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UPJO

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Editor: **Dr. Gunjan Prakash**

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What is Where

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Editorial

Dear Friends,

Greetings to you all!

Authorship is now a popular term in medical circles, mainly for those at the edges of their profession. But the question of how and when eye surgeons become authors is a difficult one; and exactly who qualifies, depends on how you define the term. What does it really mean to call an ophthalmologist, also an author?

The meaning of the word 'author' has shifted significantly through history and has been the subject of intense scrutiny over the last 40 years. The earliest definitions are not associated with writing per se, but rather denote 'the person who originates or gives existence to anything'.

Over the centuries, the relationship between the author and the text has changed. The earliest sacred texts are authorless, their origins lost in history. In fact, the ancient, anonymous origin of such texts serves as a kind of authentication. But, scientific texts demand an author's name as validation. Text has become a kind of private property, owned by the author. With the rise of scientific method, on the other hand, scientific texts and mathematical proofs were no longer seen as authored texts but as discovered truths. The ever-present pressure of promotions and name-game only muddies the water further.

In this issue we salute the great personality Charles L Schepens for his unparalleled contribution to the ophthalmic science. And then move on to the intricacies of Pterygium management, Cosmetic Reformations. Some light is thrown on the retinal arterial occlusions and so also on management of traumatic cataract.

Pupil is also enlightened upon and the Quiz is made easier this time as we received only 4 correct entries in the last issue. Practical management of Dry Eye syndrome is very well covered by the authors.

Ophthalmologists-as-author could help us to rethink a process, expand eye treatment methods and elaborate our historical frame to incorporate all forms of ocular methodologies. But while theories of medical authorship may change the way work is made, the primary concern of both the reader and the critic is not who made it, but rather what it depicts and how it depicts it.

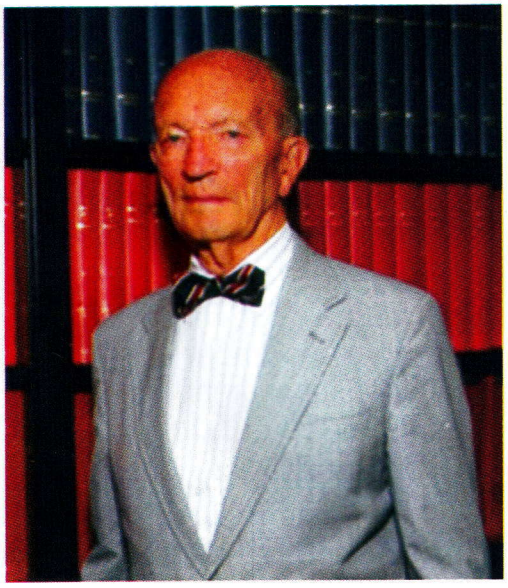
Welcoming you all to our Annual UPSOS Conference at Jhansi from 25th to 27th October, 2013.

Team UPJO

Editor
Dr Gunjan Prakash
MBBS (AIIMS), MD (AIIMS), DNB, MNAMS
B-2/22, Kamla Nagar,
Agra, Uttar Pradesh, India
Mobile : +91 9319204073
Emails: editorupjo@rediffmail.com
gunjan_prakash@rediffmail.com

Charles L. Schepens

Charles L Schepens, long considered one of the giants of 20th Century ophthalmology and the unquestioned leader in retinal detachment surgery. Schepens was born in Moeskroen, Belgium in 1912, his father was a physician. Charles Schepens first obtained a certificate in mathematics at the University of Ghent. He would have used this to enter the engineering school, his preference, but bowing to peer pressure and family tradition, he entered the medical school. He earned his medical degree in 1936 and received surgery training at the City Hospital of Ghent.



Schepens then trained in ophthalmology at Moorfields Eye Hospital in London, England prior to World War II. After the Germans invaded Belgium in 1940, he became a medical officer in the Belgian Air Force. After the fall of Belgium, Schepens escaped to France where he became active in the French Resistance smuggling documents and people over the Pyrenees to Spain during 1942 and 1943. Schepens was twice captured by the Gestapo.

Pushpgiri Vitreo Retinal Institute
Hyderabad

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While at Moorfields, working with Professor Ida Mann, Charles developed a prototype of the **binocular indirect ophthalmoscope**, which for the first time, allowed stereoscopic viewing of the eye fundus out to the peripheral retina.

The impact of Charles Schepens' contributions is immense. Before him no effective way to see the retinal periphery with an ophthalmoscope existed. The invention of the binocular indirect ophthalmoscope revolutionized ophthalmology.

The development of the indirect ophthalmoscope and the scleral buckling procedure brought the success rate for retinal reattachment surgery from 30% to 90%.

Finally, his insistence on assigning an equal value to education, research, and clinical practice pioneered an approach now so widespread that it is taken for granted. His strategy broadened and deepened the knowledge of the retina to a point that it became a subspecialty of its own.

Because of his remarkable leadership he is revered as **"The Father of Modern Retina Surgery."**

Dr. Charles L. Schepens, an eye researcher who developed sight-saving procedures and invented a device for viewing the retina that has, in turn, brought about new treatments to prevent blindness, died on March 28, 2006 at a hospital in Salem, Mass.

Life is a chain. Our bodies fail and eventually have to rest for a very long time, but our beings and our work live on through our family, colleagues and friends - ***Charles L Schepens.***

Shephali Jain

Retinal Artery Occlusion : Ocular Stroke

Five minutes worth of advice from a colleague who knows as much about the topic as anyone in the world. What could be better?

Nisha Chauhan MBBS, Gunjan Prakash MD

Occlusion of central retinal artery or its branches, severe enough to cause ischemia.

Patient profile:
Usually seen in early and mid-60s.
Males are affected slightly more than females. No racial predilection.

Risk Factors:
Diabetes, Hypertension, Lipid disorders, Cardiac disease, Systemic atherosclerotic disease

Mechanism of occlusion:
Younger patients: Thrombotic
>50 years of age: Embolic

A) Embolic: Most common cause (CRAO:1/3rd , BRAO: 2/3rd cases)

Sources of emboli-
Endogenous: Carotid Atheroma (80% cases), Cardiac valvular diseases, Cardiac tumour, Fat emboli, Amniotic fluid emboli, Leukoemboli (in pancreatitis), Septic emboli.

Exogenous: Talc emboli (i.v. drug abuse), injected steroids (nasal or periorbital), fragments of catheter tips or artificial heart valves, blood transfusion products.

Types of emboli:
Cholesterol (Hollenhorst plaque): Small glistening white. Usually carotid origin.
Platelet fibrin: Large, long and gray white.
Calcific: most severe occlusion, Large gray white.

Department of Ophthalmology
Sarojini Naidu Medical College, Agra

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B) Thrombotic: Seen in hypercoagulable states. More common in younger patients.

C) Vasculitic: Most common condition is giant cell arteritis (>55 yrs age, associated with headache, scalp tenderness, jaw claudication, anorexia and fever) Other causes: SLE, RA, Behcet's disease, and localised vasculitis in toxoplasma and bartonella retinitis.

D) Other causes: Reflex vasospasm in migraine, sickle cell haemoglobinopathy and collagen vascular diseases may play a role; elevated IOP; Peripapillary capillary loop and external compression of central retinal artery.

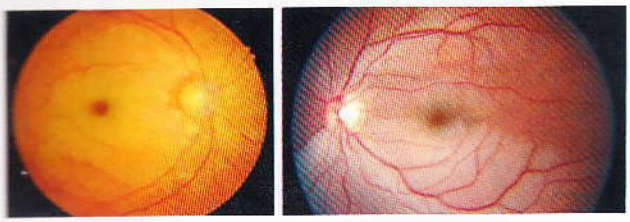
Pathophysiology: Ischemia due to vascular occlusion causes oedema of ganglion cells and they burst, imparting whitening/ opacification to transparent retina.

Presenting complaints:
1) Amaurosis fugax: prodromal symptom
   Transient mono-ocular visual loss
   Lasts 7-30 min
   Total resolution to normal
   Presence mandates complete ophthalmic examination and full systemic workup
2) Acute painless loss of Vision: CRAO
   Loss of Part of Visual field: BRAO
3) BRAO may also be asymptomatic.

Ophthalmic examination:
1) VA: CRAO: <3/60, BRAO: 3/60- 6/6
2) Pupil: CRAO: RAPD, BRAO: ±RAPD
3) Visual field: Field defects corresponding to the part affected
4) Fundus: Changes take 1-2 hrs to become evident clinically

CRAO: Ischemic retinal whitening of whole

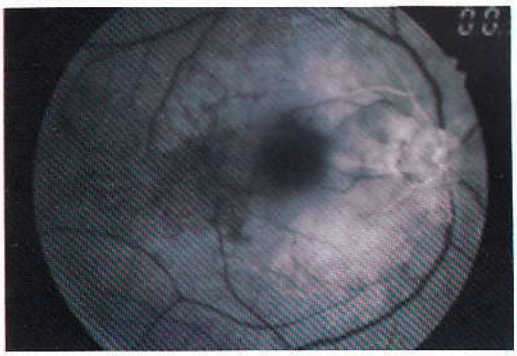
fundus with cherry red spot at fovea (no ganglion cells at fovea, no whitening, evident underlying vascular choroid). Boxcarring in retinal vessels.



BRAO: Retinal whitening in affected part

Less evident in nasal part (single layer ganglion cells).

5) FFA: Intact choroidal flush (c.f. Ocular ischemic syndrome). Absent, delayed or incomplete filling in retinal vessels with leading edge of dye.
6) ERG: Absent b wave (in ocular ischemic syndrome: both a & b wave are absent).



Management:

A) Manage acute occlusive event
- Goal is to restore blood flow.
- Manage aggressively if patients presents within 24 hrs.
- Lower IOP (Target IOP of 15 mm Hg): Digital ocular massage (because of simplicity should be tried in all patients, press globe for 5-15 seconds and sudden release), anterior chamber paracentesis, topical beta blockers, oral acetazolamide.
- Suspicion of giant cell arteritis: Start high dose steroids while biopsy report of temporal artery is awaited (saves other eye)

Other options with doubtful significance
- Medical vasodilatation: Sublingual nitroglycerine (10 mg). Other suggested options are calcium channel blockers, carbogen (inhaled) and pentoxifylline.
- Thrombolytic therapy
- Hyperbaric oxygen

B) Systemic work up
BP, Blood sugar, Lipid profile
Carotid ultrasound: especially older pts.

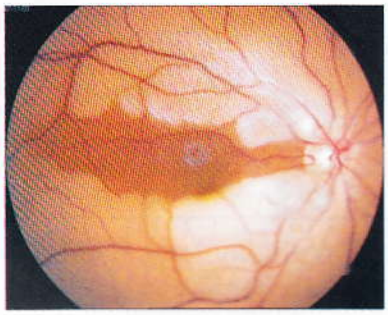
Echocardiography:
CBC, ESR, C - reactive protein, PT, PTT, Protein C, Protein S, Activated protein C, Factor V Leiden, Fasting plasma Homocysteine level, Anti phospholipid antibodies, Hb electrophoresis, VDRL

C) Complications
NVG: occurs in 2-16% cases (mainly CRAO). Most of the cases develop within 2 months. So patients of CRAO should be followed monthly for early diagnosis.

T/t: Anti VEGF, retinal laser photo-coagulation, cyclocryo therapy and anti-glaucoma drugs.

Prognosis
CRAO: Majority patients have final VA of finger counting in resolved phase. May be 6/6 in patients with intact cilioretinal artery.

BRAO: VA 6/12 or better in 80% cases. Fundus may return to near normal after months but field defects persist.



Chronic fundus changes are optic atrophy, attenuated vessels, atrophic retina.

Bilateral Port Wine Stain with Involvement of Chest Dermatomes and Unilateral Buphthalmos: A Case Report

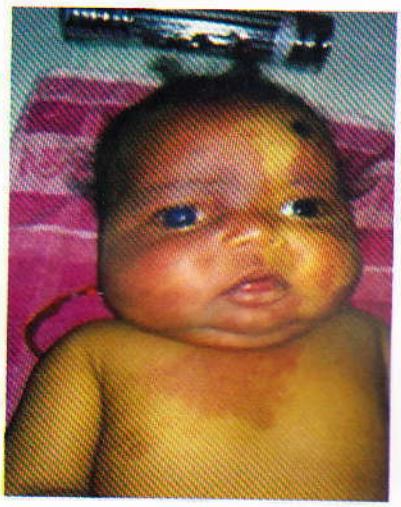
Prashant Bhushan[1] MD, Deepak Mishra[2] MS, M K Singh[1] MBBS, V P Singh[1] MBBS, R P Maurya[3] MBBS

Introduction

Sturge Weber Syndrome (SWS) is a rare Oculo cutaneous disorder, which manifests with facial capillary haemangioma, congenital glaucoma and lepto-meningeal abnormalities which in turn leads to central nervous system (CNS) afflictions like seizures, behavioural and developmental disorders. The CNS involvement is considered a hallmark of SWS.[1] SWS has been reported more commonly with unilateral port wine stains but bilateral presentations with associated chest dermatome involvement is rare.[2] We report a patient one month old with bilateral port wine stain, with buphthalmos and involvement of chest dermatomes, with no evidence of CNS involvement.

Case Report

A two month old male child presented with bilateral facial port wine stains and also in right upper chest area. The infant was a normal, full term, uneventful and unsupervised home delivery in a rural household. The right eye of the patient has a central corneal opacity, which historically has increased in size in the last two months.



The right eye had a horizontal corneal diameter of 14 mm while the vertical diameter is 13 mm; a hand held slit lamp shows the opacity to be stroma deep and presence of haab's stria, the anterior chamber was deep and the iris and pupil appeared normal, fundus details were not visible because of the opacity. The left eye had a horizontal corneal diameter of 12mm, a vertical corneal diameter of 11mm, the cornea was clear and the anterior chamber deep, the iris and pupil had no abnormalities and the optic nerve head as seen by a direct ophthalmoscope appeared normal. The intraocular pressure (IOP) of the right eye by a hand held Perkins tonometer was 22mm and 16 mm in the left eye. The port wine stain was present in the distribution area of the ophthalmic and maxillary division of the trigeminal nerve on both sides of the face; stains were also present on the chest dermatomes on the right side. A CT-scan head was done which was normal. The child was followed up for more than one year, and all developmental milestones were normal.

[1] Deptt. of Ophthalmology, BHU, Varanasi
[2] Sitapur Eye Hospital, Sitapur, UP
[3] SS Hospital, BHU, Varanasi

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A differential diagnosis of Sturge Weber syndrome and other neurocutaneous syndromes were considered. The IOP was lowered using topical beta blockers initially for a week, and later a Trabeculectomy with Mitomycin –C was done in the right eye.

Discussion

Sturge Weber syndrome is a neuro-cutaneous syndrome with variable presentations, with common manifestations like congenital glaucoma, facial and lepto-meningeal angiomas without any definite genetic predisposition. It is found in 1 in 50,000 live births.[3,4] Buphthalmos has been commonly found in patients with bilateral port wine stains and with CNS involvement[5] but in our case it is bilateral port wine stains with no CNS involvement. Onset of seizures below one year of age is more common with bilateral port wine stains and extra facial locations of port wine stains e.g. chest or torso[6] while in our case there has been no evidence of seizure, this indicates that despite presence of large angiomas, the Vascular-steal-phenomenon[7] has not been able to have its effect. The fact that the milestones were normal, also suggests that there has been no CNS involvement. The presence of corneal haziness with raised IOP indicates that glaucoma could be because of maldevelopment of structures of angle of anterior chamber than due to raised episcleral pressure. This view is also supported by the fact that glaucoma is unilateral despite facial angiomas being present of both sides of the face.

Conclusion

Sturge Weber syndrome may not present clinically in its typical form, but may have extensive port wine stains with congenital glaucoma, and no central nervous system involvement.

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In seahorses, it's the male who gives birth to the young.

Decoding the Pupil

Ankur Sinha MD, Ajay Jhinja MD

Pupillary examination is an important part of ocular examination as it often helps in identifying problems which may be local or may even involve central nervous system.

Anatomic and physiologic considerations

The size of the pupillary aperture is controlled by two opposing smooth muscles the pupillary constrictor muscle (innervated by parasympathetic autonomic nerves) and the dilator muscle (innervated by sympathetic autonomic nerves). Iris sphincter is much stronger than dilator.

Assessment of pupillary size, shape and function

Examination of pupil requires a meticulous history and a rigorous examination of pupil, which may often need pharmacologic testing.

History:

Patients with pupillary abnormalities are often not aware of their abnormalities as the symptoms are often insignificant, in most of the cases the spouse, friend or physician brings it to notice. Often, old photographs are required to identify the duration of illness. In some cases symptoms are associated with disturbance of pupil size and shape, like photophobia, blurring or "unclear" vision and difficulty in light/dark adaptation or difficulty in adapting to change in accommodative effort. Past history of pharmacologic use / misuse / accident should also be asked for. Past history of trauma, surgery, infections (Herpes etc) or migraine may point toward the cause of pupillary abnormality.

Max Vision Eye Care Centre
Jaipur

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Slit lamp anterior segment examination:

Ciliary congestion with small pupil may be due to intra-ocular inflammation.

Figure 1: Traumatic mydriasis along with hyphema in a case of trauma.

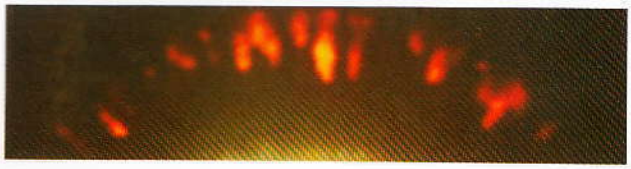

Figure 2: Transillumination defects seen in retro illumination

This also provides information regarding signs of trauma like, corneal abrasion, angle recession, transillumination defects in iris, sphincter tears, segmental defects and signs of inflammation responsible for abnormal pupil.

Figure 3: Pupillary rough irregularity and sphincter tears in case of trauma.

Measurement of pupil size:

The pupil can be measured using pupil

gauges or simple rulers in darkness, light and when the patient is focusing for near.

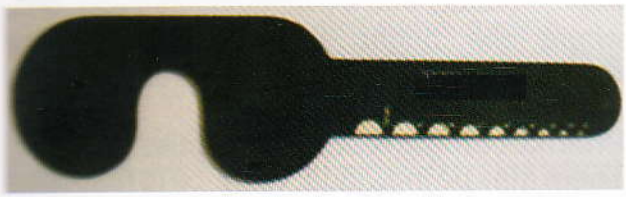

Figure 4: Commonly available pupil gauge.

A difference of more than 0.4mm between the two eyes is labeled as anisocoria and needs further evaluation. Physiologic anisocoria may be seen in 20% of general population.

Figure 5: Pupillary size being measured using pupil gauges held close to the eye.

Reaction to light:

This should be examined in dim quiet surroundings, distance fixation and there should *not* be an attempt to look for near or close the eye (this induces miosis). When the bright light is thrown in the eye being examined for a few seconds, it is the direct light reaction (Figure 6).

Figure 6: Assessment of direct light reaction.

In cases of retinal and optic nerve pathologies the pupil initially constricts and then slowly dilates to original size, "pupillary escape". This is more pronounced with a dim light. On shining light in one eye the pupil of the other eye constricts, this is consensual light response. This can be best assessed using a bright light source in one eye and dimmer light source in the contralateral eye shined obliquely. The consensual reflex is 0.1 mm less than direct reflex.

Figure 7: Assessment of consensual reflex.

Near response:

The near triad consists of pupillary constriction, accommodation and convergence. To be tested in adequately lighted room and the patient is asked to look at the accommodative target.

Assessment of Pupillary dilatation:

Pupils dilate after constriction due to light or near response, with sudden noise or pinching the back. While assessing the dilation, note "dilatation lag", which is seen commonly in defective sympathetic pathway. In such cases anisocoria is more at 4-5 seconds after constriction than 15 seconds post constriction. Rarely such a phenomenon may be seen in normal individuals.

Light near dissociation:

There is dissociation between the light response and near stimulation. In almost all cases there is impaired pupillary response to light whereas the response to near stimulation is present. In cases where the light response is present but the near response is absent, it is most likely due to lack of accommodative effort on patients part.

Argyll Robertson Pupils:
Here the pupils are very small, associated with normal pupillary constriction for accommodation, but poor response to light. This was seen more often in the past and was considered pathognomonic of neurosyphilis.

Testing for RAPD:
This can be demonstrated with alternate cover of either eye, as described by Kestenbaum. In cases with optic atrophy or asymmetrical optic atrophy, alternate cover of eyes reveal that on uncovering the normal eye or on covering the normal eye the pupil of abnormal eye dilates.

This is "Marcus Gunn or Gunn phenomenon" or "relative afferent pupillary defect". This can also be tested with a swinging flash light test, where the bright light accentuates the differences in the pupillary response.

Pharmacologic testing of pupils:
There may be individual variation in response to the pharmacologically instilled drug (due to squeezing, tearing, corneal penetration, and iris color); hence if the condition is unilateral then the drug should be instilled in both eyes so that the other eye acts as a control. When the disease is bilateral, the drop should be instilled in one eye so that the response can be compared with the other eye.

Table 1: Drugs used in pharmacologic testing for common pupillary disorders

Drug	Purpose	Dose	Time	Lighting	Measure
Dilute pilocarpine	Supersensitivity testing	0.625%	30 min	Dim light or darkness	Change in pupil diameter
Pilocarpine	Pharmacologic pupil blockage	2%	40 min	Darkness	Change in pupil diameter
Cocaine	Sympathetic defect	10%	60 min	Light	Post –cocaine anisocoria
Hydroxyampheta-mine	Detect post ganglionic sym-pathetic defect		50-60 min	Light	Absolute dilation OU or anisocoria > 1mm or change in anisocoria >1mm

Anisocoria:

Table 2: Common causes of Anisocoria

	More Anisocoria in Darkness
1	Simple (physiologic)
2	Inhibition of sympathetic pathway a. Horner's Syndrome b. Pharmacologic (thymoxamine, dapiprazole)
3	Stimulation of sympathetic pathway a. Tadpole pupils b. Intermittent dilatation caused by intermittent sympathetic hyperactivity c. Pharmacologic (adrenergic, ocular decongestants, cocaine)
4	Pharmacologic stimulation of parasympathetic pathway (pilocarpine, serine, methacholine, organophosphates etc)

More Anisocoria in Light	
1	Damage to parasympathetic outflow to the iris sphincter muscle a. Oculomotor nerve paresis b. Tonic pupil syndromes (including Adie's) c. Intermittent dilatation of one pupil caused by inhibition of the parasympathetic pathway
2	Trauma to iris sphincter
3	Acute glaucoma, siderosis
4	Pharmacologic inhibition of parasympathetic pathway (atropine, scopolamine)

Horner's syndrome:
It is caused by the interruption of sympathetic supply which causes the weakening of the retractor muscle (drooping of eye lid and lower lid to rise) & the iris dilator is weakened (small pupil). The Anisocoria is more in dark, as the normal pupil dilates well and early. Clinical features include ptosis, miosis, anhydrosis (seen only in central and preganglionic types of Horner's syndrome), apparent enophthalmos, paradoxical pupillary dilatation at the time of emotional excitement due to denervation super-sensitivity, dilatation lag, depigmentation of iris (seen in congenital Horner's).

The diagnosis of Horner's syndrome can be made using cocaine test (as described in table). Clinically, it may be useful to differentiate into central (first-order), preganglionic (second-order) and post ganglionic (third-order) variety. The actual anisocoria in cases of Horner's syndrome varies and may be affected by following conditions.

Table 3: Various conditions affecting the anisocoria of Horner's syndrome

1 Resting pupil size	2 Whether the injury is complete or not?
3 Adrenergic circulation in the blood	4 Re-innervation extent of the dilator muscle
5 Point of fixation of patient (distance or near)	6 Extent of denervation supersensitivity
7 Difference in the brightness of ambient light and examiner's light source	8 Degree of alertness or tiredness of the patient at the time of examination

Central Horner's Syndrome:
The Horner's due to damage to the central neuron is almost always unilateral and is often associated with other neurological deficits.

Preganglionic Horner's Syndrome:
This is a disorder of second order neuron in thorax and superior cervical region. Thecharacteristic anhydrosis is in the half of head, face, neck down to clavicle. The cause of this form of Horner's syndrome may be malignancy (lung/breast), accident or surgery.

Case:
A12 year old boy, presented with history of drooping right eye lid and absence of sweating in right side of face and neck for 2 years.

Figure 8: A case of 12 year old boy with mild ptosis and right pupil smaller than the left. The anisocoria increases in dark.

Ophthalmic evaluation revealed anisocoria more in dark with right pupil smaller than the left (figure 8), rest of the examination was unremarkable. He was advised MRI upper thorax, CT brain and Ultrasound Abdomen. MRI upper thorax revealed a large mass lesion (Figure 9).

Figue 9: MRI thorax showing a large 4.8 x 4.6 x 4.4 cm mass in right paraspinal area (D1-D3), in the apical region close to trachea and main vessels.

The patient underwent cardiothoracic surgery and the mass was histo-pathologically found as benign nerve sheath tumor (Schwannoma) with cystic changes. The photographs of the same patient before and after surgery show improvement in ptosis and decrease in anisocoria (Figure 10).

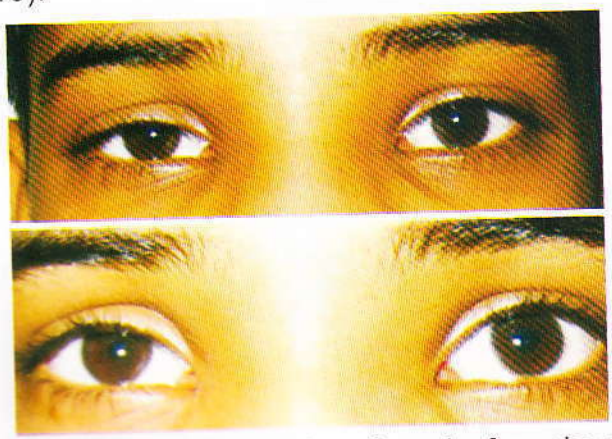

Figure 10: Before (above) and after chest surgery (below), the improvement in ptosis and decrease in anisocoria is evident.

Postganglionic Horner's Syndrome:
The is seen with involvement of third order neuron which travels "the Carotid plexus" and enters the orbit with nasocilliary nerve and divides into two long ciliary nerves and supply anterior segment of the eye and innervate the iris dilator muscle.

Pharmacologic causes of anisocoria:
Anisocoria could be caused by either stimulation of iris sphincter or stimulation of dilator muscles of the eye. Cocaine, oxymetazoline, phenylephrine may dilate pupil, but their effect is very mild and the anisocoria would be more evident in dark.

Anisocoria more in light:
In these cases the anisocoria is more in the light, this signifies the dilated or the larger pupil is abnormal and poorly constricts in response to bright light (Table 2).

Damage to parasympathetic outflow or iris sphincter:
Lesion anywhere in this pathway from the brain to the iris can produce absolute/partial paralysis of pupillary constrictor, which can result in dilated and non-reactive pupil. The pupillary constrictor paralysis may be associated with loss of accommodation in some cases.

Tonic Pupils:
Damage to ciliary ganglion or its roots may lead to poor light reaction, Paresis of accommodation, cholinergic super sensitivity, strong & tonic near reaction and slow re-dilatation this is termed as Tonic pupil. It can be of various types.

Local Tonic Pupil:
This may be caused by a variety of inflammatory, infectious and infiltrative process of ciliary ganglion which may be in isolation or as part of systemic process.

Neuropathic Tonic Pupil:
Usually a part of generalized neuropathy which may involve the ciliary ganglion or the short ciliary nerves or both.

Holmes-Adie (Adie's) Syndrome:
It consists of unilateral or bilateral tonically reacting pupils developing in otherwise healthy persons. This is more common as a unilateral disease in females between the age group of 20 to 50 years. The tonic pupils may be associated with Areflexia or hyporeflexia of deep tendon reflexes.

Patients may complain of photophobia, blurred near vision, enlarged pupil and headache. In most patients, accommodative paresis resolves over a few days to months. In few patients, aberrant regeneration within ciliary muscle- accommodative paresis that may persist until presbyopia develops and diminishes the symptoms. Segmental contraction or "vermilliform" movements are observed in all forms of tonic pupil which is a critical diagnostic observation. Almost every Adie's pupil that has any reaction to light (90%) has such segmental palsy of sphincter.

With time accommodative paresis sets and affected pupil gradually become smaller, the pupillary light reaction may even weaken, deep tendon reflexes tend to become increasingly hyporeflexic and there is tendency for patients with unilateral Adie's syndrome to develop a tonic pupil in opposite eye with time. The diagnosis is made with the use of pharmacological test with 0.1% pilocarpine. The cause of Adie's syndrome is obscure. Pharmacologic and pathologic studies indicate ciliary ganglion, short-ciliary nerves, or both as the location of lesion.

Case:
A 48 year old male presented with difficulty in reading and opening the left eye in light for 20 days. On examination, left pupil was larger than the right. The anisocoria increased in bright light and decreased in dark. On asking the patient to read continuously for 5 minutes, his symptoms improved and also the anisocoria decreased. Vermilliform movements of pupil were also noted.

Diagnosis of Adie's pupil was made and advised asymmetric near glasses and photochromatic glasses for distance.

Damage to iris sphincter:
Blunt trauma to the eyes may result in tears in the iris sphincter which may lead to dilated pupil. In such cases other tell-tale signs of trauma like Vossius ring, traumatic cataract, choroidal rupture, commotio retinae, retinal hemorrhages, angle recession etc may be seen.

Pharmacologic blockade (parasympatholytics):
The mydriasis due to drugs is extreme (usually >8mm) and may respond poorly to 1% pilocarpine solution.

Few Facts

- *Octopus has rectangular pupils.*
- *Mosquitoes like the scent of estrogen, hence, women get bitten by mosquitoes more often than men do.*
- *A person could grow 600 complete eyelashes in a life time.*

Retro-Orbital Injection of Fat Autograft in Post-Traumatic Enophthalmos

H K Bist MS, S K Satsangi MS, Manmohan Gupta MS

Introduction

- Retro-orbital fat volume reduction has been reported in patients with enophthalmos followed by trauma which could be restored by a suitable fat autograft.
- Three main steps are:

1. Harvesting of auto graft fat from donor site.
2. Transfer and purification(Refinement) of graft.
3. Placement of graft in retro-orbital space.

Case report

Thirty two year old male presented with complaints of recession of left eye ball since 2 years after trauma to the eye by wooden stick. On examination the left eye was recessed by 6 mm as measured by Hertel's exophthalmometry in comparison to right eye. Patient had no light perception in left eye. Total white cataract and posterior synechiae were present on evaluation. Retinal detachment was diagnosed on B scan in left eye. Right eye was normal.

Intervention

- Under general anaesthesia, tumescent fluid (solution of 1 mg of epinephrine, 200 mg of lidocaine, and 5 mEq of sodium bicarbonate in 1 L of normal saline) was injected at donor site.
- Autogenous fat was harvested from thigh using a 4mm diameter liposuction cannula.
- The graft was refined by 2000 rpm centrifuge for 3 minute, with 10 minute standing.
- The extremely small parcels of fat were injected by 14-gauge needle into multiple layers of intramuscular cone in retro-bulbar space.
- The volume of fat injected was 7 ml.

Outcome

- The volume of fat injected was 7 ml, with increase in exophthalmometry measurements 6mm immediate after surgery and 5 mm after 6 months. Final recession was only 1mm as compared to right eye after 6 month.
- There were no embolic complications. The procedure was well tolerated by the patient systemically.

Complication

Edema, ecchymosis and under correction was noted. Edema and ecchymosis was resolved in 2 weeks. Under correction of 1 mm was well accepted by the patient.

Conclusion

- Retro-orbital injection of fat autograft allows sustained correction of persistent post-traumatic enophthalmos.
- Two main limitation of refinement process of fat grafting are :

  a) Poor resorption if not properly refined as in standing method because of decreased surface area between graft and recipient.

  b) Adipocyte cell death if refined by centrifuged method.
- Understanding how different handling techniques affect adipocyte survival without compromising the refinement is crucial to optimizing long-term results.

Department of Ophthalmology
Sarojini Naidu Medical College, Agra

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Young Ophthalmologists : Hard Decisions

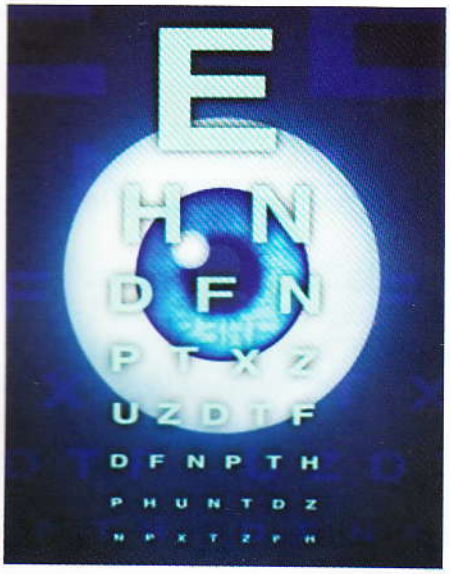
Ashish Agarwal MS

Joy of passing postgraduate examination is a feeling which cannot be described. The young doctor is elated almost to the point of euphoria. If post-graduation is done in terminal branches like Ophthalmology, hard and brain draining decisions are soon to be made. Numerous factors are to be considered before embarking on the journey of your chosen branch. They may be personal or professional. Having gone through same phase of life I would like to pen down some of my experiences, which might be of some help to you.

Your Better Half

By the time post-graduation is done, most of us get married. If she is a non-medico many of the decisions become simple. She can be with you all the time. Her job at times could be a bit of concern, but it could be managed. If she is a medico of a different branch, both of you really have to sit down and discuss.

Things like place to settle (parents might also be interested), financial investments (ours is one of the costliest branch) and kids too! I was fortunate to have an ophthalmologist as my wife. This made things much simpler. Investments were halved, we could work at same place, and we were even given preference over other candidates in interviews.



Job or Fellowship

Ours being a terminal branch and, that to surgical, we have to make a crucial decision. To go for fellowship or to go for a job. This dilemma is more if post-graduation is done from a college, where no surgical expertise is given. Unfortunately this is the condition in almost all of our medical colleges.

Jobs like senior residency in metro cities or Eye Care Centres lure us. These definitely give us financial support. After joining these places you learn that, if you do not have expert surgical hand, you are treated like a first year student. You get a decent salary, good office to sit into but almost no or minimal surgical work. This gradually leads to frustration.

What I suggest, before jumping into these setups you go for a fellowship or join a place where surgical expertise can be earned. For this purpose many good charitable hospitals are there, which could be joined (although salary and living conditions might not be to

Pakhi Eye Care Centre
Kanpur

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your satisfaction). These two or three years of hardship will definitely help you in later life.

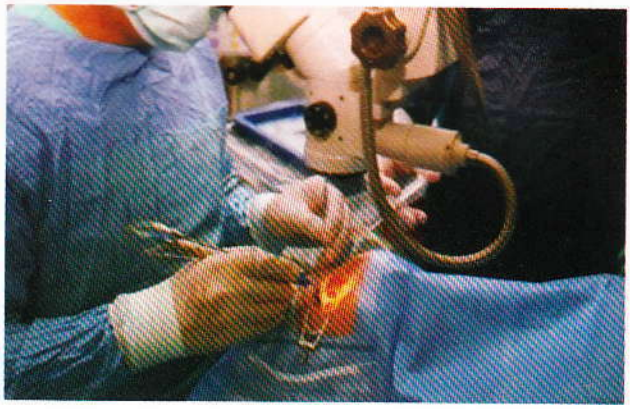
Job or Private Practice

This is a million dollar question, of which till date I am yet to find an answer. Both of prospects have their own merits and demerits. This issue is to be discussed only if you have a decent surgical hand.

First I would like to talk about the easy thing. After having sufficient surgical skill, it's the easiest way out. Just drop in your resume at different well to do eye centres (be it charitable or corporate). Salary these days is not a problem in majority of centres, if you are proficient in your field. They even are at par in majority of centres with usual perks like accommodation (in small cities). There is not much liability on your shoulders & you work peacefully. You would be given sufficient (read more than) amount of work, long working hours and holidays which your family could easily count on fingers. The problem doesn't end here.

First you would certainly not get this type of paying job in your own city, even if you get, there would be many boundations. You are working under a person/trustee who definitely would not want you to have your own identity in his hospital, which he has brought up by many years of hard work. There will always be a conflict of an employee and an employer. After spending two to three crucial years, you might realize that you could not go on like this and decide to move on. Now what?? By this time you might be having kids. Going to another place for a job will not be easy this time. Your parents (if you have to support them) will be mad at you.

Now either you compromise or take much harder decision to set up your own practice.



Starting a private practice is not an easy task. Foremost thing which comes to mind is financial backup. Ours is one of the costliest branches to set up, if you want to have a decent one.

If you are born with a silver spoon, this is not a major issue and in fact private practice will be one of the best options to consider. You have to compete with you colleagues and you would in middle of sea, full of giant big shots of your city. You can get attached to various hospitals in your city or work part time. Having a decent practice will take almost five to seven years of hard work.

Other prospect is that of a group practice. This would greatly reduce your investments and would increase the range of work that can be done on your Centre. The partners should preferably of different field to avoid clash of interests.

Income tax was first introduced in world in England in 1799 by British Prime Minister William Pitt.

Management Pearls for Traumatic Cataract

Nisha Chauhan MBBS, Richa Jain MBBS, Vishwesh Agarwal MBBS, Snigdha Sen MS

Traumatic cataract is a common cause of monocular visual loss, in children and adults, especially after penetrating injuries. The decision for management in such cases is very challenging and depends on the visual disability casued by the cataract and associated ocular injuries. The ophthalmologist is faced with the following dilemmas when dealing with the case of a traumatic cataract.

- Whether cataract removal should be done simultaneously with corneal tear repair in cases of penetrating injury or should be done as a secondary procedure?
- Even if the cataract is removed, whether or not to implant the IOL in same sitting?
- The type of IOL

Also in young children, unlike the adult patients, management of the associated amblyopia can be, at times, frustrating. But the problem of amplyopia is encountered less in traumatic cataract compared to congenital or developmental cataract because patients present early in cases of trauma.

DECISION OF SURGERY

Timing of cataract surgery and IOL implantation in the setting of trauma is still debated worldwide. Primary surgical management is often dictated by the extent of corneal and scleral injury in addition to the injury to the lens. Certain lacerating injuries of the anterior segment are particularly amenable to cataract extraction and IOL implantation at the time of primary laceration repair. This approach obviates additional operative and anesthetic risks, while affording timely visual rehabilitation.

Secondary lens removal may be indicated in cases of severe corneal injury and marked edema, which may interfere with intraocular visualization. Advantages of secondary cataract removal are better visibility, better intraocular lens calculation, anterior segment reconstruction, and stabilization of a hemato-ocular barrier.

When the lens alone is injured, delayed removal has been favored historically, but according to a recent study on traumatic cataract, the visual outcome did not differ between primary and secondary cataract extraction and between primary and secondary IOL implantation in adults, but in the amblyogenic age, primary surgery with IOL implantation should be preferred.[1]

Indications for immediate cataract removal are:

- dislocation of the lens/ lens fragments into the anterior chamber with corneal touch.
- pupillary block due to anterior lens displacement.
- angle closure secondary to an intumescent lens.
- uncontrollable inflammation.
- elevation of IOP secondary to lens-particle release.

DECISION FOR IOL IMPLANTATION

If primary IOL implantation is planned, biometry should be done on non-injured eye. In-the-bag fixation of the IOL is the preferred choice if the lens capsule and zonular support are intact. Multifocal IOLs are a

Department of Ophthalmology
Sarojini Naidu Medical College, Agra

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better alternative to monofocal pseudophakia, as they provide better uncorrected near visual acuity, stereopsis and less spectacle dependence in children with and without primary posterior capsulotomy. Acrylic foldable lenses have shown better outcome as compared with PMMA lenses in the studies.[2] Cases with sectoral zonular weakness are amenable to in the bag PCIOL implantation with haptics placed perpendicular to area of zonular weakness.

Polymethyl methacrylate (PMMA) capsular tension rings (CTR) allow easy implantation of PCIOL in cases of zonular dialysis less than 180 degrees, but the conventional capsular tension ring does not allow effective treatment of large zonular dialysis or recentration of the bag. However, the Cionni modified CTRs with scleral fixation stabilize and recenter the bag, allowing capsular preservation and in the bag implantation of PCIOL in cases of traumatic zonular dialysis.

- Sulcus fixation is safe, if the posterior capsule is compromised but zonular support is maintained.

- In children without adequate capsular support and zonular support, the decision of optimal optical correction methods is very challenging. Aphakia is avoided as there is a risk of amblyopia in these patients, thus rapid optical and visual rehabilitation is very important. Nonsurgical methods with spectacles or contact lenses and surgical methods such as scleral fixation of posterior chamber intraocular lenses (SF-PCIOLs) may also be considered. Unilateral aphakic glasses are generally not suitable for children due to aniseikonia, which may impair binocularity. Contact lenses may cause corneal problems and poor compliance in pediatric patients as well as amblyopia, due to intermittent correction of refractive error. Also, in traumatized eyes, wearing contact lenses may be intolerable or difficult due to irregular corneal surface or conjunctival scarring from the trauma or multiple operations. In such cases, SF-PCIOL is a safe alternative. There have been reports showing comparable surgically induced astigmatism in sutureless intrascleral haptic fixation with retro-pupillary iris claw fixation. Thus former can be considered in cases where no other implantation technique is possible.

- Anterior chamber placement is always an option in cases of insufficient posterior capsule support, eyes with ciliary body trauma which prevents suture fixation, no advanced glaucoma, good iris support and in adult patients.

- Aphakia may be a better choice in patients with highly inflamed eyes, as they may experience better outcomes if lens implantation is deferred.

PRE- OPERATIVE EVALUATION

Proper diagnosis and appropriate management of traumatic cataract/ subluxation/ dislocation is necessary to restore vision and prevent sight threatening complications. The methods used to evaluate the visual outcome in eyes managed for traumatic cataracts and senile cataracts are similar,but these two cataracts are different as there are associated ocular morbidity which alter the prognosis in traumatic cataract.

History:

In the setting of ocular trauma, the mechanism of injury is a critical determinant

of the type of the ocular damage sustained and is, therefore, the cornerstone of the medical history. Mechanism of injury should be noted whether blunt or perforating injury.

A perforating injury most often brings about immediate attention, many patients who have sustained blunt trauma to the globe and develop contusion cataract or lens subluxation do not seek immediate medical care. They may later develop inflammation or experience a delayed onset of fluctuating or decreased vision from subluxation of the lens or progressive cataract.

A past medical history should be obtained to establish any preexisting ocular or systemic conditions, such as glaucoma, previous ocular surgery, or diabetes, that may affect visual outcome.

Counsel Properly:

The patient and the family should be informed well about

- Possibility of uncertain visual outcome
- Challenges faced during surgery in eyes with other associated ocular injuries
- Postoperative suboptimal visual recovery
- Probability of secondary intervention

Ophthalmic Examination:

Birmingham eye trauma terminology system (BETTS) should be used to standardize the documentation of clinical findings in cases of ocular trauma.[3] The eye examination begins with assessment of the vision. In many cases of ocular trauma, the visual acuity on presentation is a predictor of visual outcome. Pupillary reaction is noted to rule out afferent pupillary defect which is indicative of optic neuropathy.

A low IOP is potentially helpful in determining the presence of a ruptured globe, whereas an elevated pressure can indicate subluxation of the lens with pupillary blockade, disruption of the angle, the inflammatory effects of lens particles, or angle closure secondary to an intumescent lens.

Imaging Techniques

Slit lamp biomicroscopy: Examination of anterior segment is done systematically both before and after pupillary dilatation and note any corneal scar, anterior chamber depth, presence of any abnormal content in anterior chamber, posterior synechiae, morphology of cataract (total / membranous / soft white / rosette) or any other abnormal finding. A subtle subluxation may only be identifiable using a retroillumination biomicroscopic view after wide pupillary dilatation. A deep anterior chamber and iridodonesis may be suggestive of subluxation, and a narrowing of the angle may indicate forward displacement of the lens. Prolapse of vitreous into the anterior chamber confirms the presence of lens subluxation.

B-scan: If corneal edema, blood in the anterior chamber or lenticular opacification precludes visualization of the lens o posterior segment, ultrasound can be usefu in determining lens position, retained intraocular foreign body, vitreous hemorrhage or retinal detachment Ultrasound biomicroscopy can also be use to avoid intraoperative surprises regardin posterior capsule integrity and lens suppo structures. This helps the surgeo preoperatively to modify his surgical pla regarding wound location, method of catara

removal and possible use of capsular tension ring or vitrectomy.

Anterior segment OCT: Its a non invasive alternative to ultrasound biomicroscopy for anterior segment evaluation. It can be utilized to identify traumatic damage to lens capsule, cortex, zonules and anterior hyaloid phase.

X-ray orbit: Can be used to localize retained foreign body in cases of penetrating / perforating injuries.

CT-scan: Computed tomography (CT) scan is ideal for defining bony anatomy of the orbit in cases with head or facial trauma and offers a greater deal of precision in foreign body localization. A CT scan can also provide information about the state of lens opacification. Signal attenuation may be seen in a lens that is cataractous, even before clinical lens opacification.

Electro-physiological tests: (ERG, EOG, VER): Can be used in higher centres to assess the co-morbidities associated with opaque lens in cases of trauma to help in the prediction of prognosis after traumatic cataract surgery.

SURGICAL MANAGEMENT

To maximize surgical outcome and minimize complications, a decision must be made between an anterior (limbal) and a posterior (pars plana) surgical approach.

Anterior (Limbal) Approach:

It is indicated in the following situations:

- No apparent zonular compromise and an intact posterior capsule
- Minimal zonular compromise, no displacement of the lens, and no vitreous present in the anterior chamber
- Dislocation of the lens into the anterior chamber.

Posterior Approach (Pars Plana Incision):

A pars plana technique and a posterior approach can be used if there is:

- Posterior subluxation or dislocation of the lens
- Preoperative / intraoperative disruption of the posterior capsule with lens fragments in the anterior vitreous. Preservation of posterior lens capsule in such cases is less important than preventing anterior vitreous traction as alternative method of IOL implantation offer similar functional results.
- Associated posterior segment injuries, where combined lensectomy, vitrectomy & IOL implantation can be planned.

POST- OPERATIVE CARE

Surgery to repair a traumatic cataract has a higher rate of postoperative complications than standard cataract surgery does.

Early post- operative complications can be corneal edema, fibrinous uveitis, hyphema and IOP rise. Fibrinous uveitis is a very common finding and can cause posterior synechiae, pupillary block glaucoma and lenticular membrane formation.Intensive topical corticosteroid, oral corticosteroid, topical antibiotics and cycloplegics usage should be there to minimize complications and should be tapered slowly over weeks. Intracameral dexamethasone can also be used intra-operatively to reduce post operative complications.

Late post- operative complications include posterior capsule opacification, IOL decentration, pupil capture, cystoid macular

edema and retinal detachment. The incidence of posterior capsule opacification in children with traumatic cataract undergoing cataract surgery with posterior chamber intra-ocular lens implantation has been reported to vary between 21% to 100%, posterior capsulotomy at the time of surgery can decrease its incidence. Primary posterior capsulotomy should be done in all children of less than 6 years age. Children with more than 6 years of age can co-operate well for yttrium aluminium garnet capsulotomy.

PROGNOSIS

Patients' age and initial visual acuity are good predictors of final postoperative visual acuity. Ocular trauma score can be used as a reliable tool for predicting the visual outcome of cataract extraction surgery. Classify the injury into open or closed globe injury and then the score calculated preoperatively after patients examination correlates well with post- operative visual outcome. About 2/3rd patients achieve visual acuity of 20/60 or better. Eyes with sharp trauma have poor visual results, as these eyes need multiple surgeries due to coexisting ocular morbidity, commonly corneal tears.

PREVENTION

A lot of ocular injuries could be prevented if proper protective wear are used, but it is difficult to convince rural communities regarding the advantages of protective wear to their children. Many house-hold items like pins, scissors, knives, pens, pencils, nail cutters and fire works are responsible for the childhood injury. Local schools should inculcate the safety habits through the school curriculum, as is being done in many urban private schools in India, along with lessons on first aid and safety precautions.

Education of the masses to avoid neglect, early referral of complicated cases and awareness of the patient to ensure compliance can avoid the blindness caused by traumatic cataract.

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Few Facts

- *The colour of a chilli is no indication of its spiciness, but size usually is - the smaller the pepper, the hotter it is.*
- *A leech has 32 brains.*
- *Marilyn Monroe had six toes.*

Management of Dry Eye

Neha Jain MBBS, Shephali Jain MS, Chaitali Deshmukh MBBS, S K Satsangi MS, Himanshu Kumar MS

Dry eye is one of the most frequently encountered ocular morbidities, a growing public health problem and one of the most common conditions seen by eye care practitioners.

The International Dry Eye Workshop (DEWS) defined dry eye as a "multi-factorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface". It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface."[1]

The risk factors for the dry eyes are: Older age, Female gender, Postmenopausal oestrogen therapy, Low dietary intake of omega-3 fatty acids, Systemic diseases as Parkinson's disease, sjogren syndrome etc, medications like antihistamines, anti-glaucoma drugs, connective tissue disease, LASIK and refractive excimer laser surgery.

The goals of management are:

- Reducing or alleviating signs and symptoms of dry eye.
- Maintaining and improving visual function.
- Reducing or preventing structural damage.

Diagnosis

Detailed Ocular history and medical history are mandatory.

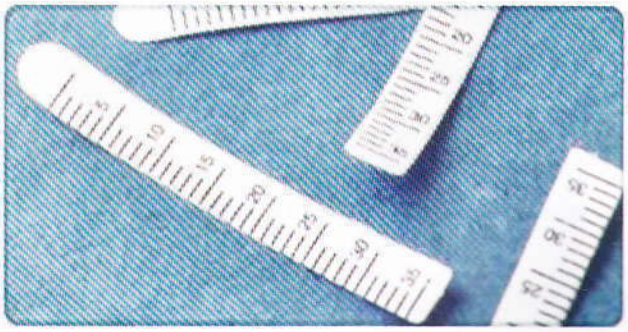
Symptoms include irritation, tearing, burning, stinging, dry or foreign body sensation, mild itching, photophobia, blurry vision, contact lens intolerance, redness, mucous discharge, increased frequency of blinking, diurnal fluctuation. symptoms worsen later in the day.

Exacerbating conditions:

Wind, air travel, decreased humidity, prolonged visual efforts associated with decreased blink rate such as reading.

Examination:

- Visual acuity measurement
- External examination: Skin, adnexa, eyelid, proptosis, cranial nerve dysfunction.
- Slit-lamp biomicroscopy: Tear film, eyelashes, eyelids, puncta, conjunctiva and cornea.



Diagnostic tests:

1) Schirmer's test: This is carried out with Schirmer's strip- Whatmann-41 paper 5mm x 35mm whose end is bent to adjust on the lid. Strip is placed between inner 2/3rd and outer 1/3rd of lid.

2) Staining: Touch the temporal conjunctiva with a wet stain dipped filter paper and asks thepatient to blink few times and then stop blinking.

Department of Ophthalmology
Sarojini Naidu Medical College, Agra

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Types of Schirmer	Measures	Method	Inferences (based on wetting)
1	Reflex and basic secretion	Strip placed for 5 minutes. The Patient is asked to keep the eye open, look straight and slightly up, blinking is permissible.	<3mm= V Severe, 3-5mm= Severe 5-9mm= Mod, 10mm= Mild >10mm= Normal
2	Reflex secretion	If above test reveals <10mm, ipsilateral nasal mucosa is irritated with a small cotton swab	no wetting or <1mm Sjogren's syndrome >1mm it is non Sjogren's syndrome.
3	Basal secretion	Conjunctival sac is anaesthetised	<5mm= Dry 5-10mm= Equivocal >10mm= Normal

3) Tear meniscus height: Height< 0.3 mm is suggestive of dry eye. It is measured with the slit lamp under a cobalt blue filter following instillation of fluorescein.

4) Lacrimal gland function test: Lactoferrin is the most abundant tear protein that is secreted by the lacrimal gland.[2] Tear lactoferrin concentrations have been reported to decrease in lacrimal gland dysfunction.

5) Other tests :

i) Slit lamp fluorophotometry:
ii) Meibometry: It is for noting the mucus gland dysfunction. Direct and indirect

iii) Ocular protection index: It is TBUT time in seconds divided by inter-blink interval in seconds. OPI <1 = Patient at risk & OPI>1 =Patient not at risk

iv) Tear Osmolarity: It is measured by taking 0.24 micro litres of tears and measuring the freezing point of dispersion. Normal = 302mOs/litre & in Dry eye = >350mOs/litre

v) Closed chamber infrared thermometry: The temperature is recorded with eye closed and then after opening the eye after five seconds at fixed point & fixed distance from the eye. The temperature increased by 0.1°C after opening the eye is normal. No increase in temperature after opening the eyes seen in dry eye

Stain	Filter	
Fluorescein Stain 2%	Cobalt blue	Time taken between the last blink and appearance of a black in the tear film is the break up time in seconds. Normal = >10 seconds Gd-1 = 10 seconds, Gd-2 = 5-10 seconds Gd-3 = 3-5 seconds, Gd-4 = < 3 seconds 

vi) Closed Chamber humidity of the eye: The humidity is measured with eye closed and then 5 seconds after opening of the eye. The difference in humidity in<1RH% is normal and in dry eye it is >1RH% (1RH% to 4RH %). It is most reliable test, non-invasive, quick test for early diagnosis of dry eyes even when other tests declare normal values.

Dry Eye Disease Treatment Guidelines Based on Disease Severity from the International Dry Eye Workshop (DEWS)[3]

Severity	Description
1	• Education • Environmental/dietary modifications • Elimination of offending systemic medications • Artificial tear substitutes gels/ointments • Eye lid therapy
2	If level 1 treatments are inadequate, add • Anti-inflammatories cyclosporine A topical corticosteroids tetracyclines (for meibomitis, rosacea) • punctal plugs • secretagogues • moisture chamber spectacles
3	If level 2 treatments are inadequate, add • Serum • contact lenses • permanent punctal occlusion
4	If level 3 treatments are inadequate, add • systemic anti-inflammatory agents • cyclosporine A • Prednisolone • Methotrexate • infliximab • surgery (lid surgery, tarsorrhaphy,mucus membrane, salivary gland, amniotic membrane transplantation)

1. Behavioural and environmental strategies

Patients should be educated about preservation of existing tears by reducing evaporation, such as learning to take breaks while reading, lowering the computer monitors to decrease lid aperture, use of protective glasses with side pieces in outdoor setting and humidification of the environment, increasing blink frequency or blinking exercises.

2. Eyelid hygiene

Washing the eyelid margin with a gentle soap decreases bacterial colonization. Bacterial colonization is believed to inhibit conjunctival goblet cell proliferation and may also increase the breakdown of meibomian lipid.[4] Reducing colonization, therefore, may improve both the mucous and lipid layers of the tear film.

3. Tear substitutes

Cellulose ethers: Hypromellose, hydroxyethylcellulose, methylcellulose, carboxymethylcellulose (0.5%) - Viscoelastic Polysaccharides:(Just tears, Refresh Liquigel, CCS, SCM).

- Increase viscosity of tears.
- Good retention time on ocular surface.
- Only of benefit in aqueous tear deficiency.

Carbomers: polyacrylic acid- Synthetic polymers: (Viscotears)

- High viscosity when eye is static, shears thin during blinking or eye movement, maximizing thickness of the tear film while minimizing drag.
- Good retention time on ocular surface.
- But tends to blur vision.

Polyvinyl alcohol- Synthetic polymer: (Liquifilm tears, D-eye, PVA Tears)

- Low viscosity but optimal wetting characteristics at a concentration of 1.4%.
- Beneficial in lipid, aqueous and mucin layer deficiencies.
- Water soluble, does not cause visual blurring.
- Has short retention time.

Sodium hyaluronate Mucopolysaccharides: (Lubristil)

- Viscous formulation
- Good retention time on ocular surface
- Beneficial in corneal wound healing

Povidone (Polyvinyl pyrrolidone) - Synthetic polymer: (D- Eye)

- Co-formulated with electrolytes.
- Beneficial in mucin layer deficiency

Acetylcysteine:

- Breaks down mucin molecule.
- Can be co-formulated with another lubricant such as hypromellose.
- Useful in severe dry eye for complications resulting from dense mucus.
- Not commercially available.

Electrolyte composition. Products that mimic the electrolyte composition of natural tears- potassium and bicarbonate appear to be the most important.[4]

Preservatives. Preservatives are added to artificial tears to reduce the risk of bacterial contamination in multidose containers, and to prolong shelf life.

There are 2 main types of preservatives: detergent and oxidative.[5]

Detergent preservatives act by altering bacterial cell membrane permeability.[5] Detergents have toxic effects on the ocular surface epithelium and, with frequent use, can cause epithelial irritation and damage.

Patients with a compromised tear film are at higher risk. E.g. Benzalkonium chloride.[4,5]

Oxidative preservatives penetrate the bacterial cell membrane and act by interfering with intracellular processes. They are sometimes referred to as "vanishing" preservatives because they dissipate on contact with the eye and, therefore, are less likely than detergents to cause ocular damage.[5] However, they may not always dissipate completely in DED patients because of decreased tear volume.[2] e.g. Stabilized oxychloro complex.

4. Anti-inflammatory therapy

Due to a newer understanding of the pathogenesis of dry eyes, use of anti-inflammatory medications is a paradigm shift in the treatment of dry eye.

- Cyclosporine (0.05%) (Restasis, Cyclomune)- Well known immuno-modulator.

Preservatives	Advantages	Disadvantages
1) Benzalkonium chloride	• Chemically stable. • Effective and fast acting against many micro-organisms. • Does not degrade easily even at high temperature.	• Can accumulate in ocular tissues causing cell death with frequent dosing. • So frequency of use must not exceed 4-6 times daily.
2) Sorbate	• Useful for sensitive eyes and contact lens wearers due to infrequent adverse reactions	• Limited antimicrobial activity • May cause punctate keratitis
3) Chlorobutanol	• Antimicrobial action	• Causes irritation in more than 50% users
4) Sodium perborate	• Low levels have good anti-microbial activity. • Changes to oxygen and water on contact with tear film.	• Causes cell death within minutes even at low concentration
5) Polyquaternium-1	• Has lesser effect on corneal epithelium than BKC.	• Causes superficial epithelial damage
5) Stabilised oxychlorocomplex	• Oxidative preservative that is converted in to natural tear components in the eye. • Antimicrobial activity. • Safe and well tolerated.	• Least cytotoxic effects

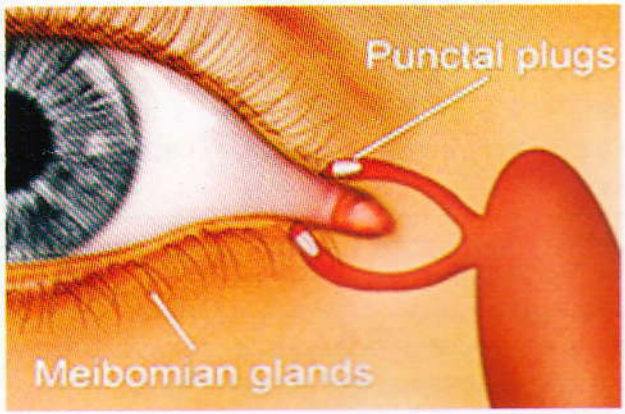
FDA approved to increase tear production in patient whose tear production may be reduced by inflammation of the eye associated with kerato conjunctivitis sicca.

It reduces inflammation by preventing T cells from releasing cytokines. Cyclosporine therapy appears to address all three tear film components (aqueous, oil, and mucin). Unit dose vials (0.4 ml). dosing=1 drop 2x daily.

- Topical corticosteroids are approved by the FDA and prescribed for corticosteroid-responsive inflammatory conditions of the conjunctiva, cornea and anterior globe—including DED.[4] Prednisolone is almost as potent and penetrates ocular tissues well. Other steroids, like loteprednol etabonate and fluoro-metholone, are less potent and have better safety profiles.
- Tetracycline: Oral doxycycline can be used in all patients with dry eyes who have significant component of meibomian gland disease.
- Essential Fatty Acids: necessary for complete health. 18 carbon omega-6 and omega-3 fatty acids.

5. Punctal plugs

- Absorbable (collagen / various polymers) last for variable periods of time (3 days to 6 months).



- Non absorbable (silicone or hydrophilic)
- Thermoplastic (thermo sensitive, hydrophobic acrylic polymer)
- Hydrogel Plugs

Punctal plugs are indicated in patients who are symptomatic of dry eyes, have a Schirmer test (with anaesthesia) result less than 5mm at 5 minutes.

Contraindications to the use of punctal plugs include allergy to the materials used in the plugs to be implanted, punctal ectropion, and pre-existing nasolacrimal duct obstruction

Intracanalicular plugs- more invasive.

Surgical occlusion of punctum can be done using electro-cautery or laser.

6. Tear stimulation Secretagogues:

They are cholinergic agonists that stimulate endogenous tear production by the lacrimal glands and/or ocular surface epithelia (e.g. oral pilocarpine).[5]

Diquafosol (one of the P2Y2 receptor agonists), rebamipide, gefarnate, ecabet sodium (mucous secretion stimulants), and15(S)-HETE (MUC1 stimulant).

7. Moisture spectacles/goggles:

Increase humidity around eye.

8. Autologous serum:

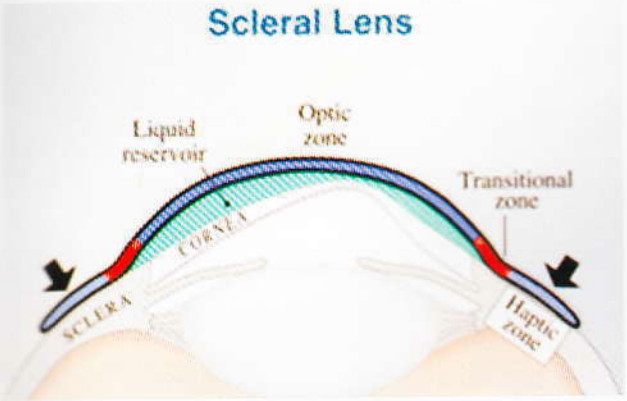
Serum and plasma contain many anti-inflammatory factors which include inhibitors of inflammatory cytokines and inhibitors of MMPs and mediators of the ocular surface inflammatory cascade of dry eye.[7]

9. Silicone hydrogel lenses:

have high oxygen permeability and relatively low water content, may be used in severe dry eye or when other therapy has failed. Highly oxygen permeable materials enable overnight wear in appropriate circumstances.[8]

The Boston scleral lens is indicated for management of severe dry eyes due to Sjögren's syndrome & autoimmune diseases. Boston Scleral provides an oxygen permeable optical shell that fits under the lids and over the front surface of the eye. It fits on the sclera and immerses the eye in a pool of artificial tears. The fluid reservoir of the device masks the distorted surface of the cornea to improve vision and provides a protective cushion that reduces pain and photosensitivity caused by inflamed and irritated corneas.

There is a small risk of corneal vascularisation and possible corneal infection associated with the use of contact lenses by dry eye patients.



10. Surgery - Tarsorrhaphy:
For severe or refractory DED. Methods include:

- Short-term tarsorrhaphy- tape, adhesive glue (lasts a few days), or botulinum toxin (lasts an average of 16 days).[9]
- Temporary tarsorrhaphy (4-6 weeks).[9]
- Permanent tarsorrhaphy. The lid margins are excised and sutured so that they heal together. The procedure can be reversed.[9]

Amniotic membrane transplantation is indicated in persistent epithelial defects unresponsive to medical treatment.

Salivary Gland Auto transplantation
It is capable of replacing deficient mucin and the aqueous tear film phase. This procedure requires collaboration between an ophthalmologist and a maxillofacial surgeon.

Parotid duct transplantation
Should be considered in dry eyes especially caused by Stevens-Johnson syndrome.

The early detection and timely management of this syndrome is important, as they can help prevent long-term sequelae and sight-threatening complications.

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Human eye blinks on average of 4,200,000 times each year.

Evaluate the efficacy of conjunctival autograft with autologous plasma in surgical management of primary pterygium

A Gupta MS, S Kumar MS, KPS Malik MS, VK Malik MS, R Jhalani MS

Introduction

Since the days of Susruta, the world's first ophthalmic surgeon, pterygia have been recognized as a triangular sheet of fibro-vascular tissue that appears on the bulbar conjunctiva & cornea, disturbing both the patient because of their appearance and the surgeon because of their tendency to recur. Pterygium are characterized by excessive fibro-vascular proliferation on the exposed ocular surface and are thought to be caused by increased light exposure, dryness, dust, heat, and wind (Rosenthal JW et al, 1953).

In the exposed population, the growth of pterygium has been seen in younger teenagers and widely prevalent in people in deserts. Pterygia are seen nearly twice often in men as in women (Cameron ME et al, 1965). Symptoms include chronic ocular surface inflammation and tearing, eventually astigmatism and blurred vision attributable to optical axis involvement. Successful management of pterygium is a constant challenge for ophthalmologists due to high recurrence rate (2.1 % to 87 %, Bardley PG et al, 1998) and sight threatening complications of different surgical techniques (Gupta VP et al, 1997).

The recent concept of the role of the corneal Limbal stem cells has lead to the development of the new concept of pathogenesis of pterygia. Accordingly, pterygium is a local limbal deficiency. Pterygium recurrence can be reduced if the limbus and limited area of the cornea are included in the conjunctival graft, as it is well recognized that limbal stem cells play a vitalrole in maintaining the ocular surface.

The function of limbal stem cells which are situated in the basal layers of the conjunctival epithelium, include regeneration of the tissue and cell replacement (Khamar Betal, 2000). Thoft introduced the concept of the ocular surface and idea of its reconstruction.

This study comprised of the use of Conjunctival autograft transplantation to the pterygia. Conjunctival auto transplant taken from the actinically unexposed conjunctiva prevents reproliferation of actinically altered cells into the cornea (Thoft RA et al, 1977). Suture is, an old, basic method to sew wound, also used to stitch the conjunctival autograft. It has some disadvantage in ophthalmic use such as irritation, infection, inflammation, and allergy.

Autologous fibrin glue has been used as an alternative method for graft fixation by some authors (Cohen & Donald, 1993; Foroutan et al, 2011). A recent cross-sectional study also describes successful outcome with sutureless and glue-free conjunctival autograft (Wit et al, 2010).

The present prospective study was then undertaken to evaluate the efficacy of sutureless and glueless conjunctival autograft with autologous plasma in cases of pterygium surgery.

Materials and methods

This prospective interventional case series included consecutive 43 eyes with primary nasal pterygium requiring surgical excision.

Subharti Medical College
Meerut, India

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The primary outcome measures included graft dislocation and pterygium recurrence. Graft success was defined as an intact graft by the end of 6 weeks after operation without need for sutures. Recurrence was defined as any growth of conjunctiva exceeding 1mm onto the cornea.

Exclusion criteria included recurrent pterygium, conjunctival scar, tumour, corneal tumour, bullous keratopathy, dry eye, pseudopterygium, glaucoma, retinal pathology requiring surgical intervention, history of previous ocular surgery or trauma.

Surgical technique

The pterygium head was held by lims forcep and was then excised from the corneal surface with the help of a crescent knife or beaver 11 no. blade. Only the thickened portions of the conjunctiva, the immediate adjacent and subjacent tenon capsule showing tortuous vasculature were excised and sharp dissection by Westcott scissors (Geuder, Germany) was done for separating the fibrovascular tissue from the surrounding conjunctiva. Care was taken to avoid conjunctival plica excision and extensive dissection of the tenon. Spontaneous haemostasis was allowed to occur without the use of cautery. The size of defect was measured in mm^2 with castroviejo callipers Dissection between donor graft conjunctiva and tenon layer was done carefully while fashioning the 2.0 mm oversized conjunctival limbal graft from the superior bulbar conjunctiva. Average graft size was 14 to 16 mm^2. The autologous conjunctival graft taken from superior bulbar conjunctiva was scraped from the under surface and a thin layer of graft was prepared. Autologous plasma was extracted pre-operatively from the patient's 3ml heparinized blood and centrifuged for 10 minutes to separate the blood cells and plasma. This autologous plasma was used after the preparation of the graft to wet the bare sclera and the graft. The graft was then carefully positioned on the host's bare sclera and care was taken to coincide the limbal portion of the graft to the limbal portion of the bare sclera. A gentle pressure was applied on the graft for 10 minutes. Antibiotic eye ointment, steroid eye ointment and lubricating eye ointment were applied in the patient's eye and the eye was patched for 48 hours.

Postoperative regimen

After removal of the patch, the patient was advised not to rub the eye and topical Loteprednol eye drops were administered four times a day which was tapered over 6 weeks. Chloramphenicol eye drops were instilled four times a day for 2 weeks along with lubricating eye drops.

The patients were followed up post operatively on day 2, 1 week, 6 weeks, 26 weeks. Refraction was performed at 6 weeks. The patients were examined for haemorrhage, wound gape, graft shrinkage, chemosis, graft dehiscence, recurrence or any other complication.

Results

The mean age of the patients was 42.8 years (range 23-61), 75% were males. All the patients were followed up for one year after surgery and there were no drop outs.

Total graft retraction occurred on the conjunctival side in 2 eyes (4.65%). In one patient, it developed following injury with a finger on the 4th postoperative day. In the other, there was lack of adhesion due to accidental inclusion of Tenon's in the free limbal conjunctival graft. The removal of Tenon's also caused delayed healing of the donor site. The graft appeared thickened and congested on the 3rd postoperative day and

the retraction was noticed on the 7th day. Both the patients were managed by securing the same graft using 9-0 vicryl suture. There was mild chemosis in all these patients. All the two patients were managed conservatively by bandaging for 48 hours.

The chemosis disappeared by the end of 7th postoperative day. At 6 weeks postoperatively, the gain in uncorrected visual acuity (UCVA) ranged from 0.18 to 0.5 logMAR in 31 (72.1%) eyes. There was no change in UCVA in rest of the patients. The BCVA showed no change following surgery.

Recurrence was seen in 1 eye (2.3%) at 6 months. None of the patients developed button hole, excessive bleeding, injury to medial rectus, dellen, pyogenic granuloma, symblepharon formation or scleral necrosis.

Fig 1: Pre-op photo Fig 2: 1st Post-op day Fig 3: same eye after 12 weeks.

Discussion

Recurrence after a successful excision continues to remain a challenge in pterygium surgery. Various adjunctive therapies like radiotherapy, antimetabolite or antineoplastic drugs, conjunctival flap, amniotic membrane, lamellar keratoplasty, conjunctival and limbal conjunctival grafts have been proposed to prevent recurrence.

Generally, the pterygium recurrences occur during the first 6 months after surgery (Adamis et al., 1990). The limbal conjunctival autograft has a recurrence rate ranging from 0-15% (Du et al., 2002; Al-Fayez, 2002). Koranyi et al (2004) demonstrated a recurrence rate of 5.3% with glue versus 13.5% with sutures and suggested that immediate adherence of the graft and lack of postoperative inflammation may inhibit fibroblast ingrowth and reduce the recurrence.

Foroutan et al (2011) prepared autologous fibrin glue and used tranexamic acid as an antifibrinolytic agent to tide over the problem of disease transmission and anaphylaxis, respectively. Fibrinogen compounds may be susceptible to inactivation by iodine preparations such as those used for conjunctival disinfection before pterygium surgery (Wit et al, 2010). In our series only one eye (2.5%) had a recurrence. Foroutan et al (2011) had a recurrence rate of 13.33% (2 eyes out of 15) in three year follow up with autologous fibrin.

Using similar procedure as ours, Wit et al (2010) had no recurrence in 15 eyes with a mean follow up of 9.2 months. Graft retraction, was seen in 3 eyes (7.5%) in our series which disappeared once the chemosis was controlled. It did not affect the final position of the graft. Graft retraction occurred in 20% cases in Foroutan et al (2011) series.

Wit et al (2010) postulated that sutureless and glue free graft resulted in an even tension across the whole of the graft interface and no direct tension on the free graft edges resulting in reduced stimulus for the formation of subconjunctival scar. Graft dehiscence is a recognized complication of using tissue glue (Uy et al, 2005; Srinivasan & Slomovic, 2007). With autologous fibrin, dehiscence occurred in 13.33% cases and was attributed to a low concentration of thrombin and fibrinogen in the autologous glue as compared to commercial preparation. Graft dehiscence occurred in two of our eyes, of which one resulted following trauma and the other was the result of accidental inclusion of Tenon's tissue in the free graft. The importance of a thin graft with

careful dissection from the Tenon's capsule is mandatory for a successful graft take up.

None of our patients developed corneal ulcer, scleral melting, conjunctivitis, dellen, symblepharon formation, excessive bleeding, injury to medial rectus muscle, secondary glaucoma, iritis, corneal perforation or corneal ulcer.

Conclusion

Sutureless and glue free limbal conjunctival autografting with autologous plasma following pterygium excision results in mild pain, no foreign body sensation with good aesthetic and functional outcomes. This surgery is a safe, effective and economical option for the management of primary pterygium requiring surgical intervention.

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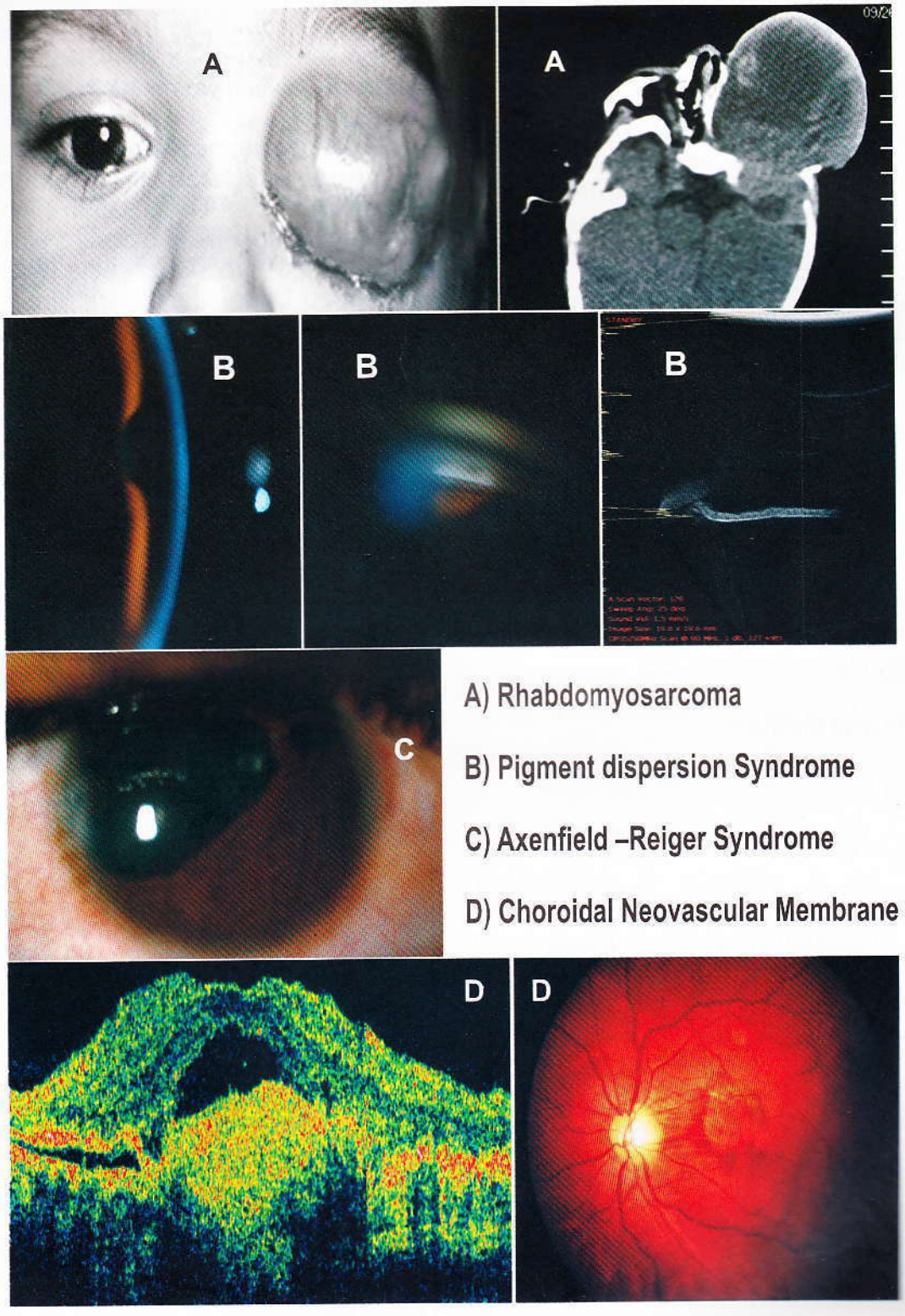
Every four days the worlds population increases by one million people.

Answers to Ophthalmic Quiz - 1



A) Rhabdomyosarcoma

B) Pigment dispersion Syndrome

C) Axenfield –Reiger Syndrome

D) Choroidal Neovascular Membrane

THE LUCKY WINNER FOR THIS QUIZ IS G-105, DR. MAUSAM GUPTA, ALIGARH. HE WOULD RECEIVE A CERTIFICATE FROM OUR SOCIETY AND A GIFT FROM TRINETRA, KAMLA NAGAR, AGRA.

Clinical Study of ACIOL & Scleral Fixated PCIOL

Akhil Agarwal DOMS, Neelima Mehrotra MS, BD Sharma MS
Deepa Nair DNB, Pankaj Kumar MS, Akanksha Singh MBBS

Introduction

Harold Ridley implanted the first intra ocular lens in 1949, it was an acrylic lens implanted in the capsular bag .

Anterior chamber lens can be broadly classified according to whether the haptics have an open loop or closed loop configuration and are rigid, flexible or semiflexible. The anterior chamber lenses may be vaulted or uniplanar.

Parry was the first person to describe suture fixation of PCIOL in 1954 by wire suturing of Ridley's posterior chamber lens.

Malbar in 1986 used a hollow needle to guide suture placement through the sclera during secondary insertion after ICCE

Materials and Method

The patients were divided into 2 groups.

Group-I Consisted of patients implanted with ACIOL, as a primary or secondary procedure.
Group-II Consisted of patients implanted with sulcus fixated (scleral fixated) IOLs, as primary or secondary procedure.

All the patients in group-I were implanted with ACIOL of optic size 5.5 mm diameter with 12.5 mm overall length.

All the patients in group-II were implanted with scleral fixated IOL's of optic size 6.5 mm with overall length of 13 mm. Its design was modified 'C' loop with eyelets for suture fixation. Sutures used were 10-0 prolene.

Eye Department, SRMS-IMS
Bhojipura, Nainital Road, Bareilly

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Technique which was used in all the patients for SFIOL:

- Superior conjunctival peritomy.
- The corneo scleral section 7mm long.
- Lamellar scleral flaps 3mm high & 2mm wide were taken at 3'o clock & 9'o clock.
- The 10-0 prolene needle was initially inserted under the scleral flap, and this needle was brought out from opposite side of sclera with the help of 26 gauge needle and then inserted in a reverse manner. So that a suture loop was formed.
- This suture loop was brought outside the eye from the sclero corneal section, after cutting the suture loop, ends were tied on both the side of eyelet of IOL.
- The suture was locked under the scleral flap and passed through the eyelet on the haptic.
- The sutures were tied in their place under scleral flaps.

Results:

Best corrected visual out come after 4 weeks

	6/60	6/36	6/24	6/18	6/12	6/9	6/6
ACIOL (25 pt)	1	1	3	2	6	8	4
SFIOL (25 pt)	2	0	2	3	7	7	4

Complication	ACIOL (25 patient)	SFIOL (25 patient)
AC Reaction	Gr II to III in all cases	Gr II to III in all cases
Hyphema	1	0
Vitreous in AC	1	0
Pupillary membrane	1	1
Decentered IOL	1	2
IOL tilt	0	1
PAS	0	0
Vitreous hge	0	2
Vitritis	1	1
Iris Prolapse	2	0
Raised IOP	4	6
CME	1	2

Conclusion

Statistical analysis of various parameters was done. We found that post operative BCVA of almost 70% of the patients in both the groups was 6/12 or better. There was no significant difference in the visual outcome of cases in both the groups.

The early and late complications in both the groups were compared and no statistically significant differences were found between them.

The advantages of ACIOL are that, the surgical procedure is safe and fast and IOL's are easy to implant in the anterior chamber. ACIOL implantation is unsuitable in case with deficient endothelial cells, large sector iridectomy, PAS, and glaucoma.

SFIOL preserves corneal endothelium, minimizes aniseikonia and produce stable, longterm fixation of the IOL. It is implanted in the same plane as crystalline lens and doesn't interfere with pupil function and isoptically physiologic. Accuracy of needle placement in SFIOL implantation is difficult as it can't be visualized behind the iris.

It is time consuming and requires elaborate skill with aggressive intra-ocular manipulations.

Our analysis shows that, one procedure doesn't offer more safety than the other in terms of post –operative visual acuity and post operative complications. But long term comparison between both the techniques is required.

Careful pre-operative evaluation of the cornea, vitreous phase and retina will allow the selection of the optimum lens type.

SFIOLs, seem to be more appropriate in young patients and in eyes with compromised cornea, shallow anterior chamber, PAS, glaucoma and lack of iris support

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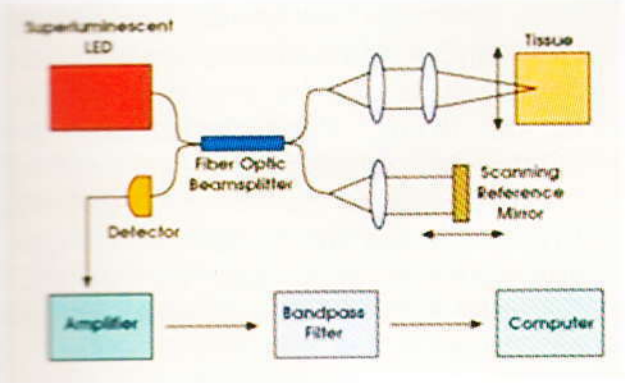
Human eyes can distinguish as many as 10 million distinct colour variations.

Optical Coherence Tomography of Anterior Segment

Snigdha Sen MS

Optical coherence tomography (OCT) is a powerful, non-invasive diagnostic imaging technology that provides high-resolution, cross-sectional images of the eye and other tissues of the body. In ophthalmology, OCT has various clinical applications both in anterior and posterior segment.



Principle of OCT

OCT performs imaging by measuring the echo time delay and magnitude of back reflected light. Conventional or Time-domain OCT, the reference mirror position and delay are mechanically scanned in order to acquire an axial scan (A-scan). In order to get a standard two dimentional cross-sectional image, the beam of light is scanned in the transverse direction and the data is displayed as a false color or grey scale image. A cross-sectional tomograph (B-scan) may be achieved by laterally combining a series of these axial depth scans.

Department of Ophthalmology
Sarojini Naidu Medical College, Agra

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Recently there have been dramatic advances in OCT technology using spectral / Fourier domain detection that enable imaging speeds of ~25,000 axial scans per second, several times faster than time-domain detection.

Time domain VS Fourier domain OCT

	Time Domain	Fourier Domain
Anterior Segment OCT	Zeiss Visante	Optovue RTVue - CAM
Axial Resolution	17 µm	5 µm
Wavelength	1310 nm	830 nm
Speed	2000 A-scans/sec	26000 A-scans/ sec

Fourier domain OCT usually require an adapter lens to study the cornea. In the cornea, OCT provides an excellent tool for diagnosis and documentation of various corneal pathologies, surgeries and response to therapy.

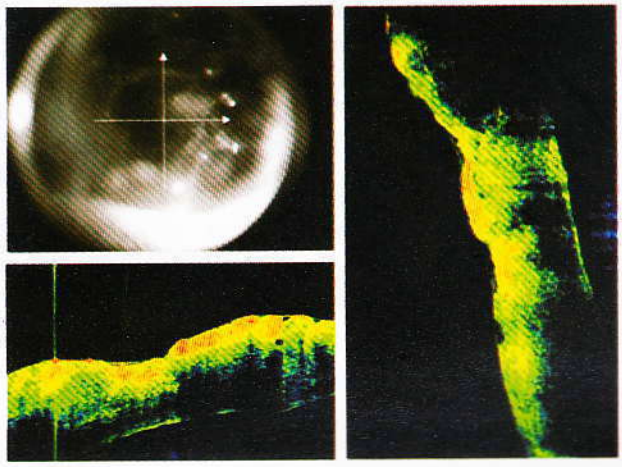
Clinical Applications of OCT:

There are various clinical applications of OCT:

1. Imaging of Cornea:
The OCT provides an high resolution imaging of cornea quite similar as in histopathological sections. One can visualize the various layers of cornea and the pathologies affecting these layers as varying degrees of reflectivity.

2. OCT Pachymetry:
OCT is superior to Orbscan technology which tends to underestimate the corneal thickness in eyes with keratoconus and pachymetry for CCT measurement.

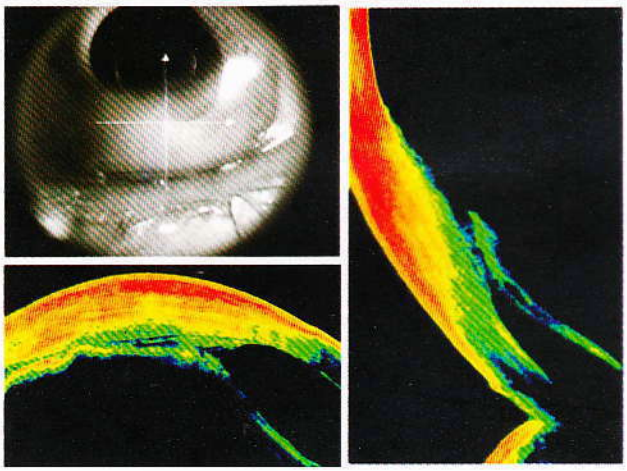
post refractive surgery. Yan Li, Maolong Tang et al[1] used OCT in eyes with keratoconus and finds it equivalent to ultrasound.



OCT picture showing Salzmann Corneal Dystrophy

3. Keratoplasty Surgeries:

As OCT allows the proper visualisation of corneal opacity depth, one can plan whether anterior or deep lamellar keratoplasty is suitable. Laurence S. Lim et al[2] uses OCT prior to DLK and provide very good results in terms of visual outcome.



Descemet's Membrane Detachment

In case, Femtosecond laser is being planned, exact depth of cut can be planned.

Prior to DSEK one can use OCT to clearly visualize the anterior cornea for any opacity which may later on hamper a good visual outcome after endothelial keratoplasty. OCT can be used to detect post-op complications like DMD, lenticule thickness, regularity and interface details which may be difficult to see on slit lamp through an edematous graft.

4. Refractive surgeries:

Prior to Photo Therapeutic Keraoplasty, OCT can be used to precisely measure the opacity depth and the amount of ablation to be done. Post-operatively the quantitative difference in the corneal thickness can be monitored for epithelial hyperplasia and anterior stromal changes. Wirbelauer C et al[3] use corneal OCT before and after PTK for recurrent corneal erosions.

Prior to LASIK corneal OCT helps to assess the suitability i.e. the thickness of the stromal bed and to screen for any other corneal disorder or pathology. Post-operatively OCT allows to measure the flap thickness, regularity, any interfacial deposit or fluid, epithelial in growth etc.

5. Keratoconus diagnosis and treatment:

David Huang et al[4] have demonstrated that OCT can precisely detect the early features of keratoconus. OCT also helps in documentation of progression and changes over time. Effect of procedures like CXL, Intacs etc. can be imaged and documented.

6. Phakic IOL'S:

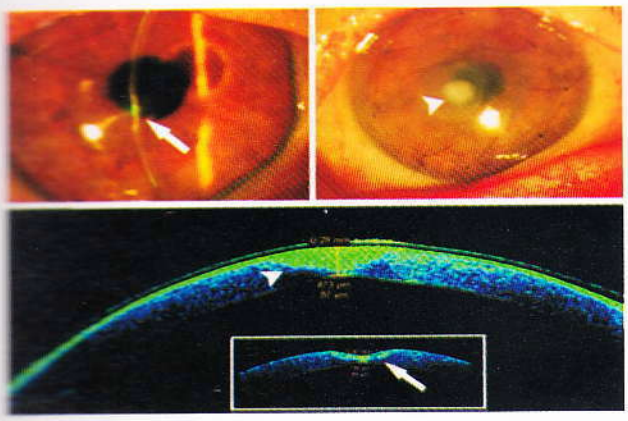
Bechmann Metal[5] described how Fourier domain OCT can precisely measure the anterior chamber depth and provide limbus to limbus imaging in one view, so one can assess the suitability of the eye for phakic IOL. After surgery one can judge the adequate separation of ICL from the crystalline lens.

7. **IOL Power Calculation:**
Minami K et al[5] studied Ray Tracing IOL power calculation using Corneal OCT in eyes that have undergone corneal refractive surgeries and in irregular cornea, as it allows for the presice calculation of the true corneal power.

8. **Dry Eye:**
Brijak MC & Huang D et al[7] studied to measure the tear meniscus height, depth and area by corneal OCT and finds it correlating well with Schirmer's test.

9. **Microbial Keratitis:**
Aris Konstantopoules et al[8] and several others use OCT in imaging of corneal infiltrate density, endothelial plaque, corneal thinning and descemetocoele. Response of therapy can be established by documentation of size & density of the reflectivity of infiltrate.

Mario nubile et al[9] analyze the integration of amniotic membrane transplantation in corneal ulcer in vivo using OCT.



OCT showing integration of amniotic membrane transplantation in corneal ulcer

Conclusion:

OCT is a complete diagnostic tool for any practice. It provides high resolution imaging of the retina, optic disc, cornea and anterior segment. It has multiple uses in corneal evaluation particularly helpful in planning and follow-up after corneal surgical procedures. With the introduction of Fourier-domain technology, the OCT has not only become faster but a more precise imaging tool for the cornea with wide ranging applications in a variety of anterior segment pathologies.

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Brief Communication - 1

Diabetic Retinopathy: What is the Physician's Role

Sandeep Parekh MD, Kamaljeet Singh MS, Sarita Bajaj MD

Diabetic retinopathy (DR) is a vascular disorder affecting the microvasculature of the retina. It is estimated that diabetes mellitus affects 4 percent of the world's population, almost half of whom have some degree of DR at any given time[1]. DR occurs both in type 1 and type 2 diabetes mellitus and has been shown that nearly all type 1 and 75 per cent of type 2 diabetes will develop DR after 15 yr duration of diabetes as shown in earlier epidemiological studies.[2,3]

Physicians Role: Regular eye check-up is a must and best way is to examine the fundus after dilating the pupils.

Recommendations for patients with type 1 diabetes include an eye examination within the first five years of onset and then at least annually. Patients with type 2 diabetes should be examined as soon as they are diagnosed and then at least annually.

American Academy of Ophthalmology recommends following goals:

- Provide visual rehabilitation for patients with visual loss.
- Identity patients at risk for diabetic retinopathy.
- Encourage involvement of the patient and primary care physician in the management of the patient's systemic disorder.
- Encourage and provide lifelong evaluation of retinopathy progression.
- Minimize the side effects of treatment that might adversely affect the patient's vision and/or vision-related quality of life.
- Provide visual rehabilitation for patients with visual loss from the disease or refer for visual rehabilitation.

Motilal Nehru Medical College
Allahabad

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Whales were once land mammals that moved to the sea and adapted to marine life.

Brief Communication - 2

Unnoticed Astigmatism: A Cause Of Chronic Headache

Rajendra Kr. Bundela, Luxmi Singh, Rubie Malhotra, Mustafa Nadeem, B. B. Lal

Astigmatism is present when refractive properties of the eye (especially cornea) are not spherical.[1] This prevents a single focal point from forming.

Accommodation may alter the nature of the image appreciably by varying the positional focal lines. To obtain distinct vision in low grades of astigmatism, efforts of accommodation put a considerable strain on the eye, and lead to headache, dull pain around the eye, nervousness and sometimes nausea[2]. We evaluated 850 patients of headache without any other complaints.

At the time of initial presentation 400 (47.06%) patients complained specifically only of headache, 220(25.9%) patients had headache with eye strain, while rest 230 (27.06%) presented having headache with blurring of vision. The mean age of patients presenting with chief complaint of headache was 23.75 ± 4.93 years in the age range of 15-35 years.

Orthoptic evaluation revealed that majority 80.35% (683 patients) had A/C ratio with in normal limit, while only 19% (167 cases) had associated convergence insufficiency.

The follow-up evaluation clearly showed gradual symptomatic relief of the prime symptom of headache. The duration of complete relief of symptom is proportional to amount of astigmatism corrected. Higher was the amount of astigmatism, earlier was the relief.

However, only 1.2% (10) patients reported worsening of symptoms which could be attributed to non-compliance of regular use of glasses, faulty glasses, disproportionate spectacle frame or patients over depending psychology.

In persons with normal 6/6 vision complaining of dull constant headache, with or without any ocular symptom, low grade astigmatism is one of the prime underlying causes for the symptom and that can be relieved by simply giving a proper astigmatism correction glasses for constant use.

Era's Lucknow Medical College and Hispital Lucknow

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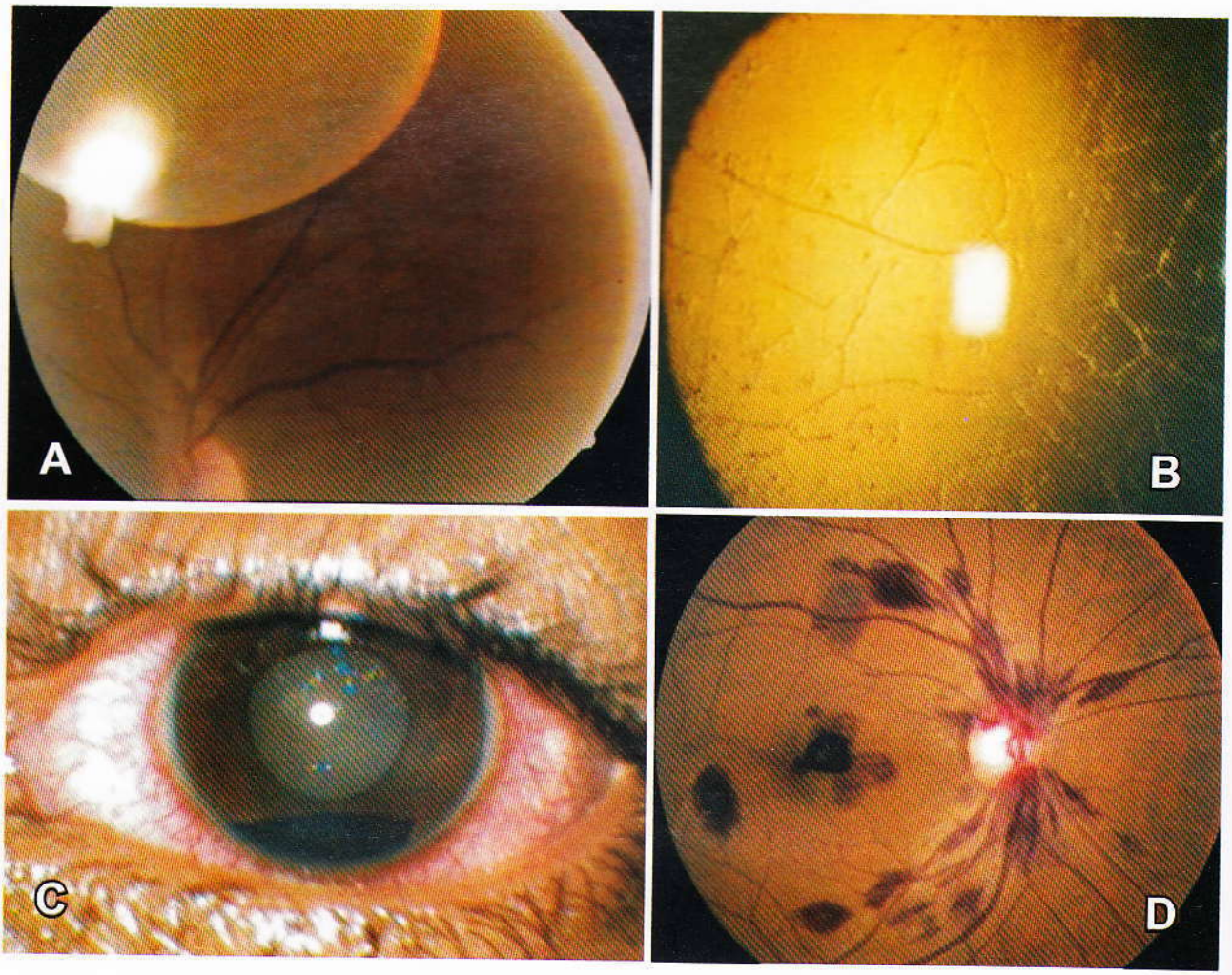
- *Twenty percent of Earth's oxygen is produced by Amazon Forest.*
- *Rats multiply so quickly that in 18 months, two of these could have over 1 million descendent*

The Ophthalmic Quiz - 2

Send your answers by post to Dr Gunjan Prakash, Editor UPJO, Department of Ophthalmology, SNMC, Agra, Mobile 9319204073 or *email* at **editorupjo@rediffmail.com** **Do not forget to mention your UPSOS Membership Number**. One of the lucky UPSOS member, contributing all the correct answers would get a **Certificate** from **UPSOS** & a **Surprise Gift**, sponsored by **Trinetra Medical Institute & Research Centre, Kamla Nagar, Agra**. Last Date 30th November 2013.



Think It Over Till Next Issue

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Answers to Ophthalmic Quiz - II

A _____ B _____ C _____ D _____

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Qualifications & Experience	Institution/University	Year
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2.	.....	.....
3.	.....	.....
4.	.....	.....

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

Proposed by
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.....
Membership No-.....
.....

Seconded by
Name -.....
.....
Membership No-.....
.....

Declaration by Candidate: I shall abide by the regulations of the society in force and any subsequent alterations made from time to time.

I am enclosing Band Draft /Multicity Cheque.No. ..... Dated.....

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Dated..... **Signature**.....

Note:
i) The society reserves all the rights to accept or reject the application.
ii) No reason will be given for any application rejected by the Society.
iii) No application for membership will be accepted unless it is completed in all details.
iv) For Life membership remit Rs. 2,000/= and Rs. 20/= for Laminated ID card, by crossed bank draft of **Rs. 2,020/-** or by crossed multicity cheque drawn in favour **of "U.P. State Ophthalmological Society"** Payable at " **Allahabad**" to **Dr. A.K, Chaddha (Mob 9415289032) 3A Drummond Road , Sukrati Vihar Colony /Allahabad / UP -211001**.

FOR OFFICE USE

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

Date:

Secretary/Treasurer
UP State ophthalmological Society

For any queries pls call **Prof Dr kamaljeet Singh –(9415214108),/ Dr.B.N.Chaudhary 9760541507**
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Mobile: 9415210136
Email id: ashishjaswal@rediffmail.com
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Editor UP Journal of Ophthalmology:
Dr. Gunjan Prakash
Mobile: 09319204073
Email id: editorupjo@rediffmail.com
Dept of Ophthalmology
S N Medical College
Agra-282 003

Editor Proceedings:
Dr. Mohita Sharma
Mobile: 09560889495
Email id: drmohita@tirupatieye.org
Chief Ophthalmologist
Tirupati Eye Centre, Noida
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