Ocular Surface Disorders - Current Concepts and Management

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Ocular Surface Disorders

Ocular surface disorders are a group of disorders of diverse pathogenesis in which, the disease results in failure of mechanisms that maintain a healthy ocular surface. (Fig. 1)

Ocular Surface

The ocular surface refers to the mucosal lining between upper and lower eyelids. The functional unit is comprised of tear film, conjunctival epithelium, limbal epithelium, corneal epithelium, lacrimal gland and meibomian gland (Fig. 2).

The 2 components essential for maintaining ocular surface health are

(1) Healthy ocular surface epithelium and
(2) Normal, stable precocular tear film

These act as a vicious cycle.

Ocular Surface Epithelium

This includes corneal, limbal and conjunctival epithelium. Of these, most important are the limbal stem cells since these maintain the dynamic equilibrium (Fig 3).

Fig. 1. OSD with unhealthy ocular surface

Fig. 2. Lacrimal gland and drainage apparatus

Fig. 3. Ocular surface epithelium

Fig. 4. XYZ Hypothesis of Thoft and Friend

The cells at the limbus are the proliferative cells. These include the SC (Stem Cells) which have the unique
property of self renewal and the TAC (Transiently Amplifying Cells) that are derived from mitotic division of stem cells and amplify by undergoing a few rounds of cell division. Cells in the non proliferative compartment are the PMC (Post Mitotic Cells) which are in the different stages of maturation by differentiation into the TDC (Terminally Differentiated Cells). This follows the limbal stem cell X,Y,Z hypothesis proposed by Thoft & Friend in 1983 (Fig. 4).

Ocular Surface Defence

The 2 factors essential for the ocular surface defence are

(1) Normal, adequate and stable tear film and
(2) Normally functioning hydrodynamic factors

The tear film consists of mucin layer, aqueous layer and lipid layer (Fig. 5).

Hydrodynamic factors include periodic, adequate and complete lid blinking to distribute an even tear film over the ocular surface and proper tear clearance to ensure adequate turnover and refreshment. Thus, the eyelids, the external adnexal glands and the ocular surface epithelia all play a major role in maintaining a normal tear film and ocular surface (Fig. 6).

Two neuronal reflex arcs function in this process. For both the arcs, the 1st branch of trigeminal nerve controls the ocular sensitivity as the afferent sensory input and the parasympathetic branch and the motor branch of the facial nerve are the afferent output (Fig. 7).

Fig. 5. Layers of tear film

Fig. 6. Normal ocular surface breaks in the tear film

Fig. 7. Neoronal feed back loop on which normal tearing depends

These 2 components are interrelated. An alteration in the quantity or quality of any of the elements of the tear film can lead to an unstable tear film and secondary changes in the epithelium. Vice versa, primary changes of the ocular surface epithelium as part of ocular surface failure can lead to a secondary dry eye. Thus, an intimate relationship exists between the two and any change in this can lead to the occurrence of various ocular surface disorders.

Ocular Surface Failure - Etiopathogenesis

2 major types of ocular surface failure have been identified based on the epithelial phenotype in impression cytology.

(1) With intact limbal stem cells
(2) With limbal stem cell deficiency

With intact limbal stem cells

Here, the normal nonkeratinized ocular surface epithelium undergoes squamous metaplasia into keratinized epithelium. This is also associated with loss of goblet cells and mucin expression. This altered epithelial differentiation renders the ocular surface epithelium non wettable. This leads to an unstable tear film, the hallmark of various dry eye disorders. This is usually due to poor ocular surface defense and dry eye may be secondary to:

(a) Aqueous tear deficiency
   - Idiopathic – Age related, Hormonal
   - Hyponutritional – Vit.A deficiency
Sensory denervation – After surgery or keratitis
Collagen vascular diseases – Rheumatoid arthritis, Wegeners, SLE
Sjogren’s syndrome and other autoimmune disorders
Drugs – Oral Contraceptives, Antidepressants, Antihistamines, Beta blockers
Lacrimal gland infiltration – Amyloidosis, Sarcoidosis, Tumors
Lacrimal gland fibrosis – Radiation
Contact lens use

(b) Lipid tear layer deficiency
Blepharitis
Acne rosacea
Contact lens

(c) Mucin tear layer deficiency
Vit.A deficiency
Trachoma
Mucocutaneous disorders
Contact lens
Conjunctival scarring – Ocular pemphigoid, Steven Johnson Syndrome, Chemical burns
Topical medications

(d) Increased tear film evaporation
Lagophthalmos
Ectropion
Computer vision syndrome
Lid retraction
Exophthalmos
Defective sensation

(e) Delayed tear clearance
Obstruction of tear outflow
Ineffective lacrimal pump

With limbal stem cell deficiency
Here, the normal corneal epithelium is replaced by conjunctival epithelium. The salient features here are conjunctivalisation, vascularisation, chronic inflammation, poor epithelial integrity manifested as an irregular surface, recurrent erosion and persistent ulcer, destruction of basement membrane and fibrous ingrowth. Conjunctivalization is the hallmark of limbal stem cell deficiency. Conditions with limbal stem cell deficiency can be classified in terms of the following.

(a) Primary limbal stem cell deficiency
These patients exhibit a gradual loss of limbal stem cell function over time. Seen in association with Peripheral keratitis
Pterygium/pseudopterygium
Aniridia
Neurotrophic keratopathy

(b) Secondary limbal stem cell deficiency
These patients have a clear pathogenic cause that is responsible for destruction of limbal stem cells.
Chemical / Thermal burns
Steven Johnson Syndrome
Ocular rosacea
Ocular pemphigoid
Contact lens wear
Multiple surgeries/Cryotherapies
Antimetabolite (5 FU) toxicity
Etiology – Common factors
Aging
Hormonal - Post menopausal females
Excessive computer use – reduced blinking
Excessive use of contact lens
Eye surgeries, injuries
Drugs
Keratoconjunctivitis sicca

Classification

The Madrid Triple Classification of Dry Eye
Dry eye classified depending upon 3 factors – Etiopathogenesis, Anatomo-pathologic and Severity
The features are:

A. Classification according to etiopathogenesis

The etiologic factors are divided into 10 groups:

1. Age related – With aging, all cellular structures of body undergo a progressive apoptosis including lacrimal glands. The lacrimal secretion begins to diminish from the age of 30 years and becomes insufficient for the necessities by 60 years.

2. Hormonal – Lacrimal secretion is affected by some endocrine gland activity, the most important of which are androgens, estrogens and prolactin. Aqueous and lipid secretions are the most affected.
3. Pharmacologic – Systemic – Antidepressants (Fluoxetine, Imipramine), Anxiolytics (Bromazepam, Diazepam, Clorazepate), Antiparkinsons (Bipiretin, Benztropine), Diuretics (Chlorthalidone, Frusemide), Antihypertensives (Clonidine, Chlorothiazide), Anticholinergics (Atropine, Metoclopramide), Antihistaminics (Dexchlorpheniramine, Cetirizine), Antiarrythmics (Disopyramide, Mexiletine)

Topical – Preservatives (Benzalkonium chloride, Thiomersal, Chlorobutanol, EDTA), Anasthetics (Tetracaine, Proparacaine, Lidocaine)

4. Immunopathic – Autoimmune disorders –

(1) Primary Sjogren’s syndrome – those preferentially affecting glands – where vasculitis by immune complex deposits, pseudolymphomas and lymphomas are sometimes associated.

(2) Secondary Sjogren’s syndrome which includes Rheumatoid Arthritis, Systemic Lupus erythematosis, Dermatomyositis, Scleroderma etc.

(3) Autoimmune attack of other tissues and secondary destruction of glands as in Steven Johnsons Syndrome, Ocular pemphigoid etc.

(4) Affecting other tissues – Thyroid and adrenal insufficiency


6. Dysgenetic – Genetic and congenital diseases that affect one or several types of dacrtyoglands – Aqueoserous (Alacrima, Dysplasia ectodermica anhidrotica), Lipid (Blepharophimosis syndrome, Keratopathy- ichyosis- deafness syndrome, First branchial arc syndrome), Mucin (Aniridia, Bietti syndrome), Ocular surface epithelium (Meesmann dystrophy, Cogan microcystic dystrophy)

7. Inflammatory/Infectious – Dacryoadenitis, Blepharitis, Trachoma, H.simplex, H.zoster

8. Traumatic – Surgical, Chemical, Radiation induced, Accidental

9. Neurologic – Lacrimal secretion is very dependent on nervous stimulation.

(1) Hypothalamic and limbic influences – Circadian rhythm of tear production that is maximum at morning and noon and minimum at night. Limbic influences such as anxiety, tiredness, psychic influences and somnolescence diminish the basal tear secretion.

(2) Afferent neurodeprivation – Any condition causing ocular surface anesthesia diminishes lacrimal secretion.

(3) Efferent neurodeprivation – Trauma, Tumors, Botulinum toxin injection.

10. Tantalic – These patients despite having enough tears, have a dry ocular surface. There are 3 types of tantalic dry eyes:

(1) Lid-eye incongruency – Lid cannot create, maintain and reshape the tear film onto the ocular surface as in lid palsy, ectropion, lagophthalmos, lid coloboma, exophthalmos, local protrusion of pterygium or dermoid cyst etc.

(2) Epitheliopathic – Epithelial dystrophies, limbal deficiency, Corneal conjunctivatisation, Endothelial decompensation etc. makes corneal epithelium less wettable.

(3) Evaporation – Environmental conditions like hot dry climates, excessive air conditioning, open car window etc.

Most of the dry eye conditions are multifactorial

B. Classification according to damaged glands and tissues

The affected parts of the lacrimal basin may be summarized in this histopathologic classification with the acronym ALMEN;

A – Aqueoserous deficiency
L – Lipid deficiency
M – Mucin deficiency
E – Epithelial deficiency
N – Nondacryologic exocrine deficiencies

C. Classification according to severity

In the initial Madrid classification severity of dry eye was divided into 5 grades:
Subclinical – Symptoms only when overexposure
Mild – Habitual symptoms
Moderate – Symptoms plus reversible signs
Severe – Symptoms plus permanent signs
Disabling – All of the above plus visual incapacity

**Recent Triple Classification of dry eye for practical clinical use**

In 8th congress of the International Society of Dacryology and Dry eye (Madrid, April, 2005), the previous Triple classification of dry eye approved in the XIV congress of European Society of Ophthalmology (Madrid, June, 2003) was modified.

Here, classification according to etiopathogenesis and affected glands and tissues are retained. Classification depending on severity was modified into 3 grades for practical use

1. Grade 1 or Mild – Symptoms without slit lamp signs
2. Grade 2 or Moderate – Symptoms with reversible signs
3. Grade 3 or Severe – Symptoms with permanent signs.

**Goals of Therapy**

Major goals include:

1. Supplementation of a deficient tear film
2. Preservation of the available tear by re-establishment of lid motility with normal lid-corneal congruity.
3. Supplementation of limbal tissue containing epithelial stem cells for the management of epithelial disease of cornea.
4. Improvement or supplementation of a basement membrane substrate.
5. Restoration of clear visual axis.

**Treatment Guidelines**

**A. Medical**

Along with tear substitutes, it is important to treat co-existing lid disease like blepharitis, trichiasis and meibomian gland dysfunction as well as to preserve the available tear by punctual occlusion.

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1. **Tear substitutes and Lubricants**

Lubricants act as a physical means of protecting already compromised ocular surface from desiccation and irritation, but the preservatives like benzalkonium chloride may counteract the benefit. Hence preservative free lubricants are preferable although more expensive.

**Guide to various types of therapy for specific tear film abnormalities**

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Forms of therapy</th>
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<tbody>
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<td>Aqueous deficiency</td>
<td>Artificial tears, Lubricants</td>
</tr>
<tr>
<td>Mucin deficiency</td>
<td>Artificial tears, Lubricants</td>
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<td>Lipid deficiency</td>
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<tr>
<td>Tear spreading/Lid problems</td>
<td>Artificial tears/Lubricants</td>
</tr>
<tr>
<td>Tear base (Epithelial)</td>
<td>Artificial tears/Lubricants</td>
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2. **Nutritional Supplements**

Boerner et al found 98% patients reported improvement in the symptoms with omega 3 supplementation. Omega 3 fatty acids produces anti-inflammatory eicosanoid acid which suppress inflammation by blocking the gene expression of pro inflammatory cytokines.

3. **Tear Stimulants (Secretagogues)**

(a) **Diquafosol tetrasodium**

This molecule acts as a uridine nucleotide analogue that acts as a agonist of P2Y2 receptor present on ocular surface. Diquafosol increases water transport via chloride channel activation and enhances nonglandular secretion of tear fluid. The drug is under clinical trial.

(b) **Pilocarpine**

Pilocarpine is a cholinergic parasympathomimetic that binds to muscarinic M3 receptors and stimulates salivary and lacrimal glands.

(c) **Cevimeline**

Cevimeline is an acetylcholine analogue and has high
affinity for muscarinic M3 receptors of salivary and lacrimal glands

d) Eledoisin
Eledoisin is an endecapeptide which when applied locally has secretostimulant effect.

(e) Mucinous stimulators
Bromhexine and N-acetylcysteine are stimulants of mucin production. Topical medicines such as geranyl farnesylacetate and hydroxyeicosatetraenoic acids have been introduced which improve the epithelium and stimulate the goblet cells.

B. Suppression of Inflammation

1. Corticosteroids
Topical corticosteroids are beneficial for ocular surface defects associated with intense inflammation as in Sjogren's syndrome, Pemphigoid, Steven Johnson's Syndrome. The anti-inflammatory effect of corticosteroids are mediated through stabilization of the cytoplasmic and lysosomal membranes, thereby reducing the release of inflammatory mediators and inhibiting chemotaxis. However, careful monitoring of these patients is essential to watch out for steroid related complications.

2. Non steroidal anti-inflammatory agents
Antiinflammatory agents like salicylates, indomethacin, flurbiprofen and progestational steroids reduce inflammation without suppressing wound repair.

3. Immunosuppressives
Immunosuppressants like antimetabolites have been effective in treating ocular surface disorders of autoimmune origin like rheumatoid arthritis, Systemic Lupus Erythematosus and ocular cicatricial pemphigoid

C. Limitation of Tissue Destruction

1. Tissue Adhesives
Tissue adhesives like isobutyl cyanoacrylate have been used as an adjuvant in the management of corneal ulcers and small perforations. It gives structural support and can arrest further stromal loss. Early application of tissue adhesive in the management of stromal melts, can postpone or reduce the need for keratoplasty or conjunctival flaps.

2. Mechanical protection of corneal epithelium

Methods:

(a) Taping of lids
(b) Tarsorraphy
(c) Botulinum induced ptosis
(d) Therapeutic soft contact lens

This is useful to protect the loosely adherent remaining transient amplifying cells or regenerating epithelium from the action of blinking eyelids has significantly improved the management of persisting epithelial defects. Soft contact lenses are undesirable in dry eye patients because of the high risk of infection. Only silicone provides adequate oxygen transmission for continuous wear, however Omafilcon A (proclear), a novel biomimetic, 59 % water content hydrogel soft contact lenses for daily wear has been found to give better comfort.

3. Promotion of epithelial wound healing and differentiation

(a) Topical autologous serum
Topical autologous serum not only moistens the ocular surface, but also provides necessary tear proteins such as epidermal growth factor (EGF), vitamin A, transforming growth factor-B (TGF-B), fibronectin and other cytokines. The effect of EGF is primarily on epithelial wound healing whereas fibronectin appears to be involved in stromal healing.

(b) Topical retinoids
These are essential for epithelial growth and differentiation. All trans retinoic acid 0.05 % in Vaseline BD has shown to increase the epithelial healing rate. Retinol palmitate ophthalmic solution has also shown an increase in goblet cells and non keratinized cells.

(c) Topical trisodium citrate 10 % and sodium ascorbate 10 %

These have been found to reduce the incidence of ulceration and perforation in the immediate treatment of alkali burns.

(d) Topical Cyclosporin A 0.05 % & 0.1 %

Its efficacy may be due to an immunomodulatory and anti-inflammatory effect on the ocular surface, thus
facilitating ocular surface healing. This has been used in severe forms of dry eye where long term use of corticosteroids are required.

D. Preservation of natural tears

(a) Punctal occlusion

Punctal and canalicular closure increases mainly the aqueous component of natural tears but also has secondary beneficial effects on goblet cell density, tear film stability, and tear osmolality. This also increases the retention of artificial tears.

(1) Thermal occlusion

(i) The hot cautery method utilizes the direct transmission of heat from a hot probe to produce a controlled burn injury to the punctal opening. It is important to treat not only the surface of punctum, but also to insert the tip of cautery gently into the proximal lumen to achieve a more effective and permanent closure.

(ii) Diathermy utilizes 455 kHz to 100 mHz radiofrequency energy to heat the tissues in the area of punctal opening and proximal lumen. A fine needle electrode is introduced into the canaliculus through the punctum and the electromagnetic current is activated until the surrounding tissues blanch and contract.

(iii) Argon laser photocoagulation has a shorter duration of effect compared to thermal cautery. Here, the punctual opening is first encircled with laser spots and then additional spots are delivered into the punctum itself.

(b) Punctal obstruction

Lacrimal punctum and canaliculi may be occluded temporarily or permanently with tissue glue or implanted foreign bodies. Temporary obstructive procedures are useful in assessing the beneficial effects of lacrimal obstruction prior to restoring to permanent occlusion.

(i) Glue

Cyanoacrylate tissue adhesive or the more recent fibrin surgical glue may be applied to the punctal opening or proximal canaliculus using 25-27 G canula or needle. Occlusion with glue lasts for only several days to week since the epithelial cells lining the lumen slough during the natural cell turnover cycle.

(ii) Absorbable implants

Collagen implants are the widely used absorbable implants. They degrade over 3-7 days, although total degradation takes up to 14 days. Catgut (2-0) or chromic catgut (4-0) sutures are alternative absorbable implants.

(iii) Non absorbable implants and plugs

Non absorbable implant materials include polyethylene, silicone and acrylic. Newer one is hydrophobic acrylic which is heat responsive and its physical dimensions undergo transition at temperatures above 320 °C. No sizing of punctal opening is required because one plug size fits all puncta before heat activation.

(iv) Surgical procedures

These are indicated in multiple punctal occlusion failures. Methods include

(i) Punctal hot cautery and suturing with nylon.

(ii) Vertical canaliculus sutured with a single 8-0 polyglactin full thickness eyelid mattress suture.

(iii) Surgical laceration of horizontal canaliculus medial to the punctum on the eyelid margin, thermal cauterization of the exposed canalicular and punctal surfaces and suture closure of both the canaliculus and punctum.

(iv) Medial tarsorraphy

(v) Bulbar conjunctival autograft from one of the fornices can be sutured as a patch over the punctal orifice after surrounding epithelial tissue is excised.
(vi) Translocation of punctal orifice anteriorly to eyelash line.
(vii) Cisternoplasty- Creating an additional skinfold at the outer angle of the eye which acts as a natural reservoir for the tears.

A. Surgical

There are many surgical approaches for treating ocular surface disorders. It is important to control inflammation before surgery, correct the precipitating problem and give prophylaxis for postoperative inflammation. Methods to restore the ocular surface epithelium include conjunctival transplantation and limbal stem cell transplantation. Amniotic membrane transplantation has been used to restore the stromal environment by replacing basement membrane for epithelial cells and stromal matrix for mesenchymal cells. Other strategies to improve basement membrane include anterior stromal puncture, excimer photo therapeutic keratectomy and lamellar or penetrating corneal grafting.

Limbal Stem Cell Deficiency

Limbal stem cells being the source of newly generated corneal epithelial cells, any injury to them can cause severe derangement of the ocular surface especially the corneal surface leading to limbal stem cell deficiency which is a serious threat to vision. The definitive treatment of limbal stem cell deficiency would be to replace those abnormal limbal stem cells with healthy one.

Algorithm of Limbal Stem Cell Deficiency Management

![Algorithm Diagram]

(LSCD-Limbal stem cell deficiency, CLAU-Conjunctival limbal autograft, KLAL-Keratolimbal allograft, Lr-CLAL-Living related conjunctival allograft, Cu-LAU-Cultured limbal autograft, Cu-LAL-Cultured limbal allograft)

Preparation of bed

Under peribulbar anesthesia, 360° peritomy is performed 3-4mm from limbus with removal of normal epithelium, pannus and symblephara and bleeding points are cauterized.

Amniotic Membrane Transplantation

Clinical properties of amniotic membrane
1. Aids tissue epithelialisation
2. Reduces inflammation
3. Reduces vascularisation
4. Reduces scarring
5. Diminishes pain
6. Protects against infection

Indications for Amniotic Membrane Transplantation

1. Reconstruction of conjunctival surface
   (a) After resection of extensive lesions (tumours, scars)
   (b) Symblepharon reconstruction
2. Reconstruction of corneal surface
   (a) Persistent epithelial defects
   (b) Partial limbal stem cell deficiency
   (c) Total limbal stem cell deficiency (prior to limbal transplantation)

Other uses of Amniotic Membrane

1. Cultivation of limbal stem cells
2. Carrier for cultivated limbal stem cell transplantation

Limitations of Amniotic Membrane

1. Absolute deficit of limbal stem cells
2. Severe stromal necrosis
3. Severe neurotrophic changes
4. Severe ischaemia
5. Absence of tear film

Surgical technique

After preparation of the bed, a processed and preserved amniotic membrane in Dulbecco’s modified Eagles medium and glycerol at -80 °C is spread over the ocular
surface with the epithelial side up. The amniotic membrane is sutured with 6-8 circumferential 10-0' nylon monofilament interrupted sutures at the limbus and with 8-0' polyglactin sutures in the periphery with the conjunctival edge.

**Conjunctival Limbal Autograft (CLAU)**

**Indications**

1. Partial / Unilateral Limbal Stem Cell Deficiency
2. Reconstruction of ocular surface following pterygium excision, excision of large tumours, symblepharon

**Complication**

Iatrogenic donor site Limbal Stem Cell Deficiency

**Surgical technique**

Donor tissue can be obtained from the same eye (ipsilateral CLAU) or from the other eye (contralateral CLAU). After peritomy, a non-contigous 6 clock hours (3 superiorly & 3 inferiorly) of donor tissue is harvested, the size being 4+4 mm conjunctiva with the limbus including 1mm of superficial clear corneal stroma. These should be placed with the epithelial side up and the limbal area of the donor near the limbus which are secured with 2 circumferential sutures 10-0' nylon monofilament and conjunctival part with 8-0' polyglactin.

**Cadaveric Keratolimbal Allograft (KLAL)**

Being an allogenic tissue, immunosuppressants are mandatory to prevent immunological rejection.

**Indications**

1. Bilateral Limbal Stem Cell Deficiency
2. Total Limbal Stem Cell Deficiency in one-eyed patients
3. Severe Ocular Surface Disorders as in Steven Johnsons Syndrome, Ocular cicatricial pemphigoid, severe chemical burns

**Surgical technique**

The related donor usually 1st degree relative should be screened for potential blood borne infectious diseases including Hepatitis B and C and HIV 1 & 2. HLA typing is performed preoperatively to find the best match. Technique is similar to CLAL and not more than 6 clock hours should be harvested.

**Post operative regimen**

Topical corticosteroid 3-4 times daily.
Topical antibiotics till the epithelium has healed (1-3 wks).
Maintenence- Tapered dose of topical corticosteroid and lubricants.
Systemic Prednisolone 1mg/kg/day, with a slow taper over 3-6 mths.
Systemic immunosuppression with oral Cyclosporine A or FK 506.

**Cultivated Limbal Epithelial Transplantation**

It is the most recent and promising technique of limbal stem cell transplantation. It can be an autograft (ipsilateral or contralateral) or allograft from a live related donor.
Indications

Autograft: Unilateral Limbal Stem Cell Deficiency
Bilateral Limbal Stem Cell Deficiency with partial Limbal Stem Cell Deficiency in one eye
Allograft: Bilateral Limbal Stem Cell Deficiency
Total Limbal Stem Cell Deficiency in one eyed patient

Surgical technique

Limbal biopsy

2×2 mm limbal tissue with 1mm into clear corneal stromal tissue at the limbus is excised and transported in human corneal epithelial medium to the tissue culture laboratory.

Cultivation of epithelia

The shredded limbal tissue bits are explanted over the central 10mm of a 3×4 cm, de-epithelialized, preserved human amniotic membrane which is the most widely used substrate for cultivation of limbal stem cells. The cells are cultured using human corneal epithelial cell medium with 10 % foetal bovine serum or autologous serum. The growth is monitored daily and medium changed in 2 days. The culture is maintained for 10-15 days, by which time a confluent monolayer of limbal epithelial cells are grown.

Transplantation

After preparing the bed, the human amniotic membrane with the monolayer of cultivated limbal epithelial cells is transplanted on the recipient cornea.

Post operative immunosuppression is required for allogenic transplants.

Other modalities

 Conjunctival flap

In conditions such as neurotrophic keratitis and sterile stromal ulceration as in chemical burns, a conjunctival flap supplies the necessary vascularity to reverse ischaemia related complications.

Buccal Mucous Membrane Transplantation

Used in eyelid position abnormalities caused by cicatrisation as in SJS, OCP, bilateral fornix reconstruction.

Keratoprosthesis

Artificial corneas are recommended in heavily scarred and vascularised corneas and in severely blind dry eyes. Osteo-odontokeratoprosthesis is a recent development in this field.

Phototherapeutic keratectomy (PTK)

Excimer laser PTK is beneficial in treating recurrent corneal erosions and persistent corneal epithelial defects by improving the basement membrane.

Guide to Dry Eye Management

First step

- Mild cases : Lubricants and artificial tear supplements- eyedrops and gels
- Moderate- Severe cases : Lubricating ointments
- Severe : Patch with lubricating ointments

Artificial tear inserts

Topical steroids

Intermediate step

- Temporary punctual occlusion with collagen or silicone
- External tarsorraphy
- Botulinum toxin induced ptosis

Final step

Very severe cases :

- Cyanoacrylate glue tissue adhesive for closure of perforation/ desemetocoele
- Corneal or corneoscleral patch/ conjunctival flap for impending/ frank perforation
- Lateral tarsorraphy in facial nerve palsy, trigeminal nerve lesions or severe exophthalmos
- Amniotic membrane graft
- Limbal stem cell transplantation

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