The Kerala Journal of Ophthalmology is the official scientific publication of the Kerala Society of Ophthalmic Surgeons and 4 issues are published every year.

It welcomes original articles, interesting case reports, update articles, book reviews, journal abstracts and research papers in Ophthalmology. Dates of the upcoming conferences and CME’s are also published. Original articles are accepted on condition that they have not been published in any other journal.

SUBSCRIPTION RATE

Annual : Rs. 600 (4 issues)
Single Copy : Rs. 150

Subscription should be sent by demand draft in favour of Kerala Journal of Ophthalmology payable at Trivandrum addressed to the Editor, KJO
KERALA SOCIETY OF OPHTHALMIC SURGEONS
(Registered under Societies Registration XXI of 1860. No.387/2003)

President
Dr. R.R. Varma
Ambikalayam, Warriam Road
Kochi - 682 016
Ph: 0484-2352010 (R)
Mob: 94471 52010

General Secretary
Dr. Sasikumar
Ambadi, Adyath Lane,
Ravipuram, Kochi 680216
Ph: 0484-2357135 (H)
Mob: 9447475101

Treasurer
Dr. Radha Ramanan
LF Hospital, Angamaly
Ernakulam
Ph: 0484 2452546 (H)
Mob: 9447006421

President Elect
Dr. B.V. Bhat
Asoka Hospital
South Bazar
Kannur
Ph: 9846139715

Vice President
Dr. A. Giridhar
Giridhar Eye Institute
Ponneth Temple Road
Kadavanthara, Kochi - 682 020
Ph: 9895377899

Scientific Committee Chairman
Dr. Sai Kumar S.J.
Giridhar Eye Institute
Kochi - 682 020
Ph: 0484-2312303 (H)
Mob: 98470 40840

Joint Secretary
Dr. Arup Chakrabarti
Chakrabarti Eye Care Centre
Kochulloor, Trivandrum 695 011
Ph: 0471-2555530
Mob: 9946410540

Immediate Past President
Dr. P. Rajagopalan Nair
Raj Bhavan
Palakkad - 676 013
Ph: 0491-2535676 (R)
Mob: 94476 45676

Immediate Past Secretary
Dr. Sahasranamam
No. 30, Vinayaka Nagar
Trivandrum 695 018
Ph: 0484-2490421 (R)
Mob: 9846020421

Managing Committee Members
Dr. Anthrayose Kakkanat
Dr. Meena Chakrabarti

Executive Committee Members
Dr. Suresh Babu
Kasargode
Dr. P.P. Kunhiraman
Kannur
Dr. Baburaj N.P.
Kozhikode
Dr. Mohammed Swadique
Malappuram

Web Site Editor
Dr. Thomas George
RIO, Red Cross Road
Trivandrum - 695 035
Mob: 93481 18711

Journal Editor
Dr. Meena Chakrabarti
Chakrabarti Eye Care Centre
Kochulloor, Trivandrum – 695 011
Ph: 0471-2555530
Mob: 9946410541

Immediate Past Secretary
Dr. Sahasranamam
No. 30, Vinayaka Nagar
Trivandrum 695 018
Ph: 0484-2490421 (R)
Mob: 9846020421

Managing Committee Members
Dr. Anthrayose Kakkanat
Dr. Meena Chakrabarti

Executive Committee Members
Dr. Suresh Babu
Kasargode
Dr. P.P. Kunhiraman
Kannur
Dr. Baburaj N.P.
Kozhikode
Dr. Mohammed Swadique
Malappuram

Dr. Rajesh Radhakrishnan
Palakkad
Dr. Babu Krishnakumar
Thrissur
Dr. Davis Akkara
Ernakulam
Dr. C.K. Mathew
Alapuzha
Dr. Varghese Joseph
Pathanamthitta
Dr. Seshadrinathan
Kollam
Dr. Biju John
Trivandrum

Immediate Past Secretary
Dr. Sahasranamam
No. 30, Vinayaka Nagar
Trivandrum 695 018
Ph: 0484-2490421 (R)
Mob: 9846020421

Managing Committee Members
Dr. Anthrayose Kakkanat
Dr. Meena Chakrabarti

Executive Committee Members
Dr. Suresh Babu
Kasargode
Dr. P.P. Kunhiraman
Kannur
Dr. Baburaj N.P.
Kozhikode
Dr. Mohammed Swadique
Malappuram

Dr. Rajesh Radhakrishnan
Palakkad
Dr. Babu Krishnakumar
Thrissur
Dr. Davis Akkara
Ernakulam
Dr. C.K. Mathew
Alapuzha
Dr. Varghese Joseph
Pathanamthitta
Dr. Seshadrinathan
Kollam
Dr. Biju John
Trivandrum
EDITORIAL
5
Human Error: Can Ophthalmic Surgery be Made Fool Proof?

MAJOR REVIEW
7
Management Strategies in Diabetic Macular Edema
Dr. Meena Chakrabarti, Dr. Valsa Stephen, Dr. Sonia Rani John, Dr. Arup Chakrabarti

ORIGINAL ARTICLE
25
Closed Globe Injuries - A Tertiary Care Experience
Dr. Raju KV, Dr. Nima CA, Dr. Anju AK
31
Visual and Anatomical Outcomes of Vitreous Surgery for Large Macular Holes
Dr. Mahesh G., Dr. A. Giridhar, Dr. Ramkumar, Dr. Alpesh Rajput
36
Population Based Assessment of Diabetes and Diabetic Retinopathy in South Kerala- Project Trinetra: An Interim Report
Dr. Manoj Soman, Dr. Unni Nair, Dr. Sheena Bilal, Dr. Raeba Mathew, Dr. Fasil Gafoor, Dr. KGR Nair
42
Awareness on Common Blinding Conditions – A Population Based Survey
Dr. Rehna Rasheed, Dr. Cini N V, Dr. Reena A
47
Comparison of Visual Field Defects in Normal-Tension Glaucoma (NTG) and Primary Open Angle Glaucoma (POAG)
Dr. Vijaya Pai H., Dr. Himabindu Veluri

OCULAR PHARMACOLOGY
51
Intravitreal Inserts
Dr. Sonia Rani John, Dr. Meena Chakrabarti, Dr. Valsa Stephen, Dr. Arup Chakrabarti

OPHTHALMIC INSTRUMENTATION
55
Fundus Autofluorescence Imaging
Dr. Meena Chakrabarti

OPHTHALMIC SURGERY
62
Phacoemulsification in Post Vitrectomy Cataracts
Arup Chakrabarti, Meena Chakrabarti, Valsa Stephen, Sonia Rani John

CURRENT CONCEPTS
69
Anterior Lamellar Corneal Replacement
Dr. Sreenivas K. Rao

CASE REPORTS
74
Solar Maculopathy- An OCT Study
Dr. Manoj Soman, Dr. Fazil Gafoor
77
Diffuse Unilateral Subacute Neuroretinitis: A Case Report
Sony Siraj E., Reena A., Thomas George
Pneumatic Displacement of Submacular Hemorrhage Combined with Intravitreal Bevacizumab Injection – An Effective Combination

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

Central Retinal Artery Occlusion following Macular Hole Surgery

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

COMMUNITY OPHTHALMOLOGY

Artificial Vision

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

PHOTO ESSAY

Asteroid Hyalosis and Diabetic Retinopathy

Dr. Meena Chakrabarti, Dr. Sonia Rani John

CONSULTATION SECTION

JOURNAL REVIEW

BOOK REVIEW

CME PROGRAMMES

PG TEAR SHEET

OPHTHALMIC HISTORY

INSTRUCTIONS TO AUTHORS
Human Error: Can Ophthalmic Surgery be Made Fool Proof?

Even when all loop holes are plugged in a systematic manner, an element of human error, ever present in whatever we do, can still result in disastrous acts of omission or commission. Especially in the field of ophthalmic surgery even if they cause little or no permanent injury to the patient, the consequences for the physician and the profession may be very serious.

Human errors during ophthalmic surgery, can be categorized into one of the 5 main categories: wrong implant, wrong eye block, wrong patient, wrong procedure or wrong transplant tissue.

Although the incidence of error is relatively low - an average of 69 cases per 1 million - this number is still 10 times the quality - defect standard accepted by the manufacturing industry according to a study published in Archives of Ophthalmology in November 2007.

The most common error is the implantation of wrong IOL and the problem can be due to wrong scan or intraoperative causes. The cause in almost every intraoperative case is failure to check the lens specification properly before implantation. Typically the circulating nurse would have pulled out the wrong lens and its parameters were not verified either by the surgeon or the scrub before implantation. In some cases intraoperative confusion is due to the surgical staff having problems reading the implant package (we are all well aware of the desire for perpetual youth and the willful denial of presbyopia).

Blocking the wrong eye can be avoided if we follow the protocol of site marking of the eye to be operated. In these cases if the human error is understood before more damage is done, the effect of the block will wear off with return to normalcy.

Operating on the wrong patient or performing the wrong procedure occur mainly due to staff confusion in identifying the patient as well as patient confusion.

The medical profession need to have a more open and proactive attitude about medical errors. Defensiveness and veil of secrecy encouraged by the authorities at the hospitals interfere with doing something intelligent. Ironically it is not inexperienced surgeons who make these errors. Typically these errors occur at busy surgical centres or in situations where more people are involved.

Factors such as switched schedules, distracted and inexperienced or new personnel inadequate preoperative verification procedures, lack of site marking and no communication between patient and surgeon are the main causes identified.
Another unusual dilemma is a confused patient who is not sure which eye is to be operated or a patient who has a right–left disorientation and will say the ‘right’ eye while pointing to the left eye.

In our busy surgical schedule ‘site marking’ helps cut down the risk of human error to a very large extent. We should also take pains and time off to explain the pre-op IOL selection procedure to the OR staff who will be helping us out in the operation theatre. A protocol for perioperative preparation is also mandatory to avoid these errors.

Planning and proper precautions are necessary before any surgical procedure. Even though your case may hold water as unintentional excusable human error in a court of law in the event of a litigation, you can rest assured no one, least of all your patient will ever forgive or forget.

Dr. Meena Chakrabarti MS DO DNB
Editor, IJO
Management Strategies in Diabetic Macular Edema

Diabetic Macular Edema (DME), a micro vascular complication of diabetes mellitus accounts for three quarter of cases of moderate visual loss due to diabetic eye disease. This entity represents a significant burden with increasing incidence and prevalence of diabetes in the Indian subcontinent, and availability of limited public health resources. In chronic poorly controlled diabetics with associated co-morbid conditions such as dyslipidemias and overt nephropathy, the edema at the macula tends to be more severe, and is characterized by the accumulation of plaques of hard exudates under the fovea, exhibiting poor response to conventional therapies and showing a tendency to becoming recalcitrant. In this situation the patient is burdened by severe visual loss, with very few management options and a poor visual prognosis. Yet DME still remains an underestimated complication of diabetes primarily due to lack of awareness among the patients and primary care physicians alike.

Diabetic retinopathy is the leading cause of blindness in patients aged between 20 and 74 years in the developed world. The Wisconsin Epidemiological study (WESDR) reported that in patients with type I DM < 5 years duration, the incidence of diabetic macular edema was nil when compared to a 32 % incidence in type I DM patients whose diabetic age exceeded 20 years.

A genetic predisposition for the development of diabetic retinopathy has been found in certain studies. A polymorphism in the aldose reductase gene has been found to be associated with an increased risk of diabetic retinopathy and other micro vascular complications after controlling all independent risk factors. In contrast there are other individuals with very long duration of diabetes, and, despite mediocre diabetic control, do not develop retinopathy indicating the presence of an unknown protective factor.

Depending on the type of diabetes, the mode of treatment and the duration of disease, significant variation in the incidence and prevalence of diabetic macular edema have been reported in several epidemiological studies. As a general rule, the lifetime risk of developing diabetic retinopathy with sight threatening complications like DME and PDR is 50 % for a patient with Type I DM and one in three for a patient with Type II DM.
In view of the rapidly increasing diabetic population in the developed and developing world, a dramatic increase in patients with diabetic macular edema is anticipated in the years to come. The reduced quality of life and dependency of patients with diabetic macular edema makes early screening and initiation of therapy very vital.

**Pathophysiology of DME :**

Vascular endothelial damage has a major contribution to the development of diabetic macular edema. The resultant breakdown of the inner blood retinal barrier causes accumulation of fluids and serum macromolecules in the intercellular space. The microvascular damage is thought to be a consequence of loss of capillary pericytes, proliferation of endothelial cells and out pouching of the vessel wall. (Fig 1)

**Grading of DME**

Diabetic macular edema is defined as retinal thickening due to fluid leakage and pooling in the macular area. The International DME severity scale grades DME based on its severity into mild, moderate and severe. (Fig: 2, 3, 4)

**Clinically significant diabetic macular edema:**

The ETDRS was one of the first large clinical trials sponsored by the National Eye Institute and this study coined the term ‘clinically significant macular edema (CSME) for edema that threatens or involves the centre of the fovea. Thus CSME includes

1. Thickening of retina at or with in 500 μm of the centre of the macula. (Fig 5).
2. Hard exudates at or with in 500 μm of the centre of macula, if associated with thickening of the retina.
3. A zone or zones of retinal thickening one disc area or larger, any part of which is within one disc diameter of the centre of the macula.

**Types of Diabetic Macular Edema**

Diabetic Macular Edema can be clinically divided into focal, diffuse, mixed, cystoid and ischemic.

**FOCAL DIABETIC MACULAR EDEMA**

In focal diabetic macular edema there are localized areas of retinal thickening caused by focal leakage from retinal capillaries or micro aneurysms. These areas are frequently demarcated by partial or complete ring of hard exudates called circinate rings. (Fig 6)
**Diffuse Diabetic Macular Edema:**
In diffuse diabetic macular edema there is more widespread thickening of the macula secondary to generalized abnormal permeability of the retinal capillaries. This generalized leakage is thought to be the result of compensatory dilation of parts of the perifoveal capillary net in response to occlusion of neighboring parts of the capillary bed. (Fig 7)

Risk factor for progression of diffuse macular edema are increasing micro aneurysm count, adult onset diabetes, hypertension, cardiovascular disease, vitreo macular adhesion and advanced retinopathy.

In clinical practice however a variety of mixed forms may be encountered.

**Cystoid Macular Edema:** is characterized by the presence of cystoid spaces filled with an ophthalmoscopically clear fluid. This is not pathognomonic of diabetic macular edema but may be associated with various other conditions. The presence of an intact attached and thickened posterior hyaloid plays a role in its pathogenesis. The presence or absence of this entity does not influence either the prognosis or management.

**Ischemic Macular Edema:** The nonperfused or the ischemic variant of diabetic macular edema is characterized by very poor central visual acuity, grossly edematous macula, and fluorescein angiographic evidence of enlarged foveal avascular zone (FAZ) and occlusion in the capillary bed of the perifoveal network. Only fluorescein angiography can document the degree of ischemia and the location of the capillary non perfusion (CNP) areas.

**Diagnosis and screening**
Diabetic macular edema in its early stages does not cause loss of visual function. The goal of therapy is rarely improvement of vision; on the contrary, in most cases, it is preservation of vision and avoidance of further damage. Hence it is necessary to detect retinopathy before visual loss and irreversible changes set in.

There is controversy regarding the modality of screening to be employed, to screen regularly the millions of diabetic patients. Though dilated fundus examination is highly sensitive and specific; it imposes a huge work load and requires a high density of qualified and trained health care providers. However a binocular stereoscopic slit lamp biomicroscopy still remains the most important diagnostic tool. In a remote area this method of detection may not be feasible. Fluorescein angiography is helpful in detecting leaking points and ischemia prior to treatment but being an invasive intervention it is not appropriate for routine screening.

Optical coherence tomography allows a non invasive quantitative and examiner independent evaluation of macular edema as well as assessment of the presence or absence of vitreo macular traction. However being an expensive modality it cannot be used for screening.

Developments of screening technique using telemedicine in remote areas have generated a lot of interest in digital fundus photography with automated grading of diabetic changes. (e.g.: micro aneurysm counting)

Although screening and early detection is vital, it does not replace a formal eye examination with patient counseling.

**Imaging in the Quantification of DME**
A slit lamp stereoscopic examination is all that is necessary in the routine diagnosis of diabetic macular edema. However further imaging studies are necessary for the following reasons:

1. To grade the severity of the macular edema.
2. To assess the presence of macular edema and the integrity of the perifoveal capillary network.
3. As a guide to decide on the modality of treatment for a specific given patient.
4. To assess the efficacy of treatment modality employed and to decide on retreatment.
5. To follow up patients with non significant DME.
6. To screen patients for diabetic retinopathy (rarely done)

The imaging modalities used to quantify diabetic macular edema includes 1) Colour fundus photographs: 30° and 50° stereo photographs 2) Fluorescein fundus
FFA has the advantage of being the only imaging modality that can assess the integrity of the perifoveal capillary net, localize areas of capillary non perfusion as well as areas of leakage of the dye. The pattern of dye leakage in a fluorescein angiogram helps to differentiate between focal, diffuse, mixed, ischemic, and cystoid varieties of diabetic macular edema. (Fig 10 a, b, c)

The earliest fluorescein angiographic changes in diabetic retinopathy in the presence of capillary microaneurysms (Fig 11). Other angiographic changes include the presence of leakage at the macula; development of capillary non perfusion areas and widening of FAZ, intraretinal micro vascular anomalies (Fig.12); neovascular fronds on the disc (NVD) and else where in the retina (NVE).

Fluorescein fundus angiography also helps to identify presence of associated lesions at the macula such as idiopathic parafoveal telangiectasia, macular drusen etc (Fig 13)

The disadvantages of relying on fluorescein angiographic findings alone are its inability to quantify the severity of diabetic macular edema and also the amount of leakage does not correlate with the degree of retinal thickening. The presence of serious side effects and its interventional nature also prevents FFA from being accepted as a 'stand alone' diagnostic and screening tool.

OCT has the added advantage of being able to reveal the presence of cystoid macular edema, sub foveal serous retinal detachment, presence of vitreomacular traction or an epiretinal membrane, which cannot be detected in a fluorescein angiogram. Moreover the macular thickness map gives us as very accurate idea of the central retinal thickness and can quantify the degree of improvement or worsening following therapy.

Several different patterns of structural changes have been demonstrated with in the retina in diabetic macular edema. These include 1. Sponge like retinal thickening (Fig 14 ) 2. Cystoid macular edema (Fig 14 b) 3. Sub foveal sensory retinal detachment (Fig 14 c) 4. Taut attached posterior hyaloid phase (Fig 14 d) 5. Presence of vitreomacular traction (Fig 14 e): and screen effect produced by a plaque of hard exudates (Fig. 14 f).

Thus OCT separates cases of diabetic macular edema with vitreo retinal interface abnormalities (such as vitreomacular traction, coincident epiretinal membrane and taut internal limiting membrane) and helps us to understand why these eyes respond poorly to pharmacological and laser therapies. It helps to selectively identify cases of diabetic macular edema, which needs surgical intervention.

Based on the central retinal thickness in the optical coherence tomogram, macular edema is classified into mild, moderate and severe.

- ≤ 200 μm: Normal
- 201 μm- 300 μm: Mild thickening
- 301 μm- 400 μm: Moderate thickening
- ≥ 400 μm: Severe thickening

Treatment Options

Treatment of diabetic macular edema combines the optimization of systemic risk factors with systemic and local pharmacological treatment, as well as laser intervention and surgical approaches. Ophthalmologists may play a key role in patient motivation, and effective co operation with the general practioners, primary care physicians, and endocrinologists is essential.

Systemic Treatment: - Effective treatment for diabetic macular edema is not possible unless all the components of the metabolic syndrome are kept under rigid control (Fig 15)
Systemic treatment for diabetic macular edema includes

1. Glycemic control
2. Blood pressure control
3. Reducing levels of blood lipids
4. Treatment of renal dysfunction
5. Systemic pharmacotherapy
   1) PKC-β inhibitors
   2) Aldose reductace and AGE Inhibitors
   3) Antioxidants

**Glycemic Control:**

Tight blood glucose control is essential for prevention of end organ damage and complications including diabetic retinopathy and macular edema. The WESDR showed a strong relationship between baseline glycated hemoglobin (Hb A1C) and the incidence of macular edema over a ten-year period. The diabetic control and complications trial (DCCT) demonstrated a 26% reduction in the risk of developing macular edema in the intensive insulin treatment group as compared with the conventional treatment group in patients with type I diabetes. Although tight glycemic control is strongly recommended for all patients with diabetes, there is no specific HbA1C value above which the risk of developing diabetic retinopathy increases. Since very rigid control in chronic diabetics carries the risk of hypoglycemic complications, an HbA1C level of 7.0% is the highest level recommended by the American Diabetes Association guidelines. In ETDRS there was a positive correlation between elevated lipid levels and an increased risk of developing hard exudates and decreased visual acuity.

**Blood pressure control**

- WESDR results have conclusively shown that in patients with type 1 DM; and a diastolic blood pressure within the fourth quartile range had a 3.3 fold increased risk of developing macular edema compared to patients with diastolic BP within the first quartile range.
- UKPDS (The UK Prospective Diabetic Study) reported a 47% reduction in the loss of three or more lines of visual acuity associated with a tight blood pressure control in patients with Type II diabetes.
- The ABCD study (The Appropriate Blood Pressure Control In Diabetes) failed to demonstrate any significant effect of intensified BP control in patients with Type II DM. This could be attributed to the lower blood pressure at baseline and poorer glycemic control in the study population of ABCD study.

**Reducing Levels of blood lipids.**

- In ETDRS there was a positive correlation between elevated lipid levels and an increased risk of developing hard exudates and decreased visual acuity.
- The DCCT show a predictive value of total cholesterol: HDL cholesterol ratio and LDL for development of clinically significant diabetic macular edema.
- However there is no concrete evidence on the efficacy of lipid lowering agents in preventing the accumulation of hard exudates at the macula.

**TREATMENT OF RENAL DYSFUNCTION AND ANAEMIA** have resulted in improvement in the diabetics retinopathy status although there are no definite controlled trials supporting these anecdotal observations.
**SYSTEMIC PHARMACOTHERAPY:** includes the use of
1. Oral PKC-β Inhibitors (Ruboxistaurine Mesylate)
2. Aldose reductase inhibitors
3. Advanced Glycation End Product (AGE) inhibitors
4. Antioxidants.

**Oral PKC-β Inhibitor:**

Proteinkinase C includes a group of iso enzymes involved in intracellular signal transduction. This isoenzyme gets activated in response to a number of stimuli, an important one being hyperglycemia. Activation of PKC-β results in endothelial cell proliferation as well as an increased vascular permeability both of which are responsible for the neovascular as well as macular complications of diabetic retinopathy. In addition, PKC-β is involved in the upregulation of vascular endothelial growth factor (VEGF). Administration of PKC-β inhibitor has a definite effect on suppressing the VEGF-induced leakage from the retinal blood vessels. The PKC-DRS2 study results show that treatment with Ruboxistaurin was associated with

1. Reduced need for initial focal photocoagulation (26% reduction)
2. Reduced visual loss in eyes that required focal photocoagulation (40% reduction of visual loss)
3. Reduced progression of diabetic macular edema to within 100 μm of the centre of fovea (26% reduction)
4. Increased chance of moderate visual gain

However, this study did not achieve its primary endpoint. Further trials are underway to study the efficacy of this drug in preventing visual loss in diabetic patients.

**Aldose Reductase and AGE Inhibitors**

The presence of hyperglycemia causes increase in the activity of the polyol pathway resulting in an accumulation of sorbitol. This build-up of intracellular sorbitol results in cellular damage. Clinical trials using aldose reductase inhibitors (to inhibit the endothelial aldose reductase activity) claim to have reduced the number of non-perfused capillaries, fluorescein leakage, and microaneurysm count. But there has been no documented effect on the progression of diabetic retinopathy.

Increased formation of advanced glycosylation end products (AGE) in diabetes have been proposed as another mechanism causing endothelial cell damage. In animal models, the AGE inhibitor, amino guanidine, effectively inhibited the development of diabetic retinopathy. Clinical trials with a novel AGE inhibitor has been successful in animal models but its efficacy in treating people with diabetes has not been proved.

**ANTIOXIDANTS**

Hyperglycemia is followed by increased production of free radicals (Reactive Oxygen species) by various mechanisms. These radicals play a key role in the development of microvascular complications of diabetes. Hence reduction of free radicals should have a beneficial effect on diabetic retinopathy.

Calcium dobesilate is a potent antioxidant registered for treatment of diabetic retinopathy in more than 20 countries. It has been shown to have a beneficial effect on vascular permeability and erythrocyte membrane properties in vitro and in animal models. Its beneficial effects clinically have not yet been conclusively proved.

**Laser Photocoagulation**

Laser PHC has been the sole modality of therapy for diabetic macular edema and even now despite the availability of vitreous surgery and intravitreal pharmacotherapy; it still remains the gold standard of treatment.

The Early Treatment Diabetic Retinopathy Study, a multicentered randomized clinical trial (1980-1989) sponsored by the National Eye Institute clearly demonstrated the effect of laser treatment in patients with diabetic macular edema, and also defined the term ‘clinically significant macular edema’ (CSME). The ETDRS showed that the risk of moderate visual loss (3 or more lines on a LOGMAR chart) was reduced by 50% in eyes treated with immediate laser photocoagulation, compared to eyes in the deferral group (15% Vs 32%) (Fig 16).
Although this study was completed almost 20 years ago, the principles of laser treatment for diabetic retinopathy and macular edema is still based on its guidelines.

**Focal Laser Photocoagulation:**

The goal of focal laser photocoagulation is to seal focal leaking micro aneurysm. The preferred end point is to obtain a darkening or whitening of the micro aneurysm. Spot size 50-200 μm/0.1s/100-200 mw is the recommended laser parameters (Fig.17). However in treating micro aneurysms close to the fovea, a spot size of 50 μm / 0.055 s / 100 mw is used. Clumps of micro aneurysms close together can be treated by a larger spot size if away from fovea. The modified ETDRS focal photocoagulation protocol recommends direct treatment of micro aneurysms and grid photocoagulation to other areas of thickening.

**GRID LASER PHOTOCOAGULATION** is applied to areas of thickened retina showing diffuse fluorescein leakage and / or capillary dropout with any associated focal leak treated as outlined above. Retinal leakage or capillary dropout identified by fluorescein fundus angiography are treated with 100-200 μm burns of light intensity with constant attention to energy levels, as the uptake in these areas can be variable. At least one burn width is spaced between burns. Areas of intense leakage are treated with grid spots one burn width apart, while areas of less intense leakage are treated by more widely spaced burns. The grid is placed within 500 μm of the disc margin, but can be placed on the papillo macular bundle. It can extend in all directions up to 2 DD from the centre of fovea, or up to the border of the PRP treatment (Fig.18).

**Micropulsed, Sub Threshold Selective Laser Therapy:**

Histological studies have shown that there may be a full thickness retinal reaction even to barely visible laser burns. In clinical pilot studies sub threshold laser coagulation (Using a green Nd: YLF micro pulsed laser) was effective in eyes with diabetic macular edema, while minimizing chorioretinal damage.

Laser photocoagulation of the macula is inherently destructive and creates small areas of irreversible damage to the retinal tissue. Furthermore, enlargement of the laser scars with progression into the central foveal area and subsequent loss of vision has been reported.

Currently much lighter intensities are used in order to obtain a barely discernible reaction of the RPE. For very central leaks observation combined with improved systemic disease control are preferred and treatment at the margin of the FAZ is avoided. If there are no appropriate targets for foveal laser photocoagulation demonstrated by FFA, laser PHC is usually deferred and other modalities of therapy suggested to the patient.

**Intravitreal Steroids:** - The role of steroids is mediated through 1) Suppression of VEGF 2) Stabilizing the leakage from retinal vessels 3) Suppression of the release of endothelial cell activators and 4) Possibly its anti-inflammatory action.

Initially uncontrolled interventional case series reported an unprecedented efficacy of intravitreal steroids (usually triamcinolone acetonide) in reducing diabetic macular edema accompanied by significant improvement in visual acuity. These uncontrolled series were followed by randomized placebo controlled trials demonstrating the efficacy of IVTA compared with standards care on both short and long term.

Several studies in eyes with persistent DME despite focal and / or grid laser photocoagulation have demonstrated the efficacy of IVTA over laser.

Although most studies have shown a beneficial effect the optional dosage of IVTA is still somewhat confusing. Audren F et al conducted a randomized prospective trial comparing the efficacy of 2 mg vs. 4 mg of Triamcinolone acetonide in the management of diffuse diabetic macular edema. Their results showed that there was no dose dependent difference in the response to intervention. However Lam DS et al and Spandau UH et al demonstrated dose dependency in the response to intravitreal injection of triamcinolone acetonide.

The high incidence of steroid related adverse effects such as (1) necessity for cataract extraction in 54 % of
phakic treated eyes (2) steroid related elevation of IOP in 44 % of treated eyes necessitates the use of caution. In order to avoid the adverse effect associated with intravitreal therapy, particularly infectious endophthalmitis, the use of periocular steroids in the management of diabetic macular edema has been studied 58, 59. The results of these trials have been contradictory to each other showing either a beneficial effect or no appreciable effect of the intervention on DME.

**Intravitreal Anti VEGF Antibodies: Bevacizumab and Ranizumab**

Vascular endothelial growth factor (VEGF) has been shown to be an endothelial cell-specific mitogen and an angiogenic inducer in a variety of in vitro and vivo models. VEGF; also known as vascular permeability factor; has been demonstrated to increase retinal vessel permeability by increasing the phos-phorylation of tight junction proteins. All variants of VEGF (particularly VEGF-A) have been implicated in the occurrence of increased vascular permeability by affecting endothelial tight-junction proteins in ocular vascular diseases such as diabetic macular edema (DME). VEGF-Levels are considerably higher in DME patients with extensive leakage in the macular region than in the patients with minimal leakage. Recent work has found elevated levels of VEGF in ocular fluids of patients with proliferative diabetic retinopathy (PDR). These studies also found that the growth of new vessels from the retina or optic nerve was thought to occur as a result of VEGF release into the vitreous cavity as a response to ischemia.

**Avastin in the management of DME**

Studies have demonstrated the usefulness of an intravitreal injection of Bevacizumab with promising effects in the reduction of macular edema secondary to central retinal vein occlusion, vascular permeability and fibro vascular proliferation in retinal neovascularisation secondary to PDR, rubeosis iridis, retinopathy of prematurity, and choroidal neovascularization secondary to AMD, and in the treatment of DME.

The use of Anti – VEGF drug is becoming increasingly more prevalent; however some unresolved issues such as the ideal regimen, duration of treatment, potential of combination treatments, and safety concerns with long term VEGF inhibition deserve further investigations.

Investigators continue to report their experience with intravitreal injections of Bevacizumab, a humanized monoclonal Ig G antibody directed against all five VEGF isoforms, in the setting of primary therapy. In a study of 51 patients, Haritoglou et al. observed that at 6 weeks after a single Bevacizumab injection, patients with DME resistant to other therapies had increased visual acuity as well as decreased central retinal thickness by OCT relative to pre-injection baseline, though the effect on visual acuity was not sustained at 12 weeks 60. The Pan – American Collaborative Retina Study (PACORES) Group studied intravitreal Bevacizumab as a primary treatment for DME in 78 eyes of 64 patients and found, at six months, over 96 % of eyes had either stable or improved visual acuity or reduction in the mean central retinal thickness by OCT. A phase II DRCR net study of 109 patients compared two does of Bevacizumab to focal laser photoagulation and demonstrated its efficacy in decreasing DME in some eyes. To date, no phase III trials have been reported that demonstrate a clear benefit for Bevacizumab in the treatment of DME.

A report by the Pan - American Collaborative Retina study Group on 101 consecutive eyes with diffuse diabetic macular edema (DDME) treated with intravitreal Bevacizumab, resulted in both anatomic and functional improvement62. Interestingly, the reduction of retinal thickness and improvement of BVCA were detected within the first 4 weeks after the injection in most of the patients. In addition, both doses (1.25 mg and 2.5 mg) were associated with improvement of BVCA and a greater reduction in central macular thickness, and no difference in between were found. Ocular tolerance of the 2 different doses of IVB was demonstrated, and no serious systemic adverse events were noticed during the study.

There are several studies in the literature on the intravitreal administration of antibodies against VEGF for DDME. However, none of them deal with anti-VEGF as a primary treatment. Haritoglou et al reported that intravitreal ranibizumab has the potential to maintain or improve BVCA and reduce retinal thickness in patients with DDME not responding to previous treatments such as photocoagulation, intravitreal
injection of triamcinolone, or vitrectomy. Their follow-up period was too short (6 weeks) to provide specific treatment recommendations. Kumar and Sinha reported results of 20 eyes with DDME treated with IVB dose of 1.25 mg that had not responded to previous photocoagulation. Their follow-up period was 6 months. They concluded that IVB resulted in a significant decrease in macular thickness and improvement in visual acuity at 3 months, but the effect was somewhat blunted, though still statistically significant at the end of 6 months. The current study compares favorably with these reports, and confirms their findings with longer follow up and a larger number of patients. Further more, at the 6 month follow up time point they also noticed a small worsening of vision as described by Kumar and Sinha. When analysis of data comparing eyes that had 1 or 2 injections against those eyes that had 3 or more injections was performed, there was a significant drop in BVCA at 6 months in the “1 or 2 injections” group, and not in the “3 or more injections” group. This suggests the need for at least 3 injections a year to maintain the BVCA results. 64 eyes (63.4 %) needed at least a second injection at a mean of 15.7 ± 11.9 weeks (range: 4 to 64 weeks).

The results of this retrospective study demonstrated the efficacy of 1.25 mg or 2.5 mg of IVB as primary treatment of DDDME, as 49.5 % of eyes showed anatomical and functional improvement. In addition, the results suggest a reduced risk of visual acuity loss in eyes with DDME treated with intravitreal bevacizumab (IVB) (82.2 % of eyes). The anatomical and visual benefit of the intravitreal Bevacizumab appears and reaches its maximum value during the first month maintains itself over 12 months. Nevertheless, statically significant differences between the 2 doses of Bevacizumab evaluated could not be demonstrated.

### Ranibizumab in DME (READ Trial)

While ranibizumab, a humanized monoclonal antibody fragment directed against all VEGF isoforms, is currently in clinical trials for DME, its off label use in DME patients is limited most likely as a result of its increased cost and less wide spread availability world wide, as compared to Bevacizumab. Two pilot studies of ten patients each suggested that it was well tolerated and may have some efficacy in promoting improvement in visual acuity and reduction in central retinal thickness by OCT.

A phase 1 study (the READ-1 Study, Ranibizumab for Edema of the macula in Diabetes, sponsored by the Juvenile Diabetes Research Foundation) of 20 patients with DME treated with intravitreal Ranibizumab (IVB). Repeated intravitreous injections 0.5 mg of ranibizumab, showed evidence of biological activity of ranibizumab in DME as well as safety and tolerability (Nguyen, et al. 2006). In the Phase 1 study, patients were given intravitreal ranibizumab at baseline and at months 1, 2, 4, and 6. Results showed that at month 7, one month after the final administration of ranibizumab and the primary endpoint of the study, the median reduction of the excess foveal thickness was 97 %, and there was a median improvement of 10 letters.

There have been no adverse events that were believed to be related to the study drug; in particular, intraocular inflammation was not observed.

The READ-2 Study is a Phase 2 randomized, multicenter clinical trial sponsored by the Juvenile Diabetes Research Foundation. The study enrolled 126 patients from 14 clinical centers throughout the United States.

Proportion of subjects who gain 15 or more letters, or achieve a final vision of 50 letters (20/25) or better if baseline VA was 40 letters (20/40) or better, at 6 months was the primary endpoint.

Patients were eligible if they had an ETDRS Visual acuity of 20/40 or worse, but better than or equal to 20/320 due to foveal thickening from macular edema secondary to diabetes (type 1 or 2) and a baseline foveal thickness of 250 μm on OCT.

Each study subject in the trail was randomized 1:1:1: to 1 of 3 treatment groups.

- **Group 1** (ranibizumab only)
- **Group 2** (Laser)
- **Group 3** (ranibizumab and laser)

The patients were followed every 12 weeks until 24 months (secondary time endpoint). At any study visit, if there is an increase of a specified amount of retinal thickness on OCT that meets re-treatment criteria, the patients will have the opportunity to receive an additional injection of ranibizumab plus laser 7 days later.
The re-treatment criteria for patients in all 3 randomized groups were an absolute retinal thickness in OCT central subfield of ≤ 250 μm (at time of study visit). Six months outcomes suggest greater improvement in visual acuity for patients receiving intravitreal Ranibizumab as compared to those receiving laser or combination therapy.

**Combination Therapy**

As diverse mechanism and patterns of DME are recognized, clinicians are using multi-model therapies to approach DME. In theory, targeting various pathologic mechanisms of DME with combination therapies may have a more lasting effect on reversing and maintaining a clinical benefit to the patients. Commonly focal laser photocoagulation is being combined primarily with Ocular Steroid therapy (either IVTA (Intravitreal injection of Triamicinolone acetonide) or PSTTA (Posterior subtenons injection of Triamicinolone acetonide) or anti VEGF agents. This strategy seeks to take advantage of the more immediate effects of pharmacologic agents while employing laser therapy for long term stabilization. Anti VEGF agents have been used to salvage eyes refractory to steroid therapy, in eyes experiencing steroid related side effects, and more recently in combination with IVTA therapy with positive results. Pharmacological agents are also being used at the time of vitrectomy surgery help to prevent recurrent DME.

**Surgical Management of Diabetic Macular Edema**

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

The vitreous gel is thought potentially to play a role in the development of DME through mechanical factors and / or physiologic mechanisms that lead to increased retinal vascular permeability. Vitrectomy has been used in the management of diabetic macular edema (DME) for many years. In many cases, this surgical procedure is performed because of macular traction and abnormality of the posterior hyaloid.

In some cases, the procedure has been performed as a “last-resort” measure in the judgment of an ophthalmologist when the eyes have been non responsive to macular photocoagulation and other modalities. Despite the fact that thousands of eyes are estimated to have had vitrectomy for DME, available data, to judge the merits and risks of surgical procedure for DME is limited. The literature consists mainly of retrospective case series.

There are at least 2 avenues of investigation that support the theoretical value of vitrectomy for the treatment of DME, based on (1) vitrectomy to relieve biomechanical traction on the macula and (2) vitrectomy to improve oxygenation of the macula leading to decreased permeability with subsequent resolution or decrease in DME.

Vitrectomy to relieve biomechanical traction on the macula has been reported widely.

The DRCR net Study evaluating vitrectomy for DME (Protocol D, available online at www.drcr.net) is designed as a prospective cohort study in patients with DME on clinical examination and a best corrected vision of 20/800 or better.

Study Objectives were:

1. To provide information on the following outcomes in eyes with DME that undergo vitrectomy: visual acuity (VA), retinal thickening, resolution of traction (if present), surgical complications.
2. To identify sub groups in which there appears to be a benefit of vitrectomy and sub groups in which vitrectomy does not appear to be beneficial.
3. To obtain data that can be used to plan a randomized trial.

The 6 months results of this clinical trial (presented at the AAO subspeciality meeting 2008 at Atlanta) showed favorable results with vitrectomy.

Triamcinolone assisted pars plana vitrectomy is preferred by many surgeons as the TA helps delineate the posterior cortical vitreous, epiretinal membrane and the internal limiting membrane. The half life of Triamcinolone acetonide in the vitreous cavity in a vitrectomised eye is only 1-6 days. The small amount of TA crystals sequestered in the vitreous cavity is not
Fig. 5: ETDRS definition of clinically significant diabetic macular edema. (Adapted from ETDRS report, Ophthalmology 1987)

Fig. 6: Focal DME characterized by focal areas of retinal thickening and a partial or complete ring of hard exudates.

Fig. 7: Diffuse DME demonstrating generalized abnormal permeability of retinal capillaries.

Fig. 8: (a) Flower petalloid appearance on fluorescein fundus angiography due to pooling of dye. (b) OCT characterized by cystoid space in an edematous retina filled with clear fluid.

Fig. 9: Ischemic Maculopathy is characterized by gross macular edema, fluorescein angiographic appearance of enlarged foveal avascular zone (FAZ) and loss of integrity of the perifoveal capillary net.

Fig. 10: a, b and c: (a) focal diabetic macular edema, (b): diffuse DME with neovascularization elsewhere (NVE) (c): ischemic maculopathy significant enough to cause a postoperative intraocular pressure spike, but may be just enough to prevent postoperative intraocular inflammation.

The concept of denuding the inner retinal surface of the internal limiting membrane promotes migration of cells, egress of extra cellular fluid and blood out of the
Fig. 11. FFA and red free photographs demonstrating the presence of macular microaneurysms which are the earliest changes in diabetic retinopathy.

Fig. 12. FFA demonstrating the presence of intravitreal microvascular abnormalities (IRMA) and proliferation of new vessels (NVE).

Fig. 13. Idiopathic parafoveal telangiectasia (IPFT) can coexist with diabetic macular edema.

retina and towards the vitreous cavity. Reduction in retinal thickening and improvement in oxygenation should theoretically improve the visual acuity.\textsuperscript{76,77}

The use of Indocyanine green (ICG) to assist internal limiting membrane peeling has been associated with reports of retinal and optic nerve toxicity. The adverse effects of ICG\textsuperscript{78} assisted ILM peeling has been reported in 46.7% of subjects who developed slowly progressive onset of optic atrophy with in 6 months of undergoing surgery, associated with irreversible peripheral visual field defects predominantly in the nasal field. Other reports on vitrectomy with ILM peeling for diabetic macular edema or macular hole surgery does not show any intraoperative or postoperative complication attributed to the use of ICG or any clinical or angiographic evidence of ICG toxicity. Other dyes have been used for internal limiting membrane peeling including Trypan blue and Brillnat Blue G.

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

Various other groups of workers have conclusively shown that PPV with ILM peeling leads to expedited resolution of diffuse diabetic macular edema and visual improvement without subsequent epiretinal membrane formation. Quantitative assessment of OCT images at the end of follow up revealed that retinal thickness in the macula appeared nearly normal with or without reappearance of foveal pit in 73.3%. The effects of
<table>
<thead>
<tr>
<th>AUTHORS/ YEAR</th>
<th>NO. OF EYES</th>
<th>MACULAR THICKNESS</th>
<th>VA</th>
<th>RESULTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>GANDORFER 2000</strong></td>
<td>12</td>
<td>Resolution 4-12 weeks</td>
<td>2 line all eyes</td>
<td>Favourable</td>
</tr>
<tr>
<td><strong>Ando F, 2004</strong></td>
<td>15</td>
<td>Resolution to Normal (20 %) Improved (26.7%)</td>
<td>OA in 46.7 %</td>
<td></td>
</tr>
<tr>
<td><strong>Shah, Patel &amp; Tomas etal 2006</strong></td>
<td>33</td>
<td>Resolution</td>
<td>Improved only in Pts with VMT</td>
<td>Unfavourable</td>
</tr>
<tr>
<td><strong>Behadir 2005</strong></td>
<td>58</td>
<td>Similar in both groups</td>
<td>Similar in both groups</td>
<td>No added benefits</td>
</tr>
<tr>
<td><strong>Parys Vanginderdeuren et al 2005</strong></td>
<td>26</td>
<td>Resolution</td>
<td>Improved</td>
<td>Good</td>
</tr>
<tr>
<td><strong>Gaurav Shah 2005</strong></td>
<td>26</td>
<td>Good resolution</td>
<td>2 line improvement</td>
<td>Good</td>
</tr>
</tbody>
</table>

Fig. 14. (a) Spongy retinal edema (b) Cystoid Edema (c) Subfoveal serous retinal detachment (d) Taut attached posterior hylaoid (e): Vitreo macular traction (f): Screen effect produced by plaque of hard exudates.
resolution of macular edema, reduction in retinal thickness and improved visual function were longer lasting than following IVTA injection. The visual improvement following PPV with ILM peel was gradual and occurred within 6 months to one year following the surgery.

**Synopsis:**

Diabetic Macular Edema is one of the major causes for moderate visual loss in a diabetic. With increasing incidence and prevalence of diabetes and limited public health resources, diabetic macular edema is becoming a major public health concern.

**Current Clinical Practice/Recommendations for DME**

- A detailed history on the duration of diabetes, adequacy of control, presence of other complications, current medications, glycaemic and blood pressure control, history of prior eye surgery, prior laser treatment and speed and duration of visual loss is taken

- Ophthalmic evaluation includes
  1. Best Corrected Visual Acuity
  2. Tonometry
  3. Slitlamp Biomicroscopic examination of the macula
  4. Assessment of Lens Changes
  5. Detailed fundus evaluation after adequate mydriasis
  6. Stereofundus photography
  7. Baseline fluorescein fundus angiography
  8. Optical Coherence Tomography

- Based on clinical evaluation, angiographic and tomographic assessment the maculopathy can be divided into
  - Focal
  - Diffuse
  - Ischemic
  - Cystoid
  - Mixed
  - VMT
  - Macular edema with subfoveal serous RD
Special attention should be paid to the vitreoretinal interface (for presence of traction, PVD, thickened taut post hyaloid, epiretinal membranes)

Depending on these findings one of the following treatment options is offered to the patient.

1. In patients with poor and improvable systemic control or diabetic renal disease with stable visual acuity or a slow decrease in vision, initiation of better systemic control (blood sugar, blood pressure, and hemodialysis, anemia) should be the first step. Specific Ophthalmologic treatment may be deferred until systemic factors are optimized.

2. For patients with **Focal Diabetic Macular Edema without significant ischemia** focal laser is still the treatment of choice. Compared with the ETDRS guidelines, use of lighter burns of longer duration and to perform more than one session of treatment in severe cases is recommended.

3. For patients with **diffuse diabetic macular edema** modified grid laser photocoagulation sparing the fovea can be performed if the OCT shows a central retinal thickness $\leq 300 \, \mu m$ and there is no evidence of ischemia.

**Micro pulsed Sub threshold Selective Laser Therapy**: using a given Nd:YLF micro pulsed laser has been shown to be effective in DME, while minimizing chorioretinal damage.

4. **Intravitreal or Peribulbar steroids** are recommended in cases where the laser treatment has not been effective or seems unlikely to improve. If the eye is phakic the risk of steroid induced cataract has to be discussed. Pre existing glaucoma or a family history of glaucoma should be taken into account.

5. Intraocular pressure has to be followed closely. Eyes with gross **diffuse macular edema and subfoveal serous retinal detachment** respond best to intravitreal injections.

6. After IVTA the situation should be re-evaluated and **laser photocoagulation** applied if appropriate.

7. **Vitrectomy combined with peeling of epiretinal membranes and internal limiting membranes** may be considered for eyes with pathology in the vitreoretinal interface. This procedure may be preceded by or combined with intravitreal steroids.

8. For all these options the other features of diabetic retinopathy should be taken into account and therapeutic approaches can be combined (**focal laser followed by PRP; IVTA followed by PRP; IVB followed by PRP, vitrectomy combined with endlaser photocoagulation etc.)**

9. In cases of uncontrollable adverse effects of steroids after IVTA, or persistent or relapsing edema with gross visual deterioration, treatment with intravitreal Anti VEGF agents is considered.

10. The efficacy of these therapeutic measures should be regularly monitored and strategies varied depending of visual acuity, clinical findings and onset of complications.

11. The risk of diabetic macular edema worsening after uneventful cataract surgery is a definite possibility. Risks and potential benefits should be
weighed against each other based on the clinical situation. Postoperative monitoring and early initiation of treatment can effectively check this risk of progression of diabetic macular oedema after cataract surgery.

12 Ophthalmologists play an important part in patient motivation. A team approach with the internist, endocrinologist, and nephrologists is absolutely necessary.

13 Systemic Pharmacotherapy: Oral PKC\(\beta\) inhibitor Ruboxistaurine has been found in experimental studies to reduce the risk of vision loss (40 % reduction), need for laser photocoagulation (26 % reduction) and progression of macular edema (26 % reduction) compared with the placebo.

14 Other systemic pharmacotherapeutic agents of use in treatment of diabetic macular edema are aldose reductase and AGE inhibitors and antioxidants

Reference


56. Spandau UH, Derse Met al. Dosage dependency of intravitreal triamcinolone acetone for diabetic


62. Jennifer Lim, Peter Campochiaro, Quan Dong Nguyen et al. The READ Trial: Ranizumab in the treatment of Diabetic Macular Edema. Presented at AAO Retina Subspeciality Meeting at the Atlanta.


Closed Globe Injuries -
A Tertiary Care Experience

Dr. Raju K V MS, Dr. Nima C A MS, Dr. Anju A K MS

Abstract

Aim: To identify risk factors, clinical presentation and analyze effectiveness of treatment and prognostic factors of closed globe injuries.

Methodology: Prospective case series conducted in 110 patients over a two year period.

Results: Out of 110 patients, 65 had a good visual acuity by medical management. Of 45 patients with initial vision <3/60, 39 regained >6/60 vision and 6 patients with <3/60 vision had posterior segment pathology.

Conclusion: Medical treatment was effective in majority of cases of closed globe injuries. More than half of patients had >6/12 vision in the end. Initial visual acuity has an important role in deciding final visual outcome. Posterior segment pathology correlated with poor final vision.

Key words: Closed Globe Injury, Prognostic Factors, Visual Outcome

Introduction

Ocular trauma is a major cause of worldwide visual impairment. It is the single most important cause of mono-ocular blindness worldwide. Ocular trauma can occur in any setting including home settings, recreational and sports-related activities, workplace and road traffic accidents. The young individuals are the main victims of trauma and the effect of it on their career and future life is devastating. The aim of our study was to determine the causes and circumstances of closed globe injuries in a tertiary care referral center, its prognostic factors and the final visual outcome and complications.

Methods

This is a prospective study conducted on patients who were admitted to the Ophthalmology Department, Medical College, Calicut from November 2004 to October 2006 with a clinical diagnosis of closed globe injuries. Cases were followed up for 6 months.

Exclusion Criteria

Those with head injuries, severe chest and abdominal trauma and those who were lost to follow up in 6 months were excluded from the study.

Clinical evaluation

The initial external examination of the eye was done with torch light. Slit lamp examination was done in all cases and findings were noted. Fundus examination
was done if possible. Visual acuity was recorded. In relevant cases and wherever possible, field charting, indirect ophthalmoscopy, 90 D lens examination and gonioscopy were done. Intraocular pressure was recorded by Schiotz tonometer.

**Investigations**

If needed, RBS, X-ray orbit, USG and CT scan were taken. X-ray orbit was taken to look for any evidence of fracture. USG was taken in certain cases where there was no fundus view and definite posterior segment pathology was suspected. CT scan was ordered especially in cases of proptosis, traumatic optic neuropathy and to rule out occult scleral rupture.

**Treatment**

All relevant medical and surgical treatment given to the patient was noted. When surgical treatment was performed, indications, procedure performed, mode of anaesthesia and post operative complications were noted.

**Follow up**

All patients were followed up at 2 weeks, 6 weeks, 3 months and 6 months. During their visits, visual acuity, IOP, fundus, slit lamp examination were recorded. In hyphema cases, gonioscopy was done after 1 month. If vision was not improving, retinoscopy was carried out and PMT was done with appropriate correction with spectacles. Final visual outcome of the patient and complications as a sequelae of closed globe injuries were evaluated and prognostic factors determined.

**Results**

The age of the patients ranged from 3 to 73 years. The commonest age group was 10-20 years (25.45 %). 17 patients were below 10 yrs and 21 patients between 20-30 years. The vast majority of patients were below 40 years, (71.82 %). The mean age of these patients was 30.05 years. There were 96 males (87.27 %) and 14 females (12.73 %). The Male: Female ratio was 6.9:1.

The most common mode of injury was with stick (19.09 %). 14.53 % had injury with tennis ball. 16 patients (14.53 %) had injury with wood, 11.82 % had injury with stone, 5.45 %, had injury with rope and rest with other agents like shuttle cock, bottle cork etc. 29.09 % were injured during play, 27.27 % injured while at home and 20 % injured at work place.12.73 % sustained sports injury and 6.36 % were injured by road traffic accidents. 5 were injured at other places. The right eye sustained trauma more often than left. 62 patients (56.36 %) had injury to right eye. 46 patients (41.82 %) had injury to left eye and 3 (1.82 %) sustained injury to both eyes.

The commonest anterior segment finding (Table 1) was traumatic mydriasis (59.09 %). Traumatic iritis was

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lid injury</td>
<td>19</td>
<td>17.27</td>
</tr>
<tr>
<td>Conjunctival tear</td>
<td>21</td>
<td>19.09</td>
</tr>
<tr>
<td>Corneal abrasion</td>
<td>38</td>
<td>34.55</td>
</tr>
<tr>
<td>Corneal FB</td>
<td>10</td>
<td>9.09</td>
</tr>
<tr>
<td>Hyphema</td>
<td>60</td>
<td>54.55</td>
</tr>
<tr>
<td>Iridodialysis</td>
<td>6</td>
<td>5.45</td>
</tr>
<tr>
<td>Iritis</td>
<td>61</td>
<td>55.45</td>
</tr>
<tr>
<td>Traumatic mydriasis</td>
<td>65</td>
<td>59.09</td>
</tr>
<tr>
<td>Cataract</td>
<td>14</td>
<td>12.73</td>
</tr>
<tr>
<td>Subluxation</td>
<td>31</td>
<td>28.18</td>
</tr>
<tr>
<td>Dislocation</td>
<td>4</td>
<td>3.64</td>
</tr>
</tbody>
</table>

Table 2 : Ocular Manifestations : Posterior segment findings

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitreous hemorrhage</td>
<td>24</td>
<td>21.82</td>
</tr>
<tr>
<td>Retinal hemorrhage</td>
<td>26</td>
<td>23.64</td>
</tr>
<tr>
<td>Retinal oedema</td>
<td>59</td>
<td>53.64</td>
</tr>
<tr>
<td>Macular oedema</td>
<td>66</td>
<td>60</td>
</tr>
<tr>
<td>Macular hole</td>
<td>1</td>
<td>0.91</td>
</tr>
<tr>
<td>Choroidal hemorrhage</td>
<td>5</td>
<td>4.55</td>
</tr>
<tr>
<td>Choroidal tear</td>
<td>10</td>
<td>9.09</td>
</tr>
<tr>
<td>Choroidal detachment</td>
<td>1</td>
<td>0.91</td>
</tr>
<tr>
<td>Retinal tear</td>
<td>2</td>
<td>1.82</td>
</tr>
<tr>
<td>Retinal detachment</td>
<td>4</td>
<td>3.64</td>
</tr>
<tr>
<td>Proptosis</td>
<td>4</td>
<td>3.64</td>
</tr>
<tr>
<td>TON</td>
<td>16</td>
<td>14.55</td>
</tr>
</tbody>
</table>

Table 3 : Causes of poor visual acuity of < 6/36

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>No.</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior capsule rupture</td>
<td>2</td>
<td>1.82%</td>
</tr>
<tr>
<td>with vitreous loss</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secondary glaucoma</td>
<td>2</td>
<td>1.82%</td>
</tr>
<tr>
<td>Irregular astigmatism</td>
<td>2</td>
<td>1.82%</td>
</tr>
<tr>
<td>Traumatic optic neuropathy</td>
<td>6</td>
<td>5.45%</td>
</tr>
<tr>
<td>Retinal detachment</td>
<td>2</td>
<td>1.82%</td>
</tr>
<tr>
<td>Vitreous hemorrhage</td>
<td>10</td>
<td>9.09%</td>
</tr>
<tr>
<td>Choroidal tear through fovea</td>
<td>1</td>
<td>0.91%</td>
</tr>
<tr>
<td>Choroidal hemorrhage</td>
<td>1</td>
<td>0.91%</td>
</tr>
<tr>
<td>Macular hole</td>
<td>1</td>
<td>0.91%</td>
</tr>
</tbody>
</table>
seen in 61 patients (55.45%). 60 patients (54.55%) had hyphema. 6 patients (5.45%) had iridodialysis. 38 patients (34.55%) had corneal abrasion, 21 patients (19.09%) had conjunctival tear and 19 patients (17.27%) had lid injury. Cataract was present in 14 patients (12.73%). 31 patients (28.18%) had subluxation of lens and 4 patients (3.64%) had posterior dislocation of lens. The commonest posterior segment finding was macular oedema (60%) (Table 2). The next common findings were retinal oedema (53.64%), retinal hemorrhage (23.64%), vitreous hemorrhage (21.82%), choroidal hemorrhage (4.55%), choroidal tear (0.91%) (Figure 1) and ciliochoroidal detachment (0.91%). 1.82% had retinaeal tear. 3.64% had retinal detachment (Figure 2). Traumatic optic neuropathy was seen in 14.55%. 1 patient had macular hole (0.91%) (Figure 3). 65 patients (59.09%) regained good vision of 6/12. 18 patients (16.36%) had visual acuity between 3/60 to PL.

The present study included 110 cases of closed globe injuries who were admitted in our department. In the present study the commonest age group involved was 10-20 years, which constituted 25.45%. 60% were below the age of 30 years. This is in concordance with study by Badrinath et al which showed 67% of patients were below age of 30 years. The mean age of patients in our study was 30.05 years. Studies by Jain et al and Banshi Krishna Malla also clearly show that children and young individuals are the most susceptible to ocular trauma.

In the present study, male preponderance (87.27%) was seen and male: female ratio was 6.9:1. Viestenz et al showed 85% were males. The male: female ratio was 2.2:1 (Badrinath S.S) and 5.4:1 (Jain et al). Male preponderance is understandable as they are more involved in outdoor activities, sports, employed in factories and industries and rash driving.
According to the present study, stick was the commonest injuring agent (19.09 %).

This correlates well with the study conducted by Michael Ilsar, Moses Chirambo, Abraham and Vitale. The mode of injury depended upon the industrialization, type of sports prevalent in the area and type of work in which most individuals are engaged. Fireworks injury was present in 3 cases and it involved both eyes. In our area it mainly occurred during the Vishu festival. In the present study, the common circumstance of injury was at play, (29.09 %) at home (27.27 %) and then the workplace. Badrinath and Banshi Krishna et al reported domestic accidents were responsible for majority of injuries.

In our study, the right eye sustained trauma (56.36 %) more often than left. In our study the most common anterior segment finding noted was traumatic mydriasis in 59.09 %. Hyphema was noted in 54.55 % of cases. Hyphema was often associated with secondary glaucoma, mydriasis and subluxation of lens. Luksza et al also found hyphema was associated with secondary glaucoma, cataract and mydriasis.

In the present study, the incidence of angle recession in hyphema was 16.67 %. (3.33 % <180° angle recession and remaining 8 had 360 ° angle recession.) This is in concordance with the study conducted by Salmon JF and Mermoud who found the prevalence was 14.6 %. However Julio et al, Ference Kuhn and Ellong A reported 60-80 % incidence of angle recession in their studies.

The present study showed the incidence of angle recession glaucoma to be 3.67 %. All patients had 360° angle recession and responded well to medical treatment. This finding correlated with the studies by Salmon JF (5.5 %) and Ellong A (2.1 %). But Julio et al found 7-9 % of patients with angle recession developed glaucoma. The difference may be because our follow up period was upto 6 months. With long term follow up, we may find more cases of angle recession glaucoma.

In the present study 20.91 % patients developed raised IOP and this correlates well with the studies conducted by Krishnan Mathew and Sreenivasan Renuka who found secondary glaucoma in 22.07 % cases.
Another finding noted in our study was that majority of patients were having normal or below normal IOP in the initial phase which can be explained by the ciliary body shock, occurring after blunt trauma.

In the present study, subluxation of lens was noticed in 28.18% cases and dislocation in 3.64%. Krishnan Mathew and Sreenivasan Renuka noticed a slightly higher incidence of subluxation (35.96%) \(^\text{11}\). The incidence of traumatic cataract in the present study is 12.73%. This is in concordance with the study conducted by Krishnan Mathew (14.92%), A Viestenz (10%) and Julio E et al (11%) \(^{11,4,9}\).

Out of the 31 cases of subluxation, 21 were managed medically and 10 managed surgically. All the 3.64% cases of posterior dislocation were managed surgically. Two cases had posterior capsule rupture with vitreous loss and were rendered aphakic. Majority of subluxated and dislocated lens had significant posterior segment pathology which accounted for their poor visual acuity even after surgery. The present study showed presence or absence of posterior capsular injury and posterior segment pathology has a definite prognostic value in predicting the final visual outcome. This finding was also noted in the Andhra Pradesh Eye Disease study by Krishnaihar et al \(^\text{12}\).

In the present study the commonest posterior segment finding noted was macular oedema in 60% of patients. The next common findings were retinal oedema (53.64%), retinal hemorrhage (23.64%) and vitreous hemorrhage (21.82%). Studies by Viestenz, Samer Said Shebl and showed an incidence of macular oedema between 30-40% \(^{3,13}\). Their studies reported high incidence of vitreous hemorrhage (57%).

Retinal detachment was seen in 3.64% cases in the present study. The incidence of retinal detachment is much higher (9-12%) in the studies conducted by Dumas J.J and Manoj Shukla et al \(^{14,15}\).

The present study showed the incidence of choroidal tear to be 9.09%. This tallies with the United States Eye Injury Registry recording where the incidence is 5-10%. A Viestenz in his study also showed 7% incidence of choroidal rupture \(^3\). In our study one patient had choroidal tear through fovea which accounted for his poor visual acuity. This suggests that tear through fovea is a bad prognostic indicator.

It was also found that there was a direct relation between traumatic mydriasis and vitreous hemorrhage, retinal oedema, macular oedema and retinal hemorrhage. The present study showed that if traumatic mydriasis is noted, it is mandatory to search for posterior segment findings. This can be explained by the fact that the force required to produce traumatic mydriasis is sufficient enough to produce posterior segment damage.

In the present study 84.55% were managed medially and 15.45% managed surgically. In the study by Banshi Krishna Malla, 50% were managed medically and 50% surgically \(^2\).

In our study according to BETTS classification 99% were Type A, 40% were having Gr. 4 visual acuity, pupil positive in 14.55% and 83.64% were zone III injuries.

Traumatic optic neuropathy was present in 14.55% patients in our study. Road Traffic Accidents was the commonest cause in our study. 50% patients had visual acuity between 3/60 and PL. Inj. Methyl prednisolone was given to all the patients. The finding noted was inspite of Inj. Methyl prednisolone, only those with initial good visual acuity regained their vision while those with initial poor visual acuity showed little response. This suggests initial visual acuity is an important prognostic indicator of final visual outcome.

Final visual outcome was > 6/36 in 75.45% of cases where as 24.55% had visual acuity of <6/36 in our study. This is in concordance with the study conducted by Jain et al who found 52.3% had visual acuity of ≥ 6/36 and 14.6% had visual acuity <6/60 \(^2\).

The cause for poor visual acuity was analysed and the most common cause noted was vitreous hemorrhage in 9.09% and, traumatic optic neuropathy in 5.45% cases. Krishnan Mathew and Renuka Sreenivasan also found that posterior segment findings like vitreous hemorrhage and macular oedema were the main causes for poor visual acuity \(^{11}\).

With the change in life style of people and the environment changes, the incidence and the causative objects for eye trauma have changed significantly. Consequently the nature and extent of the anatomical or structural lesions in ocular trauma have become more
severe. Prevention is better than cure. But once injury occurs, our aim should be to achieve maximum visual acuity and prevent the complications by medical and surgical means and with low vision rehabilitation if needed.

References

Visual and Anatomical Outcomes of Vitreous Surgery for Large Macular Holes

Dr. Mahesh G. MS DO DNB FRCSEd, Dr. A. Giridhar MS, Dr. Ramkumar DO, Dr. Alpesh Rajput DO

Abstract:

Purpose: To report the visual and anatomical results of surgery for large macular holes.

Methods: Retrospective interventional case series of 33 consecutive patients with macular hole of more than 1000 microns on Optical Coherence Tomography (OCT) who underwent vitrectomy with internal limiting membrane (ILM) peeling and intravitreal gas tamponade between January 2006 to December 2007 were included in the study. All patients had undergone preoperative OCT measurements; repeat OCT was done every 3 months thereafter. Outcome measures were visual acuity and anatomical closure.

Results: 33 eyes of 33 patients with a mean age of 62.96 years were included. Mean preoperative base diameter was 1259 microns (1006 - 1809 microns). Mean follow-up was 9.12 months (2 months - 25 months). Mean post op BCVA was 1.02 logMAR units, which had improved to 0.87 logMAR units postoperatively with a mean improvement of 0.15 logMAR units (p 0.017). Best corrected visual acuity improved or stabilized in 27 of 33 eyes (81.8 %) Anatomic closure using OCT was seen in 23 of 33 eyes (69.7 %).

Conclusion: In large macular holes vitrectomy with ILM peeling appears to be a beneficial treatment.

Introduction

The first clinical description of a macular hole was published by Henry Noyes in 1871. Since then our understanding of development and pathogenesis of macular holes has improved. However, it took more than 100 years, until Kelly and Wendell reported the first successful closure of a series of macular holes by pars plana vitrectomy and induction of posterior vitreous detachment in 1991 2. Several authors have reported significantly higher rates of anatomical closure and visual rehabilitation in many of these cases. The postoperative success rate varies between 86 % and 95 % with improvement in visual acuity in a large percentage of cases.

Recent attempts to use imaging techniques such as confocal scanning laser tomograph and the scanning laser ophthalmoscope to predict success suggest a correlation between the macular hole size and visual recovery. This study used optical coherence tomography (OCT) to measure the preoperative macular hole size and correlated this with the postoperative rate of anatomical closure.
OCT is a recently introduced diagnostic tool for high resolution, cross sectional imaging of the posterior and anterior segment of the eye with an axial resolution of 10 μm and a transverse resolution of 30 μm. OCT has been of immense use in understanding the pathogenesis and staging of macular holes, prognostication and grading of the surgical outcomes as well.

There has always been a controversy on the maximum size of hole which can be operated. In our study we report the anatomical and functional outcomes following surgery for large macular holes with a minimum diameter of 1000 microns.

Materials and Methods

A retrospective review between January 2006 to December 2007 of all eyes with an idiopathic macular hole that were examined preoperatively and postoperatively by OCT at our hospital was performed. Only eyes diagnosed as having idiopathic macular holes more than 1000 microns were included in this study. Patients with previous and or coexisting diseases such as intraocular inflammation, ocular trauma and retinal detachment, and patients who were not fit to maintain postoperative prone positioning due to systemic diseases and patients diagnosed to have macular holes due to secondary causes like post trauma etc were excluded from the study.

Patients underwent a complete ophthalmic examination including complete medical and ophthalmic history, best corrected Snellen visual acuity, Amsler grid testing, intraocular pressure measurement, slit lamp biomicroscopy, indirect ophthalmoscopy and OCT. Each patient was examined with OCT through a dilated pupil and macular holes were measured. Largest base diameter was taken for calculation of macular hole size. Informed consent was obtained prior to surgical intervention in all patients. All surgical procedures were done by a single surgeon between January 2006 and April 2008. Surgery consisted of standard three-port pars plana vitrectomy, peeling of the internal limiting membrane with subsequent intraocular gas tamponade using 13 % perfluoropropane (C₃F₈) gas. Internal limiting membrane was identified by staining using vital stains like Trypan blue (Retiblue) or Brilliant peel or IntraVitreal Triamcinolone during surgery. After surgery patients were asked to maintain a prone position for 14 days. Patients were examined on day 1, day 10, 1 month, 2 months and 6 months. Postoperative OCT was performed at 2 months following surgery.

Anatomical success was clinically defined as apposition of macular hole edges and absence of sub-retinal fluid cuff. Anatomical success determined by OCT was restoration of full or partial thickness retinal reflection over the retinal pigment epithelium and choriocapillary reflections. The primary outcome of the study was anatomical closure of macular hole. On the basis of postoperative OCT findings closure of macular hole was classified into two groups; type 1 and type 2 closures. Type 1 closure indicated that the macular hole is closed without foveal defect of the neurosensory retina as a U-type (Normal foveal contour); Type 2 closure indicated macular hole is closed with foveal defect of the neurosensory retina. Those holes where there was neurosensory retinal defect and the lips of the hole were elevated were defined as open macular holes, and considered as anatomical failure.

For statistical purposes best corrected visual acuity was which was recorded in Snellen’s was converted to logMAR. Statistical analysis was done using SPSS statistical software over 11.0.

Results

This was a retrospective interventional case series. All patients who had a macular hole of more than 1000 microns and who met the study criteria were included. 33 eyes of 33 patients who were eligible were included in the study. There were 24 women and 9 men, with a median age of 63.33 years (Range 50 – 77 years). Left eye was involved in 21 eyes while right eye was involved in 12 eyes. (Demographic data of study is shown in table 1).

Preoperative macular hole diameter ranged from 1006 μm to 1809 μm with a mean of 1259 μm. On OCT 16 holes were diagnosed as stage 3 holes while 17 holes were classified as stage 4. The mean length of visual symptoms ranged from 6 months to 36 months. Follow up ranged from 2 months to 25 months with a mean follow up of 9.12 months.

Preoperative visual acuity ranged from 3/60 (1.3 LogMAR) to 6/18 (0.5 LogMAR) with a mean of
1.02 logMAR units. Mean postoperative best corrected visual acuity has improved to 0.87 logMAR units with a mean change of 0.15 logMAR units (p = 0.017). Best corrected visual acuity had improved or stabilized in 27 of 33 eyes (81.8 %).

Anatomical closure of macular hole was achieved in 23 of the 33 eyes (69.69 %) with single surgery. Type 1 closure was seen in 19 of the 23 eyes (82.60 %). Optical coherence tomographic analysis of macular holes with type 1 closure showed regular pattern of photoreceptor layer in 7 of 19 eyes and irregular pattern in 12 of 19 eyes. (Postoperative OCT feature are depicted in Table 2).

10 patients had progression of cataract following macular hole surgery. 4 of these patients who had significant cataract affecting visual acuity underwent cataract surgery subsequently. There were no complications during the surgical procedure.

Discussion

Surgical treatment of idiopathic macular holes has given vitreo-retinal surgeons and patients an option for visual recovery for this once untreatable condition. Although the surgical results have improved over the years, controversy still exists as regards to the exact surgical timing and also case selection.

Timing of surgical intervention, depending on idiopathic macular hole staging, size and duration has shown correlation in success rate and visual recovery. Preoperative staging has been traditionally based on the classification system proposed by Gass, judging macular hole diameter on clinical and photographic evaluation using the peri papillary vein (125 μm in diameter) as reference. Moreover, conditions such as epiretinal membrane, lamellar macular hole, cystoid macular edema and macular degeneration can be misdiagnosed as macular hole on biomicroscopy. OCT helps to differentiate these conditions and also assess macular hole diameter correctly. The use of OCT may allow better quantification of macular hole diameter, as OCT measurements are reproducible with a transverse resolution of 30 μm.

The most favourable explanation for the development of macular hole is traction caused by focal shrinkage of the perifoveolar vitreous. Also glial cells and newly formed collagen may play an important part in macular hole formation by exerting tangential traction. The diameter of the hole therefore may depend mainly on traction forces and not on the duration of the macular hole.

Ultrich et al have shown that preoperative measurement of macular hole size can be used as a prognostic factor for assessing anatomical success rate. We also calculated the hole formation factor originally created by Puliafito. He considered the ratio between the overlying dimension and the hole base diameter to be of greater influence on the anatomical success rate than the base diameter alone. Puliafito found an 80 % anatomical success rate in eyes with HFF greater than 0.9 and an anatomical success rate of less than 25 % in eyes with HFF under 0.5. However in our study 30 eyes out of 33 had a HFF of less than 0.9, the 3 eyes which had a HFF of more than 0.9 closed postoperatively. Results in our study cannot be correlated to Puliafitos assumption of HFF as a predictor for macular hole closure in view of the less number of holes with HFF more than 0.9.

The closure rate following surgery in our study of 69.69 % correlated to data reported in literature but the significance of our study was that it had the largest ever reported series of eyes with macular holes of more than 1000 microns. In our study there was an improvement of best corrected visual acuity from a
mean of 1.02 logMAR to a postoperative best corrected visual acuity of 0.87 logMAR with a mean change of 0.15 logMAR which was statistically significant (p = 0.017). Best corrected visual acuity had also improved or stabilized in 27 of 33 eyes (81.8 %).

Based on ophthalmoscopy or biomicroscopic examinations, the anatomical status of the macula after macular hole surgery was classified by Tornabe et al into three types. They suggested that flat and closed outcomes have a better visual prognosis than flat and open outcomes. Imai et al categorized the successfully repaired macular hole into three patterns with OCT: U-type (normal foveal contour), V-type (steep foveal contour), and W-type (foveal defect of neurosensory retina). The authors reported that postoperative visual acuity was well correlated with these patterns (U>V>W). The visual results obtained from the two types in our study were also similar. Because the borderline between the U and V pattern in the aforementioned study was sometimes unclear, and because the ophthalmoscopic appearance of postoperative macular hole status could be easily matched with one of the two types of closure in our study, our classification system seems more clinically relevant.

Complications of vitreous surgery for idiopathic macular hole include retinal breaks, visual field defects, cataract formation and late reopening of the macular hole. Late reopening of macular holes has been reported in 5 % to 9.5 % of eyes in the previous studies. Other than cataract formation which was subsequently treated we did not encounter any complications intraoperatively or postoperatively.

**Conclusion**

In the present study we describe the anatomical and functional outcomes of surgery for large macular holes with a minimum base diameter of 1000 microns. Our study shows that even in patients with large macular holes of greater than 1000 microns surgery has resulted in anatomical closure in 69.69 % eyes with a statistically significant improvement in best corrected visual acuity with stabilization or improvement of BCVA in up to 81.8 % eyes. To the best of our knowledge our study is the largest case series of large macular holes ever presented.

**Reference**

4. Freeman WR: vitrectomy for the treatment of full thickness stage 3 and stage 4 macular hole results of multicentered randomized clinical trial Arch Ophthalmol 1997;115;11-21
10. S Ullrich: Macular hole size as a prognostic factor in macular hole surgery Br J Ophthalmol 2002; 86; 390-393
13. Duker JS: Late reopening of macular holes after initially successful; treatment with vitreous surgery; Ophthalmology; 1994; 101; 1371-78
Population Based Assessment of Diabetes and Diabetic Retinopathy in South Kerala-Project Trinetra: An Interim Report

Dr. Manoj Soman DNB FRCS*, Dr. Unni Nair MS FRCS*, Dr. Sheena Bhilal MS**, Dr. Raeba Mathew MS FRCS*, Dr. Fazil Gafoor MS*, Dr. KGR Nair MS FRCS*

Abstract

Aim : To assess the prevalence of diabetes and diabetic retinopathy in a community based screening programme in south kerala.

Methods : Between July 2007 and June 2008, 160 screening camps were conducted in 5 southern districts of Kerala. The target population underwent blood examination, comprehensive eye evaluation including dilated examination, counseling and those with vision threatening problems were referred to the base hospital for management.

Results : 7321 out of 37174 people screened (19.9 %) had diabetes including 18.9 % new diabetics. 16.2 % of diabetics had diabetic retinopathy including 4.3 % of new diabetics. Out of 1532 gradable eyes, 86.8 % had NPDR and 13.9 % PDR. Vision threatening retinopathy was seen in 39.5 % eyes. FFA was advised in 34.1 % patients. 38.9 % eyes required laser treatment and 3.5 % eyes vitreoretinal surgery. Health education was imparted to all 37174 participants.

Conclusion : Nearly 1/5th of study population had diabetes and this alarmingly high prevalence is likely to pose a public health burden in our state. Nearly 40 % of eyes have a potential to go blind and hence a concerted effort to screen and treat these eyes and spreading awareness about the disease is the need of the hour and this project is a successful effort in this direction.

Key words : Diabetic Retinopathy, Blindness, Eye Camps, Prevalence, Kerala

Introduction

Diabetic retinopathy is a major cause of visual impairment particularly in the working age group1,2 and develops in more than 75 % of diabetics within 15-20 years of diagnosis 3,4. It is estimated that 57 million people in India may become diabetic by 2025 5 and this poses a major public health problem in our country especially as rural areas in India are rapidly urbanising. Moreover compared to the west, diabetes appears at a younger age 6, is less associated with obesity 7, and genetic factors appear to be stronger in our population 8.
These clinical differences and rising prevalence of diabetes in India\textsuperscript{9} warrant well-conducted epidemiologic studies on diabetes-related complications including eye problems to assess the health service burden due to diabetes. However there is a paucity of data on the prevalence of diabetes-related eye diseases in our country. Though there are a few related studies in our country, a true picture of prevalence of this disease is not evident because of various anomalies-some studies are clinic based \textsuperscript{10}, some amongst self reported diabetics \textsuperscript{11}, rural-urban differences and differences in examination techniques-direct ophthalmoscopy \textsuperscript{12}, indirect ophthalmoscopy \textsuperscript{12,13}, photography \textsuperscript{10}, teleophthalmology screening etc. Thus the reported prevalence of diabetic retinopathy among diabetics range from 20.8 \% to 34.1 \%\textsuperscript{10,11,12,13}. Also due to diverse cultural differences, dietary patterns and religious beliefs, the prevalence of diabetes and therefore diabetic retinopathy(DR) may be different in different parts of the country. The state of Kerala, though it boasts of a high life expectancy and literacy rate, has a high prevalence (16.3 \%) of diabetes \textsuperscript{14,15} and therefore possibly diabetic retinopathy. One study on self-reported diabetic subjects revealed retinopathy prevalence of 26.8 \%\textsuperscript{11}. There are however no large scale population based studies on prevalence of diabetes and diabetic retinopathy among our population. This study was done to assess the prevalence of diabetes and diabetic retinopathy in a community based screening programme in south kerala.

\textbf{Materials and Methods}

The study area included 5 districts in South Kerala-Trivandrum, Kollam, Alleppey, Ernakulam and Kottayam. Chaithanya Eye Hospital and Research Institute along with World Diabetic Foundation a Denmark based foundation initiated this project called project TRINETRA. A total of 160 screening camps were conducted between July 2007 and June 2008. There were 2 kinds of camps- blood screening and awareness camps where eye examination was not done, and blood screening, awareness and comprehensive eye screening camps.

All the subjects underwent random blood glucose measurement. An individual with no past history of diabetes with a random blood glucose measurement >180 mg/dl was considered a diabetic. All subjects who were diagnosed to have diabetes in the past (old diabetics) or those new patients who satisfied the above said criteria (new diabetics) were examined for diabetic retinopathy.

Demographic details, diabetic history and treatment details were recorded. Ophthalmic examination included vision testing, IOP measurement and dilated fundus examination. A trained ophthalmologist performed retinal examination with direct and indirect ophthalmoscopy. Diabetic retinopathy was categorized using the modified ETDRS classification. Retinopathy was classified as Mild, Moderate and Severe nonproliferative diabetic retinopathy (NPDR), early proliferative diabetic retinopathy (PDR), high risk PDR, advanced PDR. The presence of clinically significant macular edema (CSME) was assessed using indirect and direct ophthalmoscopy. Eyes where posterior segment examination was not possible was defined as ungradable eyes. All the above information was recorded into a proforma at the camp site which was later entered into a computerized data base created at the project office in the base hospital.

Subjects who had any form of PDR or CSME were considered to have sight-threatening retinopathy. Subjects with severe NPDR, CSME and PDR were referred for further investigation and management to the base hospital. Subjects with no or minimal retinopathy were advised to schedule follow-up with their regular ophthalmologists at yearly intervals. An expert counselor focused on awareness creation giving patients information about the disease, treatment facilities, dietary advice etc. An on-site exhibition displaying diabetic retinopathy related posters was part of all these camps. Pamphlets and booklets on the disease were given to all the camp participants.

The data necessary for the present study was extracted from the data base into an excel sheet and analyzed.

\textbf{Results}

A total of 160 screening camps were conducted between July 2007 and June 2008. A total of 37174 subjects were screened for diabetes in these camps, an average of 232 per camp. The mean age of study population was 53.2 years and ranged from 20 years to 87 years.
There were 23828 males (64.09%) and 13346 females (35.9%) in the study.

Out of the 37174 people screened for diabetes, 7321 were diagnosed to have diabetes (19.69%). This included both new and old diabetics. Out of the 7321 diabetics, 5939 were old diabetics (81.12%) and 1382 were new diabetics (18.87%). The prevalence of diabetes was 19.59% in Trivandrum, 23.65% in Kollam, 20.29% in Ernakulam, 20.33% in Kottayam and 20.46% in Alleppey district. (Table1).

Out of the 7321 diabetics only 5384 patients (comprehensive eye camp patients) underwent a detailed dilated examination. The others who were not included were those who attended only the blood screening and awareness camps. Among the old diabetic subjects the duration of diabetes ranged from 1 month to 38 years. Among the above 5384 subjects, 4307 (79.99%) were previously aware of their diabetic status. Out of this group, 1647 (38.24%) were on diet control, 2428 (56.37%) were on oral medications, and 232 (5.38%) were on insulin. Out of 4307 subjects who were diagnosed to have diabetes earlier, 13 (0.30%) had evidence of past laser treatment for diabetic retinopathy. The visual acuity of the subjects ranged from 6/6 to PL.

876 (16.27%) out of the examined 5384 subjects were identified to have diabetic retinopathy. Thus a total of 1752 eyes had some form of diabetic retinopathy. 220 eyes (12.56%) although they had some evidence of retinopathy were ungradable due to media opacities including cataract, corneal opacities etc. The grade of retinopathy was defined in 1532 eyes (87.44%) on indirect ophthalmoscopy. This included 1331 eyes (86.86%) with NPDR, 213 eyes (13.90%) with PDR and 392 eyes (25.59%) with CSME. The classification of retinopathy is detailed in Tables 2 & 3. Vision threatening retinopathy defined as the presence of PDR or CSME was seen in 605 eyes (39.49%). 6 people (0.68%) were blind due to diabetic eye disease.

Fluorescein angiography was advised in 299 patients (34.13%) and laser treatment advised in 596 eyes (38.90%). The latter group included focal laser treatment for macular edema in 392 eyes (65.77%) and panretinal photocoagulation in 182 eyes (30.53%). Vitreoretinal surgery was advised in 31 eyes (3.54%). 803 eyes were advised cataract surgery (29.83%).

Among the patients with old diabetes, 52 subjects (2.38%) had been diagnosed to have diabetic retinopathy in the past including 13 subjects who had undergone laser treatment while 802 subjects (36.72%) were newly diagnosed to have diabetic retinopathy. Among the subjects with newly detected diabeties, 22 patients (4.33%) already had some retinopathy. All the 37174 participants received health education related to diabetic retinopathy in the form of either pamphlet / booklets, diabetic retinopathy poster exhibition or expert counseling etc.

**Discussion**

Although the need for a national diabetic retinopathy screening program in India is recognized, national or regional screening initiatives are yet to be launched. Though recent studies indicate that there has been an increase in the prevalence of diabetes only a few studies have attempted to assess the prevalence of diabetic eye
complications in India. In this study, we report the prevalence of diabetes and diabetic retinopathy in an mixed urban-rural population in south India based on an epidemiologic survey. The prevalence of diabetes in this study was 19.69 % which is much higher than other population based reports in the country. Considering the fact that this estimation was based on a single random blood sample value of > 180 mg % the significance of this high prevalence cannot be underestimated. Many borderline diabetics who may have abnormal GTT were not assessed in this study and this would have increased the prevalence further. Also among the subjects with newly detected diabetics, 4.33 % already had some retinopathy, which indicates that these subjects would have had undetected or uncontrolled diabetes for a considerable period of time. The above facts indicate that diabetes is highly prevalent in this study population.

The prevalence of DR among the diabetics is 16.27 % in our study. This confirms the findings of earlier studies from India. A recent study where subjects were examined by ophthalmoscopy reported a 22.4 % prevalence, whereas a similar study on self-reported diabetics revealed a prevalence of 26.8 % and another clinic-based photographic evaluation study revealed a prevalence of 34.1 %. This study found that 86.86 % had NPDR, 13.90 % had PDR and 25.59 % had CSME. Mild NPDR was seen in 60.48 %, moderate NPDR in 29.90 % and severe NPDR in 9.62 % of NPDR eyes. CSME was seen in 24.34 % of NPDR eyes. Out of the PDR eyes 47.42 % had early PDR, 36.15 % had high risk PDR, 5.63 % had vitreous hemorrhage and 10.80 % had TRD. 31.92 % of eyes with PDR also had CSME. Overall 25.59 % of the eyes were diagnosed to have CSME. This observation is similar to other studies done in this subcontinent. Narendran et al had reported that 94.12 % of the patients with retinopathy had NPDR while 6.25 % had PDR. Of the people with NPDR 62.5 % had mild NPDR, 32.81 % had moderate NPDR, 4.69 % had severe NPDR. CSME was seen in 29.41 % of affected eyes. Dandona et al had reported that 50 % had mild NPDR, 39.3 % had moderate NPDR, 7.1 % had severe NPDR, 3.6 % had PDR and 14.3 % had CSME. However these studies did not elaborate on the eyes with vision threatening retinopathy or the type of PDR. The former study reported blindness in 1.47 % people attributable to diabetes while the latter study found none compared to 0.68 % in our study. Vision threatening retinopathy defined as the presence of PDR or CSME was seen in 39.49 % in our study. These eyes have a potential to go blind if left untreated. Many of the subjects had cataract and other causes of ungradable fundi. These patients may have had some form of retinopathy associated with the cataract, which when added would increase the prevalence of diabetic retinopathy and related blindness.

Only 13 out of 876 diabetics with retinopathy (1.48 %) had undergone evaluation and treatment for diabetic retinopathy in the past. This is less than 6.1 % of those identified with retinopathy in a study in Tamilnadu. This indicates the lack of awareness on the disease or the lack of accessibility to specialized eye care facilities in our state. Moreover there is a lack of mass screening strategies in our state compared to Tamilnadu which probably could be a reason for more subjects availing diabetic retinopathy treatment in that state.

The technique of diabetic retinopathy screening used in our study was indirect and direct ophthalmoscopy. Fundus photography is considered the standard screening technique. Though mild retinopathy detection is better on photographic screening than ophthalmoscopy, some studies have reported a significantly higher specificity for ophthalmoscopy compared with nonmydriatic photography for detecting worse than mild retinopathy. Other studies have reported a much higher sensitivity for mydriatic retinal photography compared with ophthalmoscopy for the detection of worse than mild retinopathy. However Rema et al had reported that when photographic screening is used in epidemiological studies all standard photographic fields of the fundus can not be photographed and there is a possibility that some misgrading can occur among the graders eventhough they are trained. Thus in our study it is possible that we may have missed some eyes with mild retinopathy but not any of the vision threatening lesions. Although the British Diabetic Association recommends a sensitivity of 80 % and a specificity of 95 % for screening tests, investigators in most developing countries like ours may have to choose direct
or indirect ophthalmoscopy due to the relatively high costs of photography and as mydriatic and nonmydriatic fundus photography is currently not feasible financially. However photography may be ideal for screening high-risk groups within communities, clinic/hospital based screening etc.

Handling of the increasing problem of diabetes and its danger to sight includes effective education and communication with the patients on one hand, and with physicians and allied health professionals on the other. As part of this initiative all the participants in our study received health education including materials related to diabetic retinopathy.

The relatively low prevalence of diabetic retinopathy blindness may suggest that diabetic retinopathy requires less priority and attention than the other major vision impairing diseases in India like cataracts and refractive errors that account for nearly 90 % of the current burden of blindness in India. However, it has to be realised that the projected 57 million diabetics by 2025 may drastically alter the existing pattern of blindness in India. Improving healthcare facilities in India will probably translate into a large number of diabetics living longer, and thus more diabetics are at risk for developing retinopathy. Broadening the focus of existing community screening programmes to include screening for diabetic retinopathy should be considered for early detection of retinopathy especially among the underserved populations. Formulating national policy guidelines, aiming at preventing or delaying the onset of diabetic retinopathy will ensure that diabetic retinopathy does not become a major cause for needless visual impairment or blindness in the future. Further studies are required to determine the changing magnitude of diabetic retinopathy and diabetes, as well as to understand the risk factors for diabetic retinopathy and visual loss in this population.

Inspite of certain limitations this study has given an insight into the problem of diabetes and diabetic retinopathy in our state. Nearly 1/5th of study population had diabetes and this alarmingly high prevalence is likely to pose a public health burden in our state. Nearly 40 % of affected eyes have a potential to go blind and hence a concerted effort to screen and treat these eyes and spread awareness about the disease is the need of the hour and this project is a successful effort in this direction.

Reference


Awareness on Common Blinding Conditions – A Population Based Survey

Dr. Rehna Rasheed MS DO, Dr. Cini N V, Dr. Reena A MS DO

Abstract

A population based survey was conducted to assess the awareness and knowledge on common blinding conditions in an urban & semi urban population of Trivandrum. Interviews were conducted on 1600 subjects from different socioeconomic and educational strata in a two month period. Subjects were selected by cluster sampling from different wards in Trivandrum so as to include subjects with different socioeconomic, occupational and educational background. They completed a structured questionnaire on three common diseases- cataract, glaucoma and diabetic retinopathy. Analysis were done by dividing the population into three groups-general population, plus 2 and college students and health related personnels. Awareness levels of general population were divided into four groups- 80 % of the population if aware of the target eye disease -awareness considered good, 60 %-80 % satisfactory, 30 %-60 % unsatisfactory, <30 %- poor.

Conclusion - Awareness of cataract in our population is good. Except for students and health related personnels, our population is not aware of glaucoma. Awareness of diabetic retinopathy is satisfactory. Data suggest the urgent need for community based health education programmes to shift the focus from cataract to other blinding diseases of the ageing population like glaucoma and diabetic retinopathy which require early diagnosis and treatment.

Aim of the Study

To study the awareness on diabetic retinopathy, glaucoma and cataract in an urban and semi urban population of Trivandrum.

Materials and Methods

A cross sectional study was conducted in Trivandrum on an urban and semi urban population by Regional Institute of Ophthalmology, Trivandrum. Study was conducted for a period of 2 months from April 2008 to May 2008. 1600 subjects between the age group 15-75 years were enrolled in the study. Subjects were selected by cluster sampling from 25 wards in Trivandrum and 20 houses were selected from each ward. 10 ophthalmic assistant students were trained to interview and record the responses of subjects to a structured questionnaire (Table 1) on the awareness and knowledge on three common eye diseases-diabetic retinopathy, glaucoma and cataract. Questionnaire was constructed in simple Malayalam language that can be used on the target population. Questionnaire was refined during the course of the pilot study. Having
Age Distribution in the study population

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>General Population</th>
<th>Students</th>
<th>Health related personnel</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 20</td>
<td>22</td>
<td>105</td>
<td>127</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1.80%</td>
<td>49.50%</td>
<td>7.90%</td>
<td></td>
</tr>
<tr>
<td>20 - 29</td>
<td>307</td>
<td>107</td>
<td>47</td>
<td>461</td>
</tr>
<tr>
<td></td>
<td>24.80%</td>
<td>50.50%</td>
<td>31.30%</td>
<td>28.80%</td>
</tr>
<tr>
<td>30 - 39</td>
<td>290</td>
<td>47</td>
<td>337</td>
<td></td>
</tr>
<tr>
<td></td>
<td>23.40%</td>
<td>31.30%</td>
<td>21.10%</td>
<td></td>
</tr>
<tr>
<td>40 - 49</td>
<td>189</td>
<td>28</td>
<td>217</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15.30%</td>
<td>18.70%</td>
<td>13.60%</td>
<td></td>
</tr>
<tr>
<td>50 - 59</td>
<td>283</td>
<td>14</td>
<td>297</td>
<td></td>
</tr>
<tr>
<td></td>
<td>22.90%</td>
<td>9.30%</td>
<td>18.60%</td>
<td></td>
</tr>
<tr>
<td>60 - 69</td>
<td>106</td>
<td>14</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td></td>
<td>8.60%</td>
<td>0.93%</td>
<td>7.50%</td>
<td></td>
</tr>
<tr>
<td>≥ 70</td>
<td>41</td>
<td>41</td>
<td>7.50%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3.30%</td>
<td>2.60%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1238</td>
<td>212</td>
<td>150</td>
<td>1600</td>
</tr>
</tbody>
</table>

Mean age of our population: 40.0930

Most of our study population were in the 20-59 age group.

heard of the disease was considered as awareness and having understanding of the disease as knowledge. Further questions were asked on those diseases about which the subject was aware. Knowledge on diabetic retinopathy was found by assessing their knowledge.

GENDER DISTRIBUTION IN OUR STUDY POPULATION

<table>
<thead>
<tr>
<th>Gender</th>
<th>General Population</th>
<th>Students</th>
<th>Health related personnel</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>577</td>
<td>87</td>
<td>46</td>
<td>710</td>
</tr>
<tr>
<td></td>
<td>46.60%</td>
<td>41.00%</td>
<td>30.70%</td>
<td>44.40%</td>
</tr>
<tr>
<td>Female</td>
<td>661</td>
<td>125</td>
<td>104</td>
<td>890</td>
</tr>
<tr>
<td></td>
<td>53.40%</td>
<td>59.00%</td>
<td>69.30%</td>
<td>55.60%</td>
</tr>
<tr>
<td>Total</td>
<td>1238</td>
<td>212</td>
<td>150</td>
<td>1600</td>
</tr>
</tbody>
</table>

Male : Female ratio - 0.797

DISTRIBUTION OF STUDY POPULATION DEPENDING ON EDUCATIONAL STATUS

<table>
<thead>
<tr>
<th>Educational Status</th>
<th>General Population</th>
<th>Students</th>
<th>Health related personnel</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSLC</td>
<td>492</td>
<td>15</td>
<td>32</td>
<td>539</td>
</tr>
<tr>
<td></td>
<td>39.70%</td>
<td>7.10%</td>
<td>21.30%</td>
<td>33.70%</td>
</tr>
<tr>
<td>PDC/ + 2</td>
<td>180</td>
<td>73</td>
<td>26</td>
<td>279</td>
</tr>
<tr>
<td></td>
<td>14.50%</td>
<td>34.40%</td>
<td>17.30%</td>
<td>17.40%</td>
</tr>
<tr>
<td>Graduate</td>
<td>407</td>
<td>73</td>
<td>58</td>
<td>538</td>
</tr>
<tr>
<td></td>
<td>32.90%</td>
<td>34.40%</td>
<td>38.70%</td>
<td>33.60%</td>
</tr>
<tr>
<td>Post graduate/ Professional</td>
<td>159</td>
<td>51</td>
<td>34</td>
<td>244</td>
</tr>
<tr>
<td></td>
<td>12.80%</td>
<td>24.10%</td>
<td>22.70%</td>
<td>15.30%</td>
</tr>
<tr>
<td>Total</td>
<td>1238</td>
<td>212</td>
<td>150</td>
<td>1600</td>
</tr>
</tbody>
</table>

No of subjects in the study population with educational status above and below graduation almost the same.
on the importance of regular eye check up in diabetes. For glaucoma and cataract, subjects were asked to describe what they knew about the disease. The Questionnaire contained a list of possible responses. Responses given by the subject were marked by the field investigator against the response with which it closely matched on the questionnaire.

Knowledge on glaucoma and cataract were graded as good, moderate and poor depending on the responses. 4 marks were given for each correct response on a disease. If the subject scores 12-16 marks, knowledge was considered full, 8-12 was considered moderate and less than 8 as poor.

Data was analysed by dividing the study population into general population, students and health related personnel which included hospital staff and subjects whose occupation is related to health care delivery. If 80 % of the study population is aware of the target eye disease, awareness considered good, 60-80 % satisfactory, 30-60 % unsatisfactory, <30 % not aware. Similarly with knowledge; if 80 % of aware group has full knowledge of the condition, knowledge considered good, 60-80 % satisfactory, 30-60 % unsatisfactory, <30 % no knowledge.

### Results

Data analysed using computer software, Statistical Package for Social Sciences (SPSS) version 10. Data expressed in its frequency and percentage. Chi square test was used as non parametric test to compare different parameters. For all statistical evaluations, a two tailed probability value<0.05 was considered significant.

Most of our study population comes in between 20-59 age group.

| Awareness on diabetic retinopathy was found to be satisfactory in the study population (64.10 %) and in the 3 groups, general population (61.60 %), students (69.80 %) and health related personnel (77.30 %). Difference between the 3 groups was statistically significant (P<0.001). Our general population were not aware of glaucoma (27.30 %); students–unsatisfactory (59.90 %), health related personnels satisfactory (68 %), total-unsatisfactory (35.40 %); difference between the 3 groups was significant (P<0.001). The study population had good (84.30 %) awareness on cataract. Difference in awareness between 3 groups general population (83.90 %), students (82.10 %), paramedical (90 %) was not statistically significant (P>0.05)

63 % of the aware group had knowledge on the importance of regular eye examination in diabetes; the difference between the 3 groups; general population (62.60), students (60.80 %) and health related personnels (70.70 %) was not statistically significant. 28 % of the aware group had good knowledge of glaucoma; difference between 3 groups significant (P<0.001) Students had the best knowledge of the disease (57.50 %) while general population had the least knowledge (10.70 %) For cataract 29 % has good knowledge; general population (20 %), students (60.30 %), health related personnels (58.50 %) the difference was significant (P<0.001)

### Discussion

Awareness on common eye diseases plays an important part in encouraging people to seek proper eye care and treatment. A similar study was conducted by Dandona et al in Andhra Pradesh – Awareness on common eye diseases in an urban population in southern India. This

### DISTRIBUTION OF STUDY POPULATION ON AWARENESS ON COMMON EYE DISEASES (Table-5)

<table>
<thead>
<tr>
<th>Awareness</th>
<th>General Population</th>
<th>Students</th>
<th>Health related personnel</th>
<th>Total</th>
<th>Chi square</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetic Retinopathy</td>
<td>762</td>
<td>148</td>
<td>116</td>
<td>1026</td>
<td>17.921</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>61.60%</td>
<td>69.80%</td>
<td>77.30%</td>
<td>64.10%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glaucoma</td>
<td>338</td>
<td>127</td>
<td>102</td>
<td>567</td>
<td>276.22</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>27.30%</td>
<td>59.90%</td>
<td>68.00%</td>
<td>35.40%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cataract</td>
<td>1039</td>
<td>174</td>
<td>135</td>
<td>1348</td>
<td>4.591</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td></td>
<td>83.90%</td>
<td>82.10%</td>
<td>90.00%</td>
<td>84.30%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
study was conducted on 2522 subjects on an urban population in Hyderabad. But few studies have been conducted in Kerala. We conducted a study on the awareness of common blinding diseases of the aged population in the capital city of Kerala, which has 100% literacy state.

Awareness on diabetic retinopathy in our population was satisfactory (64%). Students and health related personnels were more aware of diabetic retinopathy; the difference between 3 groups was statistically significant (<0.001). Knowledge on diabetic retinopathy in our population was also satisfactory (63%), the difference between the 3 groups was not statistically significant (>0.05). The Hyderabad study showed 27% awareness on diabetic retinopathy with 34.3% subjects knowing the importance of regular eye examination in diabetes.

Awareness on glaucoma in our study population is unsatisfactory (35%) with general population not aware (27%), students unsatisfactory (59%) and health related personnels satisfactory (68%); the difference was significant. And only 10% of general population had good knowledge on the disease. Hyderabad study showed an awareness of 2.4% & knowledge 86.7%. The present study shows that students have much better awareness and knowledge (57.50%) on glaucoma compared to general population. Due to the silent nature of this disease, early detection is difficult unless the patient undergoes eye examination. So an increase in awareness of the condition is important if more people are to be screened for this condition.

The awareness on cataract is good (84%) in our study population. The difference between the 3 groups was...
Though the awareness was good, only 29% has good knowledge of the disease. General population- (20%) students-60%, health related personnels 58%, the difference was statistically significant. The Hyderabad study also showed a satisfactory awareness with poor knowledge.

Awareness on all eye diseases is more in 30-50 age group and this difference was significant (<0.001). Education played significant role in awareness of diseases. It is found to be more in subjects with an educational status of graduation and above; this difference was found to be significant (<0.001) Females were found to have better awareness than males, but the difference was not statistically significant.

### Conclusion

1. Awareness on cataract is good in our population.
2. Awareness on glaucoma is not satisfactory in our population. Students are more aware of the disease. General population has no awareness.
3. Awareness on diabetic retinopathy is satisfactory.
4. The General population has no knowledge on cataract and glaucoma.
5. Knowledge on diabetic retinopathy is satisfactory.

Our data suggest the urgent need for health education programs in order to increase the awareness and knowledge on eye diseases so that patients could seek timely eye check up, thus decreasing the burden of visually impaired. This is especially important in our state, which claims 100% literacy and a health status similar to developed countries. This study highlights the importance of shifting the focus from cataract to other blinding conditions in the aged population like glaucoma and cataract that requires early diagnosis and treatment.

### References

1. Awareness on common eye diseases in an urban population of southern India by Dandona et al. Bulletin of world health organ, 2001, 79(2) 96-102, E pub 2003 Sep 18
5. Ophthalmologica 2006; 220(2):101-8
Comparison of Visual Field Defects in Normal-Tension Glaucoma (NTG) and Primary Open Angle Glaucoma (POAG)

Dr.Vijaya Pai H. MS, Dr.Himabindu Veluri MS

Purpose: Comparison of visual field defects in patients with Normal tension glaucoma (NTG) and Primary open angle glaucoma (POAG) may reveal a difference in the pathogenesis of the two conditions.

Methods: We compared closely matched visual fields of 37 eyes with NTG and 22 eyes with POAG which were performed using the 30-2 program of Humphrey perimeter.


Results: In the mild defects 46.2 % of the NTG group showed defects close to fixation compared to 33.3 % of the POAG group (P = .687). The NTG group had a PSD of 6.78 dB compared to 5.87 dB in the POAG group (P = .648). 53.9% of eyes in the NTG group had vascular risk factors compared to 33.3% in the POAG group (P = .5).

Conclusion: Results suggest that field defects of NTG and POAG are similar and should thus not be used to hypothesize different pathogenic mechanisms.

The association between increased intraocular pressure and glaucoma is well known \(^1\), \(^2\), \(^3\). However, the exact role of intraocular pressure in the pathogenesis of glaucoma remains obscure. Since the description of a patient with glaucomatous optic nerve damage and visual field loss in the absence of increased intraocular pressure by Von Graefe, the relationship between intraocular pressure, optic nerve damage, and visual field loss has aroused controversy. However, there has been little agreement in published reports as to the similarities and differences of these characteristics in patients with normal-tension glaucoma and patients with high-tension glaucoma \(^2\), \(^3\), \(^4\). Some investigators have shown that visual field defects in patients with normal-tension glaucoma as compared to patients with high-tension glaucoma tend to be of sudden onset \(^2\), closer to fixation \(^2\), \(^4\), \(^6\), deeper and steeper \(^2\), \(^4\), more localized or less diffuse \(^7\), \(^9\), more common in the superior hemifield \(^10\), \(^11\) have a greater amount of localized visual field loss in the inferior hemifield \(^5\). The studies of Caprioli and Spaeth \(^4\), which suggested that normal-tension glaucomatous visual field defects were closer to fixation and had steeper slopes, were challenged by Phelps, Harry \(^1\), and Montague \(^1\) who pointed out the inappropriateness of the method used in making these quantitative assessments. Similarly, other investigators have found no differences between the visual field defects in patients with normal tension glaucoma and patients with high-tension glaucoma \(^12\) to \(^15\). The reasons for such controversial reports seem to be caused by
differences in study designs, including different types of perimetry, various definitions of normal-tension glaucoma regarding the highest recorded intraocular pressure, different stages of optic nerve damage and visual field loss, and different methods used in statistical analysis of the data. This study was designed to compare the visual field defects of patients with normal-tension glaucoma and patients with high-tension glaucoma by using strict criteria for defining both types of glaucoma and closely matching both groups with respect to the amount of visual field loss.

**Patients and Methods**

This study was conducted at Kasturba Medical College, Manipal. Patients seen from January 2003 to February 2006 in the Out Patient Department of Ophthalmology were selected. We used 37 eyes of 24 patients with Normal-Tension Glaucoma (NTG) and 22 eyes of 16 patients with Primary Open Angle Glaucoma (POAG) for comparison in this non-randomized comparative trial. Criteria for inclusion in the NTG group included a maximum recorded intraocular pressure (IOP) of <21mm Hg before the initiation of therapy and for the POAG group, a maximum recorded IOP of >21mm Hg by Goldmann applanation tonometry. CCT was measured with Ocuscan [Alcon] and the IOP was adjusted accordingly. All patients had glaucomatous optic nerve damage, open anterior chamber angles on gonioscopy, a diurnal IOP curve done without therapy with a minimum of 6 measurements, a visual acuity of 20/60 or better, a pupil diameter of at least 3 mm, at least two reliable visual field tests (fixation loss <20%, and false positive and false negative errors <33 %) using C 30-2 program of Humphrey perimeter, and glaucomatous visual field loss (according to Anderson’s criteria). Cup/disc ratio was determined by binocular examination of the dilated fundus using slit lamp biomicroscopy with a 90 Dioptr double aspheric lens. When binocular examination was not possible due to a poorly dilated pupil, direct ophthalmoscopy with monocular clues such as disc vessel bending was used to determine the cup/disc ratio.

Patients were excluded if they had history of trauma or any ocular disease known to affect the visual field such as diabetic retinopathy, macular degeneration and vascular occlusions. Also, patients with a visual acuity < 20/60, pupil diameter of <3mm, unreliable visual field tests, optic neuropathy other than glaucoma, local disc malformations or neurologic abnormalities were also excluded. The visual field defects were classified into mild, moderate, severe and end-stage based on Advanced Glaucoma Intervention Study (AGIS) 16 scoring system. Visual field defects of the NTG group were matched with those of the POAG group for the severity of damage, for comparison. All visual field examinations were performed by trained technicians. Mild defects in the NTG group were compared with mild defects in the POAG group, moderate with moderate, and so on and so forth. A whole group analysis was also done in which all the visual fields defects in the NTG group were compared with all the field defects in the POAG group, irrespective of the severity of the defect.

To determine if there was a difference in the frequency of visual field defects close to fixation between patients with NTG and patients with POAG, the number of patients in each group with scotomas extending to within 6° of central fixation were counted. Patients were counted if any of one of the central 4 threshold values in the pattern standard deviation plot decreased to 10 dB or more below the age-corrected normal values, or if any of the central 4 threshold values along with contiguous values decreased 5 dB or more below age-corrected normal values.

To determine if there was a difference between the two groups with respect to localized visual field loss, pattern standard deviation was compared as the NTG group and the POAG group were matched for the severity of the defect. The number of eyes in each group with a more severe involvement of either the superior or the inferior hemifield was also noted in order to determine which hemifield is more commonly involved in each group and also to determine if there are any differences in the frequency of involvement of a hemifield between the two groups. A hemifield was considered to be more involved if the PSD plot showed at least 3 or more contiguous locations with p = 0.005 in that half of the visual field 17. Chi-square analysis, t-test and Mann-Whitney tests were used for statistical analysis.
Results

The total number of eyes in the NTG group were 37, and in the POAG group, 22. The demographic data for the two groups were analyzed. In the mild defects, as reported by other investigators, the NTG group had a younger mean age and a larger mean cup/disc ratio. However, the differences were not statistically significant [Table 1]. 46.2% of the NTG group showed defects close to fixation compared to 33.3% of the POAG group. We found no statistically significant difference in the number of patients with scotomas falling within 6° of central fixation (p = 0.687). Although the NTG group had a larger PSD than the POAG group, (6.78 ± 3.25 vs. 5.866 ± 1.55), this difference was not statistically significant (p = 0.648). 53.83% of the NTG group had vascular risk factors compared to 33.3% of the POAG group. Once again, the difference was not statistically significant. The superior hemifield was more involved in both groups, although there was no statistically significant difference in the frequency of involvement between the two groups [Table 2]. A similar comparison was carried out between the two groups for each category of the field defect that is, moderate, severe, and end-stage. However, the comparison done between mild defects is given more significance as the defects tend to look similar in both groups, as they progress in severity. It is during the mild stage that any true differences can be established with more certainty.

Discussion

Since the description of an entity called normal-tension glaucoma, in which glaucomatous optic disc changes with visual field defects occur in the presence of a normal intraocular pressure, various hypotheses such as vascular ischemia have been put forward in an attempt to discover if differences exist in the pathogenic mechanisms of NTG as compared to those of POAG. Differences in the pattern of visual field loss in the two groups may lead towards such a difference. Unfortunately, due to various differences in the way previous studies were conducted, there has been little agreement between investigators, as to the similarities or differences in the visual field defects between the two groups. Some investigators found that visual field defects were closer to fixation in patients with NTG than in patients with POAG. However, others have found no significant differences in the two groups with respect to involvement of fixation. In our study, in the mild defects, we found no significant differences in the number of patients with NTG and patients with POAG with scotomas extending to within 6° of central fixation.

Studies have also shown that patients with NTG have more localized defects and those with POAG have more generalized defects. We investigated these findings by comparing the PSD values (localized visual field loss) between the two groups. Although the NTG group had a greater amount of localized visual field loss than the POAG group, this difference was not statistically significant. This finding is similar to that of Zeiter, Shin et al, who found no significant difference in the PSD values between patients with NTG and POAG.

In our study we also found a tendency toward involvement of the superior hemifield in both groups. The finding that patients with early to moderate POAG have visual field defects that involve the superior hemifield more than the inferior hemifield has been documented. In addition, many investigators have studied whether there is any difference in the distribution of visual field defects over the superior and inferior hemifields between patients with NTG and POAG. Although many have found no differences between the two groups, some have found a greater involvement of the superior hemifield in the NTG group as compared to the POAG group. We found no statistically significant difference in the distribution of field loss over the superior and inferior hemifields between the two groups.

Many authors have hypothesized that vascular factors in addition to IOP have an important role in the pathogenesis of normal tension-glaucoma. However, our results showed no significant differences in the vascular risk factors between the two groups.

In conclusion, although this study does not imply that there are no different pathogenic mechanisms between NTG and POAG, it does show that there are no differences in the visual field defects between the two groups and hence visual field defects may not point towards differences in pathogenic mechanisms.
References


Advances in biomedical engineering and ocular surgical techniques has encouraged the development of sustained release drug delivery implants to treat a variety of ophthalmic diseases. Polymer based drug delivery implants placed in the vitreous cavity have great efficacy in treating posterior segment diseases such as cytomegalovirus retinitis, uveitis, diabetic macular edema etc. Intermediate, posterior and panuveitis are typically the more severe forms of uveitis, resulting in highest incidence of visual loss. These forms of uveitis often require long-term systemic treatment with oral corticosteroids, often in conjunction with other immuno modulating therapy (IMT). Side effects of oral corticosteroids are many and can be life threatening; thus reduction of corticosteroid usage with the help of IMT is a primary goal in uveitis treatment strategy. While IMT and targeted biological agents are increasingly used as corticosteroid sparing therapy, the fluocinolone acetonide implant is the first and currently the only sustained release intraocular implant approved by the FDA for the long term control of non infectious posterior uveitis (Fig. 1). Its efficacy has been well documented. The multicentre uveitis steroid treatment (MUST) trial compares the efficacy of standardized systemic therapy vs. fluocinolone acetonide implant therapy for the treatment of severe cases of intermediate uveitis, posterior uveitis or panuveitis.

**RETISERT (Fluocinolone Acetonide Intravitreal Implant)**

Fluocinolone acetonide intravitreal implant is indicated for the treatment of chronic non-infectious uveitis affecting the posterior segment of the eye. The drug substance is the synthetic corticosteroid, fluocinolone acetonide. It is less soluble in aqueous solution than dexamethasone (Fig. 2).

The fluocinolone acetonide implant is a sustained release system (Fig. 3) designed to deliver fluocinolone...
acetonide over 30 months. Each implant consists of a silicone elastomer containing fluocinolone acetonide 0.59 mg. It delivers drug at an initial rate of 0.6 micrograms/day over the first month, decreasing to a steady rate of 0.3 – 0.4 micrograms/day over approximately 30 months. In patients with noninfectious posterior uveitis (NIPU), the implant reduces inflammation and preserves visual acuity over an extended period. Its most common side effects include elevated IOP and development of cataract.

Recently Callanan et al. published the 3-year clinical trial results from the Fluocinolone Acetonide Uveitis study group. The purpose of this multi-center, randomized study was to evaluate the safety and efficacy of 0.59 mg and 2.1 mg fluocinolone acetonide implants in NIPU. Uveitis recurrence in implanted eyes was reduced from 62% to 4%, 10.1% and 20% at 1, 2 and 3 years following implantation, respectively, for the 0.59 mg treatment group and from 58% to 7%, 17% and 41% respectively for the 2.1 mg treatment group. The rate of uveitis recurrence was low during the period corresponding to the active life of the implant but began to increase towards the end of the drug delivery period. There was no difference in the post implantation uveitis recurrence rates between the 2 groups during the first 2 years following implantation, but there was a significant difference during the third post implantation year, which was thought to be secondary to faster drug delivery with the original 2.1 mg implant. The need for adjunctive therapy to manage uveitis was significantly reduced after implantation, with 80% reduction in the use of systemic medication to control inflammation. Thus the fluocinolone acetonide implant is superior to intravitreal triamcinolone injection for uveitis. Implanted eyes had a higher incidence of IOP elevation (> 10 mm Hg) than non implanted eyes and surgical mode of intervention was required in 40% of implanted eyes vs. 2% of non implanted eyes to control the intraocular pressure.

Chich et al. reported recently that fluocinolone acetonide implant insertion can be combined safely with phacoemulsification and IOL implantation in the same surgical sitting in eyes with uveitis. Other applications for fluocinolone acetonide implants such as for macular oedema associated with central retinal vein occlusion and diabetes melitus have been reported.

Clinical Pharmacology: Corticosteroids inhibit the inflammatory response to a variety of inciting agents and probably delay or slow healing. They inhibit edema, fibrin deposition, capillary dilation, leukocyte migration, capillary proliferation, fibroblast proliferation, deposition of collagen and scar formation associated with inflammation. Corticosteroids act by the induction of phospholipase A2 inhibiting proteins, collectively called lipocortins. It is postulated that these proteins control the biosynthesis of potent mediators of inflammation such as prostaglandins and leukotrienes by inhibiting the release of their common predecessor arachidonic acid. Arachidonic acid is released from membrane phospholipids by phospholipase A2.

Pharmacokinetics: In a subset of patients who received the intravitreal implant and had blood samples taken at various times after implantation, plasma levels of fluocinolone acetonide were below the limit of detection (0.2 ng/ml) at all times. When aqueous and vitreous humor samples were assayed for fluocinolone acetonide in another subset of patients, detectable concentrations of fluocinolone acetonide was seen throughout the observation interval (up to 34 months), the concentrations were highly variable, ranging from below the limit of detection (0.2 ng/ml) to 589 ng/ml.

Medidur: The Medidur (Alimera) implant also contains flucocinolone, but it is much smaller. It is a reservoir implant, but it is not sutured to the eye wall and is allowed to float freely in the vitreous space.
Posurdex: Posurdex biogradable dexamethasone implant (Allergan) is a different concept. It’s biogradable system is not reservoir based, it uses polymer and drug mixed together and the drug releases over time. As the polymer degrades, the drug is released.

Dosage and Administration: Each vitrasert implant contains a minimum of 4.5 mg of ganciclovir and is designed to release the drug over a 6 to 8 month period of time. The rate of release is approximately 1 μg/hr. Following depletion of ganciclovir from the implant, as evidenced by progression of retinitis, the implant may be removed and replaced.

Contraindications and Complications of Intravitreal Implants:
As with any surgical procedure, potential complications accompanying intra ocular surgery to place the implant into the vitreous cavity include cataract, increased intra ocular pressure, vitreous loss, vitreous haemorrhage, retinal detachment, choroidal detachment, temporary reduction in visual acuity, endophthalmitis, hypotony and wound dehiscence. Following implantation of the implant, most patients experience an immediate and temporary reduction in visual acuity in the implanted eye which lasts for approximately two to four weeks postoperatively. All the patients should be monitored for elevated IOP regularly.

VITRASERT (Ganciclovir Intravitreal Implant)
Vitrasert implant contains a minimum of 4.5 mg of the antiviral drug ganciclovir. The implant is designed to release ganciclovir at a rate of approximately 1 μg / hr over a 6-8 month period (Fig.4).

Pharmacology: Ganciclovir is a synthetic nucleoside analogue of 2 – deoxyguanosine that inhibits replication of herpes viruses both in vitro and in vivo. Sensitive human viruses include cytomegalovirus (CMV), herpes simplex virus 1 and 2, Epstein Barr virus (EBV) and varicella zoster virus.

Emergence of viral resistance has been reported based on invitro sensitivity testing of CMV isolates from patients receiving intravenous ganciclovir treatment. The prevalence of resistant isolates is unknown and there is a possibility that some patients may be infected with strains of CMV resistant to ganciclovir. Therefore, the possibility of viral resistance should be considered in patients who show poor clinical response.

Indications: Vitrasert is indicated for the treatment of confirmed CMV in patients with AIDS.

Vitrasert implant is contraindicated in patients with hypersensitivity to ganciclovir or acyclovir.

VITRAVENE (Fomivirsen Sodium Intravitreal Implant)
Vitravene is indicated for the local treatment of cytomegalovirus (CMV) retinitis in patients with AIDS who are intolerant to or have a contradiction to other treatment for CMV retinitis.

Conclusion
Traditional treatment routes cannot obtain sufficient therapeutic concentrations for treatment of posterior
segment diseases of the eye due to anatomic and physiologic protective mechanisms such as blood retinal barrier. For ideal drug treatment, the drug should reach the target tissue directly and at the desired time and concentration. For this purpose, liposomes, biodegradable polymers and non degradable implants have been developed in the last 20 years. Liposomes did not proceed beyond limited clinical studies due to a great deal of disadvantages. The most important disadvantage of non degradable implants are the need for surgery and wide incisions. Biodegradable polymers produce the best results due to properties like ease of implantation, no need to remove and excellent bioavailability. Thus intravitreal implants provide safe and effective therapy. Promising efficacy results show a significant reduction in recurrence rate and improvement in visual acuity.

References:

Fundus Autofluorescence Imaging

Dr. Meena Chakrabarti MS DO DNB

Fundus auto fluorescence (FAF) imaging is a novel imaging method that allows topographic mapping of lipofuscin distribution in the retinal pigment epithelial (RPE) cell monolayer as well as of other fluorophores that may occur with disease in the outer retina and the sub neurosensory space. Excessive accumulation of lipofuscin (LF) granules in the lysosomal compartment of RPE cells represents a common downstream pathogenetic pathway in various hereditary and complex retinal diseases including age related macular degeneration (AMD). FAF imaging has been shown to be useful in understanding the pathophysiological mechanisms, diagnostic, phenotype-genotype correlation, identification of predictive markers of disease progression, and monitoring of novel therapies.

Most ocular media and tissues exhibit fluorescence emission upon excitation by a suitable wavelength of light. Light is absorbed by fluorophores, causing electrons to become excited to higher electronic state. The electrons remain in an excited state for a nanosecond, and their energy is emitted as they return to their ground state. Ocular fluorophores are endogenous and are visible in the cornea, lens and retinal pigment epithelium 1.

Ocular fluorophores exhibit marked changes in their fluorescence properties with regards to age and pathology. Hence they can be used as indicators of ageing or as diagnostic tools in diseases 2.

Fluorescence of the retinal pigment epithelium is mainly related to lipofuscin, a fluorescent pigment that is absent in fetal and newborn retinal pigment epithelium 3. Lipofuscin continuously accumulates in the retinal pigment epithelium as a result of incomplete digestion of spent rod outer segment disk. It is potentially noxious, acting as a photo sensitizer in blue light and generating free radicals both in isolated granules and within the retinal pigment epithelium (RPE). Lipofuscin has several distinct fluorescent components, of which A2E the red emitting fluorophore is very important as it is responsible for retinal pigment epithelial apoptosis, mediated via inhibition of lysosomal digestion of proteins and blue light mediated disruption of lysosomal membranes 4.

A 40 % increase in the fluorescence intensity of lipofuscin is commonly considered as an indicator of ageing. Abnormally high lipofuscin content in the retinal pigment epithelium has been demonstrated in a variety of inherited retinal disorders such as Bests’ disease, Stargardts disease, Fundus flavimaculatus and in age related macular degeneration.

Intact human lipofuscin granules exhibit a broad band excitation spectrum from 300 to 620 nm, and an

Chakrabarti Eye Care Centre, Kochulloor, Trivandrum 695 011
E-mail: tvm_meenarup@sancharnet.in

Fig 1 Normal fundus autofluorescence imaging.
emission spectrum that peaks in the yellow-orange region. Melanin, the other main chromophore of the retinal pigment epithelium, though nonfluorescent, exhibits age-related fluorescence properties probably due to the combination with lipofuscin. The excitation spectrum of melano-lipofuscin fluorescence is 364 nm and emission maximum at 540 nm (Fig 1).

Using a confocal scanning laser ophthalmoscope characteristic patterns of fundus autofluorescence in normal subjects and in patients with different retinal disorders have been described. Fundus autofluorescence can also be studied using the fundus camera to a certain extent.

**Fundus Auto Fluorescence Changes in early Age-related Macular Degeneration:**

Alteration in the fundus autofluorescence in age-related macular degeneration can be classified into 9 phenotypic patterns (Fig 2) including normal, minimal change, focally increased, patchy pattern, focal plaque like, linear, lace-like, reticular and speckled.

**Normal Pattern:** of autofluorescence is characterized by

1. Homogenous background autofluorescence
2. Gradual decrease in fundus autofluorescence towards inner macula and fovea due to masking effect of yellow macular pigment.
3. Normal pattern may be seen even in the presence of soft/hard drusen.

**Minimal Change Fundus Autofluorescence Pattern:** is characterized by limited irregular increase or decrease in background fundus autofluorescence without any obvious topographic pattern.

**The Focally Increased Pattern:** is characterized by the presence of at least one area < 200 μm in diameter with markedly increased fundus autofluorescence. This area will be much brighter than the surrounding background autofluorescence. This spot has well-defined borders and is usually surrounded by an area of gradually decreasing fundus autofluorescence (dark halo). These areas may correspond to areas of visible alterations in colour fundus photographs such as focal hyper pigmentation or drusen.

**Patchy Pattern:** is characterized by a larger ill defined area (>200 μm) of markedly increased fundus autofluorescence surrounded by progressively increasing background autofluorescence. These areas may correspond to a large soft drusen or areas of hyper pigmentation.

**Linear Pattern:** is characterized by the presence of at least one linear area with markedly increased fundus autofluorescence, typically well demarcated corresponding to hyper pigmented lines on fundus photograph.

**Lace-like Pattern:** shows multiple branching linear structures of increased fundus autofluorescence forming a lacy pattern corresponding to hyper pigmentation in the colour image.

**Reticular Pattern:** is characterized by the presence of multiple small areas <200 μm in diameter of
decreased fundus autofluorescence with progressively decreasing fundus autofluorescence from the centre of the lesion toward the surrounding background fluorescence. This pattern is associated with cluster of small soft drusen, hard drusen and areas of pigmentary changes (Fig. 3).

**Speckled Fundus Autofluorescence Pattern:** has the simultaneous presence of a variety of fundus autofluorescence abnormalities in a larger area of the fundus autofluorescence image.

**Geographic atrophy** in dry age-related macular degeneration is characterized by areas of retinal atrophy. Due to atrophy of retinal epithelium cells and lack of lipofuscin, fundus autofluorescence imaging in patients with geographic atrophy shows decreased fundus autofluorescence intensity over the atrophic patches (Fig 4 a & b).

Geographic atrophy (GA) represents the atrophic late-stage manifestation of “dry” AMD. During the natural course of the disease, atrophy slowly enlarges over time and the fovea itself is typically not involved until later (“foveal sparing”). Due to the distinct changes of the topographic distribution of RPE LF, the signal is markedly reduced over atrophic areas. The high – contrast difference between atrophic and non atrophic retina permits precise quantification of the atrophic areas on FAF images using customized image analysis software. This allows accurate assessment of progression of atrophy and can be used in longitudinal observations, including interventional trials. (Fig 5)

The identification of elevated levels of FAF intensities in the junctional zone of atrophy is of particular interest as these changes precede cell death. Studies using FAF imaging have reported distinct phenotypes in the distribution and appearance of these areas, while there was a high degree of intraindividual symmetry 6, 7.

Current data on spread of atrophy suggest that there is a linear growth of atrophy over time and that the best
predictor would be the growth rate in the previous year. Using FAF imaging, it has been shown that the extension of areas of increased auto fluorescence surrounding atrophy patches correlates with atrophy progression over time. Further analysis within the Fundus Autofluorescence in Age-related Macular Degeneration (FAM) Study summarizing the data of 195 eyes demonstrated that the great variability and range of atrophy enlargement between patients is largely dependent on the specific pattern of FAF abnormalities at baseline outside the atrophic patches. Eyes with the banded and the diffuse FAF pattern showed a more rapid enlargement compared with eyes without FAF abnormalities and the focal FAF pattern. Within the diffuse pattern group, eyes with a diffuse trickling pattern exhibited an even higher spread rate (median 3.02 mm²/year) compared to the other diffuse types. As there is high degree of intraindividual symmetry, genetic determinants rather than non specific ageing changes may be involved.

In the junctional zone surrounding atrophy, areas of increased fundus autofluorescence intensities and excessive retinal pigment epithelium lipofuscin load can be identified. This area of increased fundus autofluorescence has been clearly shown to precede new patches of atrophy or enlargement of preexisting atrophic patches. Atrophy enlargement varies between 0 and nearly 14 mm²/year with a mean rate of progression between 1.74 mm²/year and 2.79 mm²/year. Very small areas show slower spread of atrophy, but variations in atrophy enlargement cannot be totally explained by baseline atrophy (Fig.4).

Areas of atrophy can be accurately delineated, quantified with image analysis software and atrophy progression rates can be calculated. Hence it is an easy, feasible, noninvasive imaging technique to review patients with geographic atrophy over time. (Fig 6)

Patients with neovascular age-related macular degeneration with choroidal neovascular membranes if of recent onset shows areas of hyper fluorescence on fundus fluorescein angiography corresponding to areas of normal AF with adjacent areas of increased fundus autofluorescence. Preserved AF indicates viable retinal pigment epithelium initially which has implications for treatment interventions and long term visual prognosis. However in patients with choroidal neovascular membrane of long duration the fundus autofluorescence over the affected area is decreased indicating loss of retinal pigment epithelium and photoreceptors (Fig 6).

Theoretical consideration would suggest that FAF imaging may give important clues in choroidal neovascularisation (CNV) secondary to AMD. It may be helpful to assess the integrity of the RPE, which may influence the development and behavior of new vascular complexes as well as photoreceptor viability and potential therapeutic success.

Patients with early CNV secondary to AMD tend to have patches of continuous or normal auto fluorescence

Fig 6 Focal areas of decreased FAF is prevalent in classical CNV indicating loss of retinal pigment epithelium and photoreceptors

Fig 7. Fundus autofluorescence in chronic central serous retinopathy demonstrating inferior ‘track’ sign.
corresponding with areas of hyper fluorescence on the comparative fluorescein angiograms, implying that RPE viability is preserved at least initially, during CNV development. By contrast, eyes with long standing CNV typically exhibit more areas of decreased signal.

Comparing FAF finding with the classification of occult and classic CNV based on fluorescein angiography, focal areas of decreased FAF were reported to be more prevalent in classical CNV in comparison to larger occult CNVs\(^\text{16}\). Mc Bain and associates confirmed this findings and speculated that typical low FAF signals at the site of the CNV are related to absorption phenomena caused by the CNV growing in the subretinal space, rather than being related to severe damage to the RPE\(^\text{10}\). However a more recent study could not demonstrate any significant effect in FAF alterations between occult and classic CNVs secondary to AMD\(^\text{21}\). A continuous pattern of preserved auto fluorescence in the central macula was observed in most patients, and this was correlated with better visual acuity, shorter symptom length, and smaller lesion size.

In contrast to the data in patients with advanced atrophic AMD, the predictive value of areas with increased FAF intensities remains unclear. Looking at 125 eyes with soft drusen and no history of laser treatment, a longitudinal analysis (mean follow up: 18 months) within the FAM study reported 9 eyes with development of advanced exudative AMD during the review period. Six of these 9 eyes exhibited the so- called “patchy” FAF pattern at baseline, which may represent a high-risk marker for progression to advanced AMD.

**FAF in Acute and Chronic Recurrent Central Serous Chorioretinopathy:**\(^\text{13}\)

Definite FAF patterns can be made out in both acute and chronic CSR. AF is therefore an interesting tool to apply to differentiate between acute and chronic CSR. In acute CSR, decreased AF is due to blockage caused by oedema. Whereas in chronic recurrent forms irregular and increased AF is observed reflecting reactive RPE changes secondary to RPE defects and neurosensory detachment. Another change observed is the decreased AF at the point of leakage due to SRF blocking the AF or RPE atrophy at the leakage point itself as is presumed in chronic recurrent cases.

**FAF in STGD-FFM (Stargardts- Fundus Flavimaculatus):**\(^\text{14}\)

Although previous reports found high levels of AF in all patients with Stargardts fundus flavimaculatus disease some may have normal or low levels of FAF (Fig 8). There seems to be a relationship between patterns of AF and peripheral functional abnormalities. Patients with low levels of AF at the centre of macula, including the fovea and normal / low levels of AF...
temporally and nasally had peripheral rod and cone dysfunction. Thus it appears that patients with this pattern of autofluorescence have more wide spread disease. However there appeared to be no relationship between the degree of AF at macula and macular dysfunction as detected by PERG, since all patients had marked PERG abnormalities independent of levels of AF. (Fig. 9)

**FAF in Macular Holes:**

Autofluorescence imaging is useful for the diagnosis and staging of macular holes and is comparable with the results of fluorescein angiography. AF imaging demonstrates the bright fluorescence of macular holes with an appearance similar to that obtained in fluorescein angiography. In contrast macular pseudo holes showed no AF. The attached operculum in stage 2 and the detached operculum in stage 3 macular holes showed focally decreased AF. The associated retinal elevation and cuff of SRF were less fluorescent compared with the background AF of the normal fellow eyes. Following successful surgical treatment, the AF of macular holes was no longer visible.

Being noninvasive and rapid, AF imaging may become a useful alternative to fluorescein angiography in the assessment and differential diagnosis of full thickness macular holes.

![AF - Macular Hole](image)

**Fig. 10:** Fundus autofluorescence imaging in macular holes demonstrating increased autofluorescence at areas of the hole.

**References**


In a Lighter vein

Patients and Patience

RRV

Several attributes make up a successful practitioner of Ophthalmology. Good clinical sense and better common sense are among them. But if you ask me the most important one would be patience. It is like Patience begets Patients. Patience is most needed while asking questions and listening to the answers. But some patients very definitely test the patience of even the most patient of you.

Classically you allow the patient to talk about his/ her symptoms in his/ her own words. But there are some whose words just will not stop. The Verbal Diarrhoea Type. They will go on and on about the background of their illness till you want to interfere. “And she was telling me about that beauty parlour lady who stays in the corner house. Mind you, I never listen to gossip. It seems this lady... blah...blah... blah... and then I suddenly felt a pain in my left eye and I asked her to stop”. By that time you would want to ask her to stop. But you just grin and bear it. But sometimes real significant facts do come out of the jumble.

Then there is the Know All Type who many a time is a relative of Mrs. Malprop. “Then Dr. So and so told me that I had BP of the eye and asked me to use ‘Toilet’ eye drops twice a day. My sister also has BP of the eye but uses ‘Latrine Post’ eye drops”. She would blithely confess. Or it would be about the ‘plain glasses’ given to him for his ‘far sightedness’ by the doctor in Bangaluru. They may provide some lighter moments.

The Forgetful Type can be equally testing. In spite of you printing “Bring this with you on the next visit”, the previous prescription is invariably forgotten. And so are the present glasses. “I thought I will check my eyes while waiting for the appointment with my beautician. That is why I didn’t bring my glasses”. And when you ask if they remember the power of the glasses, fifty percent will say ‘point five’ and the other fifty percent ‘two point five’.

The Doubting Type can put gray hairs on your scalp. ‘Read the last line’, you would say. “The very last?” ‘Yes’. (Is there any other ‘last’?). “At the bottom?” ‘Yes, yes’. “The Smallest line?” ‘Yes’, you would say tearing out the (grey) hairs by the handful. And he will read ‘To be held in good light 14 inches from the eye’! And he is the one who will call you up at 11 pm to ask if he should put two or three drops each and which eye to put the drops first, right or left. Some years back I had a post-op. patient who used to come with a list (yes, list of doubts written down). ‘Can I go up the stairs?’ ‘Can I go down the stairs?’ were two of them. I was sorely tempted to answer the first in affirmative and the second in the negative.
Phacoemulsification in Post Vitrectomy Cataracts

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

Introduction

Cataract development is one of the most common complications after vitrectomy; it develops in 12.5% to 80% of eyes. The risk factors for the development and progression of cataract are older age, degree of preoperative nuclear sclerosis, intraoperative lens touch, diabetic retinopathy, and silicone oil injection. The mechanisms of development of cataract in eyes which have undergone prior vitrectomy are many. These include lens touch with intraocular instruments, intraocular tamponading agents (silicone oil, gas), crystallization process involving anterior hyaloid or posterior capsule resulting in reversible posterior capsular lens feathering and the influence of blood or inflammation leading to free radical release in the posterior segment. The indications for pars plana vitrectomy are fast expanding and the number of patients undergoing this procedure is increasing because of the improved surgical results. Therefore, the number of vitreoretinal surgeries is rising each year and hence a significant increase in the volume of vitrectomized patients, who in their vitrectomized state pose a challenge to the cataract surgeon.

Risks in Vitrectomized Eyes

The vitrectomized eyes are at a higher risk of developing intraoperative and postoperative complications due to following factors:

1. The eyes harbor sequelae of previous surgery and inflammation
2. They have associated comorbid conditions
3. Nuclear brunesence (denser cataracts) is common
4. They lack the support of vitreous gel.

The higher risks stem from the fact that these eyes are anatomically different from eyes that have not undergone prior surgery. The following factors need to be taken care of while dealing with postvitrectomy cataracts:

(a) Conjunctival scarring

Conjunctival scarring makes conjunctival dissection for fashioning the scleral tunnel difficult. If the patient has undergone prior scleral buckling procedure, scarring around the bridled extraocular muscle makes exposure difficult and insufficient. Increased episcleral scarring and bleeding should be anticipated in the vicinity of previous scleral ports. Therefore, it is preferable to perform phacoemulsification through a clear corneal incision than through a scleral tunnel. A rigid IOL implantation through the clear corneal incision may necessitate enlarging the incision and securing it with sutures at the conclusion of the procedure.

(b) Compromised corneal endothelium

In the postvitrectomized eyes, the corneal endothelium is often compromised especially in those cases where silicone oil is present in the anterior chamber

(c) Poor pupillary dilatation

In most vitrectomized eyes, the pupil does not dilate properly despite the use of mydriatics before phacoemulsification.
(d) **Zonular weakness and preexisting posterior capsular rent**

The chances of having compromised zonule, loose capsular bag, and pre-existing posterior capsular dehiscence are more in patients who have undergone lengthy vitreoretinal surgery, multiple procedures, and vitrectomy involving vitreous base dissection.

(e) **Low scleral rigidity**

Low scleral rigidity is seen in vitrectomized eyes especially in patients with high myopia. In these patients additional support using a Fleeringa’s ring or a separate self maintaining infusion may prove useful.

(f) **Cystoid macular edema**

Vitrectomized eyes are predisposed to a high incidence of cystoid macular edema.

(g) **Diabetic retinopathy**

Worsening of preexisting diabetic retinopathy necessitates meticulously scheduled postoperative follow-up for early detection of worsening and management.

(h) **Increased lens-iris diaphragm retropulsion**

An increased lens-iris diaphragm retropulsion or infusion deviation syndrome\(^\text{12}\) can occur in vitrectomized eye.

**Preoperative Considerations**

A thorough preoperative evaluation taking into account the patients’ compromised ocular health, structural changes resulting from trauma of the earlier surgical procedure and the poor visual potential is necessary in formulating a definite surgical plan. A good preoperative surgical planning helps to formulate strategies for the expected or anticipated intraoperative difficulties and goes a long way in giving the patient the best overall visual benefits.

**History**

A detailed history on the nature of the vitreoretinal pathology and the extent of previous surgery is important as they have a direct bearing on the success or complexity of the phacoemulsification procedure and its overall benefit to the patient. An eye which has undergone a limited vitrectomy and in whom the clear anterior vitreous cortex is left untouched will behave differently from one which has undergone multiple procedures involving anterior vitreous base excision for complex vitreoretinal pathology. Lengthy procedure and vitreous base excision may increase the likelihood of developing zonular dehiscence and occult posterior capsular rupture.

Patients with diabetic retinopathy should be considered for surgery only after their retinopathy is under control, since there is a good chance for worsening of the retinopathy following cataract surgery.

Cystoid macular edema may occur more frequently after cataract surgery in vitrectomised eyes and hence it is prudent to start the patients on preoperative topical nonsteroidal anti-inflammatory drops.\(^\text{13}\)

**Preoperative work-up**

**Preoperative examination** should make a note of following important findings:

The presence of conjunctival and episcleral scarring, endothelial cell count, deep anterior chamber and presence of emulsified silicone oil bubbles in it, presence of iridophacodonesis indicating compromised zonules, pupillary status, and detailed examination of retinal status especially to assess integrity of macula, and presence of open breaks.

In eyes with advanced cataract, evaluation with an indirect ophthalmoscope may not be feasible and an assessment is made using a B-scan. B-scan in the presence of silicone oil, however, gives very few relevant details and has limited utility.

**Intraocular lens power considerations**

Intraocular lens (IOL) power considerations differ in eyes without silicone oil tamponade and eyes having silicone oil tamponade.

**Eyes without silicone oil tamponade**

The IOL power considerations are the same as for a standard phacoemulsification in eyes without silicone oil tamponade.

**Eyes having silicone oil tamponade**

Silicone oil is used in vitreoretinal surgery as a tool to reduce retinal detachment and in the presence of proliferative vitreoretinopathy among other conditions.
In these eyes IOL power measurements are to be considered under two headings.

(i) Measurement of the Axial Length

Measuring the axial length (AL) of an eye filled with silicone oil can be a challenging situation because of various reasons. The low velocity within the silicone oil will cause an erroneous measurement and hence it is also necessary to correct the apparent axial length to true axial length in silicone oil filled eyes.

On B-scan examination the globe appears elongated with an unfocused appearance. On the echogram, the retinal echospike is small and difficult to display due to the sound attenuation within the liquid silicone. The system sensitivity should be increased to better identify the retinal spike.

When the silicone oil fill is incomplete, it may move around, giving rise to shifting retinal echoes, increasing the difficulty in obtaining an axial length measurement. In supine position, the silicone oil will rest on the retina while the liquefied vitreous will layer on top of it. Hence if measurement is made in the supine position (as is usually done in the immersion technique) the ultrasound beam will cross the area of liquefied vitreous first before crossing the area filled with silicone oil. Separate measurements for these two areas will be necessary to accurately measure the vitreous cavity depth. In these circumstances it is advisable to perform biometry while the patient is seated. In this position the residual liquefied vitreous will move towards the superior position of the vitreous cavity, leaving only part of the silicone oil in the optical axis where the measurement is taking place.

In silicone oil filled eye, sound travels slowly (1550 m/s in normal phakic eye vs. 1000 m/s in silicone oil filled eye). This factor makes it difficult to obtain axial length measurement.

The viscosity of the silicone oil, used as semi permanent tamponade during vitreoretinal surgery, may vary from 1000 cSt to 5000 cSt. The refraction and axial length measurements may vary with the oil viscosity. Eyes filled with 5000 cSt silicone oil tend to have higher changes in refraction and axial length than eyes filled with 1000 cSt oil. The 1000 cSt oil has a velocity of 980 m/s whereas the velocity of sound in 5000 cSt oil is 1040 m/s.

The low velocity within the silicone oil causes an erroneous measurement of vitreous cavity depth (VCD). The formula to correct AL in any silicone oil filled vitreous is:

1. VCD\textsubscript{1532} = AL - (ACD+LENS)
2. VCD\textsubscript{corrected} = VCD\textsubscript{1532} \times (1/1532) \times 980 \text{ m/s} \ (or \ 1040 \text{ m/s depending on viscosity of silicone oil used.})
3. AL\textsubscript{corrected} = VCD\textsubscript{corrected} + ACD + LENS

(1532 is the average velocity of sound in aqueous and vitreous)

The conversion factor of 0.71 multiplied by the measured axial length has been reported to correct for the apparent increase in axial length induced by silicone oil of viscosity 1000 cSt.

In case of a cataract not so advanced or mature to impair fundus examination, partial coherence interferometry (IOL Master, Zeiss) is preferred over ultrasonography to calculate the axial length of the silicone oil filled eye.

In cases of eyes filled with gas or perfluorocarbons, ultrasound echoes are blocked.

Unsuitable formulas, artifacts or large eyes beyond the machine range may cause significant errors. In certain eyes it is impossible to obtain the axial length. The following options may then be considered:

(a) Measure axial length before vitreoretinal surgery and silicone oil injection
(b) Measure axial length after silicone oil removal
(c) Measure axial length of the fellow eye (provided the patient is not one-eyed)
(d) CT-scan image can be used to measure axial length in eyes with incomplete silicone oil fill.
(e) The final option would be to consider the use of standard power IOL.

(ii) Calculation of an appropriate IOL power

Silicone oil tamponade alters the optics of the eye. The index of refraction of silicone oil (1.405) is higher than that of the vitreous gel. The higher refractive index of silicone oil makes it behave like an intraocular minus lens. Therefore, without appropriate power adjustment,
significant hyperopic overcorrection would be expected. As a result, standard theoretical and regression lens power formulas calculate a lens power which is less than needed to achieve emmetropia, resulting in a hyperopic refractive error.

The more curvature or power incorporated in the posterior surface of the lens, the greater is the postoperative error.

If the silicone oil is to be retained in the vitreous cavity at the time of IOL implantation the surgeon should consider adding 3-8 D to the calculated IOL power, depending on lens shape to achieve emmetropia. If silicone oil removal is performed at a later date patient should be forewarned that there will be a myopic shift. This shift is greater in the presence of a biconvex than planoconvex lens with the plane surface facing posteriorly. Eyes with the posterior meniscus IOLs experience the smallest change. Thus when silicone oil is filling the vitreous cavity the rule of thumb to arrive at the necessary IOL power is as follows:

(a) Use convexo-plano IOL to minimize effect of silicone oil (add 3 to the calculated IOL power).

(b) If using a biconvex lens, add 6 D to the calculated power.

(c) When silicone oil removal is performed 2-5 D of induced myopia should be expected.

In some patients the silicone oil tamponade has to be executed for long periods of time. In these cases IOL power adjustments have to be considered as silicone oil alters the refractive power of the posterior surface of the IOL.

Patel (1995) and Meldrum have suggested using the following correcting formula to find the additional IOL power to be added to the calculated IOL power to arrive at the power of IOL to be implanted in a silicone oil filled eye:

\[
\text{Additional IOL power} = \frac{(N_s - N_v)}{(A_L - A_C D)} \times 1000
\]

- \( N_s \): Refractive index of silicone oil (1.4034)
- \( N_v \): Refractive index of vitreous (1.336)
- \( A_L \): Axial length in millimeters
- \( A_C D \): Anterior chamber depth in millimeters

Choice of Intraocular Lens (IOL)

Both the hydrophobic and hydrophilic acrylic IOLs have been associated with consistently satisfactory outcome and have been well tolerated by the eye. A rigid PMMA IOL may also be considered. A silicone IOL should be avoided in an eye that has undergone prior vitrectomy. Also one piece plate haptic design lenses and lenses with small and ovoid optics should be avoided. Silicone oil can interact with the posterior surface of the IOL in patients with a posterior capsular rent impairing visual acuity as well as fundus visualization both intra and postoperatively. Postoperatively, these patients complain of defective vision and presence of rainbows or haloes around light. Silicone oil adhesion to IOL surface is maximum with the silicone IOL. However, it can also occur with hydrophobic acrylic, PMMA, and hydrophilic acrylic lenses in a decreasing order. A surface modified heparin coated IOL can reduce the postoperative reaction. A lens with a 360 degree square edge design with a large optic diameter (6-6.5 mm), which gives a greater viewing area for fundus visualization, is preferred. A plano convex configuration of the implanted IOL with the plano surface facing posteriorly ensures minimal refractive surprises. The absence of positioning holes helps reduce posterior synechiae formation postoperatively.

Preoperative Patient Counseling

Preoperative patient counseling plays a important role in mentally preparing the patient for the visual outcome after surgery. It is necessary to give the patient a realistic idea of his visual potential as well as to make him aware of the expected and unexpected intraoperative events that can complicate his surgery.

Patients with significant posterior segment comorbid conditions may have only very minimal visual improvement. The benefits of the surgical intervention to the patient may be an improved color perception, better peripheral vision, or only a better view of the fundus for the ophthalmologist. The patient may be bothered by diplopia, metamorphopsia, central scotoma or anisometropia. Paradoxial anisekonia with smaller images in the silicone oil filled eye may trouble the patient. A series of unexpected adverse intraoperative events may further mar the visual outcome of
phacoemulsification surgery in vitrectomized eyes and includes peripheral corneal injury, stripped Descemets membrane, fluctuations in AC depth, floppy iris, miotic pupil, tears in rhexis margin, marked zonular laxity/dehiscence, posterior capsular plaque, unplanned posterior capsulorhexis, unplanned AC IOL, posterior capsular rent, nucleus drop and suprachoroidal hemorrhage.

**Surgical Strategy for Phacoemulsification in Vitrectomized Eyes**

**Preoperative**

Long acting cycloplegic and nonsteroidal anti-inflammatory drops (NSAIDs) should be started at least one week prior to the surgery. This may help to maintain adequate mydriasis throughout the phacoemulsification procedure.

**Anesthesia**

It is important to decide on the type of anesthesia. Injection anesthesia is preferred by many surgeons although the procedure can be safely performed under topical anesthesia. Injection anesthesia is preferred in patients with hard cataract and associated comorbid conditions like subluxated cataract, small pupil, etc. When performed under topical anesthesia, the patient may experience discomfort during maneuvers that stretch the zonular apparatus, when there is excessive movement of the lens-iris diaphragm. This undesirable sensation may be eliminated by intracameral nonpreserved lidocaine. Enhanced posterior diffusion of the anesthetic drug through zonule may cause transient blindness due to temporary retinal block.

Injection anesthesia, by increasing the orbital volume, may negate the vitreous pressure and reduce deepening of anterior chamber. Digital massage and oculocompressive devices should be avoided as the eye may become hypotonus.

**Intraoperative Problems and Surgical Technique**

The intraoperative problems to be anticipated during phacoemulsification include: ocular hypotony, deep anterior chamber, disturbed vitreous dynamics, compromised zonule, poor pupillary dilatation, loose capsular bag, pre-existing posterior capsular rent, posterior capsular plaque, dropped nuclear fragments, and infusion deviation syndrome.

Ocular hypotony can be countered by firming the globe with viscoelastics. An infusion cannula may be placed through an inferotemporal sclerotomy port and the flow used to firm the globe. To prevent excessive deepening of the anterior chamber, it is advisable to perform the surgery at a reasonable low infusion bottle height and other phaco parameters are also adjusted appropriately. The microscope magnification (zoom) can also be adjusted at a low level to enhance the depth of focus.

1. **Incision**

A clear corneal incision is preferred to a scleral tunnel incision. In the presence of compromised zonular apparatus, the incision should be opposite to the area of zonular dialysis. Both the phaco and side port incisions should be carefully fashioned to avoid fluid leakage since fluid dynamics become increasingly important in these eyes.

A scleral tunnel incision is preferred if the patient opts for a rigid IOL or in a very challenging case where surgeon may require to convert to a large incision non phaco technique. A fornix based conjunctival flap can be dissected which may be difficult, due to the scarring. The conjunctival flap should be anchored at the periphery at the conclusion of surgery.

2. **Capsulorhexis**

Capsulorhexis may be challenging in view of the increased prevalence of anterior capsular fibrosis in many eyes. The red fundal reflex may also be compromised due to the posterior segment pathology, advanced nature of the cataract at the time of presentation and a lusterless cornea in some patients. Therefore, it is prudent to stain the anterior capsule with trypan blue dye to enhance its visibility.

A sharp cystitome should be used for capsulorhexis. It is prudent to keep a pair of microrhexis forceps and scissors handy. One may need to incise the fibrotic areas with microrhexis scissors. Every effort should be made not to deepen the anterior chamber excessively, during injection of viscoelastic. A large rhexis of about 5 to 5.5 mm should be fashioned. This facilitates nuclear emulsification, reduces the incidence of posterior
capsular opacification and capsular phimosis and promotes adequate fundus visualization during postoperative follow-up.

3. Hydrodissection steps

Hydrodissection must be slow and gentle keeping in mind the possibility of preexisting posterior capsular rent. Slow and gentle hydrodissection followed by frequent decompression should be done to avoid a posterior capsular blow-out. It is necessary to verify that adequate nuclear rotation has been achieved to prevent further stress on the compromised capsulozonular apparatus. In eyes presenting with mature white cataracts after vitrectomy, the possibility of lens touch and occult capsular rupture should be kept in mind. In these cases instead of hydrodissection, a gentle hydrodelineation and / or hydro free dissection may be performed prior to removal of nucleus.

4. Nucleus management

A technique of nuclear emulsification that is least traumatic to the capsulozonular apparatus should be employed. A direct phaco chop technique is believed to be the least traumatic. However, the surgeon may employ any technique which he/she is comfortable with and may include stop and chop or divide and conquer technique. Post pars plana vitrectomy cataracts are denser than the senile cataract and therefore, more time has to be spent in emulsifying the nucleus. Care must be taken not to cause thermal burns to the cornea and not to apply excessive force on the lens while emulsifying it.

Notable fluctuation of anterior chamber depth may occur because of increased movement of the lens-iris diaphragm. If the prior vitrectomy has spared the clear anterior vitreous cortex the fluctuations will be minimal. Excessive fluctuations can be reduced by keeping the bottle height low and maintaining irrigation whenever the phaco probe or irrigation-aspiration probes are in the eye. These patients are prone to infusion deviation syndrome wherein the fluid migrates posteriorly through the weakened zonule. It increases the volume of the vitreous compartment and causes shallowing of the anterior chamber. Raising the infusion bottle has the paradoxical effect of further shallowing the anterior chamber.

5. Cortical clean-up

Cortical clean-up should be thorough and performed using lower I/A parameters and circumferential stripping to reduce stress on zonule. A bimanual irrigation-aspiration system is very efficient for safe and complete cortex removal. Gentle posterior capsular polishing should be performed to reduce the incidence of postoperative posterior capsular opacification.

6. Small pupil strategy

As mentioned earlier, a long acting cycloplegic and NSAIDs should be instilled in the postoperative period. The surgeon should utilize a step-wise approach to small pupil management (Posterior synechiolysis, viscomydriasis, pupillary membrane dissection, stretch pupilloplasty, and iris hooks). The Malyugin ring is also a good option. Intraoperative manipulations and anterior chamber depth fluctuation should be minimized.

7. Dense posterior capsular plaques

Marked posterior capsular fibrosis or plaques are quite common in silicone oil filled eyes. Centrally located plaques may be visually significant and need to be removed. Many plaques may be removed by capsular polishing or dissection with 26 gauge needle. Once an edge is created it can be peeled off with Utrata forceps. Very dense plaques may be managed by including them in the primary posterior capsulorhexis.

8. IOL implantation

IOL should be implanted in such a position so as to ensure long-term fixation and stability as well as to optimize visualization of the posterior segment. Regardless of the IOL design, placement must be gentle, avoiding excessive rotational maneuvers.

If there is zonular dialysis, use of capsular tension ring (CTR) ensures that the capsular bag is evenly distended and the IOL is placed with one haptic oriented in the direction of the dehiscence.

At the conclusion of surgery, hypotony should be avoided by reforming anterior chamber, and ensuring water-tight closure. Altered scleral rigidity and peritalal scarring may necessitate suture placement to close the phaco and sideport incisions.
Postoperative Management

Rigorous postoperative management is necessary to prevent postoperative inflammation, secondary glaucoma, posterior synechiae and cystoid macular edema (CME). Use of topical steroid drops, nonsteroidal anti-inflammatory drops and cycloplegics are absolutely necessary. Subconjunctival injection of mydriatic agents and steroids should be considered if the intraocular inflammation is not under control. Postoperative fibrin may be managed by successful use of intracameral tPA. Vigilant management is necessary to detect and treat CME and worsening of diabetic retinopathy.

Complications

Early postoperative complications

Early postoperative complications include blepharoptosis, moderate to severe corneal edema, intraocular pressure spike, wound leak, moderate to severe postoperative iritis, peaked pupil with vitreous in the wound, iris prolapse, incorrect IOL power, IOL decentration or dislocation, endophthalmitis, macular phototoxicity, retinal detachment and vitreous hemorrhage. Silicone oil migration to the anterior chamber may occur early as well as late in the postoperative period.

Late postoperative complications

Late postoperative complications which occur more than a week after surgery include blepharoptosis, moderate to severe corneal edema, pseudophakic bullous keratopathy, chronic iritis, irregular pupil, neovascularisation of iris, capsulorhexis contraction (4.5 %), IOL decentration (4.5 %), and posterior capsular opacification (31.8 %). Posterior segment complications include new or persistent macular edema (13.6 %), persistent recurrent choroidal neovascular membrane (CNVM), proliferative diabetic retinopathy, reopened macular hole (2 - 3 %), retinal detachment, visually significant epiretinal membrane (15.9 %) and vitreous hemorrhage (4.5 %).

Conclusion

By recognizing the differences in the physiologic state of the vitrectomized eye, and keeping in mind the nature of the patients’ previous vitreoretinal pathology, the modern cataract surgeon may readily adapt a small incisional phaco technique to this challenging patient population. The principles discussed in this chapter will hopefully minimize surgical difficulty and help reduce complications.

References

Anterior Lamellar Corneal Replacement

Dr. Sreenivas K. Rao MS

1. Rationale

Although the technique of lamellar replacement of the anterior corneal layers has been in use for quite sometime and was first described in 1830 by von Walther, initial success rates were less encouraging, largely due to the presence of interface scarring. With an improved understanding of corneal anatomy and the evolution of surgical techniques and instrumentation, an increased interest in such techniques in the recent past has resulted in increasing popularity of this approach.

The idea of retaining the healthy host endothelium and limiting the surgical replacement of cornea to the diseased anterior stromal layers is obviously attractive – since most of the immunological consequences of the host immunological response affect the endothelium of the graft. While epithelial rejection is possible, it is seldom of serious import, and the occurrence of stromal rejection is quite rare. Achieving a regular stromal bed while ensuring the removal of all diseased stromal tissue and maintaining the transparency of this interface in the postoperative period however, is technically demanding and it is possible that the presence of this interface may result in a quality of vision that is slightly less than that achieved by penetrating keratoplasty.

To ensure successful lamellar corneal surgery, it is important to pay attention to the surgical tenets described by Jose Barraquer. He proposed that it was important to achieve the deepest possible interface, with a uniform posterior stromal surface, and smooth sectioning of both the recipient and the donor interfaces, and to ensure that the interface remained free of deposits. In addition it is important to ensure that the donor is of appropriate thickness, the edges coapt well, and appropriate suturing techniques are used to ensure a good refractive outcome. Improvements in the surgical instrumentation makes many of these possible today.

However, the major advance has been the understanding that the deeper the dissection in the cornea, the less the interface scarring. This is made possible by the differential structure of the posterior corneal layers which course across the entire width of the cornea without a break. In general the posterior stroma is more ordered, more hydrated, more easily swollen, and has a lower refractive index than the anterior stroma. The posterior lamellae are also wider and thicker (100-200 μm wide and 1.0-2.5 μm thick) than the anterior (0.5-30 μm wide and 0.2-1.2 μm thick). There are also differences in keratocyte morphology a more regular arrangement of the collagen layers and a lesser keratocyte density.

Extending this concept of deep corneal dissection, is the recognition that a potential plane of cleavage exists between the posterior stroma and the Descemet’s membrane. Identification of this plane facilitates the removal of essentially the entire stroma leaving only the residual Descemet’s membrane. However, a recent study indicates that this separation may occur within the banded and non-banded portions of the Descemet’s membrane in some eyes. However, achieving this plane of dissection ensures an optimum quality to the interface, and most of the recent advances in anterior lamellar corneal surgery have focused on the development of techniques that permit the safe and reproducible creation of this plane during surgery.
2. Direct surgical dissection

With this approach, the goal is often to remove the bulk of the anterior corneal stroma by manual dissection using direct visualization, initially described by Anwar.\(^9\)

Of course, it is important to ensure that the residual stromal bed does not contain any of the opacified, scarred corneal tissue. In the event that the residual stroma contains opacified tissue, further dissection is performed in layers, until clear stroma is reached. An attempt to reach the plane of the Descemet's membrane is made only in the case of very deep opacities, since the risk of perforation increases greatly as the dissection is carried into the deeper layers. Normally, about 90% of the stromal tissue is removed as this provides the advantages of working in the posterior stromal layers, highlighted previously, and also produces a bed in which the donor tissue can be easily fitted.

The surgical procedure can be performed using orbital local anesthetic infiltration and begins with marking of the corneal center. An appropriately sized trephine is then used to mark the area of corneal excision, centered on the corneal mark. Prior pachymetric mapping of the corneal surface will provide an idea of the thinnest corneal measurement and the trephine is set to cut to about 80% of this measurement. Alternatively, after the trephine makes the initial cut, the incision is deepened using a sharp knife in a free hand fashion. This allows the creation of a circular, vertical cut in the cornea following the mark of the trephine. Once a satisfactory depth has been reached, the cut central edge is held with corneal forceps and dissection is started in a horizontal plane at the base of the groove. This can be performed using a Grieshaber blade (#681.01), a crescent knife or the Bard-Parker #15 blade. When performing the dissection, it is important to ensure that the plane of the dissection remains uniform to achieve a smooth bed. In order to do this, the corneal stroma is retracted with the corneal forceps and the stretched corneal fibers are gently stroked with the blade held parallel to the posterior stromal surface. In this manner the dissection is advanced across the corneal surface and when the peripheral trephine dissected groove is reached, the central disc of tissue can be lifted free of the cornea.

Several layers of tissue can be removed in succession using the same technique in order to ensure the two goals of removing all the opacified stroma and achieving a stromal bed that is only about 10% thick. As the dissection proceeds posteriorly, it may be necessary to make a paracentesis that will ensure a low intraocular pressure. When working close to the Descemet's membrane, a high intraocular pressure can predispose to perforation of the thin posterior layers. Since the central corneal clarity is most critical for achieving a good visual outcome, once the initial dissection of the entire bed is completed, further attempts to remove tissue may be limited to the central part. Initiating the dissection can be performed by gently scratching on the exposed stromal surface using a sharp needle or knife. As the stromal fibers part, a new plane is created that can then be developed. The technique using the knife, as described above, can be used or a thin, smooth spatula can be inserted into the opening created and advanced in a sweeping maneuver to further separate the stromal layers prior to excision. Deroofing a 3 to 4 mm central window of Descemet's membrane or further thinning of the stroma in this region may suffice. A modification of this approach is the “divide and conquer” technique described by Tsubota et al.\(^10\)

3. Injection to separate Descemet’s membrane

As mentioned earlier, it is preferable to achieve a plane of cleavage between the posterior stroma and the Descemet’s membrane as this results in minimal interface scarring. A variety of techniques have been described to help achieve this endpoint in a safe and reproducible manner. Variations and refinements have been described by Archila\(^11\), Rostron\(^12\), Sugita\(^13\), Melles\(^14\) and Anwar\(^15\). Briefly, the technique requires the use of air, viscoelastic or fluid injection into the corneal stroma and depends on the ability of the injected material to enter the pre-Descemet's space cleaving the stroma from the membrane. This separation allows the safe removal of the stroma retaining the Descemet's membrane.

Anwar described what he termed the “big bubble” technique, using air injection. Stromal trephination is first performed and between 60 and 80% of the stromal thickness is trephined using a suction trephine. A 27 or 30 gauge needle is attached to an air filled syringe and bent at 60° with the bevel facing down. The needle is
introduced into the trephined groove and advanced into the paracentral corneal stroma with the bevel facing down. This helps avoid inadvertent penetration of the Descemet's membrane and also facilitates the ability of the injected air to find the pre-Descemet's plane. When the needle tip is in the desired position, air is injected and an immediate whitening of the corneal stroma is noted due to the entry of air into the stromal lamellae. In an ideal situation a large bubble with a circular outline is noted, between the Descemet's membrane and the deep stroma and this is the desired end point. If however, there is only a diffuse whitening of the stroma with no clear bubble formation, the process may be repeated at a second and third site. The injection of air often results in an increase in intraocular pressure, possibly due to the entry of air through the trabecular meshwork. A paracentesis is performed after air injection to release some of the aqueous to reduce the intraocular pressure. It is important however, to avoid performing the paracentesis before air injection, since the entry of air through the cut in the Descemet's membrane may result in enlargement of the tear. The anterior stroma is excised as described previously, exposing the roof of the “big bubble”. A sharp knife is then used to enter the bubble tangentially, and the opening is carefully enlarged to permit the introduction of a spatula or cannula. The air usually escapes during this maneuver resulting in a collapse of the bubble. At this time, a low intraocular pressure is desirable and the paracentesis is used to release more aqueous. Using the entry created by the knife, a spatula is introduced to achieve further separation of the stroma from the Descemet's after which the stromal layers are excised using scissors. Alternatively, viscoelastic can be used to achieve the same effect, and this also helps by coating the surface of the Descemet's membrane and prevents the scissors from dragging on the surface of the dry membrane.

During excision, it is important to avoid excessive traction on the corneal stroma as the attached fibers transmit the force to the delicate Descemet's membrane and can result in perforation. Making radial cuts in the roof of the bubble and removing the stroma in segments can also help. At the periphery of the stromal bed, a shelving cut is used to allow the retention of some stroma – as this allows safe suturing of the graft-host junction. The use of fluid and viscoelastic injection is based on similar principles, but is less efficient and increases the risk of Descemet's rupture, as compared to the air injection technique. An excellent review of these techniques and finer details are available in an article by Anwar and Teichmann.16

4. Closed dissection using spatulated dissectors

In an alternative approach that attempts to produce a defined depth of dissection in the corneal stroma, Melles 17 used a closed approach and semi-sharp spatulas to effect a smooth plane in the dissected bed. The essence of this technique is to determine accurately the initial depth of entry into the corneal stroma as this determines the plane of subsequent dissection as well. In order to determine this depth, Melles has described a few signs 18. After creating an initial scleral incision at about 80 % depth, a specially designed blunt spatula (DORC) is used to dissect into the peripheral corneal stroma. A paracentesis is made and the aqueous is completely replaced by air. The depth of dissection is checked by looking for the reflection of the instrument from the optical interface between the air and cornea. Two reflections are seen – one each form the anterior and posterior corneal surfaces, and the position of the posterior corneal surface is halfway between these two reflections. As the spatula is advanced into the corneal stroma, it can be used to indent the air bubble, and this results in a semicircular specular reflex. The tip of the blade is separated from the reflex by the uncut posterior stroma, which appears as a dark band. The thinner the band, the deeper the position of the dissector in the corneal stroma and when this band is a thin dark line, the position is approximately 90%. With the spatula in this position a sweeping movement is performed in a side-to-side manner to create a pocket and using specially designed semi-sharp spatulated dissectors (DORC), the dissection is progressively increased to created a lamellar dissection of the entire corneal stroma from limbus to limbus at the same plane.

After this has been satisfactorily completed, viscoelastic is injected into the dissected pocket and a trephine used to cut through the anterior corneal layers. The viscoelastic protects the posterior layer from damage. After excision of the anterior stroma, surgery proceeds
as with other techniques. With this approach however, no attempt is made to reach the Descemet’s membrane.

5. Donor preparation

Since the accent in anterior lamellar keratoplasty today is to perform deep excision of the recipient stroma – be it to Descemet’s or 90% depth, preparation of the donor button is relatively easy as the entire thickness of the button can be fitted into the stromal bed. Early attempts to use the donor button with the Descemet’s membrane intact resulted in wrinkling of the membrane and possibly a greater potential for scarring and inflammation at the interface. Histopathologic evidence also indicates that retention of the Descemet’s membrane can result in weaker graft-host bond and could contribute to the formation of a pseudo anterior chamber at the interface. It is therefore preferable to remove the Descemet’s membrane and endothelium from the donor button. This can be accomplished by staining the endothelium with trypan blue and using a dry weck cell to detach the Schwalbe’s line at the periphery and then rub off the entire membrane. Alternatively, after punching the appropriate size of corneal donor tissue, a fine forceps can be used to hold the cut edge of the Descemet’s membrane and peel the entire circular disc as one continuous sheet. In an eye in which only a partial thickness stromal removal has been performed, a dissection at a similar plane must be achieved in the donor – using either a whole donor globe, or the corneoscleral rim in an artificial anterior chamber in order to facilitate an appropriate recipient bed and donor tissue match. Such dissection is however technically more demanding and may result in greater potential for interface scarring.

It is also important to use talc free gloves during the procedure and also ensure that the interface is free of lint or other debris. After thorough flushing of the stromal bed to remove residual viscoelastic, the donor button is placed in the bed and sutures are used to secure it in position. Care must be taken to ensure that the sutures achieve an even distribution of tension and that no perforation of the Descemet’s membrane occurs. The use of air in the anterior chamber is helpful in promoting adhesion of the two layers, especially when the Descemet’s membrane has been bared.

6. Conclusions

Anterior lamellar keratoplasty has improved significantly in the past few decades and is now a viable option in the management of corneal opacities that do not involve the Descemet’s membrane and endothelium. Improvements in our knowledge of corneal surgical anatomy, wound healing, surgical instrumentation and techniques now allows the safe and reproducible use of this technique in selected patients.

References


Solar Maculopathy- An OCT Study

Dr. Manoj Soman DNB FRCS, Dr. Fazil Gafoor MS

Solar maculopathy is a rare form of photic retinopathy and is caused by sun-gazing without protection. Retinal damage is believed to be an effect of the photochemical changes that occur at the time of sun observation and in most cases appears to be reversible. The advent of newer diagnostic tools like the optical coherence tomography has enabled us to study such macular pathologies in detail.

We report a case of bilateral solar maculopathy. A 15 yr old female student presented 24 hrs after direct sun gazing with symptoms of blurred vision and central scotoma. The act was done as part of a religious ritual as advised by her friends. Total direct viewing time was approximately 20 min. On presentation, her visual acuity was 6/18 both eyes. Anterior segment examination was within normal limits. The intraocular pressures were normal in both eyes. Ophthalmoscopic examination revealed a bilateral yellowish-white spot in the center of the foveal region (Fig 1a,b). Rest of the fundus was normal on examination.

Optical coherence tomography (OCT) examination revealed bilateral full thickness increased reflectivity in the foveal region associated with normal central foveal thickness (Fig 2a,b). These findings were suggestive of a full thickness burn of the foveal region with photoreceptor damage. In view of the severity of the burn and poor vision in both eyes, she was treated with IV Dexamethasone 8 mg BD for 3 days.

The visual acuity improved to 6/9 in the right eye and 6/6 in the left eye by 10 days and fundus examination revealed RPE mottling in the fovea and OCT revealed almost normal foveal appearance with subtle hyporeflectivity in the involved part of the macula (Fig 3a,b).

By 1 month, the visual acuity was 6/6 in both eyes with normal OCT findings. At 12 months follow up the patient complained of persistent mild distortion of vision. The visual acuity remained 6/6 in both eyes and high definition 3D OCT examination revealed the presence of a focal defect in the IS/OS junction above...
the intact RPE suggestive of a microhole in both eyes (Fig. 4 a,b). The patient was reassured and advised periodic follow up.

Discussion

Solar maculopathies are rare retinal problems and are seen following direct or indirect sun gazing. Common visual complaints after an acute injury include blurred vision, central scotoma, afterimage and erythropsia. Fundus examination usually reveals a typical small yellow white foveolar lesion which may fade off after a period of 1 to 2 weeks and may be replaced by a normal looking fovea or lamellar hole. Fluorescein angiographic evaluation is usually normal but minor RPE window defects may be seen. Visual acuity is usually diminished in the range of 6/12 to 6/60 but may also be normal. Visual acuity usually recovers to 6/6 to 6/9 range over a period of 6 months.

Variability in the appearance and severity of solar maculopathy is due to the differences in exposure parameters and patients. Lengthy exposure with good fixation usually produce the most severe damage. Although solar retinopathy was initially believed to be due to retinal photocoagulation, photochemical damage appears to be the predominant mechanism. Retinal temperature rise from solar observation are usually too low for thermal damage to occur. Brief solar observation during a solar eclipse is potentially dangerous due to pupillary dilatation. However even with a constricted pupil sustained solar observation for more than 90 seconds (eg; in a religious ritual or under the effect of psychotropic drugs) will exceed the threshold for photochemical retinal damage and may cause photocoagulation. These severe foveal burns may cause more visual deficit and have a poor outcome and hence conservatively managing these situations may not be prudent. Intravenous steroids are used to treat inadvertent foveal burns during retinal photocoagulation. This was the rationale for steroid trial in this case where there was an alleged history of 20 minute direct exposure (possibly photocoagulating exposure). Moreover eyes with increased pigmentation are likely to have more damage due to the more light absorption at the pigmented RPE which is an additional risk in Indian eyes. Moreover as also in this case, clear crystalline lens in young people allow a lot of scattered light in the UV-B spectrum to reach the retina and accentuate the direct damage. This indirect damage may be the cause of solar maculopathy in young people who may deny direct sun exposure. Other patient characteristics that may increase the risk of solar retinopathy include aphakia, psuedophakia with poor UV protective IOLs, larger light adapted pupil, increased body temperature from exercise, infection, fever etc. Environmental conditions like highly reflective surroundings, reduced ozone etc may predispose to the development of solar retinopathy.

One series revealed that only 47 % of patients with visual complaints after solar eclipse viewing had a discernable fundus lesion while the rest had normal fundus. OCT may therefore be a better tool to study these pathologies and explain the visual deficit. Various OCT findings have been reported depending on the intensity and frequency of sun exposure in eyes with acute solar maculopathy. The reported OCT alterations include; a reduction in the intensity of reflectiveness of the retinal pigment epithelium, intraretinal nonreflective spaces between the inner retinal layers, increased reflectiveness of the inner retinal layers and a round hyperreflective formation in the vitreous just in front of the fovea. In our case, OCT showed a full thickness increased reflectivity including the RPE confirming a severe burn. Our observation also demonstrates that OCT appears to be potentially useful in the follow-up of these conditions and may correlate to the visual outcome better than clinical examination. Though lamellar holes have been reported following solar maculopathy, the occurrence of microholes have not been reported. Microholes are focal defects in the IS/OS junction which is better defined on high definition 3D OCT. Though this patient had 6/6 vision the presence of microhole could account for the persistent visual distortion that the patient complained. Thus OCT is an invaluable tool in the follow up of these pathologies.
Though solar maculopathy is a self resolving pathology, we preferred to use steroids in view of the prolonged exposure (possibly photocoagulating exposure) and severe burn as demonstrated by a full thickness burn on OCT. It is however debatable whether steroids had any independent effect or it just hastened the natural resolution of the condition.

References
Diffuse Unilateral Subacute Neuroretinitis
A Case Report

Sony Siraj E DO, Reena A MS DO, Thomas George MS, DO

Donald M Gass and his colleagues recognized a “unilateral wipe out syndrome” in which healthy young individuals developed insidious usually severe loss of peripheral and central vision, vitritis, diffuse and focal PED with relative sparing of macula, narrowing of retinal vessels, optic atrophy, increased retinal circulation time and subretinal electroretinographic findings. Later it was called as Diffuse Unilateral Subacute Neuroretinitis. We report here an interesting case of a 10 year old boy who was suspected to have DUSN and his condition completely resolved after a course of antihelminthic therapy.

Key Words: Dusn, Ped, Mewds, Apmppe.

Case Report

A 10 year old boy presented to us at Regional Institute of Ophthalmology, OPD with complaints of defective vision (RE) of 2 weeks duration. He was on treatment for hydronephrosis and protein energy malnutrition. He had the habit of taking non-vegetarian diet frequently from hotel and gave a history of deworming every 3 months. General examination revealed no relevant findings. Ocular examination showed a BCVA of 6/6 (RE) and 6/12 (LE). Anterior segment was within normal limits. Fundus of the LE showed a hyperemic disc with a lobulated cystic subretinal lesion of size 2DD beneath the superotemporal arcade, 1DD away from the superior border of the disc (Fig. 1). Superotemporal arcade vessels showed sheathing. Macular edema along with macular star was seen. Multiple subretinal patches were there in the superotemporal quadrant, 1DD temporal to the cystic lesion, suggestive of track lesion. A provisional diagnosis of DUSN was made. Fundus of the RE was within normal limits.

Blood investigations showed a total count of 9600 per cumm and a differential count of P49 (polymorphs) L46 (Lymphocytes) E5 (Eosnophils). CRP (C-Reactive protein) value was raised (1.2mg %). B scan revealed retinochoroidal complex thickening and oedema of the optic nerve. Patient was started on Tab Albendazole 400 mg od and after 24 hrs systemic steroids were given. Patient showed dramatic improvement on day 3 of treatment. After 1 month of treatment patient had a BCVA of 6/6 (BE). Fundus picture showed a complete resolution of the subretinal lesion and macular star.

Fig. 1. Fundus pictures of left eye. Note the hyperemic disc, subretinal lobulated cystic swelling, macular star and suggestion of a ‘track-sign’.

Regional Institute of Ophthalmology, Thiruvananthapuram.
Systemic steroids were tapered over 1 month and albendazole therapy was also stopped after 1 month.

Discussion

DUSN, also called as the unilateral retinal wipe out syndrome typically affects children and young adults. Several species of nematodes, including Toxocara canis, Baylisascaris procyonis, and Ancylostoma caninum have been suggested as the potential etiologic agent of DUSN. The nematodes have been classified into 2 different sizes. The smaller nematode, measuring 400 to 1000 μm in length, is endemic to the southeastern United States, the Caribbean islands, and Brazil. The larger nematode, measuring 1500 to 2000 μm in length, has been described in the northern midwestern United States. Usually there are no associated systemic symptoms although cutaneous and neural larva migrans have been described in a few patients. The proposed mechanism of vision loss include the host inflammatory reaction to the parasite, toxic effect of the worm’s secretary proteins, mechanical damage produced by the movement of the worm or an autoimmune reaction somehow initiated by the infection. The clinical features of DUSN manifest as early and late stages. In the early stages (vision 6/6 – 6/60), the external and slit lamp examination is often normal. Early features include retinal arteriolar narrowing, intraretinal perivascular exudates, pigment epithelial depigmentation and recurrent multifocal evanescent grey white lesion in the outer retina. The retinitis is found typically in one sector of the fundus and can provide a clue to the worm location. The retinitis resolves in 7 to 10 days with minimal or no residual retinal changes. Occasionally the worm is identified with fundus photography.

In the late stages, vision is typically 6/60 or less with a dense central scotoma. Optic atrophy and vascular attenuation are prominent features. A subretinal mass associated with choroidal neovascularization has been described in the macula and around the optic nerve. ERG shows abnormal rod and cone function in the affected eye with a reduction of b/a wave amplitude ratio suggestive of inner retinal injury. Eosinophilia is rare in DUSN. Macular cyst has been reported to be associated with DUSN as an interesting and unusual finding. In patients with diffuse unilateral subacute neuroretinitis (DUSN), the presence and, therefore, clinical visualization of subretinal nematode makes the diagnosis obvious. However when located under the retinal pigment epithelium (RPE), diagnosis is presumptive and challenging. The appearance of sub-RPE serpiginous tract, peripheral RPE hypopigmentation and good clinical response to anti-helminthics support the diagnosis. Arundhati Anshu and Soon Phaik Chee published a case report of presumed DUSN, where subretinal live worm was not seen and the patient responded well to anti-helminthic therapy. So it is important to have a high index of suspicion when patients present with a combination of above findings. This will help in early control of ocular inflammation and also in salvaging vision. Other rare presentations include a case report of diffuse unilateral subacute neuroretinitis (DUSN) that developed an acute iridocyclitis with hypopyon after a year of follow-up and resolved after treatment with systemic corticosteroid. (Cristina Muccioli et al)

Differential diagnosis to be considered include intermediate uveitis, Pars planitis, MEWDS, APMPPE, toxoplasmosis, sarcoidosis, syphilis, Behcet’s disease, atypical RP, siderosis etc.

Laser photocoagulation (Xenon or Argon) should be considered as the first line of therapy in patients in whom motile larva are identified provided the treatment will spare the macula. The role of a combination of laser treatment, systemic steroid, and antihelminthics is also proposed. For the 50% of patients in whom a worm cannot be found, a month long course of albendazole (400 mg od) along with
systemic steroids should be considered. Immobilization of the subretinal nematode has been observed following systemic antihelminthic therapy, and so it has been recommended that patients with DUSN in whom worm cannot be initially identified receive a course of such therapy in order to maximize the chances of identifying and treating the offending organism.

References
Pneumatic Displacement of Submacular Hemorrhage Combined with Intravitreal Bevacizumab Injection – An Effective Combination

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

Introduction

Sub macular hemorrhage is an important cause for acute visual loss. The visual outcome in patients with submacular hemorrhage is especially poor if the hemorrhage is thick, involves the fovea, covers a large area of the macula and is associated with an underlying CNVM especially in age related macular degeneration. Here we present a case of submacular hemorrhage in a 53 year old lady which responded dramatically to pneumatic displacement with intravitreal perfluoropropane gas in combination with intravitreal Bevacizumab (Avastin) injection.

Case Report

A 53 year old lady presented with history of sudden loss of vision in the left eye of 3 days duration. On examination vision in the right eye was 6/6, N 6 and that of the left eye was 2/60. Anterior segment was within normal limits. Dilated fundus evaluation (Fig.1a) showed a large subretinal hemorrhage over the macula in the left eye. Right eye was normal. She underwent a digital fluorescein angiogram (Fig.1b) which revealed multiple retinal pigment epithelial window defects in both eyes and blocked fluorescence in the area of hemorrhage in the left eye. An optical coherence tomography (Fig.1c) was carried out which showed a central retinal thickness of 271 μm in the right eye and 528 μm in the left eye. OCT scan in the left eye revealed a large hemorrhagic pigment epithelial detachment.

The patient underwent intravitreal injection of 0.3ml of perfluoropropane and 0.05 ml of 1.25 mg Bevacizumab (Avastin). Postoperatively, the patient maintained prone positioning for 5 days.
On review 1 month later, her vision had improved to 6/18, N 36 in the left eye. Fundus evaluation (Fig. 2a) showed adequate clearing of the submacular hemorrhage in the left eye. A repeat digital fluorescein angiogram (Fig. 2b) and OCT scan (Fig. 2c) was performed which revealed satisfactory reduction in the size of pigment epithelial detachment with a reduction in the central retinal thickness to 259 μm.

**Discussion**

Submacular hemorrhage can occur due to various aetiologies which include choroidal neovascular membrane due to age related macular degeneration, idiopathic choroidal neovascular membrane, polypoidal choroidal vasculopathy, myopic choroidal neovascular membrane and ruptured macro aneurysms.

The evolution of surgical techniques for the management of submacular hemorrhage has passed through the following stages:

2. 1987: Mechanical removal of clot along with removal of the CNVM.
3. 1991: Subretinal t-PA with removal of liquified blood and without removal of CNVM.
5. 1998: Intravitreal gas only.
6. 2001: Subretinal t-PA and pneumatic displacement.
7. 2007: Pneumatic displacement and Intravitreal Bevacizumab.
8. 2008: Intravitreal t-PA, expansile gas and Intravitreal bevacizumab.

The Herriots technique of intravitreal t-PA and gas injection can be performed in the out-patient clinic being a fast and safe procedure. Some studies have not reported good visual outcomes and it is also unclear whether intravitreal t-PA can penetrate into the retina. However animal studies on rabbits by Motohiro Kamel et al. have demonstrated the ability of t-PA to diffuse into the subretinal space after intravitreal injection.

Ohiji and colleagues reported a series of 5 patients treated with pure perfluoropropane gas and face down positioning in the management of submacular haemorrhage. Displacement occurred completely or partially in all 5 eyes. The visual outcome following displacement depends on the macular status and hence it is important to treat the macular pathology at the earliest. Excellent results of regression of choroidal neovascular membranes associated with AMD following intravitreal Bevacizumab has been reported by various authors.

Various complications have been reported in association with the procedure which includes mild vitreous haemorrhage, retinal pigment epithelial tears and non resolving vitreous hemorrhage.

**Conclusion**

Combining pneumatic displacement with intravitreal Bevacizumab resulted in dual actions of displacing the sub macular blood as well as providing intravitreal Anti VEGF monotherapy to the underlying pathology which resulted in good displacement of sub macular hemorrhagic as well as regression and resolution of the hemorrhage pigment epithelial detachment in this case.
References:


Central Retinal Artery Occlusion following Macular Hole Surgery

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

Introduction

Macular hole surgery has been found to be associated with certain complications such as iatrogenic retinal breaks, retinal detachment visual field defects, glaucoma and cataract. However, cases of central retinal artery occlusion following macular hole surgery have rarely been described. We present a case of central retinal artery occlusion occurring immediately following macular hole surgery, on the first postoperative day.

A 51 year old female was referred to our OPD with a history of defective vision in the right eye of 2 months duration. She was a diabetic and hypertensive of 8 years duration and under good control. On examination, best corrected visual acuity was 6/60 N₃₆ in the right eye, and 6/6, N₈ in the left. Her anterior segment examination was within normal limits. Applanation tonometry was 20 mm in both eyes. A dilated fundus examination revealed a macular hole with subretinal fluid in the right (Fig. 1).

The left fundus was within normal limits. FFA was done which showed a window defect corresponding to the full thickness macular hole in the right eye (Fig. 2). An optical coherence tomography revealed a full thickness operculated stage 2 macular hole in the right eye (Fig. 3).

Fig. 1: Preoperative fundus photograph shows a full thickness macular hole in the right eye.

Fig. 2: Preoperative fluorescein fundus angiography showing an rpe window defect corresponding to the hole.

Fig. 3: Preoperative Optical Coherence Tomography showing a full thickness operculated hole.
The left eye was normal. She underwent a pars plana vitrectomy with internal limiting membrane peeling in the right eye under retrobulbar anesthesia.

The routine retrobulbar block with facial block using a 5:2 combination of lignocaine with adrenaline and bupivacaine to which an ampule of hylase was added was given. A 23 gauge three port pars plana vitrectomy route was employed. Preservative free triamcinolone acetonide was injected into the vitreous cavity to delineate the posterior hyaloid face and facilitate easy PVD induction. After inducing posterior vitreous detachment, a vitrectomy was done. The internal limiting membrane was then stained with a drop of Brilliant Blue G and ILM peel was performed. A blunt spatula was used to stroke the retinal surface and once the ILM edge was obtained, a maculorhexis was done and peeling completed with an ILM forceps. The intraoperative period was uneventful. Gas tamponade was not used taking into consideration the patients’ physical inability to maintain prone positioning.

The patient was examined on the first post operative day. The eye was quite, with an intra ocular pressure of 14mm, no evidence of anterior chamber inflammation, a clear media and an attached retina. The posterior pole of the eye was pale and edematous with a cherry red spot (Fig.4).

The patient was re evaluated for any thromboembolic risks and a cardiology consultation was also performed both of which were noncontributory. The visual outcome was discussed with the patient and relatives and she was discharged on the second post operative day. Subsequent review at 1 month postoperative period showed evidence of gross arterial attenuation and consecutive optic atrophy. The macular hole appeared closed (Fig.5).

**Discussion**

Macular hole surgery has been found to be associated with several complications. The most important intraoperative complication is an iatrogenic break. Undetected or improperly managed intraoperative breaks can lead to postoperative retinal detachments that may require additional surgery or lead to further visual loss. Incidence of retinal break after macular hole surgery is reported to be 5.5 %, an incidence that is similar to that of vitrectomy for other indications. They may be caused by vitreous traction on the retina during surgical maneuvers, including instrument insertion and withdrawal. It is essential to inspect the entire retinal surface by indirect ophthalmoscopy for iatrogenic retinal breaks before fluid – air exchange. If present, breaks are treated with intraoperative retinopexy and postoperative intravitreal gas tamponade.

Intraoperative light toxicity has been reported in less than 1 % of all patients from the fibre optic endo-illuminators.

Complications of orbital regional anesthesia can occur following MHS with the same frequency as in other intra ocular procedures. Table1: gives a list of complication due to regional anaesthesia.

CRAO has been reported following retrobulbar hematoma or an optic nerve sheath hematoma. A high
incidence of CRAO has also been reported in patients having intra ocular gas tamponade when they undergo other non ophthalmic procedure in the immediate postoperative period under general anesthesia using nitrous oxide. Acute intraoperative increase in intraocular pressure can result in central retinal artery occlusion and optic atrophy. Hence it is necessary to have a tag on the patient stating the gas filled status of his operated eye.

In eyes with gas tamponade, expansion of the gas in the postoperative period can also result in acute rise of intra ocular pressure which may result in severe pain, and can cause occlusion of the central retinal artery.

Postoperative rhegmatogenous retinal detachment probably occur in 1-2 % to 14 % of all patients undergoing macular hole surgery\(^2\).\(^1\).\(^3\). Detachments may occur soon after vitrectomy probably due to intraoperative unrecognized peripheral retinal breaks, or later due to contraction of the remaining peripheral vitreous or further separation of the peripheral vitreous. These are usually satisfactorily treated by retinopexy and intravitreal gas tamponade. Peripheral visual field loss after pars plana vitrectomy with fluid gas exchange was first identified in patients undergoing macular hole surgery\(^4\). The typical field defect is a temporal wedge defect often contiguous with the physiologic blind spot. The field defect has been associated with sectoral pallor of the optic nerve and loss of corresponding nerve fiber layer indicating damage of the inner retina, nerve fiber layer and or optic nerve. Its etiology though in completely understood, is thought to be related to mechanical trauma during the surgical creation of posterior vitreous detachment, fluid –air exchange or postoperative tamponade with intravitreal gas. It is recognized by the patient within 24-48 hrs post operatively even in the presence of a large intra ocular gas bubble.

Ocular hypertension and glaucoma may also occur following macular hole surgery. Secondary open angle glaucoma may occur due to inflammation or steroid response. Incorrect mixing of gas concentrations intraoperatively may lead to an expansile gas bubble and elevated intraocular pressure.

Progressive opacification of the lens is another reported complication of vitrectomy and macular hole surgery. Other complication such as endophthalmitis/ proliferative vitreoretinopathy may occur infrequently.

Cases of central retinal artery occlusion have very rarely been reported. One case reported occurred 8 months following macular hole surgery in a highly miopic eye\(^5\).

In our patient on analysis of available data it seems likely that her arterial occlusion occurred as a complication of retrobulbar anesthesia. Although the surgery resulted in good macular hole closure, she was left with a vision worse than her preoperative status.

<table>
<thead>
<tr>
<th>SL.No</th>
<th>COMPLICATIONS</th>
<th>SIGNS and SYMPTOMS</th>
<th>MECHANISM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Venous Haemorrhage</td>
<td>Retrobulbar hematoma</td>
<td>Tearing or puncture of Orbital Vein</td>
</tr>
<tr>
<td>2</td>
<td>Arterial Haemorrhage</td>
<td>Acute massive RBH with ischemia</td>
<td>Tearing or puncture of Orbital Artery</td>
</tr>
<tr>
<td>3</td>
<td>Vascular Occlusion</td>
<td>Occlusion of CRA</td>
<td>Retrobulbar hematoma or intra sheath hematoma</td>
</tr>
<tr>
<td>4</td>
<td>ON Conduction defect</td>
<td>Transient Visual loss and VF defects</td>
<td>Conduction block by anaesthetic</td>
</tr>
<tr>
<td>5</td>
<td>ON Penetration</td>
<td>Permanent visual loss and visual field defects, ONH swelling (O.A)</td>
<td>Ischemic compression by hematoma, trauma to ciliary arteries, traumatic optic neuropathy</td>
</tr>
<tr>
<td>6</td>
<td>Globe Perforation</td>
<td>Pain, ↓ IOP, Intraocular haemorrhage, retinal tear, retinal detachment</td>
<td>Needle perforation with damage to choroid and retina</td>
</tr>
<tr>
<td>7</td>
<td>Needle penetration of Optic Nerve Sheath</td>
<td>Cardio vascular vital signs ((↑/↓)), Respiratory arrest contralateral amaurosis, III N palsy, Hemiplegia, convulsions etc</td>
<td>Central spread of local anaesthetic along submeningeal pathway</td>
</tr>
<tr>
<td>8</td>
<td>Intra venous injection</td>
<td>Bradycardia, hypotension, cardiac arrest, drowsiness, switching</td>
<td>Increased systemic levels of local anaesthetic (CNS and CVS toxicity)</td>
</tr>
<tr>
<td>9</td>
<td>Intra arterial injection</td>
<td>A/c Grand mal convulsive state</td>
<td>Acutely increased cerebral levels of local anaesthetic</td>
</tr>
<tr>
<td>10</td>
<td>Oculo cardiac reflex</td>
<td>Slowing of pulse, nausea, ↓ BP</td>
<td></td>
</tr>
</tbody>
</table>
This complication was a totally unexpected one. We should take care to counsel all patients on the occurrence of complications during local anaesthesia administration also as apart of preoperative patient counselling.

Reference:

Ophthalmic History

Johann Gottfried Zinn

(Anatomist, Ophthalmologist, Botanist………..) & the Zinnia flowers

Prof. Padmaja Krishnan, Calicut

Johann Gottfried Zinn, was born in the German town of Schwabach on the 6th of December 1727. Not much is known of his early years.

He studied Medicine in the nearby city of Ansbach, the capital of Mittelfranken in the German state of Bavaria. He then went to the college town of Göttingen with its famous University and worked under Albrecht von Haller. He was one of Haller’s best students. Obtaining his doctorate in 1749, he went to Berlin where he did extensive research into the anatomy of the eye. Here he also devoted time to the study of his other favourite subject, Botany.

In 1753, Zinn was called back to Gottingen and made director of the Botanical garden in the University. Two years later in 1755 he became Professor of Medicine.

In 1765, Zinn published his masterpiece Descriptio anatomica oculi humani. This book gave the first detailed and comprehensive descriptions of the anatomy of the human eye.

Zinn’s contributions to our understanding of ocular anatomy have been immortalised in the zonules of Zinn and the arterial circle of Zinn-Haller.

His active interest in Botany led to his writing and publishing in this field also, including descriptions of the flora around Gottingen. He described the orchid genus Epipactis that belongs to the family Orchidaceae in 1757.

To honour Zinn and his contribution to Botany, Carolus Linnaeus, the father of modern taxonomy, designated as Zinnia a genus of annual and perennial flowers in the family Asteraceae, which was native to Mexico and Central America.

Zinnia are old favourites in gardens – in pots, along borders or as background. Their flowers last more than a week, have long stems, come in various bright colours and make excellent fresh cut flowers too.

Zinn died at the age of 32 on 6th April 1759 at Gottingen, probably of lung cancer.

Despite his short life, his contributions to Ophthalmology were great and continue to live after him……
Artificial Vision

Dr Meena Chakrabarti MS, Dr Sonia Rani John DNB, Dr Arup Chakrabarti MS

Introduction

The term artificial vision comprises approaches for restoring vision in blind individuals using device or implants, interfacing with neurons of the visual system. These systems are based on the electrical stimulation of groups of neurons at several levels of the visual system with multielectrode arrays placed onto or underneath the retina, onto the visual cortex, around the optic nerve, on the sclera or in the suprachroidal space.

The history of the artificial vision began when Brindley implanted several electrical stimulators close to the visual cortex in a woman who was blind due to the retinitis pigmentosa (RP). After surgery, this patient was able to see spots of lights – electrically evoked phosphenes. Efforts were made to characterize the kind of phosphenes that were elicited with this system.

The Brindley approach was later continued by Dobelle, who implanted several patients with his cortical stimulator. The stimulator was connected to an external power source and to a visual processor with a cable. The information for the visual processor was taken from a camera chip mounted on one glass spectacles and from an ultrasound sensor giving distance information. The Dobelle group reported that the patients were able to see phosphenes, to identify obstacles and to recognize high contrast objects.

As technology advanced, new concepts were considered. Much smaller devices were designed and fabricated, devices that were remotely controlled, and devices that could be much more efficient in terms of spatial and temporal resolution compared with the historic approach of Brindley. The final goal of artificial vision is not to elicit phosphenes, but to restore vision with spatial and temporal properties similar to natural vision, vision that can be used by blind individuals to improve their daily life and performance, not only to restore spatial and temporal resolution in a picture, but also to restore the emotional content of the vision, such as the recognition of a beautiful landscape, or the face of a beloved friend.

Current Concepts for Restoring Vision Using Electrical Stimulation

Artificial vision uses electrical stimulation to drive neurons of the visual system, which are depleted of their natural input. Usually, electrical stimulation is provided in such concepts by implants consisting of an array of simulated electrodes and electronic components, e.g., for pulse generation. Two main concepts evolved, one is that the optic path of the eye is still used to transmit visual transformation. In the second concept visual information is obtained by a camera system. This information is then further processed depending on the level of the visual system where the stimulation is intended.

In the original idea of subretinal stimulation the implantation of thousands of very small microphotodiodes in the subretinal space was planned. These elements could transform light coming naturally through the optical path of the eye into electric current strong enough to drive postsynaptic cells. The microphotodiodes were intended to replace the photoreceptors. In this concept additional data processing or energy supply was not required. It was...
thought that the postreceptoral retinal data processing would be done by postsynaptic neural network, which was thought to be more or less intact. Chow et al. implanted several patients with such a system, “an artificial silicon retina” (ASR) in the subretinal space. The surgery was carried out without complications and the patients reported visual sensations in the first year. Unfortunately after a longer follow-up the patients reported that the percepts disappeared, and they were as blind as before the implantation. It turned out the devices did not generate enough power to drive postsynaptic cells. Most likely, the primary percepts were the result of an unspecified effect of mediators and other cell signal molecules released after surgery.

In approaches interfacing with ganglion cells or cells in the visual cortex, camera systems and data processing algorithms with application-specific hardware are used to obtain visual information and to calculate optimal stimulation pulses. Furthermore, in such approaches data processing algorithms will be modified by the percepts of user in a training procedure.

**Interfacing the Neurons**

In RP the photoreceptors degenerate. However, postsynaptic neurons also show considerable changes in the degenerated retina with a loss of cell bodies and a chaotic disorganization. In advanced cases of RP a certain amounts of ganglion cells remain alive, but a huge amount of remodeling occurs in which new circuits are established and neurons migrate along glial structures forming microneuromas. The typical layered structure of the retina with known functional connections is destroyed. Electrical stimulation to restore neural function uses charge delivery from a stimulating electrode to adjacent cell membranes so that their membrane potential is considerably modulated. By changing this membrane potential a neuron may fire action potential or release neurotransmitters at its synaptic terminal, thus making the neuronal chain functioning again as a response to stimulating pulses emitted from electrodes of an implanted stimulation device. However making predictions as to which cell will be stimulated and which postsynaptic cells will be activated is nearly impossible because of the structural and functional remodeling of the degenerated retina and because in the clinical situation it cannot be exactly planned where stimulating electrodes will be placed with regards to the position of target cells. If specific activation of cells is the goal, then it is desirable to have as many electrodes as possible to contact as many neurons as possible in a 1:1 ratio. Electrodes should be placed as near to the target cell as possible. Charge delivery may include adverse events in the target issue or in the material of the electrode; therefore, certain safety ranges of charge delivery should be taken into account. As consequence electrodes cannot not be made as small as possible because the charge density would be enhanced, which is the main parameter in terms of electrode material stability and safety. Currently in approaches using electrodes on the retinal surface, electrodes are fabricated in diameters of 40 -200 mm. Electrode materials are platinum or gold with or without regular or sputtered iridium oxide. Surface modification of these electrodes is used to increase the surface area of the electrode without increasing the electrode diameter in order to reduce the charge density to protect both the material and also the tissue against side-effects of chronic electrical stimulation. These large electrodes could be placed close to the outer surface of the retina as well as underneath the retina. However, compared with the cell the electrodes are still very large and single.
cell stimulation is not possible. Whole cell clusters will be activated with such large electrodes. However, by intelligent selection of stimulus parameters the activation of certain cell types may be possible even when the electrode is adjacent to a cell cluster 17, 18, 19. Technical difficulties are explained by the power needed to individually activate thousands of electrodes and by the electronics to transmit such a very high density signals within a biologically safe range of power.

**Epiretinal Stimulation**

Based on the early experiments of Dawson and Radtke 20, but also on the consideration that in RP more damage is in the outer retina than in the inner retina, strategies were developed based on multielectrode arrays fixed onto the inner retinal surface. The aim of this concept is to stimulate ganglion cells 21, 22, 23, 24. The electrodes are usually mounted on a flexible substrate; usually polyimide is used for this purpose. The electrodes are driven by power sources either outside the eye or inside the eye, then it has to be controlled remotely via inductive or optoelectronic signal end energy transfer. If the power source is out side the eye, it is necessary to connect the multielectrode array with a cable to the power source. The cable has to cross the wall of the globe, usually through the choroids and sclera. Such an implant with a transscleral cable connection to an epiretinal multielectrode array was fabricated by Mahadevappa and colleagues and has been evaluated in a pilot clinical trial 25. Theoretically, a trans retinal cable may be at risk of intraocular infection or the risk of shearing forces transmitted to the implant and its anatomical interface with the inner retinal surface when the eye is moved. However, no reports exist on such potentially adverse events. The approach being used by Horing et al. also consist of an epiretinal multielectrode array, a transscleral cable connection, and a data and energy system in which a receiver coil is mounted on to the scleral surface 23. The group of Walter and Mokwa fabricated an epiretinal device in which the transponder coil is implanted in the capsular bag, meaning that no cable passes the wall of the globe (Fig. 2) 27, 28.

A crucial problem in epiretinal stimulation is the stimulus paradigm. Usually, ganglion cells receive preprocessed data from retinal interneuron and not only information on which receptor is activated by light. Therefore camera data resembling receptor activation have to be processed in an encoder simulating retinal data processing. Adaptive spatiotemporal filters are used to process the camera data and the output of this processing is then used to stimulate the ganglion cells 29.

Because it is previously not known which ganglion cells are stimulated, the encoder properties have to be modified in a learning procedure based on the percepts of the patient, which should be as near to the input signal as possible (Fig. 3).

**Subretinal Approach**

In the subretinal approach micro photodiodes are implanted underneath the retina. The original idea of Chow et al and Zrenner et al was that thousands of
such photodiodes would act as artificial receptors and change the light into a current large enough to drive postsynaptic cells in the retina. However, it was found that the current generated by the currently available macrophotodiodes was not strong enough to drive these cells. Therefore, implants are now fabricated with an additional power supply. Zrenner and his group were able to demonstrate in patients that direct subretinal electrical stimulation can elicit phosphenes.
in patients over a certain time period and that patients were able to detect some basic geometries. At present, it is not exactly clear what cells are activated in subretinal stimulation in advanced cases of RP because the retina shows a significant amount of destruction of the original layer structure making ganglion cells reach even as far as the outer retinal surface. It may be disclosed in the future that with both approaches ganglion cells and post synaptic cells were activated from the epiretinal or from the subretinal side. That may also mean that the processing of the input may be necessary for both approaches (Fig. 4 a-e).

In both the epiretinal and subretinal approaches major surgical steps have to be taken, meaning that both approaches have a certain risk profile. Therefore, approaches are considered to minimize the surgical risk. Electrode arrays may be placed with their basic structure onto the outer scleral surface or in a transcleral pocket. Needle-type electrodes should penetrate into the supra choroidal or into the subretinal space to get close to the target cells. Such approaches are not free of surgical risk because placement of such structures at the posterior pole may cause trouble with the ciliary arteries and sharp electrodes may penetrate deep within the retina (Fig. 5). In concepts in which the electrodes remain on the scleral surface or in the suprachoroidal space the main problem remains the distance between the electrode and the target issue. However, in a pilot trial Kanda was able to demonstrate in normal volunteers that they have phosphenes with stimulus intensities similar to those reported in epiretinal stimulation and that they were also able to differentiate different sizes of phosphenes, depending on stimulus parameters.

**Optic Nerve approach**

There is some experience in connecting peripheral nerves with cuff electrodes. Therefore, such cuff electrodes were used to contact the optic nerve in two experiments by Delbeke et al. They found that they could elicit phosphenes in their patients. Patients were able to recognize objects after a long learning period and the object identification took several minutes for scanning.

The optic nerve was approached first in a neurosurgical approach at the level behind the orbit and in a second experiment within the orbit. Thresholds for electrical stimulation differed significantly and were much lower in the cranial approach than in the orbital approach.

From a theoretical point of view, a prosthesis using stimulating electrodes around the optic nerve fibres may have the problem of good spatial resolution because in the optic nerve the fibres are very densely packed and therefore a large amount of fibers could have been stimulated. Whether perforating electrodes are a solution to that theoretical problem remains unanswered at present.

**Cortical Prosthesis**

A large portion of the central nervous system is involved in the processing of visual information and the primary target of fibers from the lateral geniculate nucleus is layer 4 of area V1 at the occipital pole of the brain.

In this area good retinal topography is found with the large representation of the fovea at the most posterior parts and the peripheral representation in the more inwardly located, smaller areas of V1. Approaches in restoring vision in patients suffering from glaucoma or trauma to the optic nerve in contrast to retinal prostheses, may necessitate stimulation of neurons in V1 by passing other parts of the visual system.

Multielectrode arrays have been developed by Norman and his group based on silicon needle arrays. Such multielectrode arrays can be used for stimulation as...
well as for recording. Animal experiments showed relatively little fibrotic response around the electrode tips. The electrodes could be placed very near to the somata of the neurons in V1. Silicon–based multielectrode arrays were also implanted in trial in 6 patients who underwent brain surgery. It was shown that the stimulators could be inserted with a pneumatic shooter. Excised brain tissue showed only minor alteration such as small bleeds and deformation.

**Pixel Vision and Filters**

The possible resolution of artificial vision by using retinal implants was evaluated in animal experiments in which cortical response were obtained from the cat’s visual cortex or by optical imaging. These data show a possible spatial resolution of about 1° in space and 25 images per second. However, relatively little data are known on the percepts of patients wearing the first available prototypes with regard to picture quality. In the pilot trials that are currently running threshold measurements are taken and standardized tests are used where the single electrodes or groups of electrodes were activated. Patients are asked if they can see something, or if they can see separate spots of light or a line. Patients are also asked if they can identify the orientation of a line, whether it is a horizontal or vertical line. A systematic analysis of the presentation of the real pictures has not yet been carried out. Patients who are implanted with the 16-electrode array used by Humayun’s group and who are already connected to a camera system reported that they can identify high contrast obstacles or that they can now find the door in the wall of a house. To learn more about this kind of artificial vision with only a few implanted electrodes, simulation were performed based on certain assumptions. The simplest simulation is pixel vision where each electrode is considered as a pixel in a rectangular montage. Such stimulation can be seen in Fig. 6. It becomes obvious that the percept depends on the complexity of the real picture. For very simple pictures, e.g., a dark door in a bright room, only a few electrodes are necessary to identify the door, for complex pictures such as the face of a person, many electrodes are necessary for face recognition (Fig. 7).

To point to a person 48 electrodes are necessary, but for face recognition in the example shown in Fig. 7, 864 electrodes are necessary. In contrast, to identify the door as in Fig. 6, i.e. to move to the door, only 12 electrodes are necessary. To identify the obstacle right in front of the door to the right and to enable free movement to the door 48 electrodes are needed, but to find the door opener again 864 electrodes are necessary.

For paragraph reading Dagnelie and coworkers found that 256 electrodes placed on a 3 x 3 mm retinal implant are necessary.
Performance with such a low number of electrodes in similar test can be further improved by the design of multielectrode array. It may be useful to place electrodes with a high density in the centre of the device if it is implanted on to the macula and with a low density in the peripheral area of the multielectrode array. Performance will also depend on the size of the electrode array and the efficacy with which the electrodes make contact with retinal neurons.

Conclusion

Artificial vision comprises approaches to electrically stimulating the neurons of the visual system to bypass degenerated receptors or other neurons to restore vision in otherwise blind individuals. Stimulation can take place at the level of the retina with either subretinal or epiretinal electrodes, but also with the electrodes placed onto the scleral surface or electrodes, in the suprachoroidal space. Stimulation has also been effected at the level of the optic nerve and in the visual cortex. These concepts are currently being evaluated in pilot clinical trials providing safety data. Future developments will concentrate on increasing the number of implanted electrodes, on reducing the surgical risk, on optimizing stimulus paradigm strategies, and on modulating the degenerative process by electrical stimulation.

Reference:

Asteroid Hyalosis and Diabetic Retinopathy

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

Asteroid hyalosis (asteroid hyalopathy, asteroid hyalitis) is a monocular non-inflammatory disorder of the vitreous characterized by an accumulation of minute white spherular particles within an otherwise apparently normal vitreous gel. The condition affects ~1% of the general population and occurs in the elderly. Men are mainly affected. The condition is rarely familial and it has been associated with diabetes mellitus, hypertension, atherosclerosis, gout and hyperopia. The particles, which move within the vitreous, are composed of calcium soaps (calcium stearate and calcium palmitate). They appear gray in hematoxylin and eosin stained preparations, but lipid and calcium can be detected histochemically in appropriately prepared specimens. The asteroid bodies are moderately positive with the periodic acid-Schiff reaction and they exhibit a vivid “Maltese cross” pattern of birefringence using polarization microscopy. Asteroid hyalosis does not affect vision and patients are asymptomatic. Examination of the eye reveals countless creamy white stellate opacity within the vitreous that resemble snowballs or Christmas ornaments. Asteroid hyalosis is occasionally confused clinically with synchisis scintillans and amyloidosis.

Signs and symptoms: Asteroid hyalosis is a primarily unilateral disorder that typically occurs in patients over 60 and in men twice as often as women. Usually asymptomatic, in severe cases asteroid hyalosis can mildly affect visual acuity. Complaints of floaters are a rarity.

Ophthalmoscopically, asteroid hyalosis appears as multiple, discrete, refractile yellow or yellow-white particles suspended in the vitreous. In early stages, there are fewer bodies and they accumulate in the inferior vitreous. Advanced cases can be so dense as to impair your view of the posterior fundus.

Asteroid hyalosis (AH) is a benign condition characterized by small white or yellow-white spherical or disc shaped opacities throughout the vitreous. The frequency of this condition in the general population is about 0.042 to 0.5 % affecting all races with a male to female ratio of 2:1. In whites the prevalence of asteroid hyalosis is 1-2 %, and it is bilateral in about 10 % of cases and this prevalence seems to increase with age. Asteroid hyalosis is unilateral in 75 % cases. The aetiology of asteroid hyalosis is not clearly understood. The association of asteroid hyalosis and diabetes mellitus has been a debatable issue in ophthalmology. There have been reports which suggest an association between the two conditions while others dispute any such association.

- The controversy regarding an association between asteroid hyalosis and diabetes mellitus has been one of the longest disputes in ophthalmic literature. Multiple studies are present either indicating definite association between the two conditions or no association at all.

- Zinn reports 27 % patients with asteroid hyalosis are diabetic, while Bergren reports that 29 % of his asteroid hyalosis patients were also diabetic. Bilateral asteroid hyalosis was found in 37.5 % of our patients. There are various and differing reports regarding involvement
of both eyes. Moss\textsuperscript{11} reported approximately 9\% bilateral cases of AH, whereas according to Zinn\textsuperscript{9} it was 25\%. Jones\textsuperscript{12} has also documented a patient with acquired asteroid hyalosis in a case of early diagnosed diabetes mellitus which strongly supports association between the two conditions.

Asteroid hyalosis has been described in association with other systemic diseases such as systemic arterial hypertension and atherosclerotic vascular disease\textsuperscript{5}. Owing to association with systemic conditions, it has been suggested that asteroid hyalosis may be secondary to some form of vasculopathy in many frequencies and that diabetes mellitus is one of the conditions that may be associated with formation of asteroid hyalosis.

**Pathophysiology:** Asteroid bodies represent small calcium-laden lipids suspended within and attached to the hyaluronic acid framework of the vitreous body. While we understand the composition of the asteroid bodies, the exact genesis remains unclear. Current theories suggest that asteroid hyalosis results from aging collagen within the vitreous or a depolymerization of hyaluronic acid.

**Management:** Asteroid hyalosis is a benign condition in itself. Although it progresses, it never leads to severe vision loss, and the mild symptoms occur rarely. The vast majority of cases merely require documentation. More often than not, this disorder poses a greater challenge to the examining physician because it can obscure details of the underlying retina. Consider treatment only in patients who are also being managed for retinal disease (proliferative diabetic retinopathy, retinal tear or detachment). Vitrectomy is typically indicated in these instances. In vitrectomy for PDR with asteroid hyalosis, and in cases of simple vitreous hemorrhage, surgery should be performed with full understanding of the anatomic characteristics. Notably, if posterior vitreous detachment is not present, the occurrence of iatrogenic retinal breaks is more likely. Complete posterior vitreous detachment (PVD) occurred less often in eyes with asteroid bodies than in control eyes and partial PVD occurred more often in eyes with asteroid bodies after the age of 70 years, the prevalence of PVD, either complete or partial, was lower than in age-matched control eyes and the prevalence of liquifaction (19\%) was lower than has been reported in controls. The presence of asteroid bodies may arrest the process of vitreous collapse or contraction and that diabetes might influence the development of asteroid hyalosis.

**Clinical Pearls:**

- Asteroid hyalosis presents a picture akin to “stars in the night sky.” On eye movement, the asteroid bodies sway within the vitreous, but always return to their original position.
- Synchisis scintillans and amyloidosis are often confused with asteroid hyalosis.
- Synchisis scintillans (cholesterol bulbi) is an extremely rare condition that occurs in severely diseased eyes. This condition also presents with refractile crystals in the vitreous, although these particles are composed of cholesterol. They are not attached to the vitreal framework, so they tend to settle out inferiorly after eye movement. Because this condition occurs in end-stage eye disease, pathologists rather than clinical optometrists or ophthalmologists typically make the diagnosis of synchisis scintillans.
- Amyloidosis of the vitreous is also quite rare, and occurs typically after age 40. Patients characteristically demonstrate bilateral involvement with granular, strand-like opacities within the central vitreous. These membranes are anchored to the posterior lens surface in about half of patients. Small, yellow-white bodies dot the vitreal strands.
- Remember that the density of asteroid hyalosis does not correlate with visual dysfunction. If a patient presents with significantly diminished acuity, asteroid hyalosis is not to blame.
- Patients with asteroid hyalosis and an unknown medical status require evaluation for diabetes, hypertension, hyperlipidemia and atherosclerotic vascular disease.
- Filters used for performing fluorescein angiography can allow a better fundus view through the retinal camera in cases of severe asteroid hyalosis and may allow you to observe pathologies that conventional ophthalmoscopy does not reveal.
Photoessay of a 65 years old chronic diabetic with asteroid hyalosis and proliferative diabetic retinopathy

Fig. 1. (a & b): Color fundus photograph (a & b) showing an unclear view of the fundus details. The vitreous cavity shows specular refractable particles of asteroid hyalosis. Observe the difficulty in making out the fundus details in this 55 year old chronic diabetic with proliferative diabetic retinopathy in her right eye.

Fig. 2. (a & b): Fluorescein fundus angiography pictures of the right eye of the same patient the posterior pole with tracking microaneurysms.

Fig. 3. (a& b): Postvitrectomy color fundus photograph of right eye on the 2nd postoperative day showing pigmented laser burns and a dry macula.
Reference:

Fig. 4. (a & b): (a) Fluorescein fundus angiography performed 4 years after the retinopathy stable with a dry macula and no evidence of leakage of dry from the macula, disc or retina. (b) Fluorescein fundus angiogram of the left eye at the same period showing RPE atrophy corresponding to the PRP laser marks. Observe multiple active and leaking new vessels indicating the necessity to perform fill-in additional PRP which was performed as an outpatient procedure.
Consultation section

Dr. Suhas Huldipurkar MS 1, Dr Arup Chakrabarti MS 2, Dr. J.K. Reddy MS 3, Dr. Suven Bhattacharjee MS 4, Dr. Mohammed Naved Abdul Karim MS 5, Dr. Sai Kumar MS 6

The following post-cataract photograph along with the following questions were given to five senior cataract surgeons for opinion and comments.

1. Why has this occurred? Can a small piece of cortex which was not noticed during the primary surgery swell up to this extent?
2. What would be your preferred method of cortex removal? Is it a simcoe cannula, IA cannula or vitrectomy cutter?
3. Would you consider removal of the IOL for removing the cortex?
4. If the PC rent enlarges what is the contingency plan?
5. Is it mandatory to have VR backup while doing this case?
6. Would you reposition the lens into the capsular bag?
7. If a significant amount of cortex goes into the vitreous, would you prefer a pars plana approach?

This is the photograph of the left eye of a 65 year old lady who underwent cataract surgery 14 days back in another hospital. She came with a vision of Counting fingers 1 metre, but no other symptoms suggestive of inflammation.

According to the patient, the vision was slightly better immediately after surgery, but deteriorated after 1 week. On examination, her intraocular pressure was 16 mm Hg, dilated examination revealed a hydrophilic acrylic IOL placed in the ciliary sulcus. There was thick intumescent, cortical and epinuclear lens material behind the IOL. Although not very clear in the photograph, there is a small central rent in the posterior capsule. (Fig. 1)


Fig. 1. Anterior segment photograph of the left eye showing the thick cortical material behind IOL

Fig. 2. Pseudo PC rent seen as a relatively clear area between swollen cortical fibres
Dr. Suhas Haldipurkar

1. PC rent caused the abandoning of surgery prior to complete cortical removal. Cortical swelling of this magnitude is common after such events.

2. I would prefer vitrectomy cutter for this clean up.

3. No. I will not disturb the IOL in this instance. My approach would be by pars plana.

4. By parsplana approach the pc rent can be converted in to a controlled circular opening in the pc so that the lens can shift back in to the bag after the cortical clean up.

5. It is mandatory to have the knowledge of parsplana approach to undertake this procedure.

6. Yes . I would reposit the IOL in to the bag if the rhexis is intact and the pc rent is converted back in to a well circumscribed central PCCC.

7. Yes. My preferred approach is pars plana.

Dr. Arup Chakrabarti

Introduction

Before I proceed to address the questions raised by the editor I would like to gather further information about the patient whose case is being discussed.

(a) **The status of the corneal endothelium:** Specular microscopy can be of great help in revealing the true status of the corneal endothelium. This information will be useful in patient counseling and detailed surgical planning. Regardless of the endothelial status, the surgeon should recruit the best quality viscoelastic agent and minimize avoidable intraocular manipulations intraoperatively.

(b) **Presence of vitreous stands in the anterior chamber:** Careful slitlamp biomicroscopy may help. The presence of pupillary peaking may also indicate vitreous in anterior chamber. Intraoperative injection of triamcinolone acetonide will help to delineate vitreous in the anterior segment.

(c) **Presence and location of the PC rent:** Detecting the presence of a PC rent may be tricky in this patient. A relatively clear area between swollen cortical fibres may resemble a PC rent. On the other hand true PC rent may be camouflaged by swollen opaque cortical fibres. In many situations PC rent can be confirmed only intraoperatively (Fig. 2).

(d) **Presence of significant amount of cortex and / or nucleus in the vitreous cavity.** B Scan ultrasonography may hold a clue regarding this complication.

(e) **The integrity and size of the capsulorhexis.** In the presence of an intact rhexit (and a large PC rent) we may consider placing a suitable PC IOL in the ciliary sulcus.

(f) **A-Scan Biometry** result of the affected eye is to be obtained. An appropriate AC IOL, rigid PC IOL and a 3 piece hydrophobic acrylic IOL are to be kept as a standby.

(g) A proper **informed consent** of the patient is a must.

Now the queries raised by the editor:

1. We are dealing with a residual cortical and epinuclear sheet trapped between the intraocular lens and the posterior capsule. This cannot result from a small piece of the leftover cortex. It is important to note that a similar picture is not uncommon in the hands of a novice surgeon who may fail to detect, the presence of an epinuclear sheet intraoperatively in the presence of a bright fundal reflex. The possible reasons in the given patient are as follows:

(a) The surgeon, having attempted removal of the epinuclear sheet would have abandoned efforts at complete removal due to intraoperative miosis. Underestimating the amount of residual lens matter, he would have implanted the intraocular lens which perhaps landed in the sulcus (unintentionally). The lens appears to be in the sulcus since it is found to be decentered (nasally in this case).

(b) Intraoperative manipulations may have resulted in a posterior capsular rent. Fearing further extension of the rent with ensuing vitreous disturbance, the surgeon might have abandoned further efforts at cortical removal in a moment of panic, implanting the IOL in the sulcus.
2. All the intraocular manipulations in this case should be performed in a closed chamber environment. The key issue here is to maintain a deep anterior chamber at all stages. Hence use of Simcoe cannula, which is a co-axial system, is ruled out. I would go in with a bimanual I/A cannula and work through 2 paracentesis incisions. The cannula may have to be switched to facilitate complete cortex removal. The anterior vitrectomy unit should be kept as a standby. If I harbour the slightest suspicion as regards the presence of vitreous in the anterior chamber I would inject triamcinolone acetonide (TA) into the anterior chamber. Vitreous, if present, will trap the TA particles and will get delineated. Then I will proceed with anterior vitrectomy through a side port incision, the other side port incision being used for the infusion cannula. If vitrectomy is performed (at high cut rate and low suction levels) the same cutter could also be used to remove the residual cortex (but at a low cutting speed and high suction levels). Once the vitreous has been removed cortex aspiration can also be performed with the bimanual I/A system. The cortex should be stripped from the periphery to the centre keeping the area of the PC rent for the end. A 26 G straight and a bent J cannula will also be handy to remove wispy cortical material towards the completion of irrigation and aspiration.

3. In the given case, since the IOL appears to be in the sulcus, adequate space can be created behind the IOL in an atraumatic manner to deal with the residual lens matter and / or the vitreous. In a hypothetical case where the IOL is found within the bag, the management option may depend on the rhesis size and / or the presence of a posterior capsular rent. If the rhesis is relatively large (as compared to the optic diameter) the I/A cannula and or cutters can still be maneuvered to atraumatically aspirate the residual cortex. The problem begins when the rhesis is quite small. If there is no PC Rent the lens can be maneuvered into the sulcus and repositioned back once the residual lens matter is removed. However, in the pressure of a PC rent I would prefer to remove the cortex through the pars plana approach. Explanation of the IOL would be quite traumatic. If one does not want to extend the incision, the IOL would have to be cut with a lens cutting scissors before being extracted through the unenlarged incision.

4. Every effort should be made to prevent the occurrence of a PC rent or if it has already occurred to prevent its further extension. Vitreous disturbance also should be prevented and, if present, minimized. I would like to convert the PC rent into a posterior capsulorhexis. In that case the hydrophilic acrylic IOL can still be implanted within the capsular bag. However, the PC rent may be too large to be converted into a posterior capsulorhexis and in that case my considerations will be modified. I am not comfortable with this type of lens in the ciliary sulcus. Hence I would prefer to explant this IOL (after cutting it with dedicated lens cutting scissors under viscoelastic cover) and then inject a 3 piece hydrophobic acrylic IOL into the ciliary sulcus.

5. The surgeon performing this case must be competent in anterior vitrectomy. Every phaco surgeon’s armamentarium should include a functioning anterior vitrectomy unit. If properly performed, there should not be any loss of cortical matter into the vitreous cavity.

6. If there is no PC rent and no zonular instability I would definitely relocate the same IOL into the capsular bag. If there is a small PC rent and I have successfully converted it into a posterior capsulorhexis I will not hesitate to relocate the same IOL into the bag. However, if the PC rent is large I would exchange the hydrophilic acrylic IOL with a 3 piece hydrophobic acrylic IOL (Sensar).

7. If the surgeon is not cautious, cortex may drop into the vitreous cavity particularly if the vitreous is liquefied. A small amount of cortex may get absorbed with time with the only complaint being that of seeing floaters which diminish over time. However a significant amount of retained cortex may be associated with postoperative complications like recalcitrant glaucoma, cystoid macular oedema, uveitis etc. So a thorough removal of the residual cortex is called for which can be achieved only through the parsplana approach.
In summary, a patient with significant amount of retained cortex needs to be managed surgically after conducting appropriate preoperative workup and extensive counseling. With adherence to proper surgical strategy the postoperative results will be no different from that of a routine uncomplicated case.

Dr. J.K. Reddy

1. A small PCR might have occurred during the last nucleus piece phacoemulsification. What is seen here is epinucleus plate which is very transparent in immature cataracts and can swell and becomes opaque.

2. Fill the anterior chamber with cohesive viscoelastic (sodium hyaluronate). Aspirate the cortex with Simcoe with very low flow of BSS, a drop every second. Start the aspiration of cortex first in the periphery, as the PCR is in the centre. If the above technique doesn't work use virectomy cutter.

3. Hydrophilic IOLs in the sulcus are not very stable and produce gross myopia unless the surgeon has undercorrected intentionally. In this case IOL looks fairly well centred, so leave it.

4. IOL is on the anterior capsule. So PCR extension may not create any further problem.

5. VR back up is not necessary for handling this case.

6. Hydrophilic single piece IOLs are difficult to reposition into the capsular bag with PCR. So it is better to leave it in the sulcus or exchange it with a PMMA IOL.

7. This fine hydrated cortex will get absorbed very well. It does not produce much vitritis. So I will leave it and observe.

Dr. Suven Bhattacharjee

Since the IOL is well centred and placed in the sulcus, it would be reasonable to assume that the capsulorhexis is intact. The same should be confirmed on slit lamp examination. It would also be reasonable to assume that a PC rent had occurred with no/ minimal vitreous loss and the surgeon chose to implant the IOL in haste leaving behind a rim of epinucleus & cortex which might have been fairly transparent at the time of surgery. The possibility of a dropped nucleus should be kept in mind and ruled out by an Indirect Ophthalmoscopy (through small opening in PC) and Bscan USG. Assuming that the capsulorhexis is intact and that there is no dropped nucleus, I would prefer the following line of management:

I would prefer a Pars Plana approach and a vitrectomy cutter over a clear corneal / limbal paracentesis & I/A cannula for removal of the epinucleus and cortex. A clear corneal approach with bimanual / coaxial I/A would involve going across the pupillary edge, around the edge of the IOL and then behind it. The pupil is likely to get smaller due to handling. We will be attempting to draw out cortex and epinucleus sandwiched between the remnant PC and the anterior capsular rim & IOL. The epinucleus is likely to offer significant resistance to I/A and may drop into the vitreous during manipulation.

I would prefer a 3 port vitrectomy approach. With the sutured PP infusion in place, I would use a vitrectomy cutter through PP to remove the vitreous behind the PC rent. Thereafter, alternating between cutter ‘on’ & ‘off’ modes (in the off mode the cutter works as a aspiration cannula with a fairly wider aspiration port than the conventional 0.3 mm), I would attempt to remove the cortex & epinucleus sector by sector. While an attempt would be made to preserve the remnant PC, I wouldn’t hesitate to remove it sector wise, to gain access and completely remove the cortex and epinucleus. Once all the cortical / epinuclear matter is removed, I would use an endo illuminator with Irrigating lenses/ Landers lenses to check for any vitreous droppings. Thereafter, I would capture the Optic of the IOL in the capsulorhexis using the cutter and light pipe through the pars plana itself. While the sulcus is not the most desirable place for a hydrophilic lens, a rhexis capture of the optic would ensure stability and centration. Needless to mention, this procedure is best performed by a vitreo retinal surgeon. The next best would be an Anterior segment surgeon with a VR back up. We must remember to ensure that the patient doesn’t require a 3rd surgery.

Dr. Mohammed Naved Abdul Karim

Cataract surgery is performed in the majority of cases with a view to improve vision.[1] In this case it is unfortunate that this patient has been left with poor vision following the procedure. Cortex left behind by
the surgeon mixes with the aqueous humour and can swell up and fill the eye. The lens cortex retained in the eye after cataract extractions usually undergo lyses by aqueous but may persist as in this case.\textsuperscript{[2, 3]} Since there is no inflammation or raised IOP we can assume that it is not an infection or inflammatory response.\textsuperscript{[4]}

The plan of action is to remove the residual cortex especially from the visual axis for the patient to be able to see. My aim would be to remove the cortex without hydrating the vitreous causing vitreous prolapse. After filling the anterior chamber with viscoelastic the cortex would be removed with a simcoe cannula with a low flow or as a dry aspiration \textsuperscript{[5, 6].} In case there is vitreous prolapse I would then do an anterior vitrectomy as any vitreous left in the wound has the potential for further complications.\textsuperscript{[7, 8]} The vitrectomy can be performed with the IOL in place as the IOL is in the sulcus and has less chance of dropping into the vitreous than if it was in the bag. If I am able to aspirate the cortex without enlarging the pc rent and/or vitreous prolapse, I may try to put the IOL in the bag provided the pc rent is small. It is preferable to have the IOL in the bag but is not essential. If I feel that more manipulation is necessary then I prefer to leave it in the sulcus as this is a second surgery and less manipulation the better.\textsuperscript{[9]}

I don’t think a vitreoretinal backup or pars plana approach is necessary in this case as the cortex is usually quite soft especially after 14 days and the pc tear is very small. With a low flow/dry aspiration the cortex can be removed without difficulty. Postoperatively the patient should be on frequent doses of topical steroids and topical ketorolac to dampen the inflammation and prevent cystoids macular oedema.\textsuperscript{[10, 11]}

**Dr. S.J. Saikumar**

These are my comments and the way I managed this case.

1. Most obviously the primary surgeon must have panicked on seeing a PC rent before removal of the entire cortex, and has implanted the IOL. But the residual cortex has swelled up to such an extent that the post op vision is very similar to that of a mature cataract. This much cortex will not get spontaneously absorbed. Plugging the PC rent with a cohesive OVD and if needed a limited anterior vitrectomy would have aided in cortex removal during the primary surgery. The IOL can then be placed either in the bag or in the sulcus without compromising the final visual outcome.

2. I preferred an anterior approach initially with 2 paracentesis openings. An infusion was in place through one and a vitrectomy cutter was put through the other. After removal of the central cortex mixed with vitreous, a Simcoe cannula was used to remove the rest of the cortex. Since the cortex was hydrated and fluffy, it came out very easily.

3. There is no need to disturb the IOL in this case. The IOL can be repositioned either in the bag or in the ciliary sulcus after cortex removal. IOL removal may be needed only in rare instances like when the primary surgical manipulation has been significant and there is some underlying retinal pathology like RD or vitreous hemorrhage.

4. Even if the PC rent enlarges, the IOL can be positioned on the ciliary sulcus. If the PC opens up wide and the CCC also is not intact, one may have to consider explanting the PC IOL and replacing it with an AC IOL. But in this case, with a little care, chances of the rent enlarging are quite minimal.

5. Since there is only retained cortex anterior to the PC, a VR backup is not absolutely essential. But knowledge of the pars plana approach is advantageous.

6. If the rent is small, the IOL can be positioned back into the bag. Another method is to convert the rent into a PCCC with the cutter before positioning the IOL in the bag.

7. The take home message in this discussion would be that one should not panic on seeing a PC rent. Limited vitrectomy and cortical removal in the primary sitting itself will help avoid a second surgery.

**References**


Developments in Ophthalmology
Volume 39 - Diabetic Retinopathy

Series Editor: W. Behrens — Baumam, Magdeburg
Volume Editor: Gabrielle E. Lang, Ulm
Published by: Karger
Year: 2007

With the explosion in the diabetic population both in developed and developing countries there is an increase in the incidence of diabetic retinopathy. Despite markedly improved visual prognosis with the availability of lasers, vitreous surgery and challenging pharmacotherapy, diabetic retinopathy still continues to hold its position as one of the leading causes of blindness.

This book on diabetic retinopathy provides profound insight into the pathophysiology and new treatment options for diabetic retinopathy.

This book has eleven chapters each dealing with a specific aspect of diabetic retinopathy in a comprehensive and lucid manner. Detailed information on the latest research achievements provide a complete and updated version of the pathogenesis and management options of this disease.

The pathophysiology of diabetic macular edema though not yet fully understood is up to date with respect to the current knowledge of mechanisms of development and progression. Multi medial mapping, methods to differentiate the 3 different phenotypes of diabetic retinopathy describes options for personalized management strategies. The chapter on Optical Coherence Tomography provides additional new information on morphological findings in diabetic retinopathy.

The section on treatment options for diabetic retinopathy with emphasis on the management of diabetic macular edema elaborates on standard and novel laser therapies, current surgical options for diabetic retinopathy, and the beneficial effect of surgical options on diffuse diabetic macular edema. The next few chapters deal exclusively with the role of pharmacotherapeutic agents both systemic and intravitreal in the management of diabetic retinopathy. The use of triamcinolone acetonide intravitreal injection is discussed in detail with an emphasis on the incidence of complications, its transient efficacy and recurrence.

The use of VEGF inhibitors in the management of diabetic macular edema is presented with results of recent studies. Latest concepts on pharmacological vitreolysis are discussed highlighting the role of PVD in diabetic retinopathy and is presented by the Microplasmin study group.

Treatment of diabetic retinopathy with protein kinase C subtype \( \beta \) inhibitors Ruboxistaurine mesylate is described and study results presented.

Thus this book provides a profound insight into the pathogenesis and management of diabetic retinopathy. A recent and updated version presented in this book provides ample material and hence this book will be extremely useful for all categories- residents, general ophthalmologists and retinologists alike.

Compiled by Dr. Meena Chakrabarti & Dr. Sonia Rani John
Fundus Fluorescein and Indocyanine Green
Angiography- A Text book and Atlas

Edited by Amar Agarwal
Published by SLACK INCORPORATED. 2008

The rapid evolution of imaging has provided with a
better understanding of the patho physiologic
mechanisms in retinal disorders specifically macular
disorders. A combination of FFA, ICG and OCT has
helped us diagnose, differentiate, classify and treat
numerous retinal disorders with ever improving
accuracy. Newever treatment modalities necessitate the
use of accurate and advanced imaging techniques to
treat and follow up patients with a variety of retinal
and macular disorders.

Fundus fluorescein and indocyaninie green
angiography: A Text book and Atlas is a reference book
that comprehensively covers angiography technique
and provides essential information for an accurate
interpretation of these vital diagnostic tests.

The first 2 chapters of this book deal with the basics
and principles of FFA and ICG as well as other advanced
angiography technique. Chapters dealing with
evaluation, instrumentation, basic principles and
usually used terminology in angiography will be very
useful for the beginner. The phases of fluorescein
angiography and ICG are dealt with in detail. A chapter
on the hazards and complications of FFA provides
necessary information for planning the procedure and
counseling the patient.

Section II is on advanced angiography techniques such
as Anterior segment fluorescein angiography, confocal
scanning retinal angiography, application of fundus
autofluorescence imaging and wide field imaging
techniques. Chapter on ultra wide field angiography
and OCT helps to comprehensively depict various
retinal pathologies for accurate diagnosis and
management.

In the latter 4 sections of this book specific retinal
disorders as well as optic nerve diseases are exhaustively
covered with emphasis or pertinent pearls to diagnosis
and management.

This reference text book uses a text and atlas format
to convey its key points. It includes 550 images
and photographs to provide a visual representation
of the disease process. Also included is an
accompanying CD-ROM with over 35 minutes of
supplemental video.

About 30 top notch retinal specialties have
contributed the various chapter in this text book atlas.
Authentic current and reliable information on
interpretation of various imaging techniques provided
by this atlas will prove to be immensely useful to
one and all.
Optical Coherence Tomography (OCT) is a powerful tool for the assessment of ocular diseases. The ability to visualize cross sectional image, akin to an optical biopsy of ocular tissues, both quantitatively and qualitatively, helps in easy discrimination of health and disease as well as in assessment of changes during the course of the disease.

Being a noninvasive investigational modality OCT has gained acceptance and is now used world wide in settings ranging from, primary to quaternary care in both developed and developing countries. A handbook on OCT for clinicians and technicians has been a long felt need and this publication has helped fill up that void.

In the first chapter explicitly detailed information on how to operate OCT, tips on how to acquire images, identify problems in images and image acquisition, how to store and back up OCT data, how to retrieve patient data and how to export data and image is given. This is the most useful part of this hand book. As the language and presentation is simple and lucid, the technical aspects become easy to understand.

Charts detailing “trouble shooting” while scanning creates an awareness of the various problems that the operator faces during the procedure. Likely causes for these problem and the best solutions to circumvent the problem are detailed which are absolutely essential to all operators. These can be used as a ready reckoner by the technician and the ophthalmologist and will definitely help in acquisition of ‘perfect’ image.

The second chapter provides detailed information, beautifully depicted pictorially on how to interpret OCT images and printouts. Retinal diseases, glaucoma and neuro-ophthalmic conditions are dealt with in detail. Scan patterns and interpretation of common scans in healthy eyes provide useful information.

The last chapter on history of OCT and how OCT works is a “must-know” for all OCT users. Explanation of various commonly used OCT technologies and instrumentation to acquire retinal and anterior segment images help to simplify ‘difficult to understand’ technological aspects of this imaging technique. This chapter opens a window to where technology will take us in future.

Thus this handbook deals with all aspects of Stratus OCT and will be an useful ready reckoner to keep in your procedure room.
CME Programmes

STATE CONFERENCES
Ananthanayanam: CME 2009
Annual CME of Regional Institute of Ophthalmology, Trivandrum
26th April 2009
Organizing Secretary: Dr.Biju John
Mob:9349351689

NATIONAL CONFERENCES
Indian Intraocular implant and Refractive Surgery Convention
July 11-12, 2009
Hotel Taj Coromandel, Chennai, India
Organizing Secretary: Dr.Amar Agarwal
Ph:91-44-28 115871

Kalpavriksha 2009:
National PG CME Programme
October 1-4, 2009
Organizer: Dr.Agarwal’s Eye Hospital, Chennai

INTERNATIONAL CONFERENCES
MEACO
The Middle East African Council of Ophthalmology 2009
March 26-30, 2009
Bahrain
www.meaco.org

ASCRS 2009
American Society of Cataract and Refractive Surgeons
Annual Meeting April 3-8, 2009
SANFRANCISCO: Moscone Convention Centre
www.ascrs.org

APAO-AAO Joint Meeting
The 24th Congress of the Asia-Pacific Academy of Ophthalmology
May 16-19, 2009, Bali
secretariate@apaophth.org

World Glaucoma Congress
July 8-11, 2009
Boston, Massachusets
www.worldglaucoma.org

AAO-PAAO 2009
October 24-27, 2009
Sanfrancisco
www.aao.org/2009
The unique formulation of OPTIVE™

Goes Deep to Uniquely Tackle Hypertonicity¹

optive™
Next Generation Tears

ALLERGAN INDIA PRIVATE LIMITED
1st Floor, North Wing, Silver Jubilee Block, Unity Building Complex,
Mission Road, 3rd Cross, Bangalore-560 027  Tel: 91-80-40707070
Fac: 91-80-2229 9130   Email: allergan@agnindia.com
For the use of a registered medical practitioner, hospital or laboratory only.

FAQ’s : Performing Adequate Anterior Vitrectomy in the Event of Vitreous Loss (VL) During Cataract Surgery

Q1. Why is vitreous loss (VL) important?

Vitreous loss during cataract surgery is associated with a higher incidence of complications such as
(a) Retinal detachment: 6.8 %- 8.6 %
(b) Cystoid Macular edema: 4 %- 8 %
(c) Persistent Uveitis: 6.7 %
(d) Glaucoma: 20 %
(e) Corneal Edema 9 %.
Complications occur in 19 % to 30 % of eyes who have VL during cataract surgery.

Q2. When there is a VL what are the DON’Ts?

- Do not try to cut vitreous with phacoemulsification handpiece as the ultrasound energy will only liquefy the hyaluronic acid without cutting the collagen framework of the vitreous.
- Do not use a large bore needle to aspirate pockets of fluid vitreous.
- Do not use a Weck Cell Sponge to drag the vitreous strands out of the eye as this manoeuvre can exert traction at the vitreous base.
- This technique can mechanically chafe the iris and the retained cellulose material on the anterior vitreous cortex can incite marked postoperative inflammation.
- Do not use a full function vitrectomy probe as it has a larger size and more turbulent fluid inflow which can churn out more vitreous.

Q3. Instrumentation to perform anterior vitrectomy?

- Use a divided function probe (20 g/ 23g/ 25g)
- Use Bimanual approach.
- If there is only a small amount of vitreous prolapse a dry vitrectomy is advisable.

Q4. Why co-axial infusion should not be used?

If co-axial infusion is used, the infusion is provided by a sleeve slipped over the cutter. While using the cutter to cut the offending vitreous, the flow will hit the open posterior capsule enlarging it further. It will also hydrate the vitreous increasing the volume of contents in the posterior segment thereby increasing the vitreous prolapse. The turbulence of the infusion will also churn out more vitreous. These factors contribute to aggravating the VL and hence use of co-axial infusion is not advisable for anterior vitrectomy.

Q5. How is bimanual vitrectomy performed?

- Use separate ports for infusion and vitrectomy.
- A Bent 20 G infusion cannula; AC maintainer or the irrigating handpiece of I/A unit can be used for infusion.
- Use the paracentesis site for infusion.
- Do not use phaco wound for vitrectomy as it is too large for the cutter. Open another paracentesis site.
Q6. What is the appropriate strategy for bimanual anterior vitrectomy?
- The cutting tip of the cutter is directed through the opening in the posterior capsule and directly under the vitreous prolapse.
- The cutting port should be directed upwards.
- The infusion is directed into the AC parallel to the iris plane.
- Vitrectomy is performed until the offending vitreous in the anterior chamber is removed down to the level of and just below the PC rent.

Q7. What are the appropriate settings for anterior vitrectomy?
- Use maximum possible cut rate available on your machine.
- Use lowest suction.
- While cutting, the cutter can be held stationery or advanced.
- Never retract / pull back cutter while it is cutting.
- Do not use Low speed, High suction or cut while pulling away.

Q8. Do we need to delineate the vitreous in the AC to ensure completeness of anterior vitrectomy?
Sometimes vitreous strands in AC may be invisible. To identify these strands we can use air or triamcinolone acetonide injection.

Q9. How is air useful in anterior vitrectomy?
- We can confirm presence of vitreous if air bubble fragments in the AC
- When anterior vitrectomy is performed under air, the bubble contains the vitreous in the posterior segment and the vitreous also does not get hydrated.
- The surface tension exerted by the air bubble contains the vitreous in the posterior chamber preventing prolapse of more vitreous.

Q10. What is the role of triamcinolone acetonide in anterior vitrectomy?
Crystals of Triamcinolone acetonide help delineate the vitreous in the AC. It has the effect of “cloaking a ghost” and ensures completeness of vitrectomy. A few crystals of TA that may get left behind will help minimize postoperative inflammation.

Q11. Can the pars plana route be used for anterior vitrectomy?
The pars plana approach is the safest and ideal route for a surgeon who is comfortable using it and has experience with pars plana vitrectomy. Here the cutter is stationed with the cutting port facing upwards in the area of the PC rent.
International Society for Eternal Youthful Life!

Dr T.P. Ittyerah, Angamaly

It is the desire & dream of all persons maturing to old age that they should remain in the young youthful age. When ever you hear the sad news of the demise of your near & dear, don’t you curse the death? The unattained desire of mankind for perpetual life is to some extent attained by believing that an individual has two components, namely the BODY & the SOUL and blindly believing that the SOUL is living after your death. This belief has given sufficient strength to an individual to face death boldly. But occasionally to the extent of suicide believing that only the body (body is often compared as the dress of soul) is perished, while the soul shall be doing every thing he or she wanted to do while living (suicides by young couples when the society refuses there request for marriage.). Besides this it has discouraged research in the direction of elimination of death. Understanding death is very important for eliminating death, perhaps apoptosis & death starts as early as growth stops.

There are several efforts to maintain the appearance & vigor of young age by the use of medicines, cosmetics,& plastic surgeries,. There are several efforts for elimination of old age disabilities & problems by cataract surgery, prostate surgery, knee transplantation etc. All these activities going on in medical science are focused to a particular old age problem. We have plans & methods only to reduce disability & to postpone death but none to eliminate death!

We have a geriatric specialty in medicine to treat the problems of old age in total. This is not very popular specialty in our country perhaps because our bulk of population is sill young. Soon we will have a large older population in Kerala and looking after them shall be a big burden to the young persons.

It is every body's desire to avoid old age, but the time & money spend on this is relatively less compared to the money & time spend for life after death in heaven or for attaining ‘moksha’. The problem is so vast but we pretend that we don't see it as a problem. It is in fact the problem of every body living & going to live on this earth. Why there is not sufficient effort on the part of human beings to avoid this problem .I feel that this is because people think that, this is impossible to achieve. & accept that death is inevitable. Unlike many apparently impossible things (Landing man on the moon, Elimination of smallpox, Heart transplantation etc.) man has achieved by intense research there is not sufficient effort to achieve old age prevention & avoidance of death. To get a cure for death one should first recognize death as a disease (When mental diseases were recognized as a disease, not the invasion of body with evil spirit, we have medicines to cure them; Similarly when you recognize death as a disease, not the process of separating soul from the body, we will have remedial medicines for death) I feel that death should be considered the ‘ mother ’ of all diseases!

Another reason for this lack of enthusiasm in this research could be it may topple the whole concept of religion in the present form. Without death & life after death no religion can survive & flourish .The religious beliefs are very strong in 90% of the population right from child hood. A society to prevent natural death
shall be discouraged & mocked at by a big group of persons related to religion & benefited by death. It may be described as madness, eccentricity, impractical & unethical venture. It may even be accused that, by eliminating natural death we create new problems. But all good ideas were impractical at that point in time & severely criticized by many influential persons. If you remain afraid of criticism nothing will move. So I solicit the whole hearted support of like minded personalities to start on a program now itself. There was a concept that birth & death are in Gods domain & man should not interfere with them. There was severe criticism by priests when birth control was introduced. Now it is a widely accepted as part of a family well fair program. Can you have a good family well fair program with out death control also? In fact death control shall have more impact on the society than birth control. The new generation of couples has less interest & time to produce children and bring them up. Many developed world is on negative population growth Soon India & China also come under negative population growth. In such situation to have enough working force the solution is death control only.

There is some thinking & teaching that every organism on earth has to die one day or other. But this is not very true. The unicellular amoeba & bacteria representing animal & plant kingdom divide in to two when they mature & do not grow old & do not have a natural death. This means majority of organisms (created by the same God who has created man?) on earth do not have natural (old age) death. Man on evolution has lost this capability. Can't we recover it in one way or other? Let us join together to make an effort to recover what is lost in the process of evolution. The society can initiate research in multiplication of unicellular organisms. It can do & support research in the identification of the gene responsible for multiplication of these organisms & the use of the genes in very small multi cellular organisms for induction of division of all cells simultaneously to produce another young organism of half the size & age. When it succeeds it can be repeated in higher organisms and on humans. It means that in one stroke we can get rid of not only the pains of death but also the labor pains! The society can associate with like minded biotechnological organizations & societies to achieve its objective. I may not be there to see the results; but I am sure my grand children shall be able to see the result. Why don't you invest your time & money for your grand children &grate grand children.

I hope & believe that this society shall be the modest beginning and live till the” natural death” yields to man's will power & intelligence.

Dr Ittyerah,T.P.

Please give your opinion about this article below.
(Please tick)
1) Madness
2) Nonsense
3) Against God & God will punish
4) Impractical & impossible
5) Creates new problems & should drop the project.
6) May try but not possible in this century.
7) Hopeful & try with full effort for the success of the project.
8) Interested to be part of the program without much financial commitment.
9) Interested & willing to partly finance the project

NAME OCCUPATION PHONE MAIL ID.
GENERAL INSTRUCTIONS TO AUTHORS

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

11. All manuscripts are subjected to editorial board review.

12. Other Categories of Manuscript

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

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

    Brief reports are short communication of new instruments, new laboratory techniques or surgical techniques as well as interesting case reports with unique findings. These should not exceed 1000 words with a maximum of 2 illustrations. They should follow the format - introduction, case, and discussion. No more than 8 references should be cited. Each brief report must begin with a 75-100 word summary that highlights the significance of the articles.
UV radiation in combination with Riboflavin initiates molecular cross-linking of corneal collagen. Thus progressive corneal thinning is slowed down or even stopped. Biomechanical strength of corneal tissue is improved.

A number of clinical studies have demonstrated that progressive keratokonus and iatrogenic ectasia can be stabilized by corneal cross-linking.
You have seen the rest, now see the best!

"Aspheric Technology with ZEISS OPTIC (ZO)"

- The legendary Carl Zeiss aspheric optics on to an IOL platform for the first time
- Based on the proven, trusted and widely recognized LBE ( Liou & Brennan Eye ) Model
- Low sensitivity to decentration and tilt
- High contrast sensitivity even in low light conditions for the largest range of spatial frequencies
- Preloaded- Easy, Quick and Safe surgical workflow

Compliment your surgical skills with "ZO"

Contact for further details

Carl Zeiss India Pte. Ltd.
No: 22, Kensington Road
Ulsoor, Bangalore - 560 008
INDIA

Tel: +91-80-2557 8888
Fax: +91-80-2557 9999
E-mail: medindia@zeiss.co.in
Web: http://www.zeiss.co.in
The professional system for superb slit lamp photography

8.13 Megapixels of resolution produce clear and crisp images, for the highest quality documentation.

- Fully Integrated
- DC-3 Capture Module
- Auto Capture Mode
- High Resolution
- Movie Capability
- Right / Left Eye Indicator (SL-D7 / D82)

DC-3 can be used only with Topcon's SL-“D” Series of slit lamps
SL-02/04/07/082

Mehra Eyetech Private Limited
We care in Eye Care

Corporate Office: 54, Kaliandas Udyog Bhavan, Near Century Bazar, Prabhadevi, Mumbai-400 025.
Tel.: 022-6660 3121 Fax: 022-2437 8531 E-mail: contactus@mehraeyetech.in
http://www.mehraeyetech.in