Tear Film And Its Abnormalities

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The ocular surface, tear film, lacrimal glands, and eyelids act as a functional unit to preserve the quality of the refractive surface of the eye and to resist injury and protect the eye against changing bodily and environmental conditions. Events that disturb the homeostasis of this functional unit can result in a vicious cycle of ocular surface disease. The tear film is the most dynamic structure of the functional unit, and its production and turnover is essential to maintaining the health of the ocular surface. Classically, the tear film is reported to be composed of three layers: the mucin, aqueous, and lipid layers. A dysfunction of any of these layers can result in dry eye disease. Presence of tear film was described by Fischer in 1928 using Reflectography. Structure was described by Wolff in 1946.

REGULATION OF TEAR PRODUCTION :

The production of tears is regulated through a reflex loop. The ocular surface (conjunctiva, cornea, accessory lacrimal glands, and meibomian glands) and main lacrimal glands act with this neuronal loop to regulate the production of tears necessary for ocular surface homeostasis and repair. Stimulation of nerves at the ocular surface or in the nasal mucosa sends impulses to the brain via the fifth cranial nerve, which generate a reflex response via nerves passing to the lacrimal glands. Nerve impulses generated by emotional stimuli also feed into this reflex loop.

Similarly, ocular surface irritation from such factors as excessive evaporation, low humidity, or contact lenses results in chronic afferent stimulation and increased lacrimal secretion.

The spreading of the tear fluid on the ocular surface to form the complexly structured preocular tear film is the consequence of blinking. The sequential operation of the orbicular and levator muscles of the lids spreads the tear fluid and reconstructs the tear film architecture disturbed by the evaporation of water and by environmental contamination during the interblink period.

The movement of the lids exerts a significant pressure on the bulbar surface at each blink, with a retropulsion of the eye of 0.7-1 mm (up to 2 mm in forced blinking). If not protected by an efficient viscoelastic tear film, the ocular surface epithelia can be damaged by the applied shear forces.

TEAR FILM COMPOSITION :

LIPID LAYER : The meibomian glands, located within the tarsal plates, secrete their oily product onto the lid margins and form the outer lipid layer of the tear film. The principal function of the lipid layer is to prevent the evaporation of tears and enhance the stability of the tear film. The presence of a smooth lipid layer is also essential to provide an excellent dioptric element for light refraction into the eye and sharp retinal image formation.

The blink reflex is thought to be important in the release of secretions from the meibomian glands. Indeed, rapid and forceful blinking, such as in response to a foreign body, increases the thickness of the lipid layer. Conversely, the office eye syndrome (a peculiar eye surface pathology with clinical subjective and objective signs similar to dry eye that seems particularly frequent in office workers) appears to be associated with prolonged periods without blinking and corresponding thinning of the lipid layer.

AQUEOUS LAYER : The main and accessory lacrimal glands under different stimuli (hormonal, sympathetic, parasympathetic) produce the aqueous layer. This layer is quantitatively the most important, and is responsible for creating the proper environment for the epithelial cells of the ocular surface, carrying essential nutrients and oxygen to the cornea, allowing cell movement over the ocular surface, as well as washing away epithelial debris, toxic elements and foreign bodies. Changes in its composition occur rather quickly in response to environmental or bodily conditions and can influence the health, proliferation, maturation, and movement of the

surface epithelial cells. Many of the growth factors that are present in the aqueous phase of the tear film are derived from the lacrimal gland tissue. A significant role of growth factors in corneal physiology has been suggested. According to current models, decreased aqueous tear production results in a decreased growth factor concentration in the tears, with consequent effects on ocular surface health.

Many proinflammatory factors (e.g., HLA DR, IL-6 and IL-8), produced locally or by adjacent structures and located in the aqueous phase of the tear film, function to modulate the eye's response to changes in the condition of ocular surface.

MUCIN LAYER : The inner mucin layer is produced by conjunctival goblet cells and by conjunctival and corneal epithelial cells. The corneal and conjunctival epithelia synthesize a mucin-like glycoprotein (MUC1) at the apical surface of the epithelium to constitute the glycocalyx. This transmembrane mucin has a role in tear film spreading and is essential for proper ocular surface wetting. It prevents adhesion of foreign debris, cells, or pathogens to the ocular surface. The largest part of the mucinous content of the tear film is produced by the secretion of the goblet cells of the conjunctiva, which open on the ocular surface. The human conjunctiva also expresses the mucins MUC4 and MUC5AC, which may play an important role in forming the tear-film layer at the air and ocular surface-epithelium interface.

The mucin of the glycocalyx renders the whole of the ocular surface hydrophilic and allows the aqueous layer to spread evenly over the eye. Without the glycocalyx, tears do not properly adhere to the eye, and epithelial damage may occur, even with normal aqueous tear production. The mucins account for the fundamental non-Newtonian viscoelastic properties of the tear film, which allows the viscosity of the tears to change according to the shear rate of blinking. They maintain the dioptric integrity of the tear film in the inter-blink period and protect the ocular surface during blinking, minimizing the trauma to the surface. Recent studies suggest that tear lipocalins also make an important contribution to tear viscosity.

The actual architecture of the precorneal tear film is controversial. Wolff proposed the classical three-layered structure, but there is now evidence for a two-layered structure where under the lipid layer lies an aqueous-mucin gel, in which the mucins have a decreasing gradient of concentration from the epithelium to the surface.

TEAR FILM DYNAMICS :

SECRETION OF TEAR : Secreted throughout the day by: Accessory (basal secretion) and Main (reflex secretion) lacrimal glands. Afferent pathway is formed by 5th cranial nerve and efferent by parasympathetic supply of lacrimal gland. Most of the full term new born secrete tear within 24 hrs. and have normal secretory rate within first week. Abnormal / reflex tearing starts after 4 weeks.

DISPLACEMENT PHENOMENON : On displacement of the lower eyelid with finger over eyeball-particles in the film move upwards .Phenomenon is due to thin monomolecular layer on the surface of cornea that gets displaced and not the whole pre corneal tear film.

EVAPORATION FROM TEAR FILM : Evaporation is about 10% of production rate (0.12 microlitre/min).There is little effect of air motion on evaporation rate.

STABILITY, DRYING AND RUPTURE OF TEAR FILM : Holly and Lemp's mechanism of tear film break up: Tear film thins uniformly by evapoartion. Thinning to a critical thickness leads to attraction of lipid molecules by mucin layer and migrate down. Sufficient contamination of mucin by lipid migration, makes the mucin hydrophobic and rupture of tear film occurs.

ELIMINATION OF TEARS : On closing eyelid: Contraction of pretarsal fibres of orbicularis compresses the ampulla and shortens the canaliculi propelling the tear towards lacrimal sac.

Contraction of preseptal fibres of orbicularis pulls the lacrimal fascia and lateral wall of lacrimal sac laterally - opening of lacrimal sac - production of a relatively negative pressure with drawing of tear into lacrimal sac.

Along with increased tension on the lacrimal fascia, inferior portion closes more tightly preventing aspiration of air into nose.

On eyelid opening: Relaxation of pretarsal fibres of orbicularis causes canaliculi to expand and reopen leading to drawing of lacrimal fluid through puncti from lacrimal lake.Relaxation of preseptal fibres results in collapse of lacrimal sac and expulsion of fluid into NLD.

Drainage of lacrimal fluid from NLD into nasal cavity: Gravity helps in downward flow; Negative pressure in the NLD induced by inward or outward movement of the air current; Hasner's valve remains open till the pressure within nose is less than NLD.

TEAR FILM ABNORMALITIES :

TEAR FILM DEFICIENCY :

AQUEOUS DEFICIENCY :

CONGENITAL :

Riley Day syndrome: Disorder of ANS (sensory, sympathetic, parasympathetic);

Congenital alacrimia - hypoplasia of lacrimal gland;

Ectodermal dysplasia.

ACQUIRED :

SYSTEMIC :

Sjogren's Syndrome : autoimmune disease-immune cells attack and destroy the ductal and acinar cells of exocrine glands producing tear and saliva;

Sarcoidosis : inflammatory cells clustered over eyes;

Amyloidosis: amyloid proteins accumulated in vessels, ducts and glands;

Lymphoma, Leukaemia;

Progressive systemic sclerosis;

Haemachromatosis..

INFECTION: Mumps;

INJURY: Surgical removal and or damage to lacrimal gland; Irradiation; Chemical burns;

MEDICATIONS : Antihistaminics; Antimuscarinics- atropine, scopolamine; Beta blockers - timolol;

NEUROGENIC : Facial nerve palsy.

MUCIN DEFICIENCY :

Avitaminosis A: normal lacrimal and mucus secreting epithelium is replaced by keratinised epithelium.;

Steven Johnson syndrome and Ocular pemphigoid: affects mucus membrane of body esp of mouth and eyes. Also obstructs the lacrimal ductules;

Trachoma : chronic inflammation causing monocytic and lymphocytic infiltration- conjunctival scarring which is associated with conjunctival atrophy and loss of goblet cells;

Chemical burns : obstruction of lacrimal ductules;

Medications : Antihistaminics, beta blockers, antimuscarinics, eye drop preservatives.

LIPID DEFICIENCY :

Chronic blepharitis : cause meibomian gland dysfunction.;Acne rosacea: usually associated with meibomitis or meibomian gland dysfunction; Lid margin scarring; Seborrhoeic dermatitis; Chemical agents like turpentine.

DEFECTIVE SPREADING OF TEAR FILM;

Eyelid abnormalities: lid defects, coloboma, ectropion, entropion, keratinisation of lid margin, decreased or absent blinking due to: Hyperthyroidism, Contact lens, HSV Keratitis, Leprosy; Lagophthalmos-nocturnal lagophthalmos, hyperthyroidism, leprosy; Conjunctival abnormalities - Pterygium, Symblepharon; Proptosis

In most of these conditions the palpebral fissure width is more(usually if greater than 10mm) - resulting in significant evaporative stress on tear film.

TEAR FILM EXCESS:

PRIMARY : due to stimulation of lacrimal gland-tumours, parasympathomimetic drugs.REFLEX: stimulation of sensory branches of 5th nerve due to irritation of cornea and conjunctiva. Lids- stye, internal hordeolum, acute meibominitis, trichiasis, concretions, entropion; Conjunctiva- conjunctivitis; Cornea- abrasions, ulcer, keratitis; Sclera- episcleritis, scleritis; Uvea- iritis, cyclytis, iridocyclitis; Glaucoma; Endophthalmitis, panophthalmitis. CENTRAL OR PSYCHICAL LACRIMATION: in emotional states, voluntary and hysterical lacrimation.