth: Day: Month: Year:

Please tick(✓) to which of the following super-specialities you are most interested.
Do not tick more than two. If you are interested in more than two, please tick No.1

1. General Ophthalmology
2. Oculo Plastic Surgery
3. Refractive Corneal Surgery
4. Corneal Surgery
5. Anterior Segment Surgery
6. Intra Ocular Implant Surgery
7. Uveitis
8. Glaucoma
9. Vitreo-Retinal Surgery
10. Neuro Ophthalmology
11. Strabismus
12. Paediatric Ophthalmology
13. Contact Lens
14. Ocular Pathology
15. Any Other

----------CUT HERE----------CUT HERE----------

SPECIMEN SIGNATURES OF APPLICANT

MEMBERSHIP NO. ……..……...

SPECIMEN SIGNATURES 1. ……..……..……. 2. ……..……..……..

You Belong to: i) Ophthalmology
ii) Any other branch of medicine

Full Qualifications:
(If Post-Graduate student, mention that also)

Are you practising General medicine alongwith Ophthalmology? Yes/No

Are you in
- i) Private Practice
- ii) Serving in Private institution
- iii) Serving in Autonomous institution
- iv) Govt. Service
  - a) Non-teaching without practice.
  - b) Teaching without practice
  - c) Non teaching with practice
  - d) Teaching with practice
- v) Not covered by any of the above

You are
- i) Honorary Member
- ii) Full fledged Life member
- iii) Life member on instalments & have paid
  - a) 1st Instalment
  - b) 2nd Instalment
- iv) Annual Member & Subscription paid upto ……

(PLEASE FILL UP COMPLETELY AND SEND IT)