BDOS SCIENTIFIC HIGHLIGHTS

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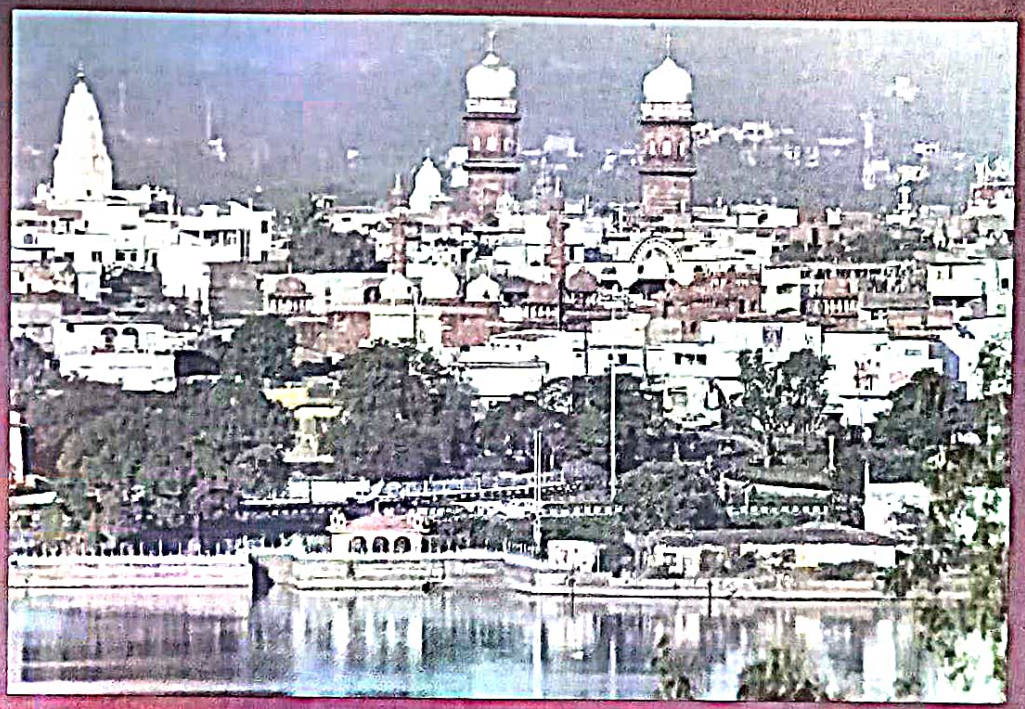

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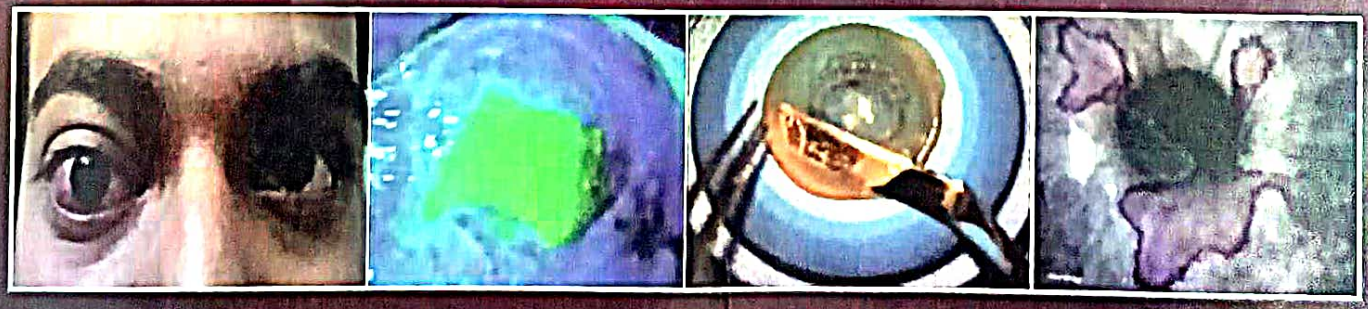

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BHOPAL DIVISIONAL OPHTHALMIC SOCIETY

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From the Editor'S Desk

Dear friends,

Happy Diwali and Prosperous New Year,

This issue of BDOS Scientific Highlight includes three articles on cornea as corneal blindness is third most common causes of blindness worldwide. It's our pleasure that peoples medical college ophthalmology department is really coming forward actively to provide articles especially the efforts from young budding ophthalmologist are encouraging. Our new member is also quite enthusiastic to share his experiences on DSEK.

One case report of rare case of cartico cavernous fistula and neurofibromatosis is also included to complete the list. I wish to extend heartfelt gratitude to all doctors for their articles and wish to encourage more and more ophthalmologists to participate in all BDOS activities.

I hope to see our BDOS to grow fast in all aspects with constant efforts, encouragement and support of all members. I am sure we can improvise further and incorporate many more activities in coming time.

With warm regards,

Dr. Vinita Ramnani
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CONTENTS:

PERSISTENT EPITHELIAL DEFECT (PED) OF CORNEA

Dr. Mary, Dr. Prakash Agarwal, Dr. V. K. Saini
Peoples College of Medical Sciences & Research Centre, Bhopal

Persistent epithelial defect (PED) of the cornea results when the anterior epithelium fails to grow over a defect within the expected time course (2-3 weeks).

Medications	Topical
	Systemic
Contact lens wear	
Chronic eye rubbing	
Traumatic	Thermal
	Radiation
	Mechanical
	Chemical (Fig. 1)
Metabolic	Intrinsic
	Acquired
Post infectious	
Immune	Rheumatoid Arthritis
	Collagen vascular diseases
	Skin disease
	Mooren's ulcer
Neurologic diseases	Neurotrophic
	Neuroparalytic
Tear film abnormality	Aqueous tear deficiency
	Mucin deficiency
	Lipid abnormality
	Lid surface abnormality
Neoplastic	Benign
	Malignant
Idiopathic	

Persistent epithelial defect (PED) results when the epithelium fails to grow over a defect within the expected time course (2-3 weeks).

Pathophysiology

As we all know the corneal epithelial are derived from the limbal stem cells residing at the limbus and conjunctiva. According to the XYZ hypothesis, the stem cells are actively dividing and then migrate circumferentially and centripetally towards the centre of the cornea. This multiplication and migration of epithelial cells is responsible for wound healing. A number of signaling and receptor mediated processes bring about the healing response.

Role of Fibrin and fibronectin: During trauma, there is damage to the hemidesmosomes of the neighboring cells. These cells then flatten and migrate. During migration of cells, focal temporary contacts are formed to stabilize the movement. These focal contacts are formed between cytoplasmic actin filament and extracellular matrix (ECM) proteins by protein vinculin. Degradation of these contacts is done by plasmin. Fibronectin is an ECM protein responsible in cell adhesion between cell surface receptors, collagen, heparin and fibrin.

Role of Plasmin: Increased activation of plasminogen leading to elevated concentration of plasmin at the defect leads to weak attachments of epithelium to matrix causing PED. Plasmin is a chemotactic substance attracting neutrophils and inflammatory cells and causes release of collangenase and lysosomal enzymes. This dissolves the basement membrane ultimately leading to sterile stromal ulceration.

Role of Growth Factors: Epidermal growth factors (EGF), transforming growth factor alpha and beta (TGF α and β), insulin like growth factor (IGF-1) and nerve growth factor (NGF) are the factors responsible for epithelial multiplication.

Role of enzyme: Collagenase enzyme which degrades type 1 collagen is produced by fibroblasts, endothelial cells and inflammatory cells. Matrix metalloproteinase enzyme(MMP) break components of extracellular matrix (ECM). In patients with recurrent erosion syndrome, MMP-2 is up-regulated. They degrade the basement membrane and lead to stromal ulceration.

Cause	Treatment
Medications	Stop all possible toxic medications. Consume preservative free Lubricants
Contact lens wear	Discontinue lens wear
Eye rubbing	Discontinue eye rubbing, lubricants, patching
Trauma	Remove offending agent, saline irrigation, supportive therapy
Infection Viral disease	Antibiotic therapy Antiviral topical systemic
Immune dz	Control of systemic dz, supportive Rx
Neurotrophic defects	Lubricants, auto serum drops, growth factors
Neurotrophic defects Meibomianitis Trichiatic eyelash	Tarsorrhaphy, Lid taping Oral Doxycycline, Lid hot fomentation and massage Removal, electrolysis
Entropion Ectropion	Lid surgery
Vernal Keratoconjunctivitis with Giant Papillae	Subtarsal Intratarsal Triceot inj. Papillae excision with AMG

Step ladder approach:

- Stop excessive topical medication, use preservative free antibiotic and lubricants.
- Remove offending cause & continue supportive therapy
- Repeated Pad/bandage for 24-48 hours with subconjunctival injection of gentamicin (0.5ml) & dexamethasone (0.5ml). (Try for one week)
- Soft bandage contact lens trial for one week.
- Topical human auto-serum in QID dose for one week (to be changed every 48 hrs) (figure 1)
- Amniotic membrane grafting (may be repeated if required, figure 2).

Newer therapy:

- Use of fibronectin, growth factors such as EGF, NGF, IGF etc
- Use of plasmin inhibitor such as aprotonin.
- Selectin blocker, intravenous fucoidin
- Steroids such as medroxy progesterone acetate.
- Non specific collagenase inhibitors such as acetylcysteine, calcium EDTA and penicillamine.
- Doxycycline (100 mg BD) and corticosteroids (oral Prednisolone 1mg/kg) – MMP inhibitors.
- Oral Ascorbic acid (Vitamin C 500 mg QID) for collagen synthesis and wound healing.

DESCEMET'S STRIPPING WITH ENDOTHELIAL KERATOPLASTY (DSEK): SHORTENING THE LEARNING CURVE

Dr. Prateek Gurjar, Sudarshan Netralaya, Bhopal

Endothelial keratoplasty (EK) has evolved tremendously in last few decades, with Descemet's stripping with endothelial keratoplasty (DSEK) as the most important breakthroughs in transplant surgery in the last 30 years. DSEK provides so many advantages compared with penetrating keratoplasty (PK) that the authors believe that all corneal surgeons should be performing it .[1,2] Earlier versions of EK like posterior lamellar keratoplasty (PLK) and deep lamellar endothelial keratoplasty (DLEK) technique involved manual recipient lamellar dissection and the excision of the posterior recipient stromal button using small curved scissors and trephine. These techinques did not provide as rapid visual recovery and were so technically difficult that few corneal surgeons were willing to perform them.[1] This was simplified by Melles by describing a Descemetorhexis technique that merely involved stripping Descemet's membrane and dysfunctional endothelium from the host cornea.[3] One further variation to DSEK involves a microkeratome to perform the donor lamellar dissection, a variation known as Descemet's stripping with automated endothelial keratoplasty (DSAEK).[4]

Surgical technique-The technique can be divided into three steps:

- Donor tissue preparation
- Recipient eye preparation
- Donor tissue insertion/Manipulation

It is highly recommended by the author to start the case with preparation of the donor tissue which allows the surgeon to make sure the donor tissue is suitable for transplantation before opening the

patient's eye. It is recommended to check for the following features in the donor corneal button before starting dissection:

Corneal/scleral rims should have a diameter of 16–17 mm to ensure firm and air-tight fixation on an artificial anterior.

The donor tissue should be examined carefully to ensure there are no divots or cuts through the peripheral cornea to the limbus that might allow the tissue to slip or depressurize during the dissection.

The donor tissue should have a good specular count preferably >2500cells/sq.mm.Lamellar dissection of the donor tissue can be performed manually or with a microkeratome, or the donor tissue can be pre-dissected by the eye bank. Artificial anterior chambers for donor tissue dissection are available from Bausch & Lomb, Moria, Katena as well as local manufacturers (fig.1).During dissection the surgeon should aim for approximately 80% dissection depth. Estimating the dissection depth can be a challenge with manual dissections. Melles recommended filling the anterior chamber with air to create a reflection that helps the surgeon gauge the dissection depth.[5] Some surgeons utilize this technique, while others prefer to use tissue storage solution, balanced salt solution, or viscoelastic in the artificial anterior chamber to help protect the corneal endothelium. To guide selection of an appropriate dissection depth, the donor thickness can be measured using ultrasonic pachymetry .The author recommends making a 3 clock hour initial peripheral incision using a 350/300 µm guarded blade. Then a series of curved dissecting blades of increasing length can be used to extend the dissection plane across the cornea (Fig 2&3). One should keep in mind that if the posterior donor button is too thin it will be difficult to manipulate and more prone to develop wrinkles that can be diffi cult to remove when applanated against the recipient cornea. Also, extremely deep donor dissections are more likely to damage the endothelium. For DSAEK, using microkeratome , the surgeon should aim for posterior donor button thickness of 0.10–0.18 mm. Depth plates (head sizes) are available that provide nominal dissection depths of 250, 300, 350, and 400µm (Fig 4).After dissection, the donor tissue is carefully removed from the artificial anterior chamber. One should avoid sudden decompression of the donor button while removing it from the anterior chamber. It is then placed endothelial side up on a standard punch trephine block, and punched to a diameter that has been selected based on the width of the recipient's cornea.The donor tissue can be covered with tissue storage solution while the recipient's eye is being prepared.

Recipient preparation

DSEK is often performed under peribulbar anaesthesia. Although some surgeons prefer general anaesthesia for a better control of intraocular pressure. With local anesthesia it is important to apply a pressure device to soften the eye. This becomes more relevant in phakic eye because back pressure can lead to forceful shallowing of the anterior chamber while the donor tissue is being inserted. This can not only damage the donor endothelial cells but can also damage the lens.The author recommends a 6 mm temporal scleral tunnel incision in the recipient eye. Temporal placement of the incision facilitates donor tissue insertion because the corneal diameter is longest in the horizontal direction. A 6mm incision allows insertion of donor tissue without folding with the technique described here.Whenever required the recipient epithelium can be removed to facilitate the view into the eye. The surface of the recipient cornea is gently marked with the trephine used to punch the donor button. Descemet's membrane is then scored in a circular pattern along the perimeter of the area to be removed, using a modified Sinskey hook. Trypan blue can be used to improve visualization of the descemet's membrane. The membrane is then grasped and removed using Mcpherson forceps. The membrane can be spread on the eye to determine whether removal was complete. During the scoring and stripping steps, the anterior chamber can remain formed by injecting viscoelastic or by intermittent or continuous infusion of balanced salt solution or air. If viscoelastic is used, it must all be completely removed before inserting the donor button because residual viscoelastic will impair donor adherence.

Donor tissue insertion--The donor tissue can be inserted in various ways

Folding the tissue in a taco fashion with endothelial side inwards. The folded donor taco is gently grasped with forceps and inserted into the recipient eye. Another way to insert the donor tissue is to pull the folded tissue into the eye from an incision 180° away using a suture.[5] Using Busin glide/ Endoglide[6,7]

Using 27 gauge needle bent at the tip to form a cystitome. This technique has small learning curve and avoids folding of the donor tissue. First the recipient bed is coated with a viscoelastic (2% hydroxyl propyl methyl cellulose), then the donor corneal tissue is placed over the bed with endothelial side down. With the cystitome the edge of donor button is engaged and gently advanced into the anterior chamber(Fig.5).Once completely inside the chamber the cystitome is disengaged and gently taken out. The chamber is then filled with air and scleral tunnel is secured with 3 interrupted 10-0 nylon sutures. The disc is then centered using modified Sinskey hook and a complete air fill is done. The sideports are also secured with 10-0 nylon sutures. The surface of the recipient cornea is then massaged to help center the donor button and move any entrapped fluid out of the donor–recipient interface. This is can even be done with Mcpherson forceps. Four small venting incisions can be made in the recipient cornea down to the graft interface to help drain any residual fluid trapped between the donor and recipient. It is recommended to remove some air at the end of 10 minutes. A residual air bubble approximately the same diameter as the donor button can then be left in the eye. This reduces the chances of pupillary block. The author recommends to have the patient lie face-up without pillow for 45-60 minutes after the procedure so that the residual air bubble can continue to press the donor tissue against the recipient cornea.The postoperative medications for DSEK remains more or less same as for PK, with topical corticosteroids continuing for several months. Visual recovery is relatively rapid and predictable after EK compared with PK, and seems to be faster with DSAEK compared with DLEK or even DSEK, probably because it produces the smoothest donor and recipient interfaces.The most frequent postoperative complications after DSEK/DSAEK includes donor tissue dislocation, Postoperative IOP rise, and graft rejection. The mean endothelial cell loss following DSEK/DASEK is around 37% at the end of 6 months.[9] Endothelial keratoplasty techniques continue to evolve to reduce these complications, with Descemet's membrane endothelial keratoplasty (DMEK) and Femto assisted endothelial keratoplasty being the latest additions.To conclude DSEK/DSAEK has significantly increased the benefits and reducedthe risks of transplantation for patients with endothelial dysfunction. The relatively rapid and predictable visual recovery and minimal postoperative restrictions make it the procedure of choice in cases of endothelial dysfunction.

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CASE REPORT : POST TRAUMATIC CAROTID CAVERNOUS FISTULA WITH EXOPHTHALMOS

DR GAJENDRA CHAWLA, DR VINITA RAMNANI-
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INTRODUCTION

Fistula is define as direct connection between arteries and veins. Carotid cavernous fistulas (CCF) are formed secondary to abnormal communications between the cavernous portion of the carotid artery and the venous plexus of the cavernous sinus. It is of two types spontaneous and traumatic. Traumatic fistulas usually result from injuries to the internal carotid artery in its course to cavernous sinus. These types of fistula frequently appears a few weeks after trauma with signs & symptoms related to increased venous pressure transmitted through the ophthalmic vein, a valve free vessel.

INCIDENCE & AETIOLOGY

1. **TRUMATIC CCF**: occurs rarely with incidence 0.2-0.3% following head and face trauma, it can be penetrating or blunt. It can be Iatrogenic following Surgical procedure like neural, ENT and vascular. Post traumatic direct CCF is common, represents 70-90 % of all cases of CCF and occurs because of single traumatic rent in the cavernous segment of ICA resulting in direct communication between ICA & cavernous sinus they are usually high flow and more common in young individuals.
2. **SPONTANEOUS:** Following Aneurysm rupture, can occur in associated with atherosclerosis, HTN, collegen vascular disorders and child birth. This dural variety is common in old females.

CLASSIFICATION:

1. According to hemodynamic – Blood velocity can be high flow/low flow
2. According to aetiology – spontaneous / traumatic (>75%)
3. Anatomical – Direct /dural CCF

Barrow (1985) classified CCF into 4 subtypes based on their communication

Type-A: Direct communication between ICA and cavernous sinus

Type-B: dural ICA branches and cavernous sinus

Type-C: Communication between dural external carotid artery branch and cavernous sinus.

Type-D: Communication between dural branches of ICA and external carotid artery branches to Cavernous sinus.

Barrow's Classification–

Typ	Pathogenesis	Arterial supply	Hemodynam
A	Head trauma aneurysm rupture (direct)	ICA direct	High flow
B	Spontaneous (Indirect)	ICA dural branches (Meningeal)	Low flow
C	Spontaneous (Indirect)	ECA dural branches	Low flow
D	Spontaneous (Indirect)	ICA & ECA dural branches	Low flow

(Precchawal et- in his study of 80 fistulas found –type B in14%, type C in15%, and type D in71% of his cases).

SIGNS & SYMPTOMS:

It can occur immediately following trauma or develops several days or week's later. Ocular presentation includes symptoms of eye pain, decreased vision, redness, bulging eyes and complaint of hearing noise in the head, diplopia, and headache. Ocular signs include conjunctiva chemosis, crockscrew episcleral vessels, arterialization of conjunctival vessels, pulsating exophthalmos, thrill and bruit, limitation of ocular movements, some time exposure keratopathy because of marked Proptosis, glaucoma and ophthalmoplegia (sixth nerve mainly). Fundus examination may reveal CRVO like picture retinal haemorrhages, retinal and disc oedema, non rhagmatogenous RD, choroidal detachment, Anterior ischemic optic neuropathy ultimately leading to blindness. Ocular symptoms and signs are because of decreased arterial **blood flow to orbit venous engorgement**. Sometime CRAO because of CCF blood in vein becomes arterilized, resulting in increased venous pressure and arterial pressure and decreased perfusion. (According to Precchawat et-all in his study of 80 patients with CCF, found more than one clinical

signs or symptoms like-43% with decreased vision,84% proptosis,93% arterialization of conjunctival vessels,42% chemosis,52% cranial nerve palsises,51% elevated IOP,13% optic neuropathy).

DIAGNOSIS: classical triad of proptosis, conjuctival chemosis and cranial bruit

1. History & high index of suspicion.
2. CT Scan/MRI Brain: Shows enlarged superior ophthalmic vein, thick extra ocular muscles, evidence of enlarged cavernous sinus with convexity of lateral wall.
3. Cerebral angiography: For confirmation of CCF. Bilateral selective angiography of both external & ICA for blood supply.
4. Colour Doppler images demonstrate arterialisation of blood flow in dilated SOV and return of normal venous flow after successful closure.

DIFFERENTIAL DIAGNOSIS–

1. Arterio venous malformation
2. Skull base tumours
3. Cavernous sinus thrombosis
4. Orbital cellulitis, mucormycosis, TB
5. Orbital tumours
6. Atypical red eye-chronic conjunctivitis.
7. Mucocele
8. Post traumatic retrobulbar haemorrhage and intraorbital foreign body
9. Peudotumor, wegners granulomatosis, polyarteritis nodosa, sarcoidosis, tolosa hunt syndrome

TREATMENT: Treatment is mainly done by neuro interventional radiologist. Various options for treatment includes-

1. Spontaneous closure: Rare mostly half of indirect low flow, in dural variety closes spontaneously, and only needed systemic diseases treatment.
2. Carotid compression therapy : successful in 17% direct, and 30% dural CCF.
3. Ligation of external & ICA
4. Fistula embolization with partical glue: Causes thrombosis of fistula by preserving ICA or Detachable balloons, Thrombogenic Microcoils (Platinum)
5. Non ballon embolization and electrothrombosis
6. Craniotomy: Direct surgical repair of CCF Direct fistulas are best treated by detectable balloons with 90% success but 30% can have ocular motor paresis for transient period.

COMPLICATIONS: Among the 80 patients treated by Preechawat et-all Intraoperative complications occurred in 3 patients- ophthalmic artery occlusion, cerebral infarction, and 8 patients experienced transient aggravation of symptoms like increased proptosis, increased IOP, choroidal detachments & venous stasis retinopathy. Ophthalmic vein thrombosis resulting in CRVO developed in 3 patients leading to profound visual loss.

Patients need to be followed up with ophthalmologist for

1. Need of transient increased symptoms & signs.
2. Persistent of any symptoms sign following embolizations like glaucoma, diplopia, exposure keratopathy.

PROGNOSIS: All sign and symptoms disappear within 6 months when fistula closes after successful embolization. Recanalization can occur. Proptosis & visual loss may never completely resolve in patients with direct CCF.

CASE REPORT- Young male presented at our centre with history of vehicular accident by motor cycle. Following accident patient was better; there were no unconsciousness or hospitalization. Ten days following accident he noticed double vision both eyes specially looking towards left side. Sixth nerve palsy on left side was detected and MRI brain was normal. One month following accident patient presented with enlarged size of right eyeball and redness, pain and sound in right ear. On examination vision was 6/6 both eyes, there was proptosis RE with bruit, marked conjuctival congestion and dilated prominent tortous vessels on temporal side. Ocular movements of both eyes were restricted on temporal sides and LE showed 20 degree convergent squint. Repeat MRI showed CARTICO CAVERNOUS FISTULA high flow post traumatic and angiogram was done, two months following trauma patient diagnosed with typical features of caratico cavernous fistula and was

PHOTOGRAPH OF PERSISTENT EPITHELIAL DEFECT (PED) OF CORNEA

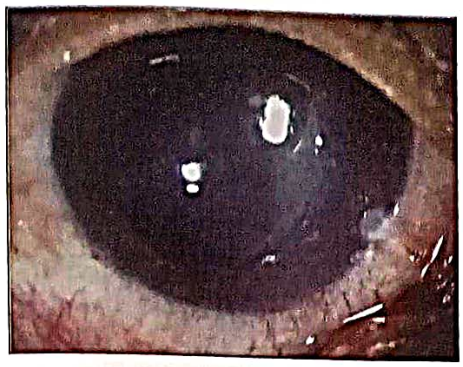


Fig. 1A: Chemical Injury

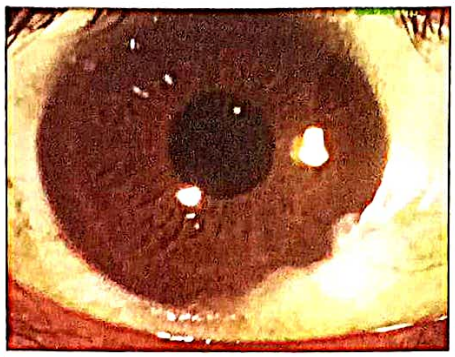


Fig. 1B: Healing with Autoserum

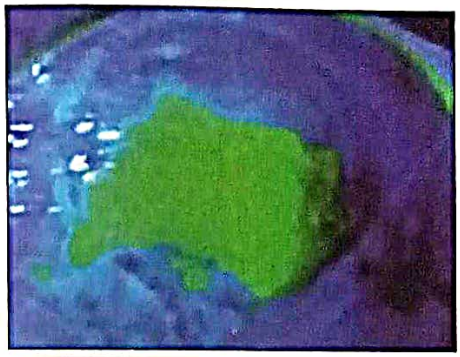


Fig 2A: Epithelial defect (HSV)

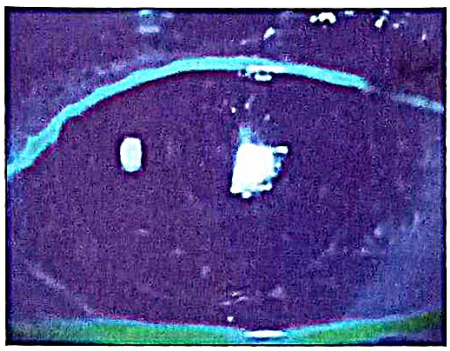
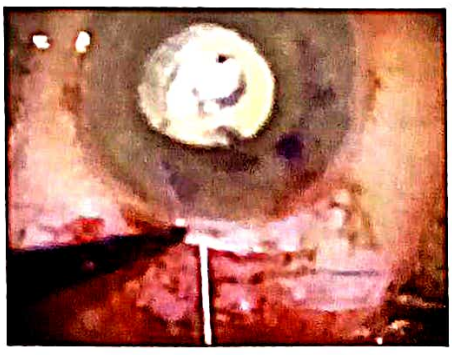


Fig. 2B: After Amniotic membrane grafting

PHOTOGRAPH OF DESCEMET'S STRIPPING WITH ENDOTHELIAL KERATOPLASTY (DSEK): SHORTENING THE LEARNING CURVE



PHOTOGRAPH OF CASE REPORT : POST TRAUMATIC CAROTID CAVERNOUS FISTULA WITH EXOPHTHALMOS

PHOTOGRAPH OF OCULAR MANIFESTATIONS OF NEUROFIBROMATOSIS

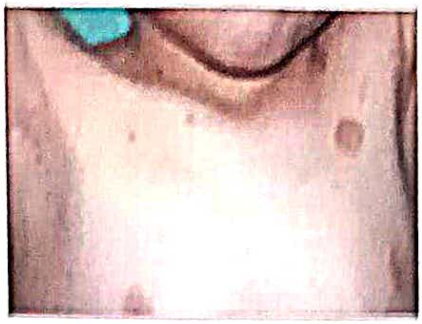


Fig- 1 , Café Au Lait spot

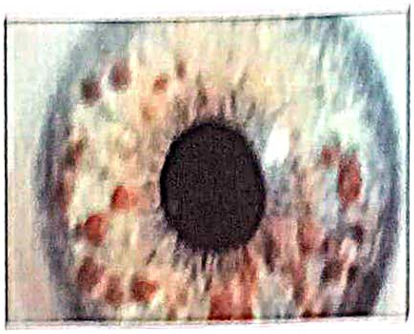


Fig- 3 Lisch nodule

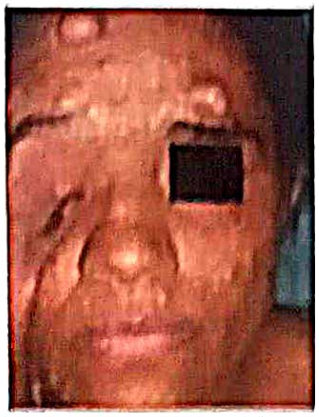


Fig -2 Eyelid neurobibroma

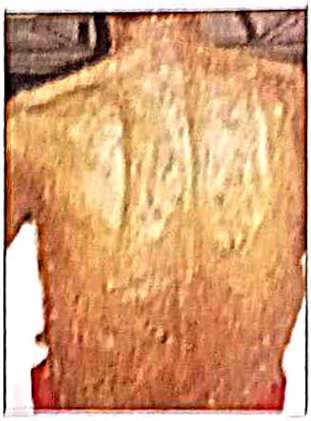


Fig. -4 Neurofibromas

Fig- 5, Multiple facial Neurofibromas

The Ophthalmic Quiz -1

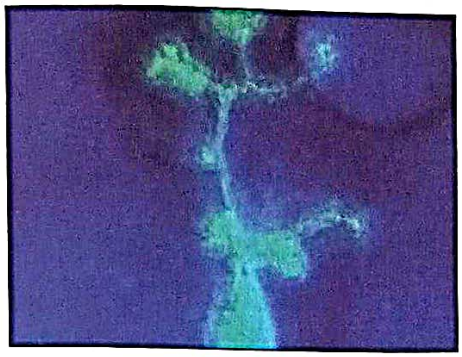
Answers– A. Corneal scraping in saline wet mount preparation showing trophozoite of a Protozoan parasite.

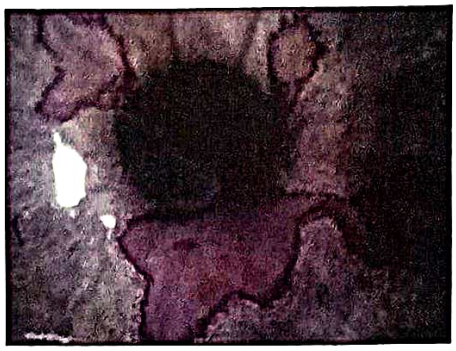
B. A case of Basal Cell carcinoma right lower eyelid

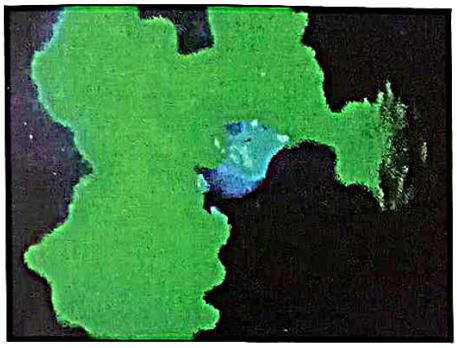
C. Proptosis left eye due to orbital cellulitis secondary to Ethamoid sinusitis .CT Scan shows subperiosteal abscess left orbit with opaque Ethamoid sinus .

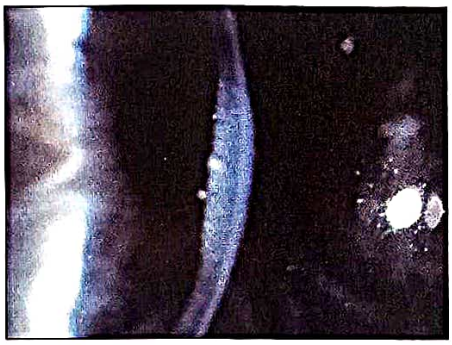
D. A case of CA maxilla left side.

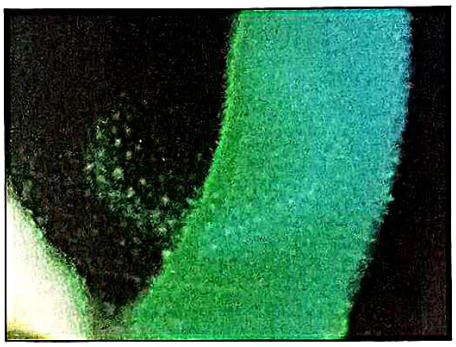
PHOTOGRAPH OF HERPES SIMPLEX KERATITIS REVISITED



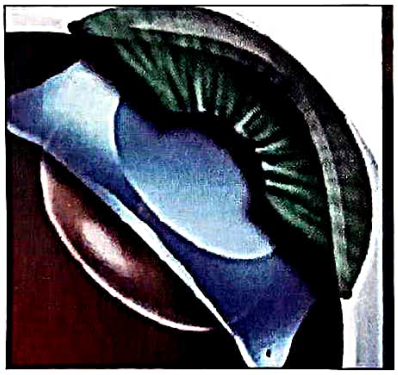


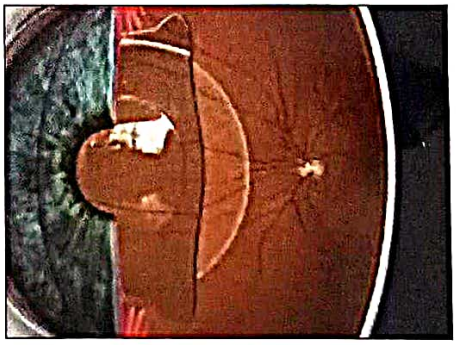


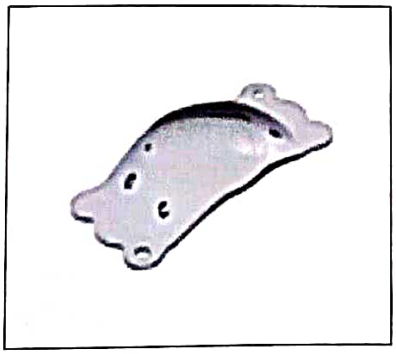


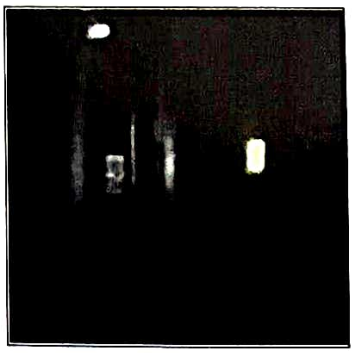


PHOTOGRAPH OF PEARLS FOR IMPLANTABLE CONTACT LENS









BHOPAL DIVISIONAL OPHTHALMIC SOCIETY

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Profile update Form

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Qualification : .....

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referred to neuroophthamic radiointerventionist. TRANS FEMORAL BALLON EMBOLIZATION was done with good results and subsequent followup after twenty days showed reduced proptosis with no obvious squint which were there in LE and full ocular movement on left side but there were little lateral restriction of movement of RE. on final followup two months following embolization patient was normal with 6/6 vision, no obvious squint and proptosis with full ocular movements and normal fundus examination.

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OCULAR MANIFESTATIONS OF NEUROFIBROMATOSIS

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Neurofibromatosis (NF) is a genetic disorder that disturbs the cell growth of nervous system[1]. NF is usually diagnosed in childhood and early adulthood.

There are three distinct types [2-5] of neurofibromatosis NF 1 is also known as Von Recklinghausen syndrome for many years it was first described by Dr Friedrich Von Recklinghausen in 1882, a German pathologist [6,8]. The disease is characterized by the growth of noncancerous tumors called neurofibroma which may be subcutaneous, involving brain, peripheral nervous system, eyes and orbit.[1]

Neurofibromatosis 1 is caused by the mutation of gene located in chromosome 17 [6,7]. The gene is finally responsible for activity of ras protein so mutation leads to abnormal cell division and formation of neurofibromatous tumors [2]. 50% cases are autosomal dominant and 50% are sporadic.

NF II is less common, sign and symptoms of NF II usually results from development of vestibular schwanomas also known as acoustic neuromas of both the ears.the gene of NF II is located on chromosome 22

Schwnomatosis-

Rare form of neurofibromatosis caused by mutation of SMAR CB I gene located on chromosome number 22.

It is caracterized by painful schwanomas of cranial, spinal and peripheral nerves. More commonly eighth cranial nerve is involved.

The diagnostic criteria for neurofibromatosis requires at least 2 of these 7 systemic signs [4,7]-

- Café-au-lait spots
- Auxillnodules of Iris
- Neurofibromas
- Plary and Inguinal freckling
- Lisch exlform Neurofibromas
- Optic nerve gliomas

Family history of distinctive osseous lesionsOther ophthalmic manifestation of neurofibromatosis 1 includes choroidal hamartomas, retinal tumors, prominent corneal nerves.

Visual loss due to optic nerve gliomas is the most important ophthalmic manifestation of neuro-fibromatosis 1.

LISCH NODULES-

It is most common type of ocular involvement in adults older than 20 years of age with neuro-fibromatosis 1 [11,12]. They are having following characteristics features-

- They are specific for neurofibromatosis 1
- Smooth usually bilateral elevated nodules
- Nearly all patients of neurofibromatosis 1 have lisch nodules by the age of 20 year

Benign hamartomas, histologically identical to iris neviOPTIC NERVE GLIOMA -15-40% of patients of NF 1 have optic nerve glioma , gliomas of visual pathway[9]. Bilateral optic nerve gliomas are pathgnomic of NF I. Patients of optic nerve gliomas usually present with headache, nausea, vomiting, blurred vision and nystagmus. On examination patients show RAPD, proptosis, squint, colour vision defect, optic disc pallor. Apart from clinical examination CT and MRI are important in making diagnosis of gliomas.

PLEXIFORM NEUROFIBROMATOSIS-

Plexiform neurofibromatosis of lid [10] is characterized by

- Thickening of upper lid
- 'S' shaped deformity
- Bag of worm sensation

Plexiform neurofibromatosis that infiltrate the orbit, temporal region and eyelids are potentially vision threatening. Patients may present with squint proptosis and amblyopia

CHOROIDAL HAMARTOMAS-

Hematomas of choroid are flat, ill defined lesions, usually present in the posterior pole and contain neur onal and melanocytic components.

RETINAL TUMOURS-

Astrocytic hamartomas, combined hamartomas of neurosensary retina and RPE, retinal capillary hemangiomas are common retinal tumors.

Patients of NF may also present with pulsatile proptosis due absence of greater wing of sphenoid. [13,14]

Diagnosis of NF is done by thorough clinical examination of patient and imaging with CT Scan and MR imaging [15,16].Treatment options include surgery, radiation and monitoring. To remove all or parts of tumor that is compressing nearby tissue or damaging organs.A patient of NF may present with variable symptoms depending on the site of lesion, a thorough systemic examination is must to make the diagnosis of NF. An individual with NF I has 50% risk of passing on the condition to an offspring but the clinical problems couldn't be predicted even in the families.[17]

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HERPES SIMPLEX KERATITIS REVISITED

Dr Nida Khan, Dr Prakash Agarwal
Peoples College of Medical Sciences & Research Centre, Bhopal

This review was done by done by Dr Nida Khan (PG student) and it lays down basics of Herpes simplex Keratitis (not Herpes Zoster). Cornea specialists Dr Ulka Shrivastav and Dr Prakash Agarwal have shared their valuable opinion on this common subject which will help all readers. Dr Ulka Shrivastav is the Head of Ophthalmology Dept at MGMC, Indore and has immense experience and expertise in corneal diseases and transplantation. Dr Prakash Agarwal has done his corneal fellowship from AIIMS, New Delhi followed by short term training at MEEI, Boston and ULCA, California.

Introduction - Herpetic eye disease is the one of common infectious cause of corneal blindness in developing countries. As many as 60% of corneal ulcers in developing countries may be the result of herpes simplex virus and 10 million people worldwide may have herpetic eye disease. At the outset we need to be clear that Herpes simplex keratitis is different from Herpes zoster ocular involvement. Both have different pathogenesis, clinical symptoms and management strategies.

Microbiology & Types of Herpes simplex virus (HSV)- HSV is enveloped with a cuboidal capsule and has a linear double-stranded DNA genome. The two subtypes are *HSV-1* and *HSV-2*, and these reside in almost all neuronal ganglia. *HSV-1* causes infection above the waist (principally the face, lips and eyes), whereas *HSV-2* causes venereally-acquired infection (genital herpes). Rarely *HSV-2* may be transmitted to the eye through infected secretions, either venereally or at birth (neonatal conjunctivitis). HSV transmission

is facilitated in conditions of crowding and poor hygiene.

Primary infection-Primary infection, without previous viral exposure, usually occurs in **childhood** and is spread by droplet transmission, or less frequently by direct inoculation. Due to protection bestowed by maternal antibodies, it is uncommon during the first 6 months of life, though occasionally severe neonatal systemic disease may occur. Most primary infections are subclinical or cause only mild fever, malaise and upper respiratory tract symptoms. Blepharitis and follicular conjunctivitis may develop but are usually mild and self-limited. Treatment, if necessary, involves topical acyclovir ointment for the eye and/or cream for skin lesions.

Recurrent infection-**After primary infection** the virus is carried to the sensory ganglion for that dermatome (for example trigeminal ganglion) where a latent infection is established. Latent virus is incorporated in host DNA and cannot be eradicated. The virus may be reactivated periodically during times of stress or otherwise. A variety of stressors such as fever, hormonal change, ultraviolet radiation, trauma, or trigeminal injury may cause clinical reactivation, when the virus replicates and is transported in the sensory axons to the periphery and clinical disease is seen. **The rate for ocular recurrence** after one episode is about 10% at 1 year and 50% at 10 years. The higher the number of previous attacks the greater the risk of recurrence.

Tip: The clinical Herpes Simplex Keratitis in adults that we commonly encounter is always secondary reactivation of a latent Herpes simplex virus.

Types or Pattern of Clinical Disease-It may be of following types:

1. Epithelial involvement
2. Stromal involvement – Disciform or Necrotising
3. Iridocyclitis
4. Neurotrophic

Epithelial keratitis--*Clinical features*

Epithelial (dendritic or geographic) keratitis involves the epithelium of cornea and is associated with active virus replication.

1. **Presentation** may be at any age with mild discomfort, redness, photophobia, watering and blurred vision.
2. **Signs** in chronological order:
   - Swollen opaque epithelial cells arranged in a coarse punctate or stellate pattern.
   - Central desquamation results in a linear-branching (dendritic) ulcer, most frequent located centrally. (Figure 1)
   - The ends of the ulcer have characteristic terminal buds and the bed of the ulcer stains well with fluorescein.
   - The virus-laden cells at the margin of the ulcer stain with rose bengal. (Figure 2)
   - Corneal sensation is reduced.
   - **Inadvertent topical steroid treatment may promote progressive enlargement of the ulcer to a geographical or 'amoeboid' configuration.** (Figure 3)
   - Mild associated subepithelial haze is typical.
   - Elevated IOP may occur.
   - Following healing, there may be persistent punctate epithelial erosions and irregular epithelium which settle spontaneously and should not be mistaken for persistent active infection. A whorled epithelial appearance can also result from assiduous, especially prolonged, topical antiviral instillation.
   - Mild subepithelial scarring may develop after healing.
3. **Differential diagnosis** of dendritic ulceration includes herpes zoster keratitis, healing corneal abrasion (pseudodendrite), acanthamoeba keratitis, epithelial rejection in a corneal graft, tyrosinaemia type 2, use of soft contact lenses and toxic keratopathy secondary to the topical medication

Treatment

Treatment of HSV disease is predominantly with nucleoside (purine or pyrimidine) analogues that are incorporated to form abnormal viral DNA. Aciclovir, ganciclovir and trifluridine have low toxicity and approximately equivalent effect. Idoxuridine and vidarabine are more toxic to the epithelium. The majority of dendritic ulcers will eventually heal spontaneously without treatment, though scarring and vascularization may be more significant with more prolonged disease.

1. **Topical**. The most frequently used drugs in India are aciclovir 3% ointment and ganciclovir 0.15%

gel, each administered 5 times daily. Trifluridine is preferred in the United States and requires instillation up to nine times a day. The drugs are relatively non-toxic, even when given for up to 60 days, acting preferentially on virus-laden epithelial cells, and penetrating effectively into the stroma. On this treatment 99% of cases resolve by 2 weeks.

2. **Debridement** may be used for dendritic but not geographic ulcers. The corneal surface is wiped with a sterile cellulose sponge 2 mm beyond the edge of the ulcer, since pathology extends well beyond the visible dendrite. The removal of the virus-containing cells protects adjacent healthy epithelium from infection and also eliminates the antigenic stimulus to stromal inflammation. An antiviral agent must be used in conjunction.
3. **Oral antiviral** therapy is probably indicated in most immunodeficient patients and may also be effective alternatives to topical treatment when the latter is poorly tolerated, or in resistant cases. If required dose is 400 mg Acyclovir 5 times a day.
4. **Skin lesions** may be treated with aciclovir cream five times daily, as for cold sores, and if extensive an oral antiviral may be given.
5. **Topical steroids** are not used unless significant disciform keratitis is also present.
6. **Slow healing or frequent recurrence** may indicate the presence of a resistant viral strain, and a combination of two topical agents with oral valaciclovir or famciclovir may be effective. A significant minority of cases are due to varicella-zoster virus.

Expert opinion / Experince (Dr Ulka Srivastav) : To prevent recurrence patient should be instructed to use lubricant liberally and avoid rubbing of eye as corneal sensation are reduced in these eyes.

Expert opinion/ Experience (Dr Prakash Agarwal): Corneal ulcer without history of trauma or any predisposing history may be a pointer towards Viral cause. Clinical findings such as central location, lack of profuse discharge, loss of corneal sensations are subtle but important pointers. The dose for HSV is 400 mg Acyclovir (800 mg generally not required.

Disciform keratitis

The exact aetiology of disciform keratitis (endotheliitis) is controversial. It may be active HSV infection of keratocytes or endothelium, or a hypersensitivity reaction to viral antigen in the cornea. A clear past history of epithelial ulceration is not always present.

Clinical features

1. **Presentation** is with a gradual onset of blurred vision which may be associated with haloes around lights. Discomfort and redness are common, but tend to be milder than in purely epithelial disease.
2. Sign
   - A central zone of stromal oedema often with overlying epithelial oedema ; occasionally the lesion is eccentric. (Figure 4)
   - Keratic precipitates underlying the oedema .
   - Folds in Descemet membrane in severe cases.
   - A surrounding (Wessely) immune ring of stromal haze signifies deposition of viral antigen and host antibody complexes. (Figure 5)
   - The IOP may be elevated.
   - Reduced corneal sensation.
   - Healed lesions often have a faint ring of stromal or subepithelial opacification and thinning.
   - Consecutive episodes may be associated with gradually worsening subepithelial and/or stromal scarring and superficial or deep vascularization.

     Mid-stromal scarring can give the appearance of interstitial keratitis.

Treatment

1. **Initial** treatment is with topical steroids (prednisolone 1% or dexamethasone 0.1%) with antiviral cover, both q.i.d. As improvement occurs, the frequency of administration of both is reduced in parallel over not less than 4 weeks.
2. **Subsequently** prednisolone 0.5% once daily is usually a safe dose at which to stop topical antiviral cover. Some patients require a weaker steroid such as fluorometholone 0.1% or loteprednol 0.2% on alternate days for many months. Periodic attempts should be made to stop the steroid altogether.
3. **With active epithelial ulceration** it is reasonable to try to keep the steroid intensity as low as possible for adequate effect, with a more intensive antiviral regimen such as five times daily, with steroid b.d. or t.i.d., titrated according to the signs of activity.
4. **Topical ciclosporin** 0.05%may be useful,

particularly in the presence of epithelial ulceration and to facilitate tapering of topical steroids such as in steroid-related IOP elevation.

5. Oral Steroids are not advocated.

Expert opinion / Experince (Dr Ulka Srivastav) : To avoid long term use of steroids topical cyclosporin is very good alternative

Expert opinion/ Experience (Dr Prakash Agarwal): Localised edema of cornea with few KP's is the most characteristic sign. Patient may require steroids under observation for 2-3 months before complete healing occurs. Oral steroids should not be used as it may exacerbate the viral disease.

Necrotizing stromal keratitis-This rare condition is thought to **result from active viral replication** within the stroma, though immune-mediated inflammation plays a significant role. It may be difficult to distinguish clinically from severe disciform keratitis and there may be a spectrum of disease, including overlap with neurotrophic keratopathy. One should be wary that a similar clinical picture may be caused by other infections.

1. **Signs**
- Stromal necrosis and melting, often with profound interstitial opacification.
- Anterior uveitis with keratic precipitates underlying the area of active stromal infiltration.
- An epithelial defect may be present.
- Progression to scarring, vascularization and lipid deposition is common.

2. **Treatment** is broadly similar to that of aggressive disciform keratitis, but **oral antiviral supplementation**, initially at the upper end of the dose range, may be beneficial. The restoration of epithelial integrity is critical.

- **Neurotrophic ulceration-**Neurotrophic ulceration is caused by failure of re-epithelialization resulting from corneal anaesthesia, often exacerbated by other factors such as drug toxicity.

1. **Signs**
- A non-healing epithelial defect, sometimes after prolonged topical treatment. The stroma beneath the defect is grey and opaque and may become thin. Secondary bacterial or fungal infection may occur.

2. **Treatment** is that of persistent epithelial defects; topical steroids to control any inflammatory component should be kept to a minimum.

Expert opinion / Experince (Dr Ulka srivastav) : It is better not to use bandage contact lenses in these cases as any irritation caused by it will not be felt by patient producing more deterioration.

Expert opinion/ Experience (Dr Prakash Agarwal):

It results in thinning of cornea. Thinning is a characteristic sign for necrosis. Treat aggressively with antivirals along with supportive therapy.

Prophylaxis-Long-term daily oral aciclovir reduces the rate of recurrence of epithelial and stromal keratitis by about 50% and is usually well tolerated. Prophylaxis should be considered in patients with frequent debilitating recurrences, particularly if bilateral or involving an only eye. The standard daily dose of aciclovir is 400 mg b.d. but if necessary a higher dose can be tried. Oral valaciclovir (500 mg once daily) or famciclovir are alternatives. The prophylactic effect decreases or disappears when the drug is stopped.

Complications

1. **Secondary Infection.** Herpetic eye disease is a major predisposing factor for microbial keratitis.

2. **Glaucoma** secondary to inflammation or chronic steroid use may progress undetected, particularly if there is a poor view of the optic disc. Corneal thinning and distortion may give rise to an inaccurate reading on applanation and a Tono-Pen™ may be superior in these cases. 1. **Topical** antivirals given during a rejection episode may reduce epithelial viral reactivation but toxicity may delay re-epithelialization.

2. **Prophylactic oral aciclovir** (400 mg b.d.) improves graft survival and should be given to patients undergoing penetrating keratoplasty.

Herpetic Eye Disease study (HEDS)-HEDS study provided guidelines to manage HSV Keratitis. The prominent conclusions of HEDS study are:

- Topical steroids have a role in treatment of stromal keratitis (disciform) and may be used judiciously as mentioned above.
- Oral acyclovir may not be required in all cases of herpes simplex stromal keratitis.
- Oral acyclovir is beneficial in iridocyclitis due to HSV.

ARLS FOR IMPLANTABLE CONTACT LENS

R. K. Gupta, Dr. Saroj Gupta Siddharth Eye Hospital, Bhopal

Introduction –

For most patients requiring correction of their refractive error, LASIK is a safe and effective method of correcting myopia, hypermetropia and astigmatism.In fact, LASIK surgery is still the most common procedure to help those who want to eliminate their dependence on spectacles and contact lenses. However, some patients are not good candidates for LASIK because they have high refractive error, have thin corneas, or have other conditions that might predispose them to complications with LASIK.

It also has drawbacks of lack of predictability, regression, corneal ectasia and induction of higher order abrasions.

In these patients, the Implantable Contact (collamer) Lens (ICL)

might be the best option for correcting their vision.

What is an ICL-

ICL was first developed in late 1980's in Russia by Dr Fyodorov and first implant was placed in Europe in 1993. Fyodorov introduced the concept of a soft Phakic lens in the space between iris and anterior surface of the crystalline lens. It is a single piece soft thin posterior chamber phakic intra ocular lens, which can be inserted in to the eye through sub 2.8mm incision.

The lens is customized according to size of the each eye separately. The power correction range is from +10 D to -25D with cylinder up to 8D. The lens is made of collamer.

Candidates suitable for ICL-

1. Age between21 and 40 years.
2. Stable refraction for one year
3. Candidate with high refractive error or thin corneas who are unsuitable for laser refractive surgery.
4. Endothelial cell count > 2000 cells/cu mm.
5. Anterior chamber depth > 2.80 mm.

Candidates not suitable for ICL-

1. Those with shallow anterior chamber depth < 2.8mm
2. Those with low endothelial cell density < 2000cells/cumm
3. Presence of anterior or posterior synechia.
4. IOP higher than 20mm Hg or Glaucoma
5. Myopia other than axial myopia
6. Any evidence of nuclear sclerosis or developing cataract
7. Family history of retinal detachment.
8. History of Diabetes Mellitus.
9. Women who are pregnant or nursing.
10. Any other associated ocular pathology.

Preoperative assessment of ICL-

1. Assessment of refractive error both subjective and objective.
2. Measurement of AC depth.
3. Detail anterior and posterior segment examination.
4. Keratometery and Corneal Topography.
5. IOP measurement.
6. White to white measurement – most important .It can be done with Pentacam, Orbscan, UBM, or digital calipers.

Indications for ICL-

1. Myopia of -3D to -25D
2. Astigmatism up to -6D
3. Hypermetropia

4. Presbyopia (Multifocal ICL)

Advantages of ICL-

- Preserves accommodation
- Excellent quality of vision
- Cosmetically good

It is implanted behind the iris so-

- Far from the corneal endothelium
- Close to the nodal point of eye
- Gain in retinal image size
- Greater effective optical zone

It is placed in sulcus so-

- Stable location
- Easy removable/exchangeable
- Not fixated to any tissue (iris)

Sizing of ICL-

Size of ICL depends on shape and size of eyeball. If the ICL is properly sized (white to white and AC depth are important) then a separation or a gap will be there between the posterior surface of ICL and the anterior surface of the human lens. This space is called vault. New studies states that in sizing the ICL a direct measurement of sulcus diameter by UBM improve vault height predictability and hence safety of ICL instead of white to white measurement . An ideal sized ICL will provide a vault within a range of 0.250 to 0.750 mm (½ corneal thickness to 1& ½ corneal thickness) . An undersized ICL < 0.125mm vault may cause cataract and an oversized ICL >1mm vault may lead to glaucoma Iris chaffing and pigment dispersion. This vault is measured with Anterior segment OCT.

The Procedure-

A peripheral Iridotomy is performed 1-2 weeks before surgery to provide an outlet for aqueous humor flow around the lens. Alternatively it may be performed intra operatively after ICL implantation with vannas scissors or a vitrecomy cutter. It should be sufficiently wide and positioned superiorly at 11 and 1 o'clock position.

The procedure is performed under topical anesthesia. After making a side port incision, a 2.8mm clear corneal incision is made in steep axis .The lens is introduced through injector and positioned behind the iris on a horizontal axis. The viscoelastic material is completely removed and incision is closed by hydrating the corneal incision.

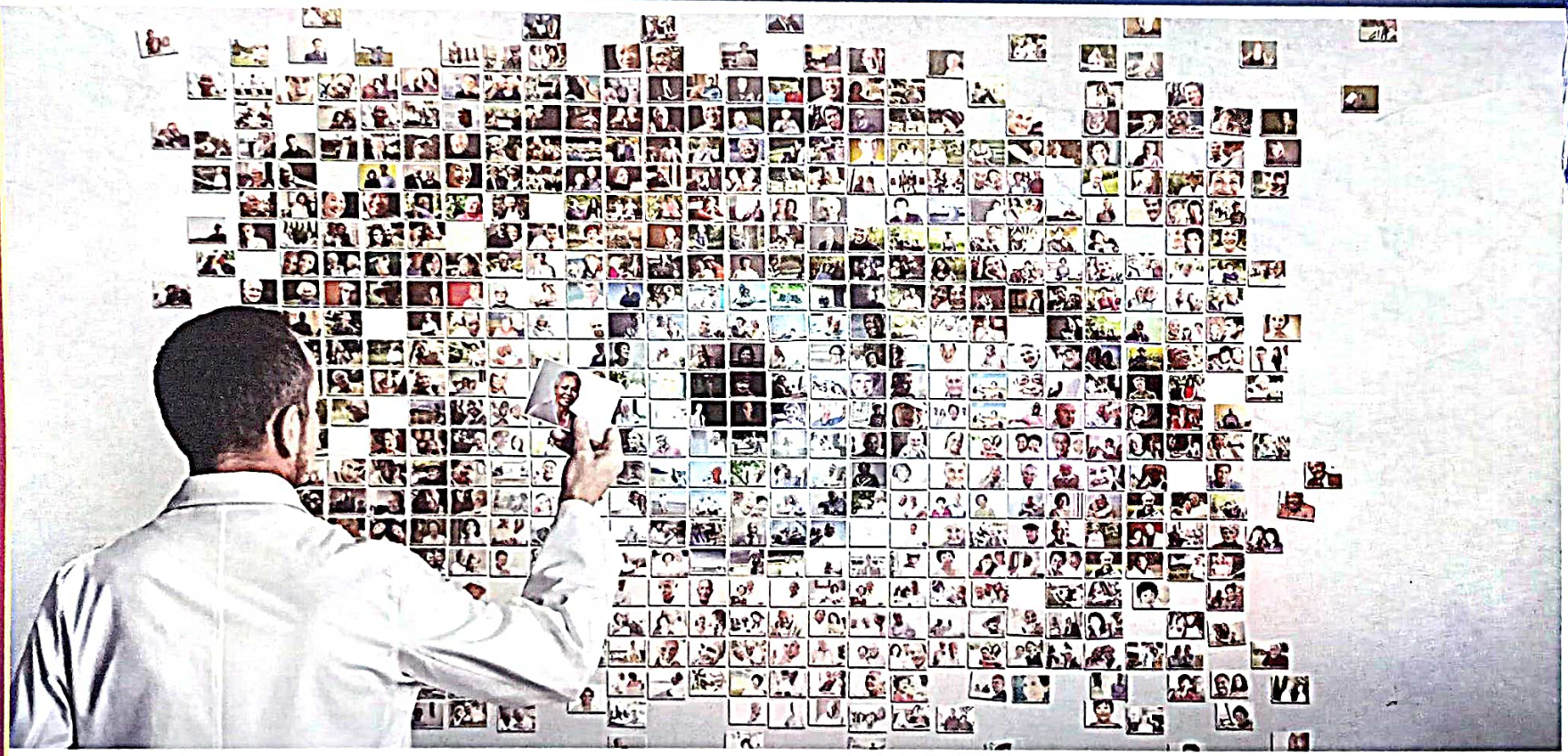
Potential ICL risks include -

1. Over correction: This complication occurs if the power of the implanted ICL is too strong. In most cases it can be corrected with corrective spectacles, contact lens or with an ICL replacement.
2. Under correction: The under correction is the result of an implantable contact lens with too weak of a prescription. Correction methods are similar to those of overcorrection.
3. Infection: During most surgeries, there is a potential risk of an infection.
4. Increased intraocular pressure: If ICL is too long then lens vault is more resulting in angle crowding and angle closure glaucoma. Pressure may build in the eye after an ICL procedure. The sooner a surgeon is alerted to this, the greater the chance of avoiding serious damage. This is detected during follow up visits with or in case there is acute blurring of vision or headaches.ICL may rarely need to be repositioned.
5. Damage to crystalline lens: Because implantable contact lenses are implanted into the Phakic eye, there is a potential that the eye's natural lens may be damaged during the procedure. If the damage is severe, the crystalline lens may need to be replaced with an intraocular lens.

If lens vault is less due to short ICL then also there is a risk of anterior capsular cataract formation.

The ICL is safe and effective modality for correction of high refractive error and for patients with thin cornea with excellent and stable post operative results.

See your success in the eyes of your patients



Defining the standard of care in the treatment of visual impairment due to DME

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