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Dry Eye Syndrome (Keratoconjunctivitis Sicca)

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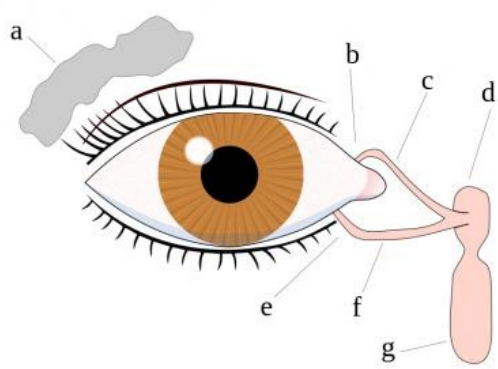
KEYWORDS :

Dry eye syndrome (DES), also known as dry eye disease (DED), keratoconjunctivitis sicca (KCS), and keratitis sicca, is a multifactorial disease of the tears and the ocular surface that results in discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface.[1] Dry eye syndrome is a common form of ocular surface disease (OSD) and may overlap with other causes of OSD, such as ocular allergy and meibomian gland dysfunction (MGD).

The ocular surface is an integrated anatomical unit consisting of 7 key interactive and interdependent components: the tear film, the lacrimal and accessory lacrimal apparatus, the nasolacrimal drainage system, the eyelids, the bulbar and tarsal conjunctiva, cranial nerve V, and cranial nerve VII.[2] Abnormalities or deficiencies in any of the 7 ocular surface components may worsen dry eye syndrome, yet they can be treated with therapeutic intervention.

The image below depicts the ocular surface anatomy.



Eye tear system anatomy- a. Lacrimal gland, b. Superior lacrimal punctum, c. Superior lacrimal canal, d. Lacrimal sac, e. Inferior lacrimal punctum, f. Inferior lacrimal canal, g. Nasolacrimal canal.

Dry eye syndrome may be subdivided into 2 main types as follows:

- Dry eye syndrome associated with Sjögren syndrome
- Dry eye syndrome unassociated with Sjögren syndrome
- Dry eye syndrome can also be subdivided into pure aqueous deficiency dry eye and evaporative dry eye.[3] Eighty-six percent of patients with dry eye syndrome also have signs of meibomian gland dysfunction.

Causes and Risk Factors for Dry Eye Syndrome-

It is not clear why some people are not able to produce enough natural tears, as already mentioned one cause of dry eye is Sjogren's Syndrome, a disease involving mild to extreme dryness in both the eyes and the mouth. The risk of Dry Eye increases with age. Other risk factors include patients who have undergone refractive surgery (LASIK), have severe allergies, are on certain medications, or are contact lens wearers. Those with rheumatoid arthritis and other diseases are also at increased risk. Women are also more likely to develop Dry Eye. Approximately 6 million women and 3 million men have moderate to severe symptoms of Dry Eye. Women who are pregnant, on certain types of birth control, hormone replacement therapy or experiencing menopause also have increased rates of Dry Eye.

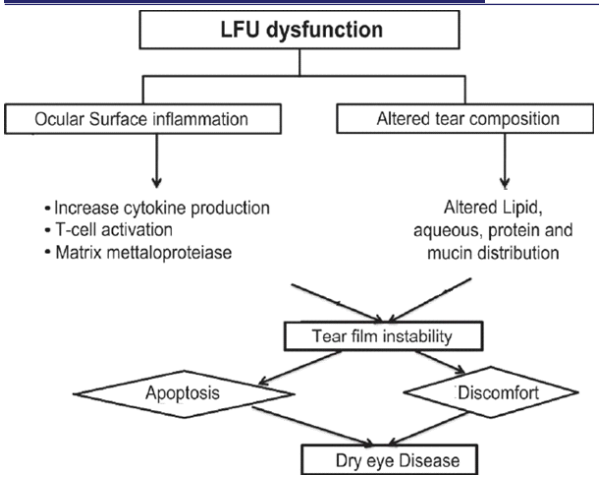
Table- Risk Factors for Dry Eye

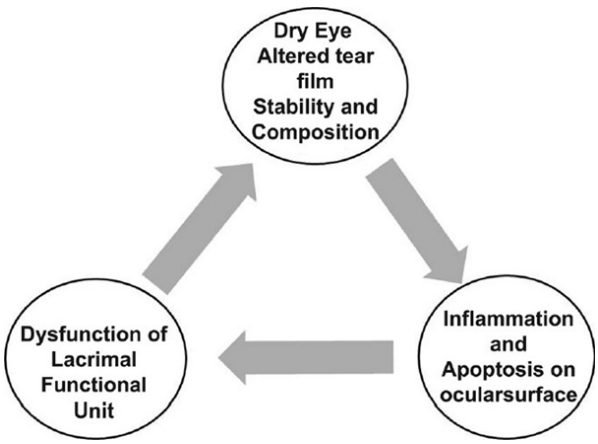
Level of Evidence		
Mostly Consistent	Suggestive	Unclear
Older age	Asian ethinicity	Cigarette smoking
Female Gender	Medcations-Tricyclic antidepressants, Selective serotonin reuptake inhibitors, Diurectics, Beta-blockers	Medications-anticholinergics Anxiolytics, antipsychotics
Postmenopausal estrogen therapy	Diabetes mellitus	Hispanic ethnicity
Low dietary intake of omega-3 fatty acids	HIV/HTLV1 infection	Alcohol use
Connective tissue disease	Systemic chemotherapy	Menopause
LASIK and refractive excimer laser surgery	Large incision ECCE and penetrating keratoplasty	Oral contraceptives
Radation therapy	Low humidity environments	Gout
Vitamin A deficiency	Sarcoidosis	Pregnancy

PATHOGENESIS

The ocular Surface and tear-secreting glands function as an integrated unit[3]. Disease or dysfunction of this functional unit results in an unstable and poorly maintained tear film that causes ocular irritation symptoms and possible damage to the ocular surface epithelium. Dysfunction of this integrated unit may develop from aging, a decrease in supportive factors (such as androgen hormones), systemic inflammatory diseases (such as Sjögren syndrome or rheumatoid arthritis), ocular surface diseases (such as herpes simplex virus keratitis) or surgeries that disrupt the trigeminal afferent sensory nerves (eg. LASIK) and systemic diseases or medications that disrupt the efferent cholinergic nerves that stimulate tear secretion[4]. Decreased tear secretion and clearance initiates an inflammatory response on the ocular surface that involves both soluble and cellular mediators.[5, 6] Clinical and basic research suggests that this inflammation plays a pathogenesis of dry eye.

Etiopathogenesis of dry eye disease





Vicious cycle of ocular surface inflammation

SIGNS AND SYMPTOMS

Depending on the severity of dry eye syndrome (DES), or keratoconjunctivitis sicca (KCS), the following are the most common patient complaints:

- Foreign-body sensation
- Grittiness
- Hyperemia
- Mucoid discharge
- Ocular irritation
- Ocular dryness
- Excessive tearing (secondary to reflex secretion)
- Photophobia
- Itching
- Fluctuating or blurry vision

These symptoms are often exacerbated in smoky or dry environments, by indoor heating, by fans, or by excessive reading or computer use and these tend to be worse toward the end of the day. Patients with meibomian gland dysfunction (MGD) may complain of redness of the eyelids and conjunctiva, but in these patients, the symptoms are often worse upon awakening in the morning.

Paradoxically, some patients with dry eye syndrome complain of too much tearing. When evidence of dry eye syndrome exists, this symptom is often explained by excessive reflex tearing due to severe corneal surface disease from the dryness. Epiphora may also accompany conjunctivochalasis, which demands consideration of surgical intervention.

Certain systemic medications also decrease tear production, such as antihistamines, beta-blockers, and oral contraceptives.

Many topical medications also decrease tear production, including antihistamines, beta blockers, and many other glaucoma medications.

The patient's medical history may be significant for coexisting connective tissue disease (CTD), rheumatoid arthritis (RA), or thyroid abnormalities. A thorough review of systems should be obtained, focusing specifically on dry mouth, arthritis, cutaneous changes, malaise, weight loss, and lymphadenopathy.

DIAGNOSTIC CRITERIA

Ohashi et al[7] suggested that a combination of (1) dry eye symptoms, (2) suggestive findings on Schirmer (< 5 mm wetting after 5 minutes) and fluorescein clearance tests, and (3) fluorescein and Rose Bengal staining (> 3+) would verify clinical dry eye. Other authors have devised different diagnostic criteria and there is no consensus in this regard to further complicate the issue, symptoms and signs do not always correlate well with each other in many patients.[8-10]

To confirm a diagnosis of dry eye, certain tests are required in the clinical setting. Tear film stability can be assessed with the fluorescein tear break-up time test (TBUT). This measures the interval in seconds between a complete blink and the first appearance of a dry spot or discontinuity in the precorneal film. Patients with TBUT less than 3 seconds are classified with clinical dry eye. If there is aqueous deficiency, the tear meniscus will appear to be thin, less than 1 mm in height.

Another clinical method for assessing the severity of dry eye is ocular surface dye staining. Fluorescein and Rose Bengal stains can be used as diagnostic dyes. Fluorescein staining occurs when the epithelial barrier is disrupted and serves as a good test for evaluation of dry eye. Rose Bengal stains devitalized epithelial cells on the conjunctiva and serves a similar purpose. However, Rose Bengal causes transient irritation after instillation and may be less comfortable. Patients with dry eye syndrome can show signs of punctate epitheliopathy and even corneal abrasions.

An important clinical test is the Schirmer test which measures aqueous tear production. This test is easy to perform in clinical settings but may be subject to errors. Strips of filter paper, called Schirmer strips, are placed on the lower lid inside the tarsal conjunctiva. The patient is allowed to blink normally and the tear strip is scored according to the degree it wets in 5 minutes. There are two ways to perform this test: (a) without topical anesthesia (Schirmer test I) which evaluates the ability of the ocular surface to respond to surface stimulation; and (b) under topical anesthesia (Schirmer test II) which evaluates basal tear secretion. Patients with tear soaking less than 10 mm are considered to have clinical dry eye and eyes with less than 5 mm wetting are diagnosed as severely dry. However, it is important to note that Schirmer tests are subject to environmental and physiologic changes with varying results over time.

Treatment

Early detection and aggressive treatment of dry eye syndrome (DES), or keratoconjunctivitis sicca (KCS), may help prevent corneal ulcers and scarring. The frequency of follow-up care depends on the severity of the signs and symptoms.

Although supplemental lubrication is the mainstay of treatment for mild and moderate aqueous-deficient dry eye syndrome, any concomitant lid disease must also be treated.

The Management and Therapy Subcommittee of the International Dry Eye WorkShop **(DEWS)** recommendations are stratified according to the severity of the disease.

Level 1 treatment consists of the following:

- Education and environmental or dietary modifications
- Elimination of offending systemic medications

- Preserved artificial tear substitutes, gels, and ointments
- Eyelid therapy

If level 1 treatment is inadequate, level 2 measures are added, including the following:

- Nonpreserved artificial tear substitutes
- Anti-inflammatory agents (topical cyclosporine, topical steroids)
- Tetracyclines (for meibomitis or rosacea)
- Punctal plugs (after inflammation has been controlled)
- Secretagogues
- Moisture chamber spectacles

If level 2 treatment is inadequate, level 3 measures are added, including the following:

- Autologous serum or umbilical cord seru
- Contact lenses
- Permanent punctal occlusion

If level 3 treatment is inadequate, level 4 treatment, consisting of the administration of systemic anti-inflammatory agents, is added.[11]

Conclusion

Dry eye syndrome consists of a wide spectrum of disorders with different causes. Clinicians should be aware of the extent of dry eye symptoms. A thorough history and investigation is necessary to identify the cause of dry eye. Simple and useful clinical tests such as Schirmer, fluorescein dye, and tear break up time tests can be used for assessing the severity of the condition. Management depends on an accurate diagnosis and the severity of the condition.

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