Dry eye syndrome

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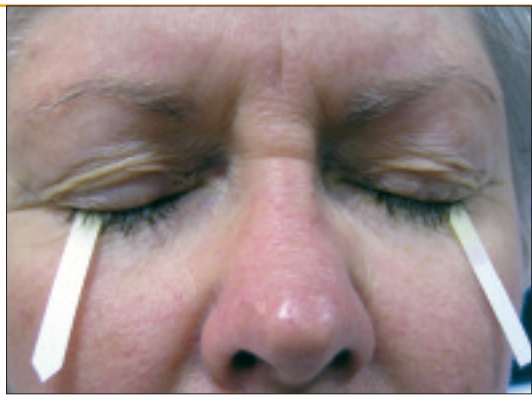

The Schirmers test tends to be inconsistent, limiting its validity and usefulness.

Dry eye syndrome (DES) is a clinical condition characterised by either **deficient tear production** or **excessive evaporation** resulting in ocular discomfort.

Tear deficient dry eye can be classified as:

- Sjogren's syndrome (an autoimmune disorder of the lacrimal and salivary glands as well as the ocular surface). Sjogrens is either primary (isolated) or secondary and associated with other connective tissue disease
- non-Sjogren's syndrome. For example, various medications are associated with DES – antihistamines, antidepressants, MAO inhibitors, antipsychotic, antihypertensives (alpha agonists, B blockers and diuretics) and NSAIDs.

Evaporative dry eye is due to:

- meibomian gland dysfunction (blepharitis with decreased abnormal lipids that are unable to retard tear evaporation)
- exposure (abnormal lids or neurological reflex arc)
- mucin-deficient states due to damage to the goblet surface cells (trachoma, chemical burns and Stevens-Johnson syndrome etc).

Pathophysiology

All forms of DES are associated with an inflammatory response at the ocular surface. There is hyperosmolar toxicity and exposure. If chronic, this leads to loss of goblet cells (mucin), epithelial dysfunction and metaplasia, as well as keratinisation.

This disruption of the epithelial barrier results in release of inflammatory cytokines and further disruption of the surface.

Inflammation may also be exacerbated or initiated by external triggers such as allergy, viral infection and preservatives in eye drops.

Symptoms

Typical complaints include burning, foreign body sensation, stinging, dryness, photophobia, ocular fatigue and redness.

People with unstable tear films may experience intermittent blurring, discomfort, excess reflex tearing and even epiphora. Symptoms are worse later in the day and in air-conditioned or heated offices or when staring at computer screens (i.e. decreased blink rate).

Inflammatory conditions, such as blepharitis, tend to be worse on waking (when inflammatory mediators accumulate) and improve as the day progresses.

Signs

Conjunctival injection, decreased tear meniscus, tear debris and staining of the interpalpebral conjunctiva with fluorescein or Rose Bengal. Meibomian gland inspissation, telangiectasia, glandular drop out, chalazions and eyelid debris are seen in meibomian gland disease.

Ancillary testing

The most practical clinical tests are:

- tear film break up time (abnormal = less than 10 seconds). After fluorescein dye is added to the eye, the interval between a complete blink and the appearance of a dry spot on the cornea (i.e. within the tear film) is measured.
- Schirmer's strips (less than 6mm at 5 minutes with local anaesthetic), and
- ocular surface staining pattern (Rose Bengal and Fluorescein).

If secondary Sjogren's is suspected anti Ro and La antibodies and rheumatoid factor and other antibodies may be sought.

Treatment: Aqueous deficiency

Tear supplementation: either as a drop, gel or ointment will lower tear osmolarity; avoid benzylkonium chloride as a preservative and avoid preparations containing vasoconstrictors; single dose non-preserved tear substitutes may be required.

Punctal occlusion is sometimes used to decrease tear drainage.

Treatment: Evaporative dry eye

Modification of the environment to lessen evaporation: humidifiers and protective glasses.

Warm compresses and lid massage improve quality and quantity of local meibomian gland secretions.

Anti inflammatory treatment – omega 3 fish oil and oral tetracyclines improve meibomian gland function. Topical steroids are controversial. Restasis (topical cyclosporin A) will soon be available for immunomodulation without the risk of corticosteroid side effects.