# DYSFUNCTIONAL TEAR SYNDROME

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Tear film can be considered as an extra cellular fluid produced by local glands and blood vessels. It has three primary layers : Mucin, Aqueous and Lipid layers.

T : Transparency of ocular surface for normal vision as anterior surface of the cornea provies 80% of the refractive power of the Eye. Tear film breakup causes optical aberrations that can degrade power of image focussed on the retina.

**E :** Epithelial cell health.

Providing mutrition and oxygen to the ocular Epithelium.

**A :** Acts against environmental and infectious hurts. These protective components which are secreted to the tear film are immunoglobin A, Lacto ferrin Lysozymes and peroxidase. Tear film washes out particulates irritants and allergens. Surface lipid layer prevents tear evaporation.

**R :** Regeneration and healing.

Providges growth factors for Epithelial regeneration and healing, helps in reduction of free radicals.

During blinking the upper lid margin exerts a shrain of 150 dynes /cm 2, on the ocular surface which may result in epithelial desquamentation and induction of apoptosis. Tear film mainly the mucin layer decrease this shear force.

The lacrimal glands and the ocular surface Epithelium function together and linked by sensory autonomic nerves and form so called lacrimal functional unit that maintains the health of the tear film.

Critical components of normal tear film includes- Electrolytes. Proteins, Cytokines, Protease immunoglobulins and phospholipids.

Unhealthy tear film cannot function to keep the ocular surface healthy.

**Pathogenesis**

Dysfuntion of the neuronal loop triggers the vicious circle.

The neuronal loop consists of lacrimal gland, the ocular surface and the interconnecting innervation. Progression of the dry Eye pathology starts with hyper stimulation of the neuronal loop. The possible cause of which is chronic irritation of the ocular surface from environmental factors. This causes activation of T cells and the production of cytokines in lacrimal tissue. Cytokines are secreted in to tears and cause disruption of the neuronal are with abnormal alteration in the quality and quality and quantity of tears.

The ensuing ocular surface inflammation can progress to ocular surface damage. In severe cases there is permanent lacrimal damage and inability to secret tears.

Changes in the tear film composition no longer nourishes and support the ocular surface epithelium, consequently factors which promote ocular surface inflammation increases. Reduction in the tear film viscosity or quntity of tears makes it thinned or spotty so it cannot provide barrier against infectious or environmental hurts.

Concentration of many tear proteins including those with antimicrobial functions are reduced. Growth factor concentration is also reduced. Solube mucin 5AC is greatly reduced in concentration because of the loss of the goblet cells from the conjectival epithelium which has impact on the viscosity of tears.

Proteases which are normaly latent and inactivated in normal tears becomes activated. Activated proteases can degrade the extra cellular matrix and the light junction between adjacent cells of the corneal surface epithelium and responsible for the cleavage of cytokines in to activated pro inflammatory forms. The osmolarity of the tear film increases. Continued osmotic stress on the cells results in continued metabolic and physiologic failure and rapid cell death.

**Signs and Symptoms**

More weight is to be given to patient's symptoms in diagnostic decision, due to its impact on quality of life.

Relevant Symptoms includes Dryness, itchiness, stinging, burning, sandy, gritty, painful blurring vision. Symptoms profile develops from mild to very severe forms.

**Signs include**

1. Conjuctive and corneal staining.
2. Tear film signs.
3. Tear breakup time.
4. Schirmer score

**Management**

Severity of thedisease indicates appropriate range of therapeutic action. There has been a trend towards the early use of Cyclosporin A as there is evidence of halt of the diseases progress. For management according to severity is classified in to four levels. (I to IV)

Level I : Patients education
(Mild) Environmental modification
Control of systemic medication.
Treserved control
Allergy control.
Level II : Unpreserved tears - Cyclosporin A.
(Moderate) Gels/Night time ointments - Secretagogues.
Level III Tetracyclines.
(Severe) Punctal plugs -Topical steroids.
Level IV Systemic anti-inflammatory therapy.
(Very severe) Acetlcysteine.
Moisture Goggles.
Surgery (punctal cautery)

**Osmoprotection**

Compatible solutes may potentially offer additional advantage to current artificial tear formulation. Acting as osmoproectants, compatible solutes have the ability to travel below the corneal epithelial surface and be absorbed by individual cells acting within the cell on a molecular level, the cells would then be able to hold on to water helping to reach osmotic balance. The cells could then shed excess salts and become protected from the salty external environment and osmotic stress. Compatible solutes like L-Carnitine and Erythritol penetrate the cells to protect corneal epithelial cells, restores osmotic balance and provides relief from osmotic stress. Such formulation will bring long standing relief to the patient.

**Conclusion**

Alteration in quality and quantity of tears triggers the disease process.

The condition is very often under diagnosed the under treated.

Symptoms should be given more weight for diagnostic decision and management.

The prevalence in creases with age.

Smokers are prone to this diseases due to frequent irritation of the ocular surface by smoke.

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