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Dr. Rajesh P
Dr. Revathy Ramesh
Dr. S. Venugopal
7 Editorial
Dr. Mahesh G.

Major Review

8 Optic Atrophy
Dr. Devendra V. Venkatramani, Dr. Gangaprasad Muthaiah Amula, Dr. Rashmin A. Gandhi

15 An Introduction to Idiopathic intracranial hypertension
Dr. Easwer HV, Dr. Suresh Nair, Dr. Vikas V, Dr. Jayanand Sudhir

26 Multifocal IOL – An Overview
Dr. Minu Mathen

Original articles

32 Relationship between structure and function of the optic nerve head-Glaucoma versus Normal
Dr. Savita Bhat, Dr. Anna Elias, Dr. Siddharth Pawar, Dr. S.J. Saikumar, Dr. Alpesh Rajput

41 Lupropros Keratoconjunctivitis in the rubber plantation area of Pathanamthitta District
Dr. Dona Susan John, Dr. Ashley Thomas Jacob, Dr. Liz Thomas, Dr. Anand S. Koottummel, Dr. Jyothi.K.

45 A Clinical study of complicated cataract in Uveitis
Dr. K.V. Raju, Dr. Sisira Sivan

50 Comparison of Peribulbar Anaesthesia with Topical Anaesthesia in Manual Small Incision Cataract Surgery
Dr. Smita Narayan, Dr. Rajini KC, Dr. Sujata N, Dr. Mallika V

Ophthalmic Surgery

56 Perioperative glucose control: what an ophthalmologist needs to know
Dr. A.G. Unnikrishnan
## Current Practice

### 60
**Contact lens in Keratoconus – An Update**  
Dr. Deepa Paulose

### News that you can use

### 66
**How to make a good powerpoint presentation**  
Dr. Biju John

## Ophthalmic instrumentation

### 72
**Optical coherence tomography - Interpretation made easy**  
Dr. Avinash Pathangay

## Systemic diseases and eye

### 79
**Retinal manifestations in systemic infections- A Ready reckoner**  
Dr. Tufela Shafi, Dr. Sandhya Menon, Dr. Gopal S. Pillai,  
Dr. Natasha Radhakrishnan

## Brief reports

### 85
**Successful Correction of Macular Ectopia and Disc Intortion with Vitreoretinal Surgery**  
Dr. Simi Manojkumar, Dr. Jyothirmayi, Dr. Vanaja Raghavan,  
Dr. Abhijit Khake, Dr. Sreeni Edakhlon, Dr. Gopal S Pillai

### 87
**Bee sting injury to eye**  
Dr. Jyotiparakash Vyas, Dr. Siddharth Pawar,  
Dr. Mahesh G. Dr. Manual John, Dr. Ivan Jacob

### 89
**FONSECAEA PEDROSOL – Unusual Cause For Corneal Ulcer – A Case Report**  
Dr. Bindu N Das, Dr. Jalal P. M

### 91
**Ophthalmic History – Sir Harold Ridley**

### 92
**What is new – Pascals Dynamic Contour Tonometry**  
Dr. Chokkalingam

## Spot Diagnosis

### 99

## Journal Review

### 101

## Book Review

### 103

## PG Corner

### 104

## Instructions to authors

### 106
Kerala Journal of Ophthalmology (KJO) has witnessed significant improvement in the quality of articles in the last few years. Currently it is one of the best rated journals among the state societies in India. The new editorial office has the added responsibility of keeping the standards high. Moreover some positive changes are considered in the layout and contents of the journal. At present this journal is read by the 1100 odd members of Kerala Society of Ophthalmic Surgeons. This includes comprehensive ophthalmologists, post graduate students and specialists. Aiming at these variable readership patterns we have made some changes in the format of the KJO.

The major reviews are related to neuro ophthalmology. Most of us encounter cases of optic atrophy and disc oedema in the general practice. Approach to these two conditions and relevant pathophysiological aspects are discussed in the first two review articles. Then there is something for the cataract surgeons regarding the status of latest multifocal intraocular lenses. Incidentally the Ophthalmic history section deals with the pioneer who introduced intraocular lenses.

Ophthalmic surgery section deals with the commonest problem faced by all surgeons. What is the ideal blood sugar level when a patient is taken for eye surgery? Article gives the current accepted guidelines for perioperative glucose control.

We have one more article for the comprehensive ophthalmologist who works in multispecialty hospitals. It highlights the possible manifestations in the eye in a setting of systemic infections. The pictures in the article can be used as a ready reckoner for diagnosis. In current practice section contact lens options for Keratoconus are discussed.

Optical coherence Tomography is now a standard method of evaluation of macula. It is important for any practicing ophthalmologist to know the basics of interpretation of OCT. The article on interpretation of OCT is a simple step by step method for interpretation which is useful for post graduate students also.

We have started a new series “News that you can use”. In the first article author has described the intricacies of making a good Power Point presentation. This will be of use to students as well as ophthalmologists preparing for some lectures.

In the “what is new in ophthalmology” section we have an article on relatively newer dynamic contour tonometry (DCT). In the PG tear sheet methods of tonometry are described in detail.

Any journal is incomplete without original article. We have original studies and case reports. Special emphasis is given to originality of the article like one on endemic keratoconjunctivitis in the rubber plantation area in Kerala. Other regular titles are continued in this issue also.

A journal is rated based on the readership and we welcome positive criticism. Editorial office can be contacted by e mail. In future the aim of this editorial team is to improve the quality further and if possible index the journal.

Expecting whole hearted co operation from each and every one of you

Mahesh G
Optic Atrophy

Dr. Devendra V. Venkatramani, Dr. Gangaprasad Muthaiah Amula, Dr. Rashmin A. Gandhi

Introduction

Optic atrophy is the ultimate end result of diseases that cause degeneration of axons of the ganglion cells, and manifests as changes in the color and the structure of the optic disc. It is associated with variable degrees of visual dysfunction.

The optic nerve contains approximately 1.2 million axons of the ganglion cells (1st order neurons) of the retina. The axons possess a myelin sheath provided by oligodendrocytes. Once damaged, the axons do not regenerate.

Light incident from the ophthalmoscope undergoes total internal reflection through the axonal fibers, and subsequent reflection from the capillaries on the disc surface gives rise to the characteristic yellow-pink color of a healthy optic disc. Degenerated axons lose this optical property which explains the pallor in optic atrophy.

Alternatively, the loss of pial capillaries which supply the optic disc may be the cause of disc pallor. The Kestenbaum index is the number of capillaries counted on the optic disc, which normally is around 10. Less than 6 capillaries indicates optic atrophy; more than 12 suggests disc hyperaemia.
Histopathologic changes in optic atrophy

- Deepening of the disc cup with baring of the lamina cribrosa
- Loss of both myelin and axons
- Glial cell proliferation
- Widening of the subarachnoid space with redundant dura
- In nerve transaction, the severed end produces bulbous axonal swellings (Cajal end bulbs).

Classification of Optic Atrophy

Ophthalmoscopic classification

- **Primary optic atrophy**: (e.g., pituitary tumor, optic nerve tumor, traumatic optic neuropathy).
  - Nerve fibers degenerate in an orderly manner and are replaced by columns of glial cells.
  - No alteration in the architecture of the optic nerve head.
  - Disc is chalky white and sharply demarcated.
  - Retinal vessels are normal.
- **Secondary optic atrophy**: (e.g., papilledema, papillitis)
  - Atrophy is secondary to prior disc swelling.
  - Excessive proliferation of glial tissue which causes the disc to appear dirty grey, and obscures the lamina cribrosa.
  - Poorly defined margins.
- **Consecutive optic atrophy**: (e.g., retinitis pigmentosa, myopia, central retinal artery occlusion)
  - Waxy pallor of the disc.
  - Marked attenuation of arteries.
- Normal physiologic cup and disc margin.
- **Glaucomatous (‘cavernous’) optic atrophy**:
  - Marked cupping along with vertical enlargement of cup.
  - Lamina cribrosa pores seen (laminar dot sign).
  - Bayoneting and nasal shifting of the retinal vessels.
  - Peripapillary halo and atrophy.
- **Temporal pallor**: may be observed in traumatic or nutritional optic neuropathy, but is common in patients with a history of optic neuritis.

Etiological classification

- **Hereditary**
  - Leber optic atrophy
  - Congenital or infantile optic atrophy (recessive or dominant form)
  - Behr hereditary optic atrophy (autosomal recessive)
- **Consecutive atrophy**: due to diseases of the retina and/or choroid
  - Chorioretinitis
  - Pigmentary retinopathies
  - Extensive retinal laser photocoagulation/long standing retinal detachments
- **Vascular**: ischemic optic neuropathy (arteritic or non-arteritic).
- **Toxic or drug-induced**: tobacco, methyl alcohol, ethambutol, sulphonamides, etc.
- **Metabolic atrophy**: nutritional amblyopia, juvenile diabetes mellitus and thyroid ophthalmopathy.
• **Demyelination**: multiple sclerosis and Devic's disease.

• **Pressure atrophy**: diseases such as glaucoma and papilledema.

• **Post-inflammatory**: optic neuritis, perineuritis secondary to meningitis.

• **Traumatic optic neuropathy**: optic nerve avulsion and transection, optic nerve sheath hematoma, and optic nerve impingement from a penetrating foreign body or bony fragment can contribute.

**Pathologic classification**

• Anterograde degeneration (Wallerian degeneration): Degeneration begins in the retina and proceeds toward the lateral geniculate body (e.g., toxic retinopathy, chronic simple glaucoma). Larger axons disintegrate more rapidly than smaller axons.

• Retrograde degeneration: Degeneration starts from the proximal portion of the axon and proceeds toward the optic disc (e.g. optic nerve compression by intracranial tumor).

**Epidemiology**

Optic atrophy can be seen in any age group. There is no sex predisposition noted.

**Differential diagnosis**

Non-pathologic disc pallor is seen in axial myopia, myelinated nerve fibres, optic disc pit, tilted disc, and disc drusen. Viewing the disc in a pseudophakic eye, or using a brighter ophthalmoscope than usual can cause the disc to look paler.

**Clinical Work-up**

**Visual acuity**

It is measured using Snellen’s optotypes or using a LogMAR chart. Visual acuity is reduced, occasionally to no light perception.

**Color vision**

Color vision is more decreased in patients with optic nerve disorders than in those with retinal disorders especially in patients with ischemic and compressive optic neuropathy.

Color vision may be assessed with pseudoisochromatic tests (e.g., Ishihara color blindness test, Hardy-Rand-Rittler polychromatic plates, Dvorine plates) or the Farnsworth-Munsell 100 Hues test or the Farnsworth panel D-15 test.

**Pupillary evaluation**

Pupil size should be noted, as well as the magnitude and the latency of the direct and consensual responses to light and near stimulation.

A relative afferent pupillary defect (RAPD) is a hallmark of unilateral or asymmetric afferent sensory abnormality. Occasionally it is the only objective sign elicited. RAPD can be quantitatively graded by balancing the defect using neutral density filters.

Clinically, it is graded as follows:

• Initial constriction, but greater escape to a larger intermediate size than when the light is swung back to normal eye (trace).

• No changes in initial pupillary size, followed by dilation of the pupils (1-2+)

• Immediate dilation of the pupil, instead of normal initial constriction (3-4+)

**Contrast sensitivity test**

This test measures the ability to perceive slight changes in luminance between regions that are not separated by definite borders, and is a sensitive test for optic nerve function.

It can be tested using Pelli-Robson contrast
sensitivity chart, Cambridge low-contrast grating test or Arden gratings.

**Pulfrich phenomenon**

In optic nerve damage, the transmission of impulses to the occipital cortex is delayed. In patients with unilateral or markedly asymmetric optic neuropathy, when an oscillating small target in a frontal plane is viewed binocularly, the target appears to move in an elliptic path rather than in a to-and-fro path.

**Extraocular movements**

Restriction can be obtained in cases of compressive optic neuropathy due to either the mass effect or the involvement of the nerve supplying the muscle.

**Cranial nerve examination**

All cranial nerves are examined to rule out associated nerve involvement to help determine the site of the lesion.

**Ophthalmoscopic features**

**Optic disc**

Optic disc changes can present with temporal pallor, focal pallor or bow-tie pallor (as seen in compression of the optic chiasma), or cupping (glaucomatous damage).

In the early stages of the atrophic process the optic disc loses its reddish hue. The substance of the disc slowly decreases, leaving a pale, shallow exposed lamina cribrosa. In the end stages of the atrophic process the retinal vessels of the normal caliber still emerge centrally through the otherwise avascular disc.

Focal notching or diffuse obliteration of the neuroretinal rim with preservation of color of any remaining rim tissue is characteristic of glaucoma.

Optic disc cupping also develops in patients in non-glaucomatous eyes due to ischemia, compression, inflammation, hereditary disorders or trauma.

**Peripapillary retinal nerve fiber layer**

Early focal loss of axons produces dark wedge-shaped defects (best seen with a red-free filter on slit-lamp bio-microscopy) in the peripapillary retinal nerve fiber layer.

**Retinal vessels**

In most cases of optic atrophy, the retinal arteries are narrowed or attenuated. In cases of non-arteritic anterior ischemic optic neuropathy, the vessels may be focally narrowed or completely obliterated.

**Investigations**

**Visual field testing**

In optic neuropathy, visual field changes can include enlargement of the blind spot, caeco-central scotoma, altitudinal defects (e.g. anterior ischemic optic neuropathy, optic neuritis), and bitemporal defects (e.g. compressive lesions, similar to optic chiasma tumors).

**Neuro-imaging**

Neuro-imaging is indicated to find the cause of atrophy.

- Ultrasonography is recommended when orbital tumour is suspected.
- For post-traumatic optic neuropathy a non-contrast CT scan is preferred.
- In optic neuritis or multiple sclerosis, a gadolinium-enhanced MRI/fluid-attenuated inversion recovery (FLAIR) sequence is useful to detect hyperintense areas of demyelination.

**Electroretinogram (ERG)**

Abnormal electroretinogram (ERG) results that
can be seen are as follows:

- **Subnormal**: Potential less than 0.08 microvolts; seen in toxic neuropathy
- **Negative**: a preserved a-wave but absent b-wave. May be seen in arteritic AION or central retinal artery occlusion.
- **Extinguished ERG**: seen in complete optic atrophy.
- **N95:P50** ratio in pattern ERG is low in optic neuropathy and normal in maculopathy.

**Visually evoked potential (VEP)**

In optic neuritis the VEP has an increased latency period as compared to the normal eye which persists even after visual recovery. Compressive optic lesions tend to reduce the amplitude and cause waveform changes of the VEP.

**Unexplained optic atrophy**

As optic atrophy is a sign of end-stage optic nerve damage and not a diagnosis in itself, further investigation is required if the above tests do not reveal its cause. These include:

- Blood glucose level
- Blood pressure, cardiovascular examination
- Carotid Doppler ultrasound study
- Venereal Disease Research Laboratory (VDRL)/Treponema pallidum hemagglutination (TPHA) tests
- Serum vitamin B-12 levels
- Anti-nuclear antibody levels
- Sarcoid work-up
- Homocysteine levels
- Antiphospholipid antibodies
- ELISA for toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus (TORCH panel)

**Treatment**

No proven treatment exists to reverse optic atrophy. At present, the best defense is early diagnosis.

If specific treatment of the cause is initiated before the development of optic atrophy, useful vision may be salvaged. For example, early diagnosis and prompt treatment can help in compressive and toxic neuropathies.

Neuro-protective agents like gingko biloba have been tried with anecdotal success.

Research in stem cell therapy may provide answers in the not-too-distant future.

Low-vision aids should be considered for occupational rehabilitation.

**Suggested further reading**

Figure 1  Healthy Optic Discs

Figure 2  Primary Optic atrophy

Figure 3  Secondary (post-papilloedema) atrophy

Figure 4  Glaucomatus atrophy

Figure 5  Consecutive optic atrophy (post pan-retinal photo coagulation)
An Introduction to Idiopathic Intracranial Hypertension

Dr. Easwer HV, Dr. Suresh Nair, Dr. Vikas V, Dr. Jayanand Sudhir

Introduction

The constellation of raised intracranial pressure without ventricular enlargement along with an elevation of cerebrospinal fluid pressure with a normal composition of the same constitutes Idiopathic Intracranial Hypertension (IIH). The many names it bears viz. pseudotumor cerebrii, serous meningitis, otitic hydrocephalus benign intracranial hypertension underscores the enigma that it is in terms of etiology and pathogenesis apart from it’s uncertain clinical course. Owing to it’s significant morbidity in terms of possible visual loss and chronic headache IIH needs early diagnosis and management.

Pathogenesis of Idiopathic Intracranial Hypertension

Though modern imaging studies have demystified our understanding of this disease, much is yet to be known about the exact pathogenesis. As per the Munro Kellie doctrine (1), there is a constant relationship between blood, CSF and brain contents with one increasing at the cost of raised pressure beyond a point of physiological limit. An increase in CSF production, proposed in the pathogenesis of IIH by Quincke (2) has since been discounted. Dandy (3) proposed increased blood volume though later investigators could not find evidence for the same. The recent studies with MR imaging reveal increased white matter water with an increase in apparent diffusion coefficient of the same (4).

The more acceptable theory includes one of brain’s venous out flow obstruction at the large cortical venous sinuses. IIH involves elevated venous pressure, leading to increased resistance to CSF absorption and subsequently increased ICP (5). Though an obstruction is not always evident, the pressures in these venous channels are found to be elevated in patients with IIH. Here again controversy is courted as to whether the sinus obstruction or resistance to venous outflow is secondary to elevated intracranial pressure affecting the cortical venous system (6).
Clinical features

The common symptom that brings the patient to the physician is headache, which is often more in the early hours of the morning (7). Vomiting though this is not a common feature at times relieves this. Pain along the occiput and trigeminal regions of the face also has been reported.

It is the onset of visual obscurcation with the headache that often necessitates a comprehensive ophthalmological evaluation for the victims of IIH. Another visual phenomenon is the presence of double vision due to abducens nerve involvement (8).

Tinnitus is present in some due to augmented blood flow through the venous sinuses is often experienced by its sufferers (9). Ataxia and vertigo also accompany the visual disturbances. Often there may be features of endocrine disturbances in the form of pituitary deficiency due to empty sella turcica.

It is important to obtain a detailed history of previous drug therapies as many a time the IIH may be secondary to pharmacological interventions. This list includes Vitamin A (10,11) and its derivatives, Tetracyclines(12), Fluoro quinolones, sulfa derivatives, growth hormone(13), and steroids including oral contraceptives(14).

The list of systemic disorders associated with IIH includes SLE (15), anemia(16), underlying malignancies, (17),Addison's disease (18), thyroid disorders(19), uremia (20) etc. It is important to rule out the same prior to embarking on surgical treatment.

The Dandy's criteria (21) modified subsequently holds good to identify cases of IIH as it seeks out those with features of raised intracranial pressure in an alert patient without neurologically localizing features or false localizing features .The CT/MRI subsequently rules out mass lesion and the CSF study shows elevated pressure without other causes being found for the same.

Investigating a case of IIH

The investigations into a case of suspected IIH includes a visual evaluation including optic fundus examination and perimetry to evaluate the severity of papilledema and extent of visual loss. Rarely papilledema may be absent too (22). The Snellen’s visual (23) chart may not reveal the extent of visual loss. Visual Evoked Potential often show disturbances. Endocrine evaluation involving cortisol and thyroid evaluation is also required (18,19).

CT scan is often the commonest investigation sought for IIH as the physician tries to rule out a case mass lesion in the brain. The widened perioptic space and the presence of empty sella turcica (arachnoid membrane hernia ting into the pituitary fossa) are often present. A slit-like ventricle, though classically described, is seldom found in patients with IIH (25).

Cerebrospinal Fluid (CSF) evaluation

Once the presence of mass lesion is ruled out the neurologist often embarks to rule out any chronic meningitis by doing a lumbar puncture in a guarded fashion due to raised intracranial pressure. The CSF often shows very high pressure but significantly the chemical and cytological composition does not show any significant alteration suggestive of absence of chronic meningitis, which often mimics IIH.

MRI in IIH

MR imaging especially with angiography has become the cornerstone of any investigation into IIH.

Magnetic resonance Imaging of the brain may reveal (not always) empty sella turcica with arachnoid invagination, patulous optic nerve
sheaths, flattening of the posterior sclera of the globe and papillary projections on the optic nerve sheath in varying frequencies (26,27,28).

The presence of dural venous sinus stenosis is being increasingly identified in cases of IIH thanks to improvements in the Magnetic Resonance Imaging techniques and technology. One report yielded 27 cases of sinus stenosis in a series of 29 cases imaged using the auto-triggered elliptic-centric-ordered three-dimensional gadolinium-enhanced MR venography (ATECO MRV)(6). This has often changed the management principles from CSF diversions and other surgeries to endoluminal procedures dilating the narrowed venous systems.

References:

6. Idiopathic intracranial hypertension: The prevalence and morphology of sinus venous stenosis. R.I. Farb, MD; I. Vanek, MD; J.N. Scott, MD; D.J. Mikulis, MD; R.A. Willinsky, MD; G. Tomlinson, PhD; and K.G. terBrugge, MD. Neurology 2003; 60:1418–1424.
Medical management of IIH

Carbonic anhydrase inhibitors (e.g., acetazolamide) are the only effective medications for treatment of IIH. Acetazolamide was originally demonstrated to variably decrease CSF production by 6 to 57% among human subjects [1]. A standard dosage is a 500-mg time-release capsule twice daily. The effective dose must be individualized; a dose of 0.5 to 1g per day usually suffices for resolution of papilledema. Caution must be exercised in prescribing Acetazolamide in pregnancy for reported teratogenicity, including limb malformations and cortical dysgenesis in animals [2]. Case reports have documented neonatal metabolic acidosis and electrolyte abnormalities [3]. Furosemide, another diuretic has little effect on CSF production; it may be used for patients who cannot tolerate acetazolamide.

The role of corticosteroids in the treatment of papilledema is controversial. A short course of high-dose corticosteroid therapy may be helpful for patients with acute visual loss resulting from fulminant papilledema [4]. However, corticosteroids should not be used chronically for treatment of papilledema.

Weight loss can be beneficial for patients with papilledema resulting from IIH. It is difficult to prove that weight loss improves papilledema in IIH because

1) acetazolamide administration is often initiated concurrently[5]
2) few patients lose enough weight to test the theory, and
3) the disease can remit spontaneously.

However, a history of recent weight gain often accompanies the initial presentation of IIH.

Some neurologists advocate serial lumbar punctures (e.g., twice weekly) as an alternative to
surgery for patients with papilledema that cannot be controlled medically. However, serial lumbar punctures are a poor approach because

1) most obese patients with chronic papilledema are difficult to treat with lumbar punctures and

2) patients generally dislike serial lumbar punctures.

At best, lumbar puncture and drainage of a large volume of CSF are useful emergency measures for patients with severe papilledema and sudden deterioration of vision. In some cases, it is necessary to hospitalize patients with lumbar subarachnoid drains until surgery can be scheduled. This is a good strategy if lumboperitoneal (LP) shunting is planned, although it may increase the risk of infection. Optic nerve sheath fenestration is easier to perform when the sheath is turgid; therefore, the drain should be removed or clamped before surgery.

Surgical Treatment

Surgical intervention is warranted when medical treatment fails. A common management error is to delay too long before recommending surgery. Corbett [6] emphasized that "there is no ‘acceptable’ level of visual field or acuity loss which one should wait for—visual loss which continues despite optimum medical therapy is sufficient reason to turn to decompression". Surgery should also be considered for patients who are unlikely to return for follow-up visits or who are unable to cooperate with medical therapy. Surgery should be considered not only for treatment of visual loss but also for treatment of intractable headaches.

Surgical manoeuvres include some form of shunt procedure such as lumboperitoneal shunt, ventriculoperitoneal shunt or optic nerve sheath decompression. Ventriculoperitoneal shunts are quite effective in reducing intracranial pressure but this is technically challenging because of the normal or small-sized ventricles in these patients.

LP Shunting

Ventriculoperitoneal (VP) shunts initially advocated for IIH were difficult to insert among patients with IIH, because the ventricles were often small. The development of LP shunting circumvented this problem. Vander Ark et al. [7] published the first description of LP shunting for patients with IIH. Soon thereafter, Spetzler et al. [8] developed a method for percutaneous insertion of the shunt into the lumbar sac, greatly facilitating placement of LP shunts. A lumboperitoneal shunt is considered the preferred neurosurgical procedure for intracranial hypertension. But this procedure has a high failure rate and other complications like sudden severe visual loss and simultagnosia. The mortality from lumboperitoneal shunting, primarily from infection has been reported to be as high as 7% to 16% [9]. In spite of all these limitations, lumboperitoneal shunt remains an acceptable procedure for those patients with severe headache and for those with conjoined visual loss.

After the introduction of LP shunting, few studies documenting its efficacy for the treatment of papilledema were published. Anecdotal reports of success suggested that it was a curative procedure, as long as the shunt functioned properly. In 1981, Johnston et al. [10] published a major review of 134 cases of IIH treated between 1942 and 1979, with a mean follow-up period of 11.6 years. Fourteen patients received shunts (six VP shunts and eight LP shunts). Of the six patients who received VP shunts, four demonstrated resolution of all symptoms within 6 months. One patient developed a shunt obstruction that necessitated revision, and another patient experienced a shunt infection that necessitated removal. Of the eight patients who received LP shunts, all demonstrated resolution of all symptoms within 6 months. One patient developed a shunt obstruction that necessitated revision, and another patient experienced a shunt infection that necessitated removal. Of the eight patients who received LP shunts, all demonstrated improvement within 1 month. One patient experienced a shunt infection, and one patient exhibited severe low-pressure symptoms as a result of overdrainage. In a follow-up study, Johnston et al. [11] reported
on 36 patients who received shunts for treatment of IIH. The patients required a total of 86 shunting procedures, with a complication rate of 52% and a failure rate of 48%; the lowest revision and complication rates were associated with LP shunts.

A multicenter review of the outcomes of shunting for 37 patients was performed in the late 1980s [12]. In that study, 37 patients received a total of 73 LP shunts and 9 VP shunts, and only 14 patients remained “cured” after a single surgical procedure. Sixty-four percent of shunts lasted less than 6 months, with shunt failure (55%) and low-pressure headaches (21%) being the most common reasons for reoperation. The vision of most patients either improved (13 patients) or stabilized (13 patients) postoperatively. That report initially led to a resurgence of interest in optic nerve sheath fenestration among ophthalmologists. However, the finding that many optic nerve sheath fenestrations fail within 1 year, as well as mounting evidence of serious complications, has restored LP shunting as the favored surgical treatment option.

Two major studies demonstrated the efficacy of LP shunting for treatment of IIH. Eggenberger et al. [13] conducted a retrospective study of 27 patients with IIH, who were monitored for a median of 47 months after shunting. Visual loss was the main reason for surgery for 14 patients; headaches were the reason for the remaining 13 patients. Vision improved or remained the same for all 14 patients, and headaches improved for all patients. There were no serious complications, except for shunt failure. Fifteen patients (56%) required shunt revision, sometimes more than once (range, 1–13 revisions). The average number of revisions per patient was 2.4, with one revision being performed every 2.6 years. The authors concluded that LP shunting was a satisfactory treatment for the majority of patients.

Burgett et al. [14] reported data for 30 patients who underwent LP shunting for treatment of IIH. The mean follow-up period was 35 months. Of 14 eyes with impaired acuity, 10 eyes (71%) improved by at least two chart lines; only 1 eye experienced a decline in vision. Goldmann perimetry documented improvement for 64% of eyes with abnormal fields, and no eyes exhibited any worsening. Again, the only complication was frequent shunt obstruction. Twelve patients required no shunt revision. The remainder underwent a mean of 2.5 revisions/patient. These two studies provided encouraging data regarding the efficacy of LP shunting; the operation seems effective, as long as the shunt remains patent. As in most large reviews [15, 11, 12], obstruction was the most common complication of LP shunts (accounting for 65% of revisions in the study by Eggenberger et al. [13]. In all patients with suspected shunt obstruction, lumbar subarachnoid pressure should be measured. Neuroimaging findings may not be revealing, because the ventricles are not enlarged in IIH [16]. Technetium-99 shunt function studies can provide valuable data by demonstrating tracer flow into the abdomen and providing a halftime for radionuclide clearance [17, 18]. Other complications associated with LP shunting in those studies were less common. Secondary intracranial hypotension caused by CSF overdrainage accounted for 15% of revisions in the study by Eggenberger et al. [13], and lumbar radiculopathy accounted for 4.5% of all revisions. Shunt infections occur in only approximately 1% of cases of LP shunting [19]. Tonsillar herniation (acquired Chiari malformation) [20, 21] and syringomyelia [22] are other recognized complications of LP shunting, but they only rarely necessitate revision [23]. Tonsillar herniation may create a “new” headache syndrome. A problem common to all obese patients is technical difficulty with excessive subcutaneous abdominal fat, which necessitates large incisions. In this respect, the use of laparoscopic techniques for insertion of the peritoneal catheter is potentially advantageous.

LP shunt valve pressure mechanisms with external pressure control are now being developed. The current preference is for LP shunting with an inline
horizontal-vertical valve. For patients with repeated LP shunt obstructions, the option of VP shunting should be considered. Although VP shunting is more invasive, the long-term outcomes may be better [24]. Technical innovations in stereotactic surgery enable accurate targeting of the lateral ventricle. A recent study of seven patients treated with stereotactic VP for IIH demonstrated successful uncomplicated shunt placement in all cases [25]. Five of the seven patients experienced resolution of papilledema and six of the seven experienced resolution of headaches postoperatively. Another study demonstrated the application of frameless stereotaxy and intraoperative fiberoptic endoscopy for precise ventricular catheter insertion [26]. Those studies support the idea of routine stereotactic VP shunting in IIH, with either frame-based or frameless stereotaxy. VP shunting may facilitate noninvasive assessment of shunt function, because it provides a reservoir for isotope shunt function testing; noninvasive analysis of LP shunt function has been limited to radiological findings [27].

**Complications of lumboperitoneal shunting**

- Obstruction
- Infection
- Low-pressure headaches
- Radiculopathy
- Tonsillar herniation (acquired Chiari I malformation)
- Syringomyelia
- Subdural hematoma
- Shunt migration
- Shunt dependency

**Optic Nerve Sheath Fenestration**

Optic nerve sheath fenestration, introduced by de Wecker [28], was the first treatment devised for the surgical relief of papilledema. The operation involved insertion of a guarded neurotome into the orbit to slit the optic nerve sheath via a conjunctival incision. However, subtemporal decompression, which was introduced by Dandy [29] in 1937, became the operation of choice for papilledema. Dandy performed a right subtemporal craniectomy for decompression and reported excellent initial results in alleviating headaches and preventing visual loss. However, the longer-term efficacy was uncertain and morbidity and complications were significant, including seizures, infections, focal brain damage, cosmetic disfigurement, intracranial hematomas, and further visual deterioration [30]. Subtemporal decompression rapidly became obsolete after the introduction of intracranial shunting procedures by Ingraham et al. [31] and Matson[32].

The failure rate associated with LP shunting renewed enthusiasm for optic nerve sheath fenestration among ophthalmologists in the 1980s. The procedure had continued to be performed by a few surgeons [33, 34, 35] but only sporadically. In 1988, three major reports appeared in the ophthalmologic literature, describing the outcomes of optic nerve sheath fenestration for treatment of IIH in large series of patients [36, 37, 38]. The results were surprisingly good; the operation seemed to provide effective treatment of papilledema and maintained or improved visual acuity for 85 to 100% of patients. However, the follow-up periods were short in those studies.

In a study of 53 patients (101 eyes), Spoor et al. [39] reported that optic nerve sheath fenestration improved visual function for 69 eyes with acute papilledema and 10 eyes with chronic papilledema. In a later report with longer follow-up periods, Spoor and McHenry [40] described the outcomes of optic nerve sheath fenestration for 75 eyes of 54 patients with IIH. After initial improvement in visual function, 24 eyes (32%) required repeat optic nerve sheath fenestration because of deteriorating visual function. Deteriorating vision was detected a mean of only 10.4 months after surgery, and 25% of eyes continued to lose vision even after repeat surgery.
In 1989, Sergott et al. [41] reported improved visual function for 12 of 14 patients with progressive nonarteritic AION who were treated with optic nerve sheath fenestration. In 1995, the National Eye Institute-sponsored Ischemic Optic Neuropathy Decompression Trial Research Group reported the results of a multicenter study of optic nerve sheath fenestration for treatment of AION [42]. The study found no benefit of optic nerve sheath fenestration for treatment of AION, contradicting the study by Sergott et al. [41], and documented significant complications of the procedure, including optic nerve injury during surgery. The incidence of catastrophic visual complications was 3 cases/115 patients, or 2.6%. Another study reported postoperative blindness for 3 of 200 patients (1.5%) [43]. A 2% risk of outright blindness has discouraged patients and ophthalmologists. Plotnik and Kosmorsky [44] emphasized that the complication rate may be as high as 40%, including vascular compromise (11%, central retinal artery occlusion, branch retinal artery occlusion, or outer retinal ischemia), transient ocular motility disturbances (29%), and papillary dysfunction (11%). Although enthusiasm for optic nerve sheath fenestration has moderated, the procedure remains a viable option for the prevention of visual loss resulting from papilledema. Optic nerve sheath decompression has emerged as the preferred surgical treatment for progressive visual loss in patients with intracranial hypertension when medical therapy fails. Visual field improvement occurred in 55% [37] to 100% [36] of eyes. The appearance of optic disc, particularly in relation to optic pallor, and the magnitude of visual loss should not dissuade the physician from surgical therapy. Marked improvement in visual function occurs after surgery in patients with preoperative disc pallor [38]. Although most patients with intracranial hypertension either improved or stabilised their visual function after optic nerve sheath decompression, some patients seemed to regress after an initially successful operation.

In a few cases, optic nerve sheath decompression needs to be repeated. Technically, this is a difficult procedure due to scarring changes around the optic nerve, mainly involving fat adhesion [47]. Other complications of optic nerve sheath decompression include macular changes such as chorioretinal striae, pigmentary disturbances, exudates and subretinal hemorrhage or scar. These changes are due to transudated fluid that emenates from swollen optic disc and may cause permanent central visual loss. Visual loss can occur despite a technically well-performed optic nerve sheath decompression and can result from hemorrhage or infection. Progressive visual loss despite optic nerve sheath decompression can sometimes be reversed by a lumbo-peritoneal shunt [45]. The mechanism of optic nerve sheath decompression has not been entirely elucidated but it is likely that a bleb, similar to a trabeculectomy bleb is created during the healing phase and allows a low-grade leak of CSF into the retro-orbital tissues, thereby reducing the amount of fluid mechanical force that is exerted on the neuro-ocular junction.

Success of therapy is judged by the relief of headache, elimination of transient visual obscurations and diplopia, and the reduction and elimination of papilloedema. Stabilisation or improvement of visual field indicates adequate therapy. The discovery of a relative afferent papillary defect is an important finding and may either indicate worsening of a particular eye or improvement in the opposite eye. It should be remembered that as many as 40% of the optic nerve axons may be lost before there is any detectable field defect, implying that once a field defect is found, therapy must be aggressive and effective [46, 47].

Complications of Optic nerve sheath fenestration
- Central/ Branch retinal artery occlusion
- Central retinal vein occlusion
- Choroidal infarction
- Traumatic optic neuropathy
- Hemorrhage (intrasheath or intraorbital)
- Diplopia
- Pupil dilation resulting from sphincter denervation
- Anterior segment ischemia
- Compressive optic neuropathy resulting from orbital cyst
- Infection

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Multifocal IOL—An Overview
Dr. Minu M Mathen

Advances in intraocular lens (IOL) design have significantly improved the visual outcomes of cataract surgery. Multifocal IOLs are designed to reduce dependence on spectacles after cataract surgery, and IOLs are gaining acceptance as potential refractive surgical options in selected patients.

Monofocal IOLs provide excellent distance visual function. But because of their limited depth-of-focus, they do not provide clear vision at near without spectacle correction. Monovision techniques may be helpful for some patients but can sacrifice binocularity.

The introduction of the multifocal IOL in the early-mid 1980s provided the potential for a range of uncorrected vision from near to far. Providing distance and near vision increases the depth of field and improves visual quality at near- that improves with time. Multifocality is the brain’s natural ability to adapt to near and far vision as it chooses, based on the object being viewed, between the 2 images (near and far) produced by the optical elements of the IOL. These multifocal IOLs provide distance, intermediate, and near correction.
Principle

When a person is viewing a distant object, a sharp retinal image is provided by the parts of the IOL within the pupillary area that have the distance correction; a somewhat blurred image is provided by the other parts of the IOL as the images are superimposed on the retina. The decrease in contrast of the in-focus image is produced by the split of the total light energy between the far and near focus, while the simultaneous presence (superimposition) on the retina of the in-focus image and out-of-focus image can produce a sort of retinal confusion; however, this is overcome by the brain’s capability to use multifocality.

Multifocality theoretically implies that more straylight reaches the retina. However, psychometric measures show that perceived straylight is not different in eyes with monofocal IOLs, thus the importance of brain adaptation.

The optical quality of the eye depends on defocus, light scattering, eye aberrations, and diffraction. After lens extraction and IOL implantation, this quality will also be affected by aberrations and scattering induced by the IOL. Reduced image contrast and unwanted visual phenomena, including glare and halos, have been associated with multifocal IOLs. One possible solution to improve the performance of multifocal IOLs is to direct different amounts of the refracted–diffracted light to the different foci, thus giving preference to distance or near vision. Another was to direct different amounts of light to the different foci depending on pupil diameter.

Refraction and diffraction principles have traditionally been used to create multifocality from near to distance. In refractive optics, the different zones of equal refractive power have a mutual focus. Phases of incoming light are incoherent, creating some destructive interference. This interference affects the intensity of the focus light and thus leads to a reduction in brightness and visual acuity. The retinal image with multifocal refractive IOLs depends on pupil diameter because of the IOLs’ power profile. Diffractive IOLs are less pupil dependent and have advantages over refractive IOLs in near vision.

The profile of the most recent and commonly used multifocal IOLs.

The AcrySof IQ ReSTOR (SN6AD1&SA6AD3) (fig 1) multifocal IOL combines the functions of the apodized diffractive region and the refractive region. The apodized diffractive optics are within the central 3.6 mm optical zone of the IOL. This area comprises 12 concentric steps of gradually decreasing (1.3 to 0.2 μm) heights, creating bifocality from near to far (2 foci). The refractive region of the optic surrounds the apodized diffractive region. This area directs light to a distant focal point for a larger pupil diameter and is dedicated to distance vision. The overall diameter of the IOL is 13.0 mm, and the optic diameter is 6.0 mm. The IOL power varies from +10.0 to +30.0 D and incorporates a +4.0 D near addition (add) and recently a +3.0 D near add which provides better intermediate vision. The asphericity...
was incorporated into the IOLs to prevent optical contrast reduction and to compensate for the physiologic spherical aberration inherent in the average cornea. IOLs are single piece with an anterior aspheric optic surface and are composed of an acrylate–methacrylate copolymer containing a blue light–filtering chromophore.

The ReZoom multizone IOL (fig 2) has 5 concentric refractive zones that refract light toward the main foci. Zones 1, 3, and 5 are distance dominant, and zones 2 and 4 are near dominant. The transitions between the zones are aspherical to provide balanced intermediate vision. The difference in intensity on the focal points makes the ReZoom a distance-dominant IOL with better intermediate visual function. The overall diameter of the IOL is 13.0 mm, and the optic diameter is 6.0 mm. The IOL power varies from +6.0 to +30.0 D and incorporates a +3.5 D near add.

The Acri.LISA 366D (fig 3) is a bifocal biconvex refractive–diffractive single-piece IOL with a 6.0 mm foldable acrylate aspherical optic, an overall diameter of 11.0 mm, and 0-degree haptic angulation. The surface is divided into main zones and phase zones; the phase zones assume the function of the steps of diffractive IOLs. The phase zones have a mean refractive power corresponding to the zero diffractive power of the main zones. The IOL power responsible for distance vision is refractive and diffractive at the same time. The first diffractive power used for near vision is formed by in-phase interference of waves from the main zones. The 2 focal points are created by phase zones on the anterior surface of the IOL. The incident light is distributed with 65% to distance focus and 35% to near focus. The diffractive structure has a soft transition of the phase zones between the main zones. The adjusted phase zones were designed to reduce disturbing light phenomena (eg, scattered light, halos) to improve retinal imaging quality and visual performance. The IOL has an aspherical profile to correct positive spherical aberration of the cornea. The optic is made of acrylate (refractive index 1.46) with 25% water content and ultraviolet wavelength–absorbing properties (Acri.Lyc material). The hydrophobic surface of the Acri.LISA 366D IOL, has sharp edges to reduce posterior capsule opacification. The IOL power varies from
Surgical considerations

Pre operative

Premium IOLs maximize patients’ outcomes and satisfaction by combining cataract and refractive surgery. Patient selection is probably the single most important determinant of success for the surgeon as well as for patients’ satisfaction. A thorough preoperative counselling is very important before choosing the right patient for a multifocal IOL. Let the patients view a video (which most IOL manufacturers have now) regarding the principles of a multifocal IOL. A highly demanding patient is not a right candidate for a multifocal IOL.

We need to explain the advantages (minimal spectacle dependance & spectacle independence improves with time) and disadvantages (possible reduction in contrast sensitivity, mild glare and halos around light sources, need for bilateral implantation for maximum effect of neuroadaptation, need for a refractive laser procedure for a possible residual refractive error and a possibility of need for near vision glasses for very fine print or for intermediate distance). Knowing all these, if the patient has the drive to go for these IOLs, they would be more willing to overlook these minor problems. We need to make sure that the patient does not go in for the surgery with the idea of getting perfect results as there is always a possibility of varied human response to surgery and these IOLs are not the perfect replacements for the vision one would have enjoyed in the second or third decade of life.

Patients who have corneal irregularities, limited BCVA, and macular pathology which would compromise the final visual outcome should be excluded. Patients who have a corneal astigmatism of more that 0.75 D should be told about the persistence of this residual refractive error postoperatively. We could always reduce it by performing limbal relaxing incisions according to the astigmatism if it is less than 1.5 D but their predictability is not always same. The option of a laser correction for this also can be put forward like bioptics, where a LASIK flap is cut 1 to 2 weeks before proceeding with the cataract surgery and when the refraction is stable postoperatively, 3 to 6 weeks after surgery, the flap can be lifted to perform LASIK in order to treat the preexisting refractive astigmatism or any residual sphere.
So always set realistic expectations postoperatively. The ideal candidate for a multifocal IOL is one who understands the technology’s abilities and limitations, with minimal astigmatism and an otherwise healthy ocular surface, retina, macula, and optic nerve.

**Intra operative**

A successful outcome with any multifocal IOL requires great attention to detail on the part of the surgeon. An emmetropic result and centration of the lens must be obtained to maximize quality of vision, facilitate neuroadaptation, and minimize dysphotopsia. If these endpoints are not obtained, aberration will result, which will lower patients’ satisfaction.

The IOL needs to be centered over the visual axis versus over the geographical center or slightly nasal to it. To identify the visual axis ask the patient to fixate on the microscope’s filament and mark the anterior Purkinje image with methylene blue. Then center a circular capsulorhexis around it so that there is a 360 degree CCC overlap on the IOL optic. Single piece models should always be implanted inside the capsular bag (restore and acrylisarn). We should always keep a back up of multi piece IOLs for sulcus placement in case of a posterior capsule rupture.

**Post Operative**

Causes of suboptimal results after multifocal IOL implantation are monocular implantation, minimal residual refractive error, posterior capsular opacities, ocular surface disease, cystoid macular edema (CME), and a decentered IOL.

The patient needs to be encouraged to start reading and using their eyes for near work as early as possible to get used to the multifocality. We should avoid giving the patient a near vision addition during the initial postoperative weeks. Also encourage the patient to undergo a multifocal IOL implantation in the other eye also within a few weeks to enhance the neuroadaptation which improves the multifocal visual function.

YAG laser capsulotomy might be required earlier and more frequently in these patients whose quality of vision can be affected by early capsular fibrosis. But if an IOL exchange is deemed necessary (because of unacceptable residual power or dissatisfied patient), it is advantageous to perform it prior to YAG laser capsulotomy.

Emmetropia must be obtained to maximize outcomes and meet patients’ expectations (as many of them are highly sensitive to minor residual powers) for which we could perform either LASIK or surface ablation. It is usually required for eyes with residual sphere of 0.50 D or greater and cylinder of 0.75 D or greater which is not accepted by the patient.

Dry eye disease is common in older patients. Even a mild breakdown of the corneal epithelium reduces the tear film’s ability to smooth out the ocular surface. A more regular tear film and ocular surface improve quality of vision.

The loss of contrast sensitivity associated with a multifocal IOL is worsened by CME. Once the normal architecture of the retina is lost, visual quality is degraded for life. Snellen visual acuity will improve, but contrast sensitivity will be permanently reduced. The best way to look for CME after cataract surgery is with OCT. In addition, OCT is a very effective preoperative screening tool for foveal membranes, which will reduce quality of vision after cataract surgery. The pre and post operative use of topical NSAIDs is very beneficial in multifocal IOL cases.

**Conclusion**

The goal of any refractive surgery is primarily to meet or exceed the patients’ expectations. Making sure that they have a realistic expectation of the
limits of refractive IOL surgery is important, as no surgery is perfect. With very careful patient selection, adequate preoperative patient counseling and attention to detail during surgery and proper post operative follow up and treatment can make the vast majority of our multifocal IOL patients happy.

References


Relationship Between Structure and Function of the Optic Nerve Head—Glaucoma versus Normal

Dr. Savita Bhat, Dr. Anna Elias, Dr. Siddharth Pawar, Dr. S.J. Saikumar, Dr. Alpesh Rajput

Abstract

Purpose: To evaluate the relationship between retinal nerve fibre layer (RNFL) thickness measured by optical coherence tomography (OCT) and light threshold values obtained with the Humphrey Field Analyser (HFA).

Methods: Thirty-one normal subjects and 72 glaucoma patients were included. Around the optic disc, RNFL thickness was measured with Stratus OCT scans and sensitivity evaluated with the Swedish Interactive Threshold Algorithm (SITA) Standard strategy at the same visit. The RNFL thicknesses at the inferior, superior, nasal and temporal regions were compared to retinal sensitivity values in the same areas.

Results: Correlation between RNFL thickness and retinal sensitivity in the regions of the optic nerve head using the Karl-Pearson’s correlation coefficient for the two groups showed significant correlation between structure and function loss at the inferior, superior and nasal regions in the glaucoma group.

Conclusions: There is significant structural and functional correlation for values around the optic nerve head in the glaucoma group.

Key words: Visual field, Optic nerve head, retinal nerve fiber layer, Optical coherence tomography
Introduction

The diagnosis of glaucoma despite the plethora of newer machines and imaging techniques rests on recognizable glaucomatous field changes on the gold standard automated perimetry. The hallmarks of glaucomatous progression are visual field deterioration and morphologic changes of the optic disc, including narrowing of the neuroretinal rim accompanied with deepening or widening of the optic cup, or both (1).

Structural changes occur before functional alterations in many types of glaucomas (2,3). To assess the structural changes several different modalities are available. Optic nerve head photography, retinal nerve fibre layer photography, Heidelberg retinal tomogragh (HRT), Nerve fibre analyzer GDx, Retinal thickness analyzer and the Optical coherence tomography (OCT). Several studies with follow up show progression of retinal nerve fibre layer changes before visual field changes in glaucoma and ocular hypertensive patients (4,5). Similar studies have demonstrated significant correlation between structural parameters obtained with HRT and visual field indices either globally or regionally (6,7,8,9). With the GDx, equipped with variable corneal compensation (VCC), scanning laser ophthalmoscopy correlates better with mild glaucomatous changes correlate better with perimetry than those with fixed corneal compensation (FCC) (10,11).

Soliman et al studied the relationship between RNFL loss on OCT and visual field damage. This is non-linear, exponential and shows that a considerable amount is lost before development of detectable field damage. In early stages of glaucoma, field changes occurs before RNFL loss detected on OCT (12, 13).

White on White (W/W) perimetry is a generally accepted method for monitoring visual field damage in glaucoma patients and suspects. Glaucoma patients suffer a loss of about 40% of their retinal ganglion cells before this loss is picked up on W/W perimetry.

To detect the structural changes on the RNFL, we used OCT in this study and compared it to visual functional sensitivity on HFA using the Swedish Interactive Thresholding Algorithm (SITA) Standard strategy.

Aim of the Study

To evaluate the relationship between retinal nerve fibre layer (RNFL) thickness measured by optical coherence tomography (OCT) and light threshold values obtained with the Humphrey Field Analyser (HFA) using the Swedish Interactive Threshold Algorithm (SITA ) Standard strategy.

Materials and Methods

Subjects

Seventy – two glaucoma patients and 31 normal control subjects participated in a longitudinal, prospective study on visual field and optic disc change. Patients were recruited consecutively from the glaucoma clinic with the following inclusion criteria: (1) clinical diagnosis of open-angle glaucoma with notching or progressive thinning of the neuroretinal rim, (2) baseline visual field mean deviation between −2 and −10 dB, (3) open angles by gonioscopy, and (4) best-corrected visual acuity of 6/18 or better.

Normal control subjects were recruited among volunteers with the following inclusion criteria: (1) clinically normal appearance of the optic disc and fundus, (2) intraocular pressure of less than 22 mmHg, and (3) best-corrected visual acuity of 6/12 or better. Common exclusion criteria were: (1) systemic disease like diabetes, neurological diseases or systemic medication known to affect the visual field, (2) refractive error exceeding 5 diopters (D; equivalent sphere) of myopia or hyperopia or 3 D of...
astigmatism, and (3) contact lens wear. Additionally, patients were excluded if there was concomitant ocular disease, and controls were excluded if there was any ocular disease. One eye was chosen randomly as the study eye for the controls and also for the patients if both eyes were eligible.

**Methods**

A detailed medical and surgical history was elicited from the patients, all of whom underwent a complete ophthalmic examination that included slit-lamp biomicroscopy, visual acuity testing with refraction, ONH examination with slit lamp biomicroscopy, applanation tonometry, gonioscopy, HFA perimetry using the Swedish Interactive Thresholding Algorithm (SITA) standard strategy and OCT evaluation of RNFL.

**Instrumentation**

**Optical Coherence Tomography**

Optical coherence tomography uses low-coherence interferometry to image intraocular structures cross-sectionally. It is analogous to ultrasound B-mode imaging, except that it uses light rather than sound and provides in vivo tissue sampling with axial resolution of the current commercially available unit in the range of ~10 μm. Cross-sectional images of tissue microstructure are obtained by measuring the echo time delay and magnitude of light backscattered from internal tissue microstructure. Optical coherence tomography has been shown to obtain accurate and reproducible NFL and retinal thickness measurements. Detailed descriptions of OCT have been previously published (14,15). Optical coherence tomography enables cross-sectional imaging of the macula, peripapillary and ONH regions. The peripapillary scan is a circular scan with a diameter of 3.4 mm centered on the ONH.

Optical coherence tomography was performed by using Stratus OCT, model 3000 (Carl Zeiss Meditec Inc, Dublin, CA, USA). The results were analyzed with Version 4.0.1 software. After dilatation to a minimum of 5 mm, a patch was placed over the other eye. Three hundred and sixty degrees circular scans with a diameter of 3.4 mm, centered on the optic disc were performed using the Fast RNFL thickness protocol.

The RNFL thickness was defined as the number of pixels between the anterior and posterior edges of the RNFL. Each scan consisted of 100 individual A-scan samples evenly distributed along a circle circumference. Three circular scans, each 3.4 mm in diameter centered on the optic disc, were obtained from each test eye. The best quality, properly aligned scan was chosen for analysis. Average RNFL thickness was calculated globally and separately for superior, inferior, temporal and nasal quadrants. Good quality OCT scans were defined as scans with a signal-noise ratio of 40 dB.

**SITA Standard HFA 30-2**

W/W perimetry was performed with HFA (Zeiss Humphrey Systems, Model 750) by using SITA-standard test strategy 30-2 program. A reliable test was defined as having fewer than 33% false-positive or false-negative scores and fewer than 20% fixation losses. Test was repeated to establish baseline in most subjects. The dB threshold values around the blind spot were taken at the inferior, superior, nasal and temporal for sectoral analysis.

**Statistical analysis**

The parameters compared were average RNFL thickness of the entire circumference of the optic disc and quadrant thickness consisting of superior (46 to 135 degrees), nasal (136 to 225 degrees), inferior (226 to 315 degrees) and temporal (316 to 345 degrees) quadrant areas between the three groups.

To compare similar areas on the fields, decibel light threshold sensitivity at the superior, inferior,
nasal and temporal quadrants were obtained thus. Three points are obtained for superior and inferior areas of the disc and four points for nasal and temporal sides. For instance, the peripheral two points for superior are halved. Therefore, sum of one central point and two halved peripheral points divided by two gives threshold value at superior quadrant and similarly for the inferior area. For the 4 nasal points, sum of two central and two peripheral points divided by three gives threshold nasal sensitivity.

Paired comparisons for all significant mean defects were conducted. To evaluate the strength of the association of W/W perimetry, correlations between RNFL thickness and visual field parameters were assessed by correlation coefficients (Karl-Pearson’s r) and significance calculated using the Student t-test. Data were reported as mean ± standard deviation (SD). A P value of less than 0.05 was considered statistically significant.

**Results**

Seventy two patients and 31 normals were enrolled. There was no difference between the groups with regard to gender as depicted in Table 1.

**Table 1** represents the demographic features of the two groups.

<table>
<thead>
<tr>
<th></th>
<th>Glaucoma</th>
<th>Normal</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>60.17 ± 12.27</td>
<td>47.96 ± 12.35</td>
<td>0.003</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>1.05</td>
<td>0.93</td>
<td>0.81</td>
</tr>
<tr>
<td>CD ratio</td>
<td>0.61 ± 0.18</td>
<td>0.49 ± 0.15</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Mean age of normals was 47.96 ± 12.35 years. The mean age of patients in the glaucoma group was 60.17 ± 12.27 years. The mean vertical CD ratio was 0.49 ± 0.15 and 0.61 ± 0.18 in normal and glaucomatous eyes respectively (P <0.05)

The RNFL thickness was greatest in the inferior and superior quadrants and thinner in the nasal and temporal quadrants in the normal group. The RNFL profile demonstrated the “double hump” pattern. These results are consistent with those of earlier studies. (16)

The RNFL thickness in glaucomatous eyes differed significantly from normal eyes in all parameters (P <0.001). Representative OCT recordings in patients with glaucoma and normal subject are depicted Figure 1 and Figure 2 respectively.

Mean RNFL thickness was thinner in glaucomatous eyes (78.35 ± 19.46 mm) than the normals (94.75 ± 12.6 mm) and this was statistically significant at p<0.001. The RNFL was thinner in glaucomatous eyes in the inferior (98.5±31.25 mm), superior (93.34±27.58 mm), and temporal (56.51±13.92 mm) quadrants when compared to normals (P <0.01) except in the nasal aspect as tabulated in Table 2.

**Table 2** shows RNFL thickness average and in each sector for the two study groups.

<table>
<thead>
<tr>
<th>RNFL Thickness</th>
<th>Glaucoma(mm)</th>
<th>Normal(mm)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>78.35 ± 19.46</td>
<td>94.75 ±12.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Inferior</td>
<td>98.5±31.25</td>
<td>128.58 ± 15.06</td>
<td>0.001</td>
</tr>
<tr>
<td>Superior</td>
<td>93.34±27.58</td>
<td>115.71±22.62</td>
<td>0.007</td>
</tr>
<tr>
<td>Nasal</td>
<td>66.44±21.65</td>
<td>70.55 ± 16.71</td>
<td>0.942</td>
</tr>
<tr>
<td>Temporal</td>
<td>56.51±13.92</td>
<td>66.32 ±12.08</td>
<td>0.002</td>
</tr>
</tbody>
</table>
With reference to global indices in glaucoma W/W perimetry, mean deviation (MD) was -7.94 ± 5.93 and pattern standard deviation (PSD) was 5.23±3.89; and for normal group mean deviation (MD) was -3.01 ± 1.31 and pattern standard deviation (PSD) was 1.84 ± 0.49. The mean RNFL thickness on OCT correlated to the global indices on the W/W perimetry- mean deviation (MD r= 0.68, p<0.01) and did not show correlation to the PSD (r= -0.61). For the normal group, RNFL did not show any correlation to the MD (r= -0.08) and PSD (r= -0.03). This is depicted in Table 3.

### Table 3 correlates RNFL thickness on OCT to the global indices on the perimetry for the two groups using Karl – Pearson’s correlation.

<table>
<thead>
<tr>
<th></th>
<th>RNFL (mm)</th>
<th>MD</th>
<th>Correlation</th>
<th>PSD</th>
<th>Correlation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glaucoma</td>
<td>78.35 ± 19.46</td>
<td>-7.94 ± 5.93</td>
<td>r= 0.68, p&lt;0.01</td>
<td>5.23±3.89</td>
<td>r= -0.61</td>
</tr>
<tr>
<td>Normal</td>
<td>94.75 ±12.6</td>
<td>-3.01 ± 1.31</td>
<td>r= -0.08</td>
<td>1.84 ± 0.49</td>
<td>r= -0.03</td>
</tr>
</tbody>
</table>

The correlation between RNFL thickness on OCT to the corresponding retinal sensitivity values obtained on the white on white SITA standard perimetry in the four common sectoral regions of the optic nerve head using the Karl-Pearson’s correlation coefficient for the two groups are as depicted in Table 4.

### Table 4 correlates RNFL thickness of each sector to the corresponding retinal threshold values for both the groups.

<table>
<thead>
<tr>
<th></th>
<th>Glaucoma</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>r = 434, p = 0.001</td>
<td>r = 114, p = 0.54</td>
</tr>
<tr>
<td>Superior</td>
<td>r = 466, p = 0.001</td>
<td>r = 126, p = 0.50</td>
</tr>
<tr>
<td>Nasal</td>
<td>r = 416, p = 0.001</td>
<td>r = -0.061, p = 0.745</td>
</tr>
<tr>
<td>Temporal</td>
<td>r = 255, p = 0.028</td>
<td>r = -0.4, p = 0.05</td>
</tr>
</tbody>
</table>

**Discussion**

Optical coherence tomography helps in obtaining objective and reproducible measures of RNFL thickness and detects focal defects independent of the visibility of RNFL. Sommer et al (17) in a 10-year follow-up study reported that RNFL thinning is a sensitive indicator of the extent of glaucomatous damage and that RNFL loss precedes measurable ONH and visual field damage approximately six years before any detectable visual field defects. Thus, the possibility of detecting these defects in areas of physiological decreased visibility is enhanced when OCT, rather than a conventional method, is used.

In our study, mean RNFL thickness was thinner in glaucomatous eyes (78.35 ± 19.46 mm) than the normals (94.75 ± 12.6 mm) and this was statistically significant at p<0.001. (Table 2). Hoh et al (18) reported that the mean RNFL thickness measured with OCT was significantly less in glaucomatous eyes (56.9±21.5 mm) than in ocular hypertensive (83.70±16.57 mm) and normal (90.86±14.17 mm) eyes; although RNFL thickness tended to be greater in normal than in ocular hypertensive eyes, this difference was not statistically significant.

The RNFL was thinner in glaucomatous eyes in the inferior (98.5±31.25 mm), superior (93.34±27.58 mm), and temporal (56.51±13.92 mm) quadrants.
when compared to normals (P <0.01) except in the nasal aspect as tabulated in Table 2. Guedes et al (19) reported that the inferior RNFL was the only parameter in which a statistically significant difference was observed between normal subjects and glaucoma suspect groups. Pieroth et al reported a specificity of 81% and sensitivity of 65% in detecting focal defects solely through statistical analysis of OCT measurements and also noted that focal RNFL defects are located in the inferotemporal and superotemporal regions of the RNFL.

Teesalu et al. demonstrated that among patients with glaucoma, 38% of apparently normal W/W perimetry hemifields were classified as abnormal using SWAP hemifield data while 52% were classified as abnormal using HRT data, thus suggesting that eyes with seemingly healthy W/W perimetry hemifields may, in fact, already be affected by glaucoma. Subjects with abnormal SWAP values had thinner RNFLs than those with normal SWAP values. Therefore, assessment of RNFL by OCT may be as sensitive as SWAP in early detection of glaucoma and before a specific W/W perimetry defect has occurred (4).

The mean RNFL on OCT correlated to the global indices on the W/W perimetry- mean deviation (MD r= 0.68, p<0.01) and did not show correlation to the PSD (r= -0.61) (Table3). Soliman and associates 12 reported a significant correlation (correlation coefficient r = 0.557) between average RNFL thickness and mean deviation on W/W perimetry. Parisi et al and Zangwill et al have also reported a significant correlation between average RNFL thickness and MD. Kanamori et al showed that the highest correlation coefficient in all parameters was 0.729 at the average RNFL thickness, suggesting that average RNFL thickness was most useful for monitoring glaucoma.

Table 4 represents sector –wise correlation of RNFL thickness and retinal sensitivity values for glaucoma subjects and normals. Statistically significant correlation occurs for the former group where as not so for the normal subjects. This is possibly due to smaller number of patients in the normal group. Localized RNFL defects can be clinically detected if more than 50% of the thickness of RNFL is lost (20).

Therefore, we conclude that OCT gives fairly good structural correlation to the visual sensitivity functional loss in the glaucomatous optic nerve head. This can form an adjunct to diagnosis and management of glaucoma patients especially, in certain individuals who are unable to perform conventional standard perimetry.

References


6. Weinreb RN, Shakiba S, Sample PA et al. Association between quantitative nerve fibre layer measurement and visual field loss in


Luprops Keratoconjunctivitis in the rubber plantation area of Pathanamthitta District

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Dr. Ashley Thomas Jacob MS, DNB, FICO, MRCOphthal
Dr. Liz Thomas MS, DNB, FICO, MRCOphthal
Dr. Anand S. Koottummel MS
Dr. Jyothi K DO

Abstract

Objective: To present our experience of a specific type of keratoconjunctivitis in the rubber plantation area surrounding Kozhencherry in Pathanamthitta district.

Methods: We describe 94 patients who presented to our OPD from 2004 January to 2009 June with the typical symptoms and signs. Symptom is waking up from sleep with pain in the eye and diminution of vision. Signs include diffuse conjunctival congestion, ropy discharge, variable amount of corneal epithelial defect, punctate epithelial erosions and striate keratopathy. Seasonal increase in the number of Luprops was documented.

Results: The occurrence of keratoconjunctivitis correlates very well with the seasonal increase of Luprops. The peak time of presentation was from March to July-August every year. The treatment protocol which was found effective is in the line of acid chemical injury since the beetle produces a phenolic secretion.

Conclusion: Luprops keratoconjunctivitis is a common, but improperly diagnosed and treated condition in the rubber plantation areas, which has not been reported before.

Keywords: Luprops, keratoconjunctivitis, rubber plantation.
**Aim of study**

To present our experience of a specific form of acute keratoconjunctivitis in the rubber plantation area surrounding Kozhencherry in Pathanamthitta district.

**Materials and methods**

This is a hospital based study on 94 patients who presented to us from January 2004 to June 2009 in Kozhencherry with a peculiar type of acute keratoconjunctivitis characterized by the typical symptom of the patient waking up from sleep with severe pain and foreign body sensation in the eye and diminution of vision, the affected eye showing variable amount of signs like lid and periorbital edema, diffuse conjunctival congestion, tearing, discharge, punctate epithelial erosions, corneal and conjunctival epithelial defects, fluorescein staining and striate keratopathy. Depending on the extent of corneal involvement, the cases were divided into mild, moderate and severe. Mild keratoconjunctivitis (Figure: 1) will have only punctate epithelial erosions of cornea. Moderate keratoconjunctivitis (Figure: 2&3) will present with corneal epithelial defect involving less than 50% of cornea while severe keratoconjunctivitis (Figure: 4&5) involves more than 50% of cornea.

All patients were seen within 24 hours of affliction and were followed up until complete healing was achieved. All patients were asked whether their house is near rubber plantation and also for the presence of a rubber plantation litter beetle locally known as ‘Mupli beetle’ (Figure: 6) in their house.

The extraction and analysis of the beetle’s secretion was done in vitro. Whatman’s filter paper impregnated with acidified aqueous solution of potassium iodide and concentrated starch solution was dried in sunlight for half an hour. The beetles were placed on the paper and pressed on dorsal side so that the secretion was spread on the paper, which stained it black (Figure:7) The conjunctival sac of the cases with keratoconjunctivitis was tested for this chemical. Swab from the lower fornix was taken and tested on the indicator paper.

Physical examination was done for any hyperpigmented skin lesions of new onset on the exposed body parts. On presentation, 20 patients who initially presented were treated with topical antibiotic and tear substitute. 74 patients received saline wash, topical antibiotic-steroid combination and preservative free tear substitute. The antibiotic-steroid combination was tapered and stopped depending on the clinical response.

The causative genus of beetle was identified by local entomologists.

**Results**

Of the 94 cases, four (4.25%) were bilateral, 36 (38.29%) in right eye and 54 (57.45%) in left eye. 69 (73.4%) cases were males and 25 (26.66%) females. The age range was 3–81 years. The follow-up period was 3 to 12 days.

The cases presented all round the year, but the peak time of presentation was from March till July-August every year. All patients resided near or within one kilometer of rubber plantation area. 54 cases had new onset, hyperpigmented skin lesions on exposed parts. All patients noticed the beetle in and around their residential area. The beetle invades residential areas in the months of March-April, remain dormant for many months and leave settlements during November-December for breeding in the rubber plantations. The occurrence of keratoconjunctivitis correlated very well with the seasonal increase in the number of beetles.

The secretion was found to be quinonoid
in nature and was also demonstrated in the conjunctival sac of the patients. So the treatment protocol which was found effective was in the line of acid chemical injury since the beetle produces an acidic secretion. The initial use of topical steroid hastened recovery. 20 patients who were treated as corneal epithelial defect with topical antibiotic and tear substitute recovered in 5 to 12 days. 74 patients who were given topical steroid in addition to topical antibiotic and tear substitute recovered in 2 to 7 days.

The recovery period also depended on the extent of corneal epithelium initially involved. Mild cases recovered faster than moderate and severe ones.

**Discussion**

* Luprops, (1) locally known as ‘Mupli beetle’ is a rubber plantation litter dwelling beetle. Seasonal mass invasion of huge aggregations of this beetle, numbering 0.5 to 4 million on residential buildings following summer showers is a regular event in the rubber tracts along the western slopes of South Western Ghats of Kerala. Attraction of these beetles towards light, following overnight invasion into buildings is a frustrating nuisance for the residents. Clusters of several hundred to thousands crawl inside living rooms and fall off into beds and food from ceilings. Subsequently they congregate in dark, undisturbed areas such as attics and wall voids and remain dormant for several months, mainly March-April and leave the settlements during the favorable season of November-December for breeding in the rubber plantations (2). Though the beetles do not sting or bite, when disturbed by picking them off the walls or when they are squashed or pressed while sleeping, they release an irritating, odoriferous phenolic secretion leading to hyperpigmented patches on skin.

* Keratoconjunctivitis is usually secondary to transfer by patient’s finger of the phenolic secretion of the beetle, when it is accidentally crushed while sleeping.

* The treatment protocol which was found effective was in the line of acid chemical injury since the beetle produces a phenolic secretion. The recovery period also depended on the extent of corneal epithelium initially involved.

* Similar kind of beetle induced keratoconjunctivitis has been reported as ‘Nairobi Eye’ caused by blister beetle (genus Pederus) in Southwest America and Tanzania (3, 4).

**Conclusion**

This is the first report of a keratoconjunctivitis caused by a litter dwelling beetle. The injury is caused by the phenolic secretion of the beetle. The recovery depended on the severity of injury and the treatment given. There was speedy recovery with topical steroids.

* Ophthalmologists who are working in the afflicted areas may be confused about the etiology and treatment of this condition. Many cases are being treated as viral with topical acyclovir which is again epitheliotoxic and do more harm to the already compromised epithelium. So it is important to be aware of this type of keratoconjunctivitis to help the patients get a speedy recovery and to inform the patient regarding the etiology since the patient himself will not be aware of it.

**Acknowledgement**

* To Dr. Joseph Mar Dionysius for the support in extraction and analysis of the beetle’s secretion and entomological diagnosis.

* To Dr. Noela Marie Prasad for providing guidelines for the study
References


2. Fr. Dr. T. M. Joseph. 2007. A preliminary study on defensive glands of rubber beetle, Luprops curticollis fairmaire (Coleoptera: Tenebrionidae) (Unpublished)


A clinical study of Complicated Cataract In Uveitis

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Corresponding author: Dr K.V. Raju, Director

Abstract

This clinical study of complicated cataract in uveitis was conducted at Regional Institute of Ophthalmology Kozhikode.


METHODS:

Cases of complicated cataract secondary to uveitis, attending the OPD during a period of 2 years were included in the study. Those cases of senile and traumatic cataracts were excluded.

RESULTS

The peak incidence of uveitic cataract was between the age group 31 – 40 years. Younger age groups had a rapid progression of cataract. Uveitic cataracts were most commonly associated with chronic anterior uveitis. Most of the patients who underwent cataract surgery for complicated cataract had a substantial improvement in vision.
Introduction

Uveitis is a common problem encountered in diverse forms. It is a chronic and usually protracted condition, requiring long-term treatment with corticosteroids or other immunosuppressive drugs. Cataract formation is a common finding in these patients. The management of cataract associated with uveitis requires special precautions and has its own attendant problems. The challenge lies not only in the technical difficulty of surgery but also in the ability to control the inflammation in the perioperative period.

Aim of the Study


To evaluate the final visual outcome in patients undergoing surgery for complicated cataract.

Materials and Methods

Cases of complicated cataract secondary to uveitis, attending the OPD during a period of 2 years were included in the study. Diagnosis of uveitis was made on the basis of systematic ocular examination. Routine laboratory investigations were done in all cases & specific investigations were done if indicated.

The final diagnosis was based on history, clinical findings and results of laboratory investigations. Morphological appearance of cataract was assessed by slit lamp.

Patients were followed up regularly to assess the control of uveitis and the progression of cataract. Those with visually significant cataract and quiet eyes for a period of at least 3 months underwent cataract surgery. Pre-operative visual acuity and visual acuity at 6 weeks, 6 months and the final follow-up was noted.

OBSERVATIONS AND DISCUSSION

1. Age distribution (Figure 1)

It was found that the peak incidence of uveitic cataract was in the age group 31-40 years and the progression of cataract was found to be more rapid in the younger age group.

2. Sex distribution

Females outnumbered males in this study by 2%. It was also found that anterior uveitis was more common in males and posterior & intermediate uveitis were more common in females.

3. Type of uveitis (Figure 2)

In the present study the type of uveal inflammation most commonly associated with complicated cataract was anterior uveitis (62%), followed by intermediate uveitis (20% ), pan uveitis (10% ) and posterior uveitis (8%).

4. Pattern of uveitis

Majority of cases with complicated cataract had a chronic form of anterior uveitis (64%). 24.6% had recurrent anterior uveitis. 10% patients showed evidence of cataract after an attack of acute severe anterior uveitis.

5. Duration of history

The duration of history of uveal inflammation in this study varied from 3 weeks to as long as 10 years in this study. Another feature observed was that in cases of panuveitis that caused complicated cataract, the duration of uveal inflammation was very short.

6. Type of Anterior uveitis

83% of cases with complicated cataract had non granulomatous type of anterior uveitis.
7. **Type of cataract: (Figure 3)**

<table>
<thead>
<tr>
<th>Systemic illness</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Juvenile rheumatoid arthritis</td>
<td>1</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>4</td>
</tr>
<tr>
<td>Behcets disease</td>
<td>1</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>1</td>
</tr>
</tbody>
</table>

In the present study, the most common morphological type of complicated cataract was posterior subcapsular type (74%). 18% were total cataracts, 6% were of anterior subcapsular type. One case (2%) had a suture cataract.

8. **Initial visual acuity: (Figure 4)**

36% of cases with complicated cataract had a best corrected visual acuity of 6/6 - 6/18. 25% had a BCVA of 6/24 - 6/60, 24% the vision was reduced to less than 6/60. 11% of cases had a vision of as low as Hand movements and 4% PL vision.

9. **Associated systemic illness**

Systemic disorders were associated with 18% of uveitic complicated cataract.

10. **Associated ocular complications**

<table>
<thead>
<tr>
<th>OCULAR COMPLICATIONS</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secondary glaucoma</td>
<td>5</td>
</tr>
<tr>
<td>Exudative RD</td>
<td>3</td>
</tr>
<tr>
<td>Epiretinal membrane</td>
<td>2</td>
</tr>
<tr>
<td>Band keratopathy</td>
<td>1</td>
</tr>
<tr>
<td>Secondary vasculitis</td>
<td>1</td>
</tr>
<tr>
<td>Neovascular glaucoma</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TYPE OF SURGERY</th>
<th>TYPE OF IOL</th>
<th>PREOP VISION</th>
<th>POSTOP VISION – 6WEEKS</th>
<th>POSTOP VISION – 6 MONTHS</th>
<th>ASSOCIATED COMPLICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>2MCF</td>
<td>1MCF</td>
<td>6/36</td>
<td>Immediate post op uveitis</td>
</tr>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>PL</td>
<td>PL</td>
<td>PL</td>
<td>Retinal detachment, Neovascular glaucoma</td>
</tr>
<tr>
<td>Phaco</td>
<td>Single piece PMMA lens</td>
<td>6/36</td>
<td>6/12</td>
<td>6/12</td>
<td>Epiretinal membrane, Posterior capsular opacification</td>
</tr>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>6/60</td>
<td>6/18</td>
<td>6/18</td>
<td>Posterior capsular opacification</td>
</tr>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>HM</td>
<td>HM</td>
<td>HM</td>
<td>Retinal detachment</td>
</tr>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>3MCF</td>
<td>6/12</td>
<td>6/6</td>
<td>---</td>
</tr>
<tr>
<td>SICS</td>
<td>Single piece PMMA lens</td>
<td>HM</td>
<td>6/12</td>
<td>--</td>
<td>Lost follow up</td>
</tr>
</tbody>
</table>
11. Surgical outcome: (Figure 5)

Cataract surgery with PCIOL implantation was done in 18% of cases. Single piece poly methyl methacrylate (PMMA) intraocular lens were implanted in all cases.

Among those who underwent cataract surgery, 71% had substantial improvement of visual acuity.

Two cases received preoperative anti-inflammatory medications including topical and systemic steroids starting 3 days prior to surgery. All eyes received topical corticosteroids in tapered doses over 6 weeks postoperatively. Only one case developed severe postoperative uveitis.

Conclusion

Fifty eyes with complicated cataract were studied. The peak incidence of uveitic cataract was between the age group 31 – 40 years. Younger age groups had a rapid progression of cataract. Uveitic cataracts were most commonly associated with chronic anterior uveitis.

(Duration ranged from 3 weeks to as long as 10 years). The most common morphological type of complicated cataract was posterior subcapsular plaque type. In all cases where uveal inflammation was well controlled, the lens opacity remained stationary. Most of the patients who underwent cataract surgery for complicated cataract had a substantial improvement in vision. The causes of poor visual outcome in the rest were presence of posterior segment pathology like cystoid macular edema, epiretinal membrane and retinal detachment.

ILLUSTRATIONS

Figure 1: Age distribution

Figure 2: Type of uveitis

Figure 3: Type of cataract
References


Comparison of Peribulbar Anaesthesia with Topical Anaesthesia in Manual Small Incision Cataract surgery

Dr. Smita Narayan, Dr. Rajini.K.C, Dr. Sujatha.N, Dr. Mallika.V.

Abstract

PURPOSE: To compare the safety and efficacy of Peribulbar Anaesthesia with Topical Anaesthesia in Manual Small Incision Cataract Surgery using a Randomized Controlled Clinical Trial.

METHOD: 150 patients were randomized to Peribulbar and Topical groups with preset criteria after informed consent. All surgeries were performed by two surgeons. Pain during administration of anaesthetic, during surgery and 4 hours after surgery was graded and compared for both techniques. The surgeon also scored for surgical ease/discomfort in terms of unwanted ocular movements, patient cooperation and anterior chamber stability. The patients were followed for 1 month.

RESULTS: 66 patients of peribulbar group and 64 patients of topical group had no pain during surgery. 70 patients in peribulbar group had grade 0 pain while only 24 had grade 0 pain in Topical group. The surgeons experience was the same in both the groups. The average time taken for surgery was 7 min.

CONCLUSION: MSICS under Topical anaesthesia is safe, patient friendly and as effective as Peribulbar Anaesthesia.

Key Words: Manual Small Incision Cataract Surgery, Topical Anaesthesia, Peribulbar Anaesthesia.
Introduction:

MSICS (Manual small Incision Cataract Surgery) is a very suitable procedure for high volume surgeries in an unequally developed country like ours. It has been conventionally performed under peribulbar or retrobulbar anaesthesia, though at some centers, it is also done under subtenon or subconjunctival anaesthesia.

MSICS is the most cost-effective of all the surgical interventions in terms of quality of life restored.

The self-sealing incision of MSICS has shortened the duration of surgery considerably. This has resulted in the use of shorter acting anaesthetic agents with less invasive methods of administration.

Topical anaesthesia has been used in ECCE with IOL implantation (with subconjunctival supplementation) and also in clear corneal cataract surgeries. It has also been described along with intracameral Lignocaine 2% for MSICS. Peribulbar anaesthesia is a time-tested popular procedure, but it has a higher risk of injury to intraorbital structures and also a palpable fear of the “needle near the eye”. This can be completely avoided in topical anaesthesia.

We here describe a technique of using only topical Lignocaine 2% jelly in MSICS.

Materials and Methods:

All the patients opting for cataract surgery with PCIOL implantation were asked to participate in this trial. The first 150 who agreed to informed consent were randomized to either peribulbar or topical techniques of anaesthesia.

The exclusion criteria were:

1. Age <30 or >90 years.
2. Sensitivity to Xylocaine.
3. People who preferred Phacoemulsification.
4. Previous intraocular injury, inflammation or surgery.
5. Pupil <5mm in diameter.
6. Inability to understand the visual analog pain scale.
7. Inability to understand and comply with verbal commands (causes including deafness, dementia and aphasia).

They were operated upon by two surgeons of reasonably good experience (of 5 years and 1000 surgeries with the peribulbar technique and 100 surgeries with topical anaesthesia).

Assuming 90% power and 5% level of significance and assuming that there would be no pain in 40% and 60% of cases by either technique (difference of proportions), each arm should have a minimum of 58 patients. Assuming loss of 20% to follow up the study aimed to randomize 150 patients (although 144 would have been sufficient). Informed consent was obtained from all the participating patients.

The randomization schedule for each surgeon was obtained from a table of random numbers.

Each patient was randomly assigned a chit on entering the Block (preanaesthetic area) Room. The peribulbar anaesthesia was given by a senior postgraduate student. The topical anaesthesia was given by the nurse as 4% Lignocaine drops, 5 minutes before the surgery and by the surgeon on the table as 2% Lignocaine jelly after draping and insertion of the eye speculum.

The patient was asked to gauge for pain during surgery and 4 hours after surgery using the modified visual analog pain scale. After each surgery, the
surgeon evaluated her experience based on four parameters. Patient’s cooperation, difficulty due to ocular movements and anterior chamber stability were graded on a scale of 1 to 3, thus giving a cumulative range of 3-9 points. The fourth parameter was adverse results, which was mentioned as and when they happened.

The patients were followed on first postoperative day, first week and six weeks after surgery. The best-corrected visual acuity was noted.

**PERIBULBAR ANAESTHESIA:**

5ml of 2% Lignocaine with 1:10,000 adrenaline was injected using a 24G needle at the junction of middle and outer third of lower orbital margin with the needle directed parallel to the floor of orbit. A supplementary injection of 1-2 ml was given at the supraorbital notch with needle directed parallel to the orbital roof, if necessary. The eyelid was closed and pressure applied for 5 minutes.

**SURGERY UNDER TOPICAL ANAESTHESIA:**

At the start of the surgery, the patients were instructed to hold the hand of paramedical staff and to squeeze the hand whenever they felt pain, which was recorded together with the surgical step during which they felt pain.

The instillation of the local anaesthetic was done as described above. The patients usually reported a stinging sensation. After about 1 minute, they were advised to look at the operating microscope light and the surgery was started. No superior rectus suture was taken. The sclera was exposed by making a fornix based conjunctival flap and cauterizing the bleeding vessels with a bipolar wet field cautery. The rest of the steps were the same as in routine MSICS.

**MODIFIED VISUAL ANALOG PAIN SCALE:**

<table>
<thead>
<tr>
<th>Grade 0</th>
<th>Grade 1</th>
<th>Grade 2</th>
<th>Grade 3</th>
</tr>
</thead>
</table>

**Results:**

150 patients underwent MSICS between Feb 2009 and May 2009 and completed the 6 weeks follow up. They were operated by 2 surgeons. [Table 1]

<table>
<thead>
<tr>
<th>Surgeon</th>
<th>Peribulbar</th>
<th>Topical</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>43</td>
<td>35</td>
<td>78</td>
</tr>
<tr>
<td>B</td>
<td>35</td>
<td>37</td>
<td>72</td>
</tr>
<tr>
<td></td>
<td>78</td>
<td>72</td>
<td></td>
</tr>
</tbody>
</table>

93 patients were females, 50(64.10%) in peribulbar group and 43(59.72%) in topical group. Average age in the two groups was 67 and 65 years respectively. There was no statistically significant difference between the two groups w.r.t age (p=0.143) and sex (p=0.213).

Type of cataract according to morphology was nuclear in 52, nuclear and subcapsular in 76 and posterior subcapsular in the rest. Nuclear density ranged from Grade I to V and correlated with age.
That there is a significant difference in pain during administration of anaesthesia, between the two techniques, is entirely obvious. [Table 2] shows the various grades of pain during surgery in both the groups. Average for pain during surgery was 0.15 for peribulbar and 0.11 for topical in a range of 0-4. Most of the patients felt pain during prolapsing of the nucleus into the anterior chamber and during stretching of the wound while delivering the nucleus in the topical anaesthesia group.

**Table 2: Pain during surgery**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Peribulbar</th>
<th>Topical</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>66</td>
<td>64</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

The 3 patients who felt Grade 2 pain in the peribulbar group had increased positive pressure.

[Table 3] describes pain 4 hours after surgery. 3 patients in peribulbar group (average for pain=0.102) reported Grade 2 pain 4 hours after surgery compared to 70 patients in topical group (average for pain=0.67) reporting up to Grade 1 pain and 2 patients reporting Grade 2 pain. There were 2 posterior capsular rents in the topical group and 1 in the peribulbar group. 3 patients in peribulbar group had increased positive pressure during surgery while none in the topical group had so. One person in each group had buttonholing of the scleral tunnel. The incidence of postoperative complications in each arm was similar.

**Table 3: Pain 4 hours after surgery**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Peribulbar</th>
<th>Topical</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>70</td>
<td>24</td>
</tr>
<tr>
<td>1</td>
<td>5</td>
<td>46</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

**Table 4: Frequency distribution of surgeon’s score for surgical ease/discomfort during MSICS during peribulbar and topical anaesthesia.**

**Peribulbar Anaesthesia:**

<table>
<thead>
<tr>
<th>Surgeon’s score</th>
<th>Patient cooperation</th>
<th>Unwanted ocular movements</th>
<th>Anterior chamber stability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>74</td>
<td>57</td>
<td>72</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>14</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>7</td>
<td>5</td>
</tr>
</tbody>
</table>

**Topical Anaesthesia:**

<table>
<thead>
<tr>
<th>Surgeon’s score</th>
<th>Patient cooperation</th>
<th>Unwanted ocular movements</th>
<th>Anterior chamber stability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>69</td>
<td>70</td>
</tr>
<tr>
<td>2</td>
<td>2</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

There was no significant difference in both groups with regards to uncorrected and corrected visual acuity postoperatively. 73/78 (93.58%) patients in peribulbar group and 67/72 (93.05%) patients of topical group had BCVA >6/9.
The average time taken for surgery from insertion of speculum to taking off the speculum after subconjunctival injection was 7 minutes.

**Discussion:**

At the time of anaesthetic administration, topical anaesthesia scores much higher than peribulbar anaesthesia. The risk of globe perforation, optic nerve injury, pain and fear perceived because of peribulbar anaesthesia are all eliminated with topical anaesthesia. Further benefits include non-interference with the visual function (risk of retrobulbar haemorrhage including), unlimited ocular movements and absence of increased intraorbital volume. Patients under topical anaesthesia also had good analgesia intraoperatively, but the surgeon had to operate with incomplete akinesia, which some may find disconcerting. Surgeon’s evaluation of the technique indicates that the patient cooperation was very good in 96.5% cases. The importance of sticking to the selection criteria is hence emphasized. The incomplete akinesia is not exactly unwanted as the patient is able to follow the surgeon’s instructions very well. The anterior chamber stability is good in topical anaesthesia as there is no increase in intra orbital pressure. The slippery nature of the jelly along with the fact that surgery is done without a superior rectus support may lead to some discomfort to the surgeon.

The mean pain score of 0.11 (SD: range 0-4) is comparable to studies done on topical anaesthesia for phacoemulsification. The mean pain scores are similar to the studies for topical group but none of the patients in our study needed subtenon lignocaine supplementation as was required by some patients in all the mentioned studies. Similarly we did not have to resort to intracameral Lignocaine in any patient.

Fichman has found no major changes in pulse rate, blood pressure and respiration rate of patients during surgery under topical anaesthesia. There is also no significant change in plasma cortisol levels during surgery under topical anaesthesia. This shows that the patient is not under any undue stress when surgery is done under topical anaesthesia.

The relaxation of the ciliary body using Tropicamide (0.8%) with Phenylephrine (5%) prior to surgery is a very important tool to make the surgery pain free under topical anaesthesia.

The fact that the time taken for surgery is only <7 minutes is also contributory to the success of surgery under topical anaesthesia.

There have been no unwanted effects of Lignocaine gel 2% on extracapsular surgery and phacoemulsification.

The limitations of this study include the subjective nature of the modified visual analog pain scale and the fact that surgeries were done by only two surgeons.

**Conclusion:**

In MSICS, topical anaesthesia using Lignocaine 2% jelly is safe and as effective as peribulbar anaesthesia and is more comfortable to the patient at the time of administration.

**References:**


2. Parkar T, Gogate PM, Deshpande M, Adenwala A, Maske A, Verappa K. Comparison of subtenon anaesthesia with Peribulbar anaesthesia for


Perioperative glucose control: What an ophthalmologist needs to know
Dr. A.G. Unnikrishnan

Why control glucose in patients undergoing surgery?

Glucose control in the perioperative period is important for several reasons: firstly, hyperglycemia has been associated with perioperative mortality and morbidity. Secondly, hyperglycemia can lead to infections and impaired wound healing. Finally, there is sufficient clinical trial evidence to suggest that achieving normoglycemia can prevent minor and major complications in the postoperative period.

What are the targets for perioperative glucose control?

The targets for glucose control in the perioperative period are summarized in table 1. Targets must be institution-specific. In other words, before setting targets for a particular institution, the following issues must be taken into consideration: the patient’s affordability, availability of glucometers and nursing staff, and availability of doctors in the hospital. Intensive glucose control requires repeated glucometer-based testing, without which there is a risk of hyperglycemia. These targets hold good even if the patient is not a known diabetic, as glucose control has been shown to improve outcomes even in this particular subset. For minor surgeries, a short period of euglycemia before surgery is acceptable. However, for prolonged surgeries requiring general anesthesia, at least a fortnight of good control before surgery is essential, as this is required to reverse neutrophil dysfunction in diabetic subjects. A HbA1c value <7% also indicates good control.
Table 1. Targets for in-hospital perioperative glucose control

Before and After Surgery (monitor glucose every 2-4 hours):

- Fasting Plasma Glucose: 90-110 mg/dl
- Postprandial Plasma Glucose: 90-180 mg/dl
- In patients not eating, Random Plasma Glucose: 90-180 mg/dl

During Surgery/ in the Surgical ICU (monitor glucose every 1-2 hours):

- Random Plasma Glucose: 140-110 mg/dl

*Modified From: Guidelines of the American Association of Clinical Endocrinologists (AACE)*

Achieving targets in cataract surgery/ similar procedures

A simple flow chart is displayed in figure 1. In general, it is not advisable to make the diabetic patient fast for more than 4-6 hours. This is because fasting causes stress, and this releases hyperglycemia-inducing hormones like cortisol, catecholamine and growth hormone. In addition, fasting leads to catabolism, and causes the breakdown of adipose tissue to free fatty acids (FFAs). The FFAs, in turn, inhibit insulin secretion and interfere with insulin action- this triggers a vicious spiral of hyperglycemia.

**Figure 1. An algorithm for managing hyperglycemia on the day of cataract surgery.**

<table>
<thead>
<tr>
<th>Patient gets admitted the night before surgery</th>
<th>Same dose of oral drugs/ insulin is taken at night</th>
</tr>
</thead>
<tbody>
<tr>
<td>Next day, early morning, a random plasma glucose is checked</td>
<td></td>
</tr>
<tr>
<td>Glucose &lt; 180 mg</td>
<td>Glucose &gt; 180 mg/dl</td>
</tr>
<tr>
<td>Glucose &gt; 180 mg/dl</td>
<td>Regular Insulin 8u s/c</td>
</tr>
<tr>
<td>Check Glucose After 3 hrs</td>
<td>Glucose &gt; 180 mg/dl</td>
</tr>
<tr>
<td>Glucose &gt; 180 mg/dl</td>
<td>Regular Insulin 8u s/c</td>
</tr>
<tr>
<td>Glucose after 3 hours still &gt; 180mg/dl</td>
<td>Start insulin infusion (see figure 2), control glucose</td>
</tr>
<tr>
<td>Start insulin infusion (see figure 2), control glucose</td>
<td>Take up for surgery</td>
</tr>
</tbody>
</table>

Note: After Surgery, i.e. that day itself, the patient should take the same dose of oral drugs/ insulin, which he/she had been taking earlier.
Achieving targets for major surgeries

In this case, the patient is likely to require prolonged general anesthesia, and it is also more likely that the patient is more ill. In these situations, an insulin infusion from the morning of surgery is useful (see figure 2). This can be delivered either by adding insulin in a pint of dextrose/dextrose normal saline and infusing it. However, delivering it via an insulin infusion pump is a better strategy, and is outlined in figure 2. Combining insulin infusion with dextrose means that the patient will be obtaining calories and will not be starving: thus the stress response as well as catabolism associated with starvation can be prevented, and post-operative outcomes can be improved.

The ideal regimen and the inappropriate sliding scale

The sliding scale in a descriptive term for a fixed insulin protocol that is stuck on the notice board of some wards. The sliding scale essentially means that small doses of short acting insulin are given intermittently, depending on a table that says “x” units for a particular glucose level. The sliding scale is dangerous, as it does not take into account post meal excursions, and can result in a rollercoaster-like sugar fluctuations. It has been estimated that about half of in-hospital hypoglycemia is related to sliding scale use. It is desirable that the sliding scale is replaced by more physiological insulin regimens like the insulin infusion protocols or the basal-bolus regimen. The basal bolus regimen consists of giving long (or intermediate) acting insulin once daily (usually at bedtime) to set the base and a bolus dose of short acting three times a day before each meal. The fasting glucose is corrected by adjusting the long acting insulin, while the postprandial glucose is corrected by titrating the dose of short-acting insulin. Among the various insulin regimens, twice daily premixed insulin is the most practical, and can be used to achieve preoperative control

Figure 2. Intravenous insulin infusion for major surgeries

Step 1. Load insulin by adding 50 units short acting insulin in 50 ml of normal saline in a syringe pump

Step 2. Give a bolus dose of insulin. To decide the bolus dose, divide the current blood glucose by 50. For e.g. if the glucometer reading is 350 mg/dl, 350/50 = 7 units is the bolus dose of insulin to be given IV

Step 3. Set the hourly infusion rate on the infusion pump. To decide the rate, divide the current blood glucose by 100. For e.g. if the glucometer reading is 350 mg/dl, then the infusion rate is 350/100 = 3.5 units per hour. If the patient is fasting or will be fasting for more than 4-6 hours, also add 5% dextrose at 100 ml/hr with 10-20 mmol of potassium/500 ml.

Step 4. Readjust dose by Checking Glucose levels every 1-3 hours. Assume that the target range is 90-180 mg/dl. If the glucose level is below 90 mg/dl, decrease infusion rate by 1-3 units/hour. If the glucose is > 180 mg/dl, increase the infusion rate by 1-3 units/hour.

Step 5. Prevent hypoglycemia: If the glucose falls below 80 mg/dl, stop the infusion and give 1 ampoule of 25% dextrose. Recheck sugars hourly and restart infusion if sugars rise to > 180 mg/dl

Step 6. Stopping Infusion: If the insulin infusion is being stopped; give a dose of s/c insulin half an hour before stopping the infusion.

Note: This infusion is not based on any particular published protocol, and has been tailored to the Indian setting by the author. An internationally accepted protocol is the Yale insulin infusion protocol; visit: www.glycemiccontrol.net/pdf/Yale%20Insulin%20Infusion%20Protocol4.pdf for a free download.
The ideal regimen should be individualized for each patient. The success of any regimen for perioperative glucose control requires teamwork between the surgeon, physician, anesthetist as well as paramedical personnel.

**Figure 3. Insulin patterns**

*The top figure represents endogenous insulin secretion of by the pancreas, which all regimens must mimic. There is a “basal” insulin secretion throughout the day, on which is superimposed bolus secretions at the time of each meal. The twice daily (using 30: 70 premixed insulin) and the thrice daily treatment regimens (using one long acting insulin and three short acting insulin injections are both popular regimens for in-hospital control.*

**References**


The goal of any contact lens is to provide adequate vision with maximum comfort over a prolonged period of time. With rare exception, a rigid gas permeable material is the lens material of choice. Currently, there is some balance needed in choosing a material with high oxygen flux versus a material with greater durability and deposit resistance. A number of contact lens designs for fitting keratoconus have been suggested. The advantages and disadvantages of the major lens designs are discussed in the succeeding sections.

Rigid Gas Permeable Lenses

Rigid gas permeable corneal lenses are the lenses of first choice for correcting the irregular astigmatism which occurs as the cornea changes shape. The aim is to provide the best vision possible with the maximum comfort so that the lenses can be worn for a long period of time. A mild to high Dk/t material is preferred as it provides the stability required for these high powered lenses.

Fitting Methods

1. Three-point touch design

The three-point-touch design is the most popular and the most widely fitted design for keratoconic patients. The aim is to distribute the weight of the contact lens as evenly as possible between the cone and the peripheral cornea. Three-point-touch refers to minimal apical clearance in the center, minimal area of touch /bearing in the mid periphery with adequate edge
clearance. This type of fitting works well for small central cones.

2. Apical clearance fit
(Steep fitting)

In this type of fitting technique, the lens vaults the cone and clears the apex, resting on the para central cornea. This type of lens was suggested as it was argued that apical clearance would minimize trauma to the central cornea. Apical clearance lenses are small in diameter (8.0 mm) and have small optic zones (5.8 mm). The apical clearance method works well on cones which have central apexes or on displaced apexes which are only slightly inferior to the visual axis.

This method, best for smaller cones, is impractical for large cones, such as a sagging oval cone or globus cone. The potential advantages of reducing central corneal scarring are outweighed by the disadvantages of poor tear film, corneal oedema and poor visual acuity as a result of air bubbles becoming trapped under the lens.

3. Apical bearing fit
(Flat fitting)

This apical bearing fitting philosophy is useful for displaced apexes.

As keratoconus develops, the apex of the cornea is generally displaced inferiorly. If a small lens is placed on an inferiorly displaced apex, the lens is generally positioned low, and the lid often dislocates the lens with each blink. In such cases, a lens of larger diameter (9.0 to 9.8 mm) is preferable. The fitting method positions the upper edge of the lens under the upper lid to prevent lens dislocation. Good visual acuity is obtained as a result of apical touch. It has been reported that flat fitting contact lenses can lead to progression or acceleration of apical scarring.
Different types of RGP lens designs

Mild Keratoconus (<45D)
Aspherics or multicurve lenses
Kera 1 and II (No.7)
Acutity K
Rose K (David Thomas)

Moderate Keratoconus (45-52D)
Kera II
Quasar KNO7
Rose K (David Thomas)
Woodward KC3

Advanced Keratoconus (53-62D)
Kera II/III
Rose K (David Thomas)
Profile K (J Allen)

Severe Keratoconus (>62D)
Boston Scleral Lenses
Large diameter lenses
S-Limm (J Allen)
Dyna-intra limbal (No.7)
Rose K Lens (David Thomas)

The Rose K is a unique keratoconus lens design with complex computer generated peripheral curves based on data collected by Mr. Paul Rose, an optometrist, from New Zealand. It is the worlds most frequently prescribed gas permeable lenses for keratoconus which is FDA approved. The system (26 lens set) incorporates a triple peripheral curve system—standard, flat, steep—in order to achieve the ideal edge lift of 0.8mm.

Rose K fitting guide

Step One: Start the trial fitting 0.2 steeper than the average of the K readings. Initial base curve selection should be based on either keratometry or by corneal topography. For topographies, choose a first lens equivalent to the corneal curvature at the 3mm ring, temporal side.

Step Two: If this is a new rigid lens wearer, it may be advisable to use an anesthetic. This quickens the fitting process and allows a more accurate assessment of the fluorescein pattern because of reduced tearing.

Step Three: Too much fluorescein will give false patterns - so be careful when instilling it. Accurate fluorescein assessment is the only method of fitting this lens.

Step Four: Trial fit for the base curve – First assess the central fit. Ignore the peripheral fit at this stage. A light feathery touch at the apex of the cone is best. The rest of the pattern should look as close to alignment fit as possible.

Step Five: Next consider the peripheral fit. The trial lenses have a standard edge lift, but an increased or decreased edge lift on the same base curve can be ordered. If the central base curve looks good but you have inadequate peripheral edge lift, then order increased edge lift. Conversely, order decreased lift if there is excessive peripheral lift.

The design starts with a standard 8.7mm diameter and works by decreasing the optic zone diameter as the base curve gets steeper. It is available in base curves of 4.75-8mm and diameters of 7.9-10.2mm. Toric curves are available on the front and back surfaces as well as in the periphery. The practitioner has a choice of peripheral curves (standard lift increased or decreased edge lift). Standard lift lenses should work 70% of the time. Peripheral curves can be configured to a toric design.

The Rose K lens achieves an 85% first fit success in the UK. The Rose K lens design is a fully flexible lens that works well on early to advanced keratoconus patients. Complex lens geometry, combined with the enhanced material benefits of Boston ES™, makes the Rose K lens the good fit enhancing patient comfort and visual acuity. Multiple parameters make fitting the Rose K lens possible for most keratoconic eyes.
The peripheral band of fluorescein should be 0.5 to 0.7 mm wide.

**Step Six**: The standard diameter is 8.70mm but any diameter is available. Smaller diameters of 8.10 - 8.30 work well on steep corneas and on highly astigmatic corneas. A larger diameter will tend to make the lens ride higher. Palpebral aperture width also influences the diameter of the lens. For large palpebral apertures, large diameter lenses are required.

**Step Seven**: Do a careful over-refraction using 1.00D steps initially and then refine. If the final over-refraction is greater than 4 Diopters, vertex distance compensation has to be done.

**Follow-up examinations**

Prior to a follow up examination, the contact lenses should be worn for at least three continuous hours and the patient should be asked to identify any problems related to contact lens wear. Patients should be re-examined two weeks after dispensing their lenses. Regular routine six monthly examinations are important for good patient management and successful contact lens wear. Lenses should be re-polished at least annually and ideally every six months. Protein removal systems are also prescribed.

**Rose K2 Lens**

The Rose K2 lens was created to address two critical areas of performance for the keratoconic patient - spherical aberration and small optical zones. The base curve of the Rose K2 lens has an aspheric (non-spherical) optical zone unlike the spherical optical zone found on the original Rose K lens. This aspheric optical zone controls spherical aberration found on all contact lenses in higher minus powers, typically present with keratoconus lenses. The incorporation of aspheric optics into the lens design improves vision performance and enhances wearing comfort. The aspheric optical zone is larger than that of the original Rose K reducing glare, haloes and flare, common for many keratoconic patients in dim illumination (night time).

**Rose K2 IC (Irregular Cornea) and K2 Post Graft lenses** are large diameter, reverse geometry, intra limbal lenses (diameters that extend to the outer edge of the cornea), with aberration control aspheric optics for applications in specialty GP lens fitting. Primary applications for Rose K2 IC lenses include: Pellucid Marginal Degeneration, Keratoglobus, Post Graft, and LASIK- induced Ectasia, Post PRK, large oval, sagging cones and patients with irregular corneas due to trauma. Nipple and oval cones, found in some keratoconus patients, are secondary applications.

**ACT (Asymmetric Corneal Technology)**

By nature, the keratoconic cornea is asymmetric, where the inferior Quadrant is frequently significantly steeper than the superior portion, causing the GP lens to lift off at 6 o’clock. Rose K lenses incorporating ACT are designed to accommodate this asymmetry (good edge lift at 3, 9 and 12 o’clock but lift at 6 o’clock). The inferior quadrant of the lens is steeper than the superior quadrants, providing a more accurate fit at 6 o’clock making the lens more comfortable and often providing superior vision.
Piggyback lenses

Piggyback lenses are used when the Rose K lens has to be fitted very flat to attain good vision. Fit the Rose K lens 0.3 mm flatter and 0.5 mm larger than if the lens was fitted directly onto the cornea. Use a low minus soft spherical lens carrier which does not hydrate easily. The Rose K lens must move freely over the soft carrier. Always fit flatter than normal to ensure good Rose K lens movement.

Soft Lenses

Soft contact lenses have a limited role in correcting corneal irregularity as they tend to drape over the surface of the cornea and result in poor visual acuity. Silicone hydrogel lenses can be relevant here since the increase in rigidity compared to conventional hydrogels helps to mask the astigmatism and silicone hydrogel torics such as Pure vision (Bausch & Lomb) may be successful. However, soft lenses designed specifically for keratoconus such as Kera soft (Ultra-vision) or Soft K (Acuity contact lenses) have a useful role in early keratoconus or where a patient may be intolerant of RGP. They afford higher levels of comfort and longer wearing times.

One custom-made soft lens for keratoconus, manufactured by Flex lens is made of material with 45% water content and can be made in any power or curvature. When high-molecular-weight fluorescein is instilled in the eye of a patient wearing this lens, the fluorescein pattern under the lens is similar to that of a rigid lens. Lens movement is vital for a successful fit. Movement of this lens can be maximized by varying the secondary curve.

Disadvantages of soft lenses are corneal edema and neo vascularisation if the lenses are overworn.

Hybrid Lens System

The Soft perm lens (Ciba vision) is a hybrid lens with an RGP centre surrounded by a soft hydrophilic skirt. These lenses tend to be used in cases of RGP lens intolerance or for patients with displaced corneal apexes. There are many advantages to the soft perm lens as it provides better comfort than the RGP lens, better centration and visual acuity. It provides the comfort of a soft lens and visual acuity of a rigid lens.

The Synerg Eyes hybrid contact lenses are the first FDA-cleared hybrid contact lens specifically designed for keratoconus vision correction. Utilizing revolutionary hybrid technology, Synerg Eyes® has developed a family of lenses that provide keratoconus patients with the all-day comfort of soft contact lenses and the excellent visual clarity of rigid gas permeable lenses. SynergEyes contact lenses for keratoconus are custom designed to meet your vision correction needs. There are two different lens designs, SynergEyes® KC, and ClearKone™, to address all stages of keratoconus.

Disadvantages are the frequent breakage, giant papillary conjunctivitis and peripheral corneal neovascularisation.

Piggyback Lenses

Piggyback lenses are used in cases of RGP lens intolerance, proud nebulae in keratoconus, apical dimpling and recurrent corneal erosion. The system consists of a rigid lens fitted on top of a soft lens. The aim is to maintain the same level of visual acuity as with a single lens.

The RGP lens should be fitted first. Good centration is important and a slightly larger area of apical touch is usually acceptable as the RGP lens will be cushioned by a soft lens. A silicone hydrogel soft lens should be used where possible, with good movement and coverage/centration as in a normal soft lens fitting.

Caring of the two types of lenses can be difficult long term. The cornea should be observed carefully for dryness and neovascularisation.
Scleral lenses

Scleral lenses play a very significant role in cases of advanced keratoconus where corneal lenses do not work.

The Boston Scleral Lens

Boston Scleral Lens (BSLPD- Boston scleral lens prosthetic device) is a specially designed fluid ventilated gas permeable contact lens device that provides a non-surgical means of restoring vision in eyes affected with corneal disorders. Invented by Dr Perry Rosenthal of Boston, FDA approval was obtained in 1994. The lens is about 18 to 23 mm in diameter and it rests entirely on the sclera and arches over the damaged cornea, thereby creating a space that is filled with normal saline or artificial tears. This fluid reservoir masks the distorted corneal topography and improves vision. It also functions as a unique liquid bandage by protecting the corneal surface from the desiccating effects of exposure to air and the friction of blinking. The presence of corneal oedema is a contraindication to wearing this device. The need to customize and design individual lenses makes these lenses expensive.

Conclusion

Patients with keratoconus are a challenge. A wide range of contact lens designs and materials is available. Contact lens management is often a compromise between the quest for an ideal fit and the patient’s requirements for comfort and best vision. Keratoconics can live a normal life with the help of a good contact lens practitioner.

References

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5. Keratoconus-Boston foundation for sight.
How to Make a Good Power Point Presentation

Dr Biju John.C MS; DNB; FRCS

Introduction

The purpose of any presentation is to present information in an interesting and effective way. Microsoft’s Masterpiece software “MS Power Point” allows us to combine text, graphs, charts, relevant photographs and diagrams and of course Video so as to make the presentation content more interesting and easier to understand. If you can put in one word what an ideal power point presentation should do, well arguably it is “Communicate”. The software should help the presenter to communicate effectively with the audience by enhancing and accentuating the presentation without overwhelming it. The key to success is to make certain your slide show is indeed a visual aid and not a mere “teleprompter”. At the same time it should not be a visual distraction, because of overuse of lots of “bells and whistles” available in the software.

Vint Cerf—widely regarded as one of the “father’s of Internet” had this to say about Power Point. “Power corrupts and PowerPoint corrupts absolutely.” So it is important that we handle this software the right way so as to make our presentations meaningful and useful to our audience and at the same time keep them focused and interested.
Planning your presentation

The first step of any successful presentation is planning. Planning helps you decide the content and the order in which the information has to flow. Collect all the information you need. Make sure that your sources are current and also well accepted eg standard journals, text books etc. The presentation should always be tailored to the type of audience and also to the subject. This requires some amount of homework. All your hard work may go waste if you end up giving the presentation which you prepared keeping in mind a group of Ophthalmologists, to a group of General Practitioners.

Read and Research your subject thoroughly. After a good presentation it would look embarrassing if you are not able to answer some basic questions about the topic. You don’t want to leave the audience with the impression that “His knowledge about the subject is only Power Point Deep”. So know your presentation so well that you will be able to do the presentation even if there is no computer available. Just to bring into perspective the amount of preparation needed for a good presentation or speech let me quote Mark twain. “It usually takes more than 3 weeks to prepare a good impromptu speech”

Arranging the Content in the Slides

The most important ingredient of a good presentation is the quality of its scientific content. At the same time even the most thoroughly researched and up to date content may go waste, if it does not come out in a structured and orderly fashion. The information flow should be progressive and clear. For eg: You are explaining life cycle of Toxoplasmosis. Consider 2 scenarios

1. You show a picture like the one below and explain in one go. Best way to beat the timer.

2. Consider an alternate scenario. The slide has only the picture of that part of the life cycle in cat’s intestine at first appearing successively as the presenter explains it. Then on mouse click the next part appears viz the Oocyst and its sporulation and this is explained. The next part ie ingestion of Oocysts by the intermediate hosts then appears and so on.

The audience will definitely benefit better by the 2nd mode of rendering as here the flow of information is progressive and easier to focus on. On the other hand in the first scenario all the information is presented simultaneously and so is cluttered and difficult to focus on.

This holds good in the relation between different slides also. They should be arranged such that it flows out as a coherent story. It is also a good idea to have an introductory slide highlighting the key points you want to discuss and stick to that order so that the audience will be aware what path they are expected to follow in the presentation.

The KISS principle

Cramming up your presentation with a lot of
information is not going to improve its quality. After all the idea of any presentation is to convey a set of ideas effectively to the audience in such a way that they are able to grasp most if not all. Be aware that the attention span of your audience is short. Always remember the “KISS principle” when designing your presentation ie “Keep it Short and Simple”. Try to focus on 4-5 main points. Make sure that your slides emphasize the main points. Try to have only one main idea in one slide and not more than 4-6 points per slide. In corporate world ideal presentations are supposed to obey the 10/20/30 rule which means a good presentation should have 10 slides, should finish in 20 minutes and the font size of the body text should not be below 30 point. O.K that rule cannot be applied exactly like that in our case, but this is how generally it should be.

Ensuring Clarity and Readability of the Slides

Consider this scenario. You are explaining the Glycolytic pathway. You included all the steps in a single slide as you don’t want to break the continuity and after all it is one single process. PowerPoint had warned you when you were typing the slide that you better split this up into 2 different slides. But you ignored it. So now you have everything in one single slide and you begin reading from 1st step. The audience especially those in the back rows starts reading from their handouts as the strain of trying to decipher what is written on the screen becomes too much for them. If no hand outs are available, they would be probably reading their program sheet to find out what follows or what is going on in the other halls. Lack of Clarity and readability of the slides is one of the biggest PowerPoint killers. Solution: The “666 rule”

The 666 rule

This is a good rule suggested by “Presenter’s university” for simplicity of design and clarity of the content in PowerPoint.ie

Use No more than

- 6 words per bullet
- 6 bullets per slide
- 6 Text slides in a row

If you adhere to the above principles, the readability and visual impact of your slides will improve greatly.

The fonts should not be too big or too small. If you observe the 666 rule then power point will automatically adjust the font size to about 30 to 36 points which is ideal for body text and between 36 to 48 points for title. Another idea suggested by a veteran to determine the optimal font size is “find out the age of the oldest person in your audience and divide it by two. That’s your optimal font size”. The reason people use a small font is twofold: first, that they don’t know their material well enough; second, they think that more text is more convincing.

Don’t forget to Format

Always remember to format whatever text you put in the slides appropriately. Imagine reading a slide filled with 1 complete paragraph of text. Neither you nor your audience will be able to extract any reproducible information from that. You might try but your audience is sure to give up. So format the text as Bullet points, limiting it to 6 bullets a slide and each bulleted line containing not more than 6 words. This will make the major ideas stand out, which the audience can easily grasp and you can also expand upon the ideas by adding more information. The audience loves it when you add your own experiences to emphasize the points. Occasional stories or jokes can really liven things and you can put the audience under your spell. But be careful that you keep all these things within the context of that particular point or idea that you are trying to convey to the audience. Clear sans serif fonts like Arial, Lucida sans, Helvetica, Comic Sans
MS etc are easier to read from a screen whereas script type (Serif fonts) is difficult to be read from a screen. Again italics are also difficult to be read from screen. So use them only sparingly. Avoid All Caps. Better to use different colors and bolding for emphasis than underlines as underlines may signify hyperlinks. Use numbered lists when you have a list with priority or sequence. Otherwise use bullets.

Use colors having good contrast between background and text (Light on Dark /Dark on light) and also complementary colors. Selecting built in power point templates and sticking on to the colour combinations inside them will be safer.

Don’t Read from slides

Many presenters in conferences use the slides as a mere teleprompter and just read what is written on the slides. This is not good communication. An oral presentation should focus on interactive speaking and listening, not reading by the speaker or the audience. Reading text ruins a presentation. Your slides should attempt to reinforce your words and not repeat them.

Graphics Really counts

No body can keep concentrating on Text alone slides for long. Also if you intend to show only text then why do you need power point? An OHP would be more than sufficient. Adding suitable and appropriate graphics (pictures, graphs, charts, animations) add life to your presentations and will help to keep up the interest of the audience. Also we all know that “A Picture is worth thousand words”. So graphics can convey a lot of information and in a much more effective way than words. Never lose an opportunity to use them, but make sure that they are appropriate and won’t seem totally out of context. Illustrate but don’t decorate. They should be used only to emphasize key points of your presentation. For eg everybody is tired of those usual windows clipart pictures and many cant bear the sight of those popping up on slides just for the sake of having a graphic there. Also do you really need the picture of your family in the midst of Management options for that disease? If you so badly want to show it then let it be your Thank You slide. At the same time when somebody is explaining the Anatomy of Orbit with the help of two slides full of text and then shows you the diagram as the next slide and tells you that “this is what I have just explained”-All you want is to close your eyes and doze off. This is a situation where text is totally unwanted or to be used only along with the graphics so as to emphasize your point.

Consistency

Use the same colors and fonts throughout. Better not to use more than 2 types of fonts in your presentation- one for the titles and the other for the text.

Select graphic images in the same style. Templates go a long way toward helping to maintain consistency. If you are not using templates then it is better to alter the fonts, colour, title format etc in the Master Slide so as to preserve consistency.

Differences on the other hand can be used effectively to draw attention to something, to imply importance. The difference in the word “Difference” in the previous sentence is to illustrate this. But be very careful not to overdo this as you may end up distracting and confusing the audience. Use SURPRISES to attract and not to distract the audience. If I had used different word art for the different words in the previous sentence, I would have ended up only distracting the readers.

Slide Transitions, Sound Effects &Text Animations

“PowerPoint is designed for making a slide show a little more attractive with images and text that
move, but when an idiot makes them all move, interest is lost.” –Anonymous.

It is not known who contributed this quote, but it brings out the good and bad of Power Point very effectively. Adding movement to text, graphics, and images creates a dynamic, engaging experience for your audience and enhance visual interest and impact. But if they become the focus of attention rather than the content then that ruins the presentation. They can be used effectively to stress a point, to bring something into focus etc. Edward Tufte really hits the nail on its head when he says-

“If your words or images are not on point, making them dance in color won’t make them relevant.”

The corollary is if it is a key point then making it stand out from the rest of the text can only make the presentation better, provided you do it subtly and without distracting the audience. Indeed, good PowerPoint animation is so seamless that you are unaware of it. It reaches its zenith when it allows audience to become lost in the story you are telling.

Knowing when and where to draw the line between dynamic content and distraction is essential to creating clear and compelling presentations.

If you are using Custom Animations, add them with care. Too many will distract your audience. They will be more interested in what is flying in from where, than in your topic. Typical examples of distracting animations are the “letters flying in from top one by one”; swivel; swish etc. However, little or no animation in your presentation might cause them to snooze if your topic is dry. Different text animations and transitions on different slides is also not a good idea.

Animation is at its best when it is used to

1. Reinforce Key Concepts;

2. Illustrate processes
   a. Eg: Pathogenesis; Examination Techniques; Working of Instruments

3. Conveying concepts of change
   a. E.g.: Stages of Disease

However the custom animations available in Power Point are highly inadequate for this sort of an application. There are other excellent software which will help you in designing such animations and insert them into power point. My favourite is Macromedia Flash. It not only helps you to create excellent animations to help you explaining processes, pathogenesis etc, but better still you can create entire presentations in the software itself without using power point at all. And once exported as a windows executive file, the presentation can be played in any windows computer even if it hasn’t heard of Microsoft Office.

Practice and Rehearse Well

“Time and that Timer will wait for none” – Well that is of course my own modification of the famous proverb. The Timers are here to stay in the conferences. High time we accept that and stop our fight with them. William Shakespeare’s words “Better be three hours too soon than one minute too late” should guide us here. Of course we are talking in minutes here and not hours. It is important that we rehearse our presentations a number of times with the “Rehearse timings” facility in Power Point and make modifications until we are sure that it will finish well ahead of the allotted time. This is all the more important for beginners and ideally they should practice in front of an audience.(Just in case somebody new to PPT is doing this-After the rehearse timings show is over when Power Point asks you that stupid question-“Do you want to keep the new slide timings to use …………….?” Make sure that it is the “no” button that you click.)
“Absolute faith in Technology can land you in trouble”

I remember one of my professors who used to take a few OHP notes along with his CD ROM as a back up. In his words “you never can trust these computers”. Well even though now days these occasions when the Laptop at the podium becomes allergic to a particular presentation especially the videos are quite rare, they do happen. So it is always better to be have a back up plan. I usually take at least 2 CD ROMs and one USB pen drive having my material. Also make sure that the presentations are working perfectly from all these media before taking them to the venue. Videos and Hyperlinks are notorious in giving the presenters problems by refusing to behave properly at the right time. Make sure that you copy the entire folder along with the video, animations and the PPT presentation to the computer to be used for presentation and make sure that they are working properly in the preview room. In worst case scenario if your beautiful video refuses to play during the presentation, Be ready to come out of the Power Point, go to the folder and make the video play by directly double clicking on the video file. Of course after it plays out you will have to manually close the media player and once again resume the slideshow from where you stopped. Still much better than not being able to play the video at all.

Summary

In summary a good Power Point presentation should be Simple; Factual; Focused; Clear; Progressive; Consistent and Easily readable. Content and Communication holds the key for excellence here. The text should be to support the communication. Graphics including animations are there to simplify complex concepts and explain complex relationships. These and other visuals including video should be used to support and not to distract. Use sounds only when absolutely necessary.

Disclaimer

Different people have different expectations from a particular presentation and hence what seems as an excellent presentation to one may be perceived as a useless and dull presentation by another, depending on their general outlook, attention span, and expectations. Naturally an article like this definitely will have a personal bias. So the opinions expressed here should be viewed only as a set of loose guidelines endorsed by many but not all experts in the field. The reader is free to accept or reject it.
By providing both ultra-structural and quantitative information of the macula, OCT has become the diagnostic investigation of choice for macular disorders.

The basics of OCT interpretation can be achieved by answering 4 standard questions.

Understanding what a normal macula on OCT looks like is of primary importance before we understand its abnormalities. It is characterized by 2 high reflective structures, the nerve fibre layer and the retinal pigment epithelium choriocapillaries complex.

In between the two red layers are various layers of the sensory retina represented with shades of green. In between the two red layers are various layers of the sensory retina represented with shades of green.

Interpretation of abnormal OCT images is better understood by answering the following 4 questions.

1. How does the vitreo retinal interface appear?
2. What is the foveal contour like?
3. Is Retinal architecture altered?
4. **Whether the uniformity of retinal pigment epithelial choroiocapillaries layer is disrupted?**

Now let us see how to answer these questions by going through the various abnormalities that we may come across when we are answering these questions.

1. **How does the vitreo retinal interface appear?**

   **Optically** clear vitreous above and a high reflective nerve fibre layer below represent the normal vitreo retinal interface.

   The vitreo retinal interface abnormalities can be a membrane that can be a single or a double membrane.

   The attachments of these membranes vary from no attachment, partial attachment or total attachment.

2. **What is the foveal contour like?**

   The normal foveal contour is a V shaped depression.

   Abnormality in the foveal contour can be in the form of obliteration of the foveal contour which can be either due to pulling mechanism from an overlying membrane or by pushing mechanism from underlying intraretinal fluid.
Widening of the foveal contour is another abnormality which when seen signifies foveal thinning.

Macular holes can be full thickness or lamellar which can be either inner lamellar or an outer lamellar hole.

The steep configuration of the walls noted in pseudo holes helps one to differentiate from the inverted mushroom shape of inner lamellar hole.

3. Is Retinal architecture altered?

Abnormality of the retinal architecture can appear as accumulation of fluid which can be either intraretinal or sub retinal fluid.

The intra retinal fluid accumulation can be diffuse or with cystic spaces.

Hard exudates appear as spots of increased hyper reflectivity with a trail of shadowing behind it.

Schisis which can be identified as a split in macula can be rarely seen and easily diagnosed.
4. Whether the uniformity of retinal pigment epithelial choriocapillaries layer is disrupted?

The uniform red layer of the retinal pigment choriocapillaries complex can be disrupted by a bumpy appearance due to Drusen, fusiform thickening depicts a possible choroidal neovascular membrane.

Elevations seen due various retinal pigment epithelial detachments can be better understood by this composite picture. **Serous PED** is characterized by optically clear space with a distinct reflectivity from the underlying choroid underneath it. **Hemorrhagic PED** appears similar to serous PED but there is absent reflectivity from the underlying choroid due to shadowing effect of the blood beneath the RPE. **Fibrovascular PED** is characterized by multiple medium reflectivity echoes. Distinct reflectivity of underlying choroid is absent.

To summarize if one sees a distinct reflectivity of choroid **serous PED** should flash in ones mind.

With this basic understanding let us see how we can interpret OCT images by answering these four questions.

**Case 1** is an OCT image of a patient with macular thickening.

The vitreo retinal interface shows a double membrane which is partially attached to the retinal surface.
The foveal contour is obliterated to pulling mechanism.

Retinal architecture is altered due to intraretinal accumulation of fluid in cystoid spaces.

The retinal pigment epithelial and choriocapillaries complex is uniform.

To summarize this image is characterized by a cystoid macular edema due to a vitreo-macular traction and with a presence of antecedent epiretinal membrane.

**Case 2** is an OCT image of patient with diabetic maculopathy.

The vitreo retinal interface shows no abnormality. The foveal contour is partially altered due to pushing mechanism.

Retinal architecture is characterized by diffuse accumulation of intraretinal fluid and sub foveal fluid. Intraretinal accululation of hard exudates are also noted.
The retinal pigment epithelial and choriocapillaries layer are normal.

To summarize this image is characterized by diffuse macular thickening with subfoveal serous detachment.

**Case 3** is an OCT image of a patient with a macular hole.

The vitreo retinal interface shows a single membrane which is partially attached.

The foveal contour is lost due to a full thickness foveal defect with a presence of a normal retinal pigment epithelial choriocapillaries layer.

To summarize this image is characterized by the presence of an epiretinal membrane with a full thickness macular hole.

**Case 4** is an OCT image of a patient with Central serous chorioretinopathy.

The vitreo retinal interface is normal.
The foveal contour is partially obliterated due to the pushing mechanism of intraretinal fluid.

The retinal architecture is characterized by the presence of intra and sub retinal fluid.

The vitreous interface is normal with partial obliteration of foveal contour with intraretinal and subretinal fluid altering the retinal architecture.

The Retinal pigment epithelium and the choriocapillaries is elevated with a optically clear space with a distinct reflectivity form the choroid.

To summarize this image shows the presence of cystoid macular degeneration in a patient with central serous retinopathy with a small subfoveal serous PED.

**Case 5** is an OCT image of a patient with choroidal neovascular membrane.

The vitreal retina interface is normal with partial obliteration of foveal contour with intraretinal and subretinal fluid altering the retinal architecture.

The Retinal pigment epithelium shows a fusiform focal thickening.

To summarize this patient has a CNVM causing subretinal fluid.
OCULAR TUBERCULOSIS

Focal choroiditis (Fig 1) in a 28 year old lady who presented with mild blurring of vision OD with multiple systemic problems including cranial nerve palsies. HRCT was done which revealed the presence of miliary tuberculosis.

Subretinal abscess with optic disc granuloma in a 38 year old female who presented with periorbital swelling and pain on extraocular movements. (Fig 2a)

Mantoux was done which showed necrotising reaction. Resolution was seen following ATT and supportive systemic steroids. (Fig 2b)
Focal retinochoroiditis (Fig 3a) with vitreous haze in a 19 year old boy who presented with recent onset blurring of vision with a significant history of weight loss and evening rise of temperature. Systemic evaluation showed caseating hilar lymphadenopathy, following which he was put on ATT. The retinochoroidal lesion subsequently scarred (Fig 3b).

Multifocal choroiditis with optic nerve head granuloma and papillitis (Fig 4) in a 40 year old male, who presented with painful, sudden onset decrease in vision, lid edema and erythema, a picture simulating orbital cellulitis. Systemic workup showed presence of tuberculosis.

Solitary choroidal granuloma (Fig 5) discovered in a 50 year old male of pulmonary tuberculosis who had no eye symptoms but came for routine ophthalmic evaluation.

Ocular syphilis is relatively uncommon and can have myriad presentations like focal or multifocal choroiditis, chorioretinitis, neuroretinitis, occlusive vasculitis (Fig 6) ,optic neuropathy and even extensive pigmentary in late stages changes simulating retinitis pigmentosa. It should essentially be suspected in any case of uveitis resistant to conventional therapy. Ocular syphilis should be equated with tertiary syphilis and managed like neurosyphilis.

PARASITIC INFECTIONS

OCULAR TOXOPLASMOSIS

Focal necrotizing lesion adjacent to an old chorioretinal scar. (Fig 7 a, b)- representing
congenital infection. This is a classical presentation of Toxoplasmosis as compared to tuberculosis where fresh activity is seen within an area of old scar. Systemic toxoplasmosis can present with multiple lymphadenitis and pyrexia of unknown origin and can even be mistaken for lymphoma.

Focal necrotizing retinochoroiditis patch without an adjacent chorioretinal scar (Fig8) – active toxoplasma retinochoroiditis representing an acquired infection.

Dense vitreous haze with underlying active focal retinitis patch (Fig 9a) - Head light in Fog appearance

Inactive vasculitis with an adjacent retinochoroidal scar (Fig 9b)

Kyrelias arterialitis - non occlusive non inflammatory, non leaking arterial plaque (Fig 10)
Franceschetti’s syndrome-traction band extending from old scar to disc. These bands can also extend between two scars (Fig 11)

Inactive lesion- Punched out chorioretinal scar at posterior pole (Fig 12)

OCULAR TOXOCARIASIS

Ocular toxocariasis is caused by infestation with the toxocara canis more commonly known as the intestinal roundworm of dogs. It typically presents in healthy adults as a solid peripheral or posterior pole granuloma with associated traction bands resulting in disc drag(Fig 13a, b), vascular distortion, macular heterotopias, retinal stress lines and even retinal detachment. All these findings can be seen in the absence of uveitis. A much less common presentation as chronic endophthalmitis in a younger age group.

OCULAR GNATHOSTOMIASIS

Gnathostoma spinigerum- unusual intraocular worm presenting as anterior uveitis with secondary glaucoma, subretinal track with adjacent vasculitis (Fig 14 b), in a 44 year old male. Repeat examination 1 week later showed the presence of worm on the iris and iris holes (Fig 14 a).

OCULAR DIROFILARIASIS

Intraretinal dirofilarial worm with diffuse retinal pigmentary changes (Fig 15 a, b)-(Fundus photo courtesy Dr Biju Raju.)

INTRA OCULAR CYSTICERCOSIS

Cysticercosis is caused by the pork tapeworm, it is a relatively rare infection, and presents as a cystic lesion inside the eye (Fig 16). Ultrasound demonstrates a cystic lesion with central
hyperechoic, highly reflective scolex. Surgical removal is the treatment of choice. Here we can see a subretinal cysticercosis pre and post op pictures and USG. CT scan showed that there were cerebral cysticercosis as well. Always look for neurocysticercosis in cases of ocular cysticercosis.

48 year old male patient of alchoholic liver disease and suspected hepatocellular carcinoma presented with diminution of vision both eyes. Fundus examination showed vitreous exudation with foci of retinitis in both eyes (Fig 18 a,b). He was diagnosed to have metastatic endophthalmitis. Vitreous biopsy culture was positive for Candida.

**MUCORMYCOSIS**

Aspergillus endophthalmitis in immuno compromised patient, who gave a history of diabetes and alcoholic liver disease for which he had underwent a liver transplant and was on immunosuppression. Fundus examination showed large yellowish infiltrates in posterior pole which progressed rapidly over few days (Fig 17 a,b). Vitreous biopsy was positive for Aspergillus.

43 year old male ,known HIV positive with low CD 4+ counts who presented with floaters .Fundus examination showed whitish area of retinal opacification with adjacent haemorrhage along the retinal blood vessels(Fig 20 a,b) suggestive of CMV infection.

**VIRAL DISEASES**

**CMV RETINITIS**

Mucormycosis- in an HIV positive Manipuri lady who later succumbed to infection. (Fig 19)
The same patient after treatment with intravenous gancyclovir-inactive CMV retinitis characterized by decreased haemorrhages, sheathing of vessels and a more granular appearance (Fig 20 c,d)

In the absence of treatment, these lesions show a relentless extension along the retinal blood vessels with eventual involvement of the optic nerve head.

**ACUTE RETINAL NECROSIS**

Acute retinal necrosis (Fig 21a,b,c) typically affects healthy individuals and is characterized by retinal necrotic infiltrates with periarteritis which begin in the retinal periphery and eventually result in full thickness retinal necrosis. These may be accompanied by vitritis, retinal haemorrhages and optic disc edema. The posterior pole is involved in late stages.

This 45 year old gentleman came with history of lump in the throat, fever and diminution of vision. Biopsy from the neck revealed lymphocytic infiltration, further investigations proved that he had systemic CMV infection (Fig 22)
Successful Correction of Macular Ectopia and Disc Intortion with Vitreoretinal Surgery

Dr Simi Manojkumar, Dr Jyothirmayi, Dr Vanaja Raghavan, Dr Abhijit Khake, Dr Sreeni Edakhlon, Dr Gopal S Pillai

This 30 year old lady had complaints of decrease of vision in the left eye of 3 years duration. There was progressive diminution of vision and on examination, her best corrected visual acuity was 6/6 in the right eye and CFCF in the left eye. There was a left exotropia of about 30 degrees. Anterior segment examination was within normal limits. Retinal examination revealed normal disc, macula and retina in the right eye. Left eye showed an abnormal disc with 90 degrees of intortion. (fig 1) There was a falciform fold arising from the inferior retinal periphery, right till the disc, and the macula was entangled within it. (fig 2) There was a macular drag and the macula was lying inferior to the disc. The retinal periphery was screened and found to be normal. There were no cells or any other evidence of active uveitis.

The patient underwent a pars plana vitrectomy and dissection of the falciform fold. Underneath the fold, there was a choroidal granuloma and the connection between the fold and the granuloma was severed. There was an epiretinal membrane on the surface of the falciform fold which was peeled out. Postoperative period was uneventful.

Figure 1 Disc left eye                                           Figure 2 Disc and macula left eye

Fig 3 Post op 1 month left eye
After 1 month, the disc remained intorted, but the falciform fold showed signs of opening up and the macula got free from the fold. The fovea and macula were seen separately from the fold, but there was still retinal detachment involving the macula. The vision had improved to 1/60 in the left eye.

After 3 months, the disc started to show signs of extortion. The macula further moved towards its original location. The retinal detachment around the macular region was showing signs of settlement. Vision improved to 2/60 and the squint disappeared.

After 6 months, the disc almost completely became normal with nearly 80 degrees extorted from the first picture. The macular morphology was nearing normalcy and the fluid around the macula had dried up significantly. Her vision had improved to 6/60 and the squint was not to be seen.

**Discussion**

Macular ectopia and disc tortions or drags can be seen in certain cases like ROP, Toxocariasis, high myopia etc. Macular ectopia may be a cause of severe visual loss and in most cases, it is irreversible. Here we are presenting a case where there was a macular ectopia and disc intortion, probably secondary to a peripheral retinal toxocara granuloma.

Vitrectomy and dissection of the epiretinal membrane has freed the macula from the traction completely and this has over a period of time moved out from the ectopic location to the central location. This was also associated with an improvement of visual acuity.

A search of the literature has shown that macular ectopia is a common accompaniment of ROP, but seldom has anyone tried vitrectomy in the management of ROP induced macular ectopia, because it is usually long standing and visual prognosis is guarded. In cases with diabetic macular traction and macular heterotopia, the results of vitrectomy has been excellent. In such cases, as patients undergo vitrectomy early, the visual prognosis is much better. However in this case, we had attempted vitreo retinal surgery because the history dated only a few years.

Thus vitrectomy and epiretinal membrane dissection can be of help in cases with macular ectopia especially of a short duration of onset. The squint causd by the same was corrected spontaneously as the eye took up foveal fixation.

**References:**


A 63 year old male presented to our hospital with a history of bee sting injury to right eye 1 day back. On examination his BCVA was perception of light with inaccurate projection OD and 6/6 OS. The IOP was unrecordably low. There was no history of any systemic disease. On slit lamp examination the right eye revealed a diffuse stromal corneal edema with coarse descemet’s folds and the anterior chamber was totally flat. [Figure1] B scan ultrasound revealed multiple point echoes of moderate intensity in the vitreous cavity suggestive of vitritis. [Figure2] Ultrasound biomicroscopy was performed which revealed ciliary body effusion. [Figure3]. Laboratory investigations revealed: Hb 13.4 g%, TC 16100/dl, DC P65, ESR 5, RBS 133, B.Urea 55mg%, S.Creatinine 1.3mg%, Cholesterol 133mg%, SGOT 80 u/l, SGPT 224, ALP 85, Total protein 7.1 g%, Albumin 4.8mg%, Globulin 2.3 g%, Sodium 139 meq/l, Potassium 3.5meq/l. The patient was then started on Systemic steroids initially IV methyl Prednisolone with systemic antibiotic followed by topical antibiotics, steroids and cycloplegic. On the next review after 1 week, on examination the corneal edema had partially resolved residual descemet’s membrane folds were seen. The anterior chamber was shallow. The pupil was oval, mid dilated and fixed. The anterior lens capsule showed pigments. The lens showed nuclear sclerosis. [Figure4]. B scan ultrasound OD revealed multiple dome shaped elevations with a moderately reflective membrane which depicted a double spike at the corresponding location on the A scan and the interior of the elevations showing low intensity hyperechoic signals with corresponding low intensity spikes on the A scan suggestive of multiple serous ciliochoroidal detachments (Fig 5).
Discussion

The bee sting is a modified ovipositer in female bee and wasp. It has toothed lancets and poison glands. The venom acts as a toxic agent, and the sting itself has a mechanically damaging effect on the structures of the eye (Grant, 1962; Bucherl and Buckley, 1971). Toxic reaction is due to biological amines which are protoplasmic poisons. It contains neurotoxin, hemolitin, mellitin, hyaluronodase, phospholipase A and histamine (Gilboa et al; BJO 1977, 61, 662-664).

Dose of venom and adverse events are unknown and these could be toxin related or immunologically mediated. Case reports show VEP to be prolonged but pattern ERG is usually normal. The retina may not be affected. In our case it was a direct sting to globe. Various reported clinical presentations in cases of bee sting include Cilio choroidal detachment (Pal et al Eye 2005), Optic neuritis (Choi et al Korea 2003), Mimics viral keratitis (Jain et al Cornea 2007), Cataract and corneal decompensation, (Arcieri et al Cornea 2002), Cataract (Agarwal et al IJO 1995), Uveitis, glaucoma and optic neuropathy (Teoh et al Ophthalmology 2005), Disc oedema, Scotoma (Maltzman et al Ophthalmology 2000) and bilateral optic neuritis( AJO 1994) Papilloedema of 1 dioptre and much retinal venous engorgement with normal visual acuity have been observed 9 days after bee stings (Goldstein and Rucker, 1964). Optic atrophy and papillitis secondary to bee sting have been described (Walsh and Hoyt, 1969; Goldstein and Woltman, 1960).

In this case there is cataract, low IOP and choroidal detachment with no perception of light. This is probably due to the optic neuritis element which was underlying. The devastating effect in this case may be due to direct inoculum of toxin into the eye unlike in many published data where there is bite in the periorbital region.

References:

FONSECAEA PEDROSOI – Unusual Cause for Corneal Ulcer – A Case Report

Dr Bindu N Das MS, Dr Jalal P M DO

Introduction:

Fonsecaea pedrosoi, a dermataceous fungi is the commonest causative agent of chromoblastomycosis – a chronic mycotic cutaneous and subcutaneous infections which primarily occurs in the humid tropical regions.

Corneal infection due to Fonsecaea pedrosoi is very rare and a few cases of infection are reported in the literature. Treatment of this mycosis is challenging not only because of scarcity of effective antifungals but also due to the need for the prolonged periods of treatment, either with medications or surgery.

Here we present a case of corneal ulcer due to Fonsecaea pedrosoi which showed necrotizing features, both in cornea and conjunctiva which responded to topical and systemic antifungals

Case Report:

A 59-year old male patient was referred to our OPD in June 2009 with a corneal ulcer of the left eye 3 weeks after trauma with a tree twig. Patient had been treated with topical antibiotics, antifungals and cyclopentolate for two weeks without any clinical improvement.

His distant visual acuity was 6/36 in the left eye and 6/9 in the right eye at the time of presentation. IOP measured was normal. There was a 3mm X 3mm sized corneal ulcer over the temporal cornea (Figure 1) with regular borders with infiltration up to the anterior stromal area. Surrounding cornea was hazy.

Anterior chamber showed mild inflammatory reaction with few KP’s over the endothelium with mild flare. Posterior segment examination was within normal limits.

Because of clinical features and findings – a diagnostic possibility of non healing corneal ulcer due to fungi or low virulent bacteria were made. Topical antibiotics was changed to fortified cefazolin and gentamicin, antifungal Natamycin.
Corneal scrapings were collected for fungus and bacteriologic microscopic examination and culture. Wet mount of KOH were positive for fungal filaments. No clinical improvement was noticed. There was associated severe conjunctival reaction, chemosis and lid signs on the subsequent days. There was no progression or worsening of ulcers but corneal thinning. Topical medication was reduced in frequency. Ulcer perforated which was managed with pressure bandage and other symptomatic measures. Meanwhile on Sabouraud’s agar culture growth of Fonsecaea pedrosoi was confirmed as slow growing, velvety, spreading olivaceous green colonies with a blackish reverse (Figure 2, 3).

Treatment was modified with addition of Itraconazole topically and Ketoconazole orally twice daily. Patient improved well with newer medications, conjunctival signs decreased, ulcer started healing and the surrounding cornea also cleared very well. At the time of discharge his visual acuity was 6/12 LE – 6/9 RE with corneal thinning and pigmentation over the healed ulcer (Figure 4).

Patient was put on Natamycin and Itraconazole and oral Ketoconazole, and was kept under regular follow up. He improved during follow up. Last visit showed pigmented macular corneal opacity with visual acuity of 6/12 LE and normal surrounding cornea.

Discussion:

Fonsecaea Pedrosoi is a dermataceous fungi isolated in 1913 as an etiological agent for chromoblastomycosis. Very few cases of corneal chromoblastomycosis were reported in literature. Usually presents as features of fungal corneal ulcer. Treatment of this type of corneal ulcer is a challenging problem not only because of the non-availability of good penetrated antifungals but also due to need for prolonged treatment. Common antifungal medication sensitive to this organisms are Amphoterin B, Flucytosine, Itraconazole, Ketoconazole, Voriconazole. In view of the resistance of Fonsecaea Pedrosoi to the current antimycotic agents it is better to consider the possibility of combining the best available drugs in order to obtain a synergic effect.

Combination of systemic and topical antifungal may provide best option for cure in corneal chromoblastomycosis.

References:


Sir Harold Ridley
Compiled by: Dr Dhiresh K.

“A Pioneer in the quest to eradicate world blindness”, Sir Nicholas Harold Ridley was described by World Health Organization in 2003 in their bulletin two years after his death.

Sir Nicholas Harold Ridley an English Ophthalmologist was born on 10th July 1906, in Leicestershire to Nicholas Ridley and Margaret. He had a younger brother Olden. In his childhood Ridley had a stammer which he largely managed to cure.

He was educated at Charterhouse School before studying at Pembroke college, Cambridge from 1924 -1927. He completed his medical training in 1930 at St.Thomas Hospital. He worked as a surgeon at Moorfield eye hospital and St Thomas Hospital in London, specializing in Ophthalmology. In 1938 Ridley was appointed full surgeon and consultant at Moorfield eye hospital.

While working with Royal Air Force casualties during world war II, Ridley noticed that when splinters of perspex from aircraft cockpit canopies became lodged in the eyes of wounded pilots they did not trigger rejection, leading him to propose the use of intraocular lenses. He implanted first intraocular lens on 29th November 1949 at St Thomas Hospital, London. His implantation, inarguably one of the most important innovation in the history of ophthalmology and a blessing to society was widely criticized. He worked hard to overcome complication and had refined the technique by the late 1960s.

Ridley had a special and sincere interest in the field of tropical ophthalmology. His father was an ophthalmologist in British Royal Navy of the 20th century, a position providing his family with worldwide outlook. His wife was raised in India. He led important research into Onchocerchiasis when he was stationed in Ghana for part of his war service in World War II. His work in Africa ranks among most important of his non-intraocular lens contributions. He not only performed clinical examinations on affected patients but also completed histopathological analysis of the microorganism causing the disease. He himself painted a figure showing this disease fundus, now universally termed as Ridley fundus.

He was a fellow of the Royal College of surgeons and a fellow of the Royal society. In February 2000 he was knighted by Queen Elizabeth at Buckingham palace in London.

INTRODUCTION

Accurate measurement of intraocular pressure is a fundamental prerequisite in any ophthalmic examination. Over the last five decades, Goldmann Applanation Tonometer (GAT) reigned as the “gold standard” and became the standard for the routine measurement of intraocular pressure yielding results with largely low intra and interobserver variability. However, the accuracy of GAT depends on many factors like corneal thickness, corneal curvature, corneal structure and axial length (1). This is due to the fact that Goldmann simplified the eye to be a perfectly spherical, dry, flexible and infinitely thin while in reality the human eye and cornea fulfill none of these criteria. The shortcomings of the GAT were more clearly revealed in the Ocular Hypertension Treatment Study (OHTS) (2). While it is accepted that central corneal thickness (CCT) must be recorded along with intraocular pressure (IOP) as part of glaucoma work up( 3), no reliable nomogram is yet available to convert GAT readings and CCT into true IOP (4 - 6).

DYNAMIC CONTOUR TONOMETER

To address the shortcomings of GAT, the Pascal Dynamic Contour Tonometer (DCT) was developed. This tonometer was named in honour of Blaise Pascal a 17th century mathematician and physician who formulated the “Pascal’s Law of Pressure.”

PRINCIPLES OF DYNAMIC CONTOUR TONOMETRY

In Pascal Dynamic Contour Tonometer (Fig 1), the so called contour – matched tonometer tip has a concave surface (Fig 2) that allows the cornea to acquire the shape that it naturally assumes when the pressure on both sides of the cornea is equal and distortion is minimal. Exposing a miniaturized pressure sensor closely to the contour of such
The cornea is thought to measure IOP directly without systematic errors resulting from force – to – pressure translation. The contour surface is devised in such a way to generate minimum distortion of the cornea and to direct all forces acting within the cornea to the pressure sensor surface.

A contact surface which matches the contour of the cornea is shown to have the desired property of creating equilibrium between capillary force, rigidity force, appositional force and force exercised on the cornea by the IOP. Therefore, the pressure sensor integrated into the contoured surface will measure IOP with no systematic errors introduced by such forces or by changes in corneal properties.

**MECHANICAL AND GEOMETRICAL CALCULATIONS IN DCT**

The cornea may be considered as a spherical shell constructed from a material that resists stretching but is fairly flexible to bending deformation. The contour tonometer features a cylindrical tip with a concave contact surface. Once contour matching has taken place, the four major effects acting on the contact surface and on the cornea – capillary force, rigidity force, appositional force and force exercised on the cornea by the IOP – are at equilibrium (Fig 3).

\[ F_{iop} + F_c + F_r + F_{ap} = 0 \]

where:

- \( F_{iop} \) is the force exerted by the effective IOP, acting on the tonometer’s contact surface.
- \( F_c \) is the capillary force of adhesion created within the tear film, caused by the negative capillary pressure within the tear film.
- \( F_r \) is the rigidity force responding to the deformation of the cornea.
- \( F_{ap} \) is the appositional force applied externally to the tonometer.
The two forces with negative signs, $F_c$ and $F_{ap}$, attract tip and cornea, whereas the forces with positive signs $F_{iop}$ and $F_r$ push the tip and cornea away from each other.

Strictly speaking, each individual cornea would require a custom – made contour matched tip to fulfill this contour – matching condition. However, the mathematical and geometrical formulae evolved show that a “standard contour” is acceptable which permits faithful measurement of true IOP for a wide range of corneal dimensions. In fact, the condition for a precision measurement is fulfilled as long as the contact area is larger than the pressure sensing element and the outer diameter of the tear film annulus is smaller than the diameter of the tip itself. In contour tonometry it is not necessary to know the diameter of contact or the appositional force. This makes the method much less susceptible to operator bias or error (7).

The results obtained by DCT are not affected by variations in corneal radius, curvature, astigmatism, central corneal thickness, hydration and rigidity. The only variable modulating the intraocular pressure recorded by DCT is the ocular pulse amplitude (OPA). The cardiac cycle causes a volume displacement of the choroidal bed and hence a pulsatile change giving rise to the OPA (Fig 4). Precise determination of the OPA is possible with DCT, since it records 100 samples per second and is capable of detecting static pressure as well as dynamic pressure fluctuations on a time scale from approximately 0.1 to 10 seconds.

**TAKING MEASUREMENTS WITH DCT**

The Pascal DCT is slit lamp mounted and operated in fashion similar to the GAT.

1. Set the patient on the slit lamp. Have the patient blink a few times and then looking from the side, move the sensor tip close to the patient’s eye and align the sensor tip on the apex of the cornea.

2. Switch Pascal unit on by turning the blue knob gently in the clockwise direction. Wait for the LCD to activate before the unit touches the eye.

3. Looking through the left ocular, carefully advance the slit lamp until the surface of the sensor tip touches the cornea. continue advancing until the cantilever is in the approximately upright position.
4 Using the joystick on the slit lamp, adjust the position of the sensor tip slightly until opaque spot enclosing the blue – green square of the pressure sensor is concentric with the contact zone. The circular contact zone is darker the surrounding area. The opaque spot in the center is the pressure sensor obstructing the view of the eye. For proper alignment, the pressure sensor should be centered on the contact zone and not necessarily centered on the pupil (Fig 5)

5 Count approximately five to seven consecutive undisturbed oscillations of sound. (The pitch corresponds to the IOP level) If during measurement the cantilever is not deflected away from its forward position enough, the oscillating sound will be intermittent and irregular. If this occurs, push the joystick slowly towards the patient until the intermittent oscillating sound will become continuous. If the cantilever is deflected back too far, an alert (persistent repetitive beeps) will sound.

6 Swiftly retract the slit lamp and the sensor tip away from the patient’s eye

7 The PASCAL DCT will compute the IOP and OPA from the pressure curve wave recorded and give a numerical display of results (Fig 6)

FIGURE 6 – DISPLAY OF READINGS
AS SHOWN IN THE DCT

<table>
<thead>
<tr>
<th>TECHNICAL SPECIFICATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>IOP Measurement Range</td>
</tr>
<tr>
<td>OPA Measurement Range</td>
</tr>
<tr>
<td>Accuracy</td>
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<tr>
<td>Recording Time</td>
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<tr>
<td>Mounting</td>
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<td></td>
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<tr>
<td>Result display</td>
</tr>
<tr>
<td>Appositional force</td>
</tr>
<tr>
<td>Sensor Tip Diameter</td>
</tr>
<tr>
<td>Tip cleaning / sterilization</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Contact surface</td>
</tr>
<tr>
<td>Power</td>
</tr>
</tbody>
</table>

PERTINENT FEATURES OF DCT

All functions in the DCT are assessed with the unique blue knob which is an easy, single button
operation. The Pascal DCT employs disposable tip covers to avoid contamination and infection hazards. The tip is transparent for visual control of the corneal interface. Numerical display of results avoids operator bias and reading errors. Audio feedback helps operator to record high quality data. Each measurement comes with a quality score which helps in avoiding erroneous readings due to poor quality. The instrument is self-calibrating and battery operated. No fluorescein staining is required.

The DCT measures the ocular pulse amplitude which in several studies has been found to be reduced in patients with normal tension glaucoma.\(^8\) The measurement of ocular pulse amplitude provides an indication of ocular blood flow and its influence on various ocular pathologies.\(^9\)

**STUDIES COMPARING DCT AND GAT**

In a study, Intraocular pressure obtained by DCT was 1.7 mm Hg higher than the readings

<table>
<thead>
<tr>
<th>APPLANTION TONOMETER</th>
<th>CONTOUR MATCHING</th>
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<tbody>
<tr>
<td>Electricity operated</td>
<td>Battery operated</td>
</tr>
<tr>
<td>Needs periodical calibration</td>
<td>Self calibrating</td>
</tr>
<tr>
<td>Applanation tip needs disinfection</td>
<td>Disposable sensor tip covers used</td>
</tr>
<tr>
<td>Cornea is distorted in process of measurement</td>
<td>Cornea is tension free</td>
</tr>
<tr>
<td>Force measurement done (Pressure measured indirectly)</td>
<td>Direct pressure measurement</td>
</tr>
<tr>
<td>Force dependant</td>
<td>Force independent</td>
</tr>
<tr>
<td>Systematic errors due to force – to – pressure translation</td>
<td>No systematic errors due to force – to – pressure translation</td>
</tr>
<tr>
<td>Static recording of IOP</td>
<td>Dynamic recording of IOP</td>
</tr>
<tr>
<td>Applanation area is critical</td>
<td>Contact area independent</td>
</tr>
<tr>
<td>IOP estimate arrived at</td>
<td>IOP measurement arrived ate</td>
</tr>
<tr>
<td>Mechanical end point</td>
<td>Electronic, digital end point</td>
</tr>
<tr>
<td>Flourescein needed</td>
<td>No flourescein needed</td>
</tr>
<tr>
<td>No feedback on quality of measurement</td>
<td>Audio feedback available</td>
</tr>
<tr>
<td>Operator bias</td>
<td>No operator bias</td>
</tr>
<tr>
<td>Accuracy of readings affected by corneal properties like curvature or CCT</td>
<td>Accuracy of readings not affected by corneal properties like curvature or CCT</td>
</tr>
</tbody>
</table>
obtained by GAT.10 This is in good agreement with studies that found intraocular pressure readings by applanation tonometry to be 1.2 – 2 mm Hg lower than the true IOP, as measured manometrically in human eyes in vivo.11 Hence the higher readings obtained by DCT compared with GAT readings are not unexpected because DCT is calibrated against a manometrically controlled pressure standard rather than a GAT pressure reading.

**DCT MEASUREMENT IN LASIK**

DCT records intraocular pressure accurately independent of corneal thickness in patients before and after corneal refractive surgery (LASIK) (12).

**DISADVANTAGES OF DCT**

Despite its introduction, DCT is yet to be used widely due to the cost factor. Measuring IOP with the DCT requires the tip of the tonometer to rest on the patient’s cornea for approximately 5 seconds. This is slightly longer than the contact time that an experienced examiner would require with the GAT. Hence accurate assessment may be difficult in uncooperative patients. However, the acoustic signal of the DCT that informs the examiner about the correct alignment of the tonometer tip encourages the patients to remain still for the time needed.

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Identify the condition from the colour fundus photograph and optical coherence tomography picture

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Randomised Controlled Trial of Posterior Sub-tenon Triamcinolone as adjunct to Panretinal Photocoagulation for treatment of Diabetic Retinopathy.


The study was designed to evaluate the efficacy of a single posterior subtenon capsule injection of triamcinolone acetonide before panretinal photococoagulation [PRP]. It was a randomised, contralateral eye controlled, open label, parallel group study.

BCVA, slit lamp biomicroscopy, applanation tonometry, fundus examination, fluorescein angiography, OCT at baseline were recorded. Type 1 or 2 diabetes was eligible. Selected patients had to have severe NPDR or PDR with clear ocular media and no other disease in either eye. Those who had history of panretinal or focal photocoagulation, previous vitrectomy, presence of vitreous haemorrhage, signs of vitreomacular traction, history of periocular or intraocular steroids within the past 6 months were excluded. Eyes were allocated to one of the 2 groups, PSTA or no injection. 82 eyes of 41 patients with symmetrical severe NPDR or PDR were enrolled. Of the 41.98% [40] completed the study.

Posterior sub-tenons injection of 20 mg triamcinolone acetonide in a volume of 0.5ml was injected in the inferotemporal quadrant under topical anaesthesia 1 week before the first PRP session. PRP done 4 times, at 2 week intervals in both eyes.

The mean changes in BCVA at 6 months compared with the baseline were a worsening of 0.010 in the control group and an improvement of 0.072 in the PSTA group [p 0.04]. In the control group the mean logMAR BCVA tended to drop from the baseline to 1 month, rise slightly from 1 month to 3 months and drop again from 3 months to 6 months. The mean changes in foveal thickness at 6 months compared with baseline were an increase of 32.8micrometer in the control group and a lessening of 9.7 micrometer in the PSTA group [ p-0.03]. In summary visual acuity and retinal thickness in the PSTA group tended to be superior to those in the control group, without regard to CSMO. Hence it is possible that PSTA prevents PRP induced macular thickening and hence less visual loss. The progression of logMAR BCVA and retinal thickness of the 2 groups differed widely until the 3 month examination. Hence advantage of PSTA is the control of visual decline and retinal thickening until a few weeks after PRP completion.

Intravitreal Bevacizumab Treatment for choroidal Neovascularization in Pathologic Myopia: 12- month Results


The study was designed to evaluate the short term efficacy and safety of intravitreal bevacizumab for the treatment of myopic choroidal neovascularisation. It was a prospective, non-randomised, interventional case series. 20 EYES from 20 patients with myopic CNV were consecutively enrolled. Pathological Myopia was defined as a spherical equivalent greater than −6.0 dioptres or axial length more than 26.5mm. CNV was classified as juxtafoveal if the lesion was closer than 200 microns but not under the geometric centre of the FAZ.

Standardised refraction protocol was followed. Complete ophthalmic examination was done. Foveal centre thickness was measured for all eyes using OCT. Leakage from the CNV was evaluated on FA by a trained photographer blinded to the study. Recurrence was defined as evidence of
leakage from a previously closed CNV. 1.25 mg of bevacizumab was injected with a 30 G needle at 3.5 to 4mm of inferotemporal limbus. All patients were scheduled for monthly intravitreal injections for 3 months. Follow up visits at 4 week intervals. Monthly additional injections were given until absence of any leak in FA or absence of any fluid on OCT.

At baseline the mean BCVA was 24.8 +/- 11.86 letters. At 12 months after treatment the mean BCVA was 43 +/- 12.38 letters. This improvement in visual acuity resulted highly significant p=0.00001. At 12 months BCVA improved 10 letters or more in 18 out of 20 eyes and improved 15 letters or more in 14 out of 20 treated eyes. No treated eyes experienced a worsening of BCVA from baseline. The mean FCT at baseline was 223 +/- 47.43 microns. At 12 months the mean FCT reduced to 207 +/- 50.87 microns. Reduction in FCT was not statistically significant.

All patients received 3 scheduled monthly injections. 8 out of 20 treated eyes required more than 3 injections. Monthly additional injections were performed until absence of leakage from the cnv and absence of any fluid collection on OCT were obtained. Patients younger than 50 years experienced a significantly better visual outcome after treatment BCVA. Outcome was less favorable in eyes that required more than 3 injections. At month 12 mean BCVA change from baseline in eyes that underwent 3 injections was 21.5 +/- 9.42 letters while in eyes that required more than 3 injections it was 13.25 +/- 6.56 letters[p=0.03] None of the patients experienced systemic complications related to intravitreal bevacizumab.

Visual improvement might result not only from the drying up of fluid collections around the lesion but also from an actual reduction of CNV size because of the known fibrotic changes associated with closure. A significant correlation between ageing and need for additional injections was found. And less favorable visual outcome in these eyes was also found. In conclusion intravitreal VEGF inhibitors provide new treatment strategies in treating CNV associated with pathologic myopia.

Viscocanalostomy versus Trabeculectomy for Primary Open Angle Glaucoma: 4- Year Prospective randomised Clinical Trial.

Gilmour DF, Manners TD, Devonport H, Varga Z, Solebo AL, Miles J. Eye 2009; 23(9):1802-7

In this randomized clinical trial, the authors compare the effectiveness and safety of viscocanalostomy with trabeculectomy in the management of primary angle glaucoma.

Patients were selected from the ophthalmology outpatient clinic at York District Hospital. 50 eyes of 43 patients were enrolled from March 2000 to Feb 2004. Patients were divided into 2 groups. First group underwent trabeculectomy and second group viscocanalostomy. All surgeries performed by a single surgeon and followed up prospectively. Patients were examined preoperatively, at day 1, day 3 if required, day 6, week 2 and thereafter as near as possible to 1,3,6,12,18, 24, 30, 36, 48, 54, and 60 months. IOP were recorded, presence or absence of any complications, presence and description of bleb, visual acuity with glasses, and full examinations as routine to monitor any progression of the glaucoma. Bleb interventions including needling and antimetabolites were allowed and recorded in both groups.

Mean follow up was 40 months (SD 15), with a range from 6 to 60 months. Forty two percent (n=10) of the patients in the trabeculectomy group had a successful outcome (IOP less than 18 mm of Hg with no treatment) at last follow up visit, compared to 21% in the viscocanalostomy group. IOP was lower in the trab group with differences in IOP being statistically significant at month 12 (P=<0.001), 24 (P=<0.001), 30 (P=0.030), 36 (P=<0.001), and 48 (P=0.018). The trabeculectomy group required less postoperative topical IOP lowering medication (P=0.011).

The authors conclude by saying that trabeculectomy is more effective at lowering IOP than viscocanalostomy in POAG patients.

Compiled by Dr Seema KM, Dr Sharmila CP.
Little flower Hospital. Angamally
Uveitis – Text And Imaging

Edited by: Dr Amod Gupta, Dr Vishali Gupta, Dr Carl P Herbor, Dr M. Moncef Khairallah

This 830 pages book with 71 contributors from major eye centers of India as well as different parts of the world comprises 3 parts.

Part-I covers imaging techniques including Slit Lamp photography, Laser, Flare photometry, Angiography (Fluorescein as well as Indocyanine Green) of anterior segment and posterior segment, Ultrasound B-Scan and Biomicroscopy, Optical Coherence Tomography (time domain as well as spectral domain), Confocal Microscopy, electrophysiology, Visual Field Test in Uveitis and systemic examination and imaging in Uveitis.

Part-II covers specific Uveitis entities and includes chapters on infective, parasitic, vasculitic and inflammatory uveitis, chorio capillaropathies, specific granulomatous uveitis and masquerade syndromes. This section has covered important entities in great detail and has also included various conditions prevalent in all the different parts of the world.

Part-III covers the complications associated with Uveitis and their surgical management as well as certain surgical techniques used for diagnostic purpose such as vitreous biopsy and chorio retinal biopsy. The book is well organized and has been extensively illustrated with black and white as well as colour photographs of good quality, with a few instances of repetition of texts. Though there is no separate chapter on pharmacotherapeutic agents used in uveitis, various chapters in Part-II of the book have included the current therapeutic agents. On the whole it is well written and illustrated book which deserves to be part of any ophthalmology library and will be of immense value to Post Graduate students as well practicing Ophthalmologists who handle Uveitis cases.

Compiled by Dr. Thomas Thachil, Giridhar Eye Institute
CURRENT MANAGEMENT STRATEGIES OF NEOVASCULAR GLAUCOMA

- Neovascular Glaucoma is a potentially devastating secondary form of glaucoma that results from the growth of fibrovascular membrane over the trabecular meshwork in the anterior chamber angle.
- The delayed diagnosis or poor management can result in complete loss of vision or loss of the eye. The current impetus is therefore on early diagnosis followed by immediate and aggressive treatment.
- The availability of effective anti-VEGF drugs, better cyclo-destructive procedures and newer glaucoma implants has brought about a drastic change in the management of neovascular glaucoma.
- Most common causes of Neovascular Glaucoma are: Retinal Ischaemic Disorders – 97%, (Diabetic Retinopathy CRVO (common) irrespective of aetiology), extra ocular vascular disorders, carotid artery occlusive disease, ocular tumors, irradiation, post surgical, inflammatory disease, miscellaneous. The risk of NVG in CRVO is 16% ranging from 10% in perfused eyes to 40% in non-perfused eyes.
- The various causes of Neovascular Glaucoma follow a common path.
All causes result in retinal hypoxia which releases various angiogenic substances viz. VEGF, FGF, into the vitreous. This diffuses from the posterior to the anterior chamber and results in new vessel formation at the angle and pupil.

**WORK-UP OF NEOVASCULAR GLAUCOMA**

- High index of suspicion is required for proper diagnosis.
- In all patients, visual acuity, pupil with RAPD more than 0.7 log units and slit lamp examination to rule out rubeosis along with undilated gonioscopy should be performed in the follow-up of cases with a risk of neovascular glaucoma.
- 6-12% of eyes have neovascularization of the angles (NVA) with neovascularization of the iris (NVI).
- The hallmark of neovascular glaucoma is elevated intraocular pressure + neovascularization of iris/neovascularization of angles.

**AIM OF TREATMENT**

Aim of treatment is lowering the elevated intraocular pressure, treatment of retinal ischaemia, maintaining visual acuity, prevention of painful blind eye and regression of NVI/NVA.

**MANAGEMENT OF NEOVASCULAR GLAUCOMA**

This includes topical steroids, cycloplegics usually Atropine and anti-glaucoma medications preferably beta-blockers and carbonic anhydrase inhibitors. Prostaglandins and pilocarpine are not a drug of choice. Following are the flow charts for management of neovascular glaucoma in patients with and without useful vision.

Various drainage implants can be used if multiple trabeculectomy augmented (with MMC + Injection Intravitreal Bevacizumab) fails. Intravitreal and intracameral injection Bevacizumab have been used widely in the treatment of neovascular glaucoma.

With currently available modalities, different forms of laser, newer anti-VEGF treatment, better cyclo-destructive procedures it should be possible to maintain useful vision and prevent the loss of globe in most patients with neovascular glaucoma.

Compiled by Dr Savita Bhat
Consultant, Giridhar Eye Institute, Cochin
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.

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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: Author(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 copyright 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.

11. All manuscripts are subjected to editorial board review.

12. Other Categories of Manuscript
   a) Original Articles should generally not exceed 3,000 words or 12 double-spaced pages.
   b) Review Articles: can be on topics of relevance to clinical practice, research methodology, community ophthalmology or investigative work, of relevance to visual science. These articles should include up to date review of existing literature, and summarize the current status / preferred practice for that particular topic.

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