Management of Dry Eyes in primary care

This factsheet was produced under a collaboration between the UK Vision Strategy, RNIB, and the Royal College of General Practitioners.

Key learning points

• Dry eye is a very common condition presenting to GPs, and is probably underdiagnosed. It is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, watering and visual disturbance resulting from tear film instability [1]. Dry eye is synonymous with keratoconjunctivitis sicca.

• The normal tear film has 3 components
  a) pre-corneal mucin layer (produced by conjunctival goblet cells)
  b) aqueous layer (produced by lacrimal gland)
  c) outer protective oil film (produced by eyelid meibomian glands).

It maintains the transparency of the cornea in the face of environmental, endocrine and cortical influences.

• Dry eye symptoms are common; reported by 15% of adults in population Surveys [2] and result from an inflammatory response initiated by tear hyperosmolarity and other factors that stimulate corneal nociceptive nerves.

• The aim of treatment is to restore the normal tear film as far as possible. Most dry eyes can be managed in primary care.

• Symptoms include eye soreness, grittiness, blurring, watering especially in windy conditions (called paradoxical tearing thought to be the body’s
reaction to drying of the cornea).

- Dry eyes may not resolve, and symptom control is the mainstay of treatment.

**Evidence based treatment for dry eye**

- Since dry eye represents a disease spectrum in the majority of cases contributed to by excessive evaporative tear loss and aqueous tear deficiency [3], treatment should aim to address both [4].

- Increasing tear volume with artificial tears reduces tear hyperosmolarity which reduces friction between the upper lid as it spreads tears across the cornea in blinking [5] and dilutes inflammatory mediators on the corneal surface.

- Improving oily layer volume with lid hygiene is regarded as the mainstay treatment for evaporative dry eye (blepharitis, meibomian gland dysfunction: MGD). It has three components: application of heat to eyelids, mechanical massage of the eyelid glands, and eyelid margin cleansing with moistened lint or commercial lid scrubs. Good results for symptoms and signs are reported [6] but consensus on technique standardisation is lacking.

**Risk factors for dry eye**

- Low androgen and high oestrogen levels are risk factors for dry eye. Men on anti-androgen therapy and women with androgen insensitivity develop meibomian and goblet cell dysfunction. Premature ovarian failure and postmenopausal oestrogen therapy are risk factors for dry eye. Dry eye symptoms are more common in women than men [6].

- Aging is associated with physiological changes that predispose to dry eye, including decreased tear volume and flow, increased osmolarity, decreased tear film stability and alterations in lipid composition.

- Contact lens wear: reduce corneal sensory innervation.

- Eye drops: Glaucoma and other long-term topical medications containing preservative cause inflammatory changes in the ocular surface and
exacerbate dry eye [6].

- Eye surface diseases that damage goblet cell numbers (reduced mucin production) or corneal epithelium e.g. allergic eye disease, chemical injury, Steven’s Johnson Syndrome.

- Previous eye surgery or injury: Post-operative Lasik refractive surgery has a reported dry eye incidence of 75% [8].

- Systemic drugs: Systemic retinoids reduce meibomian function. Reduced lacrimal secretion has been linked to antihistamines, beta blockers, antispasmodics, diuretics, and with less certainty, tricyclic antidepressants.

- Reduced blink rate, incomplete blinking: using computer screens [9], reading for long periods, Parkinson’s disease [10].

- Auto immune disease infiltrating the lacrimal gland (primary or secondary Sjogren’s syndrome), is an important cause of aqueous deficient dry eye, 12% in one prospective study [11], though an uncommon cause of dry eye overall.

- Non-Sjogren’s aqueous deficiency: reduced or absent stimulated tears induced by emotion or in response to potent stimuli eg cutting onions. Occurs with lacrimal gland ductal stenosis Rarer causes include gland infiltration e.g. sarcoidosis or lymphoma. Reduced sensory innervation is also considered to be the mechanism for aqueous deficient dry eye in diabetes and longstanding contact lens wearers.

**Non-drug treatments of dry eye**

- Cleansing lid margins: to remove local irritants, cell debris and pollutants. Eye service manuals recommend cleansing the lid margins in blepharitis and meibomitis using mild (baby) shampoo and cotton buds or a clean moist facecloth.

- Soften meibomian oils: Hot compresses (40-45oC) on the eyelids reduce blockage of meibomian ducts by liquefying meibum.

- Improve blink rate and extent: use of video display units (VDU) is associated with both reduced blink frequency and tear film instability. Conscious complete blinking may alleviate dry eye symptoms by spreading tears more
effectively and encouraging lipid secretion.

- **Diet**: there is evidence to support the use of omega-3 dietary supplementation (for example, 2g tds) to improve symptoms and signs of dry eye by improving meibomian gland function [12].
- **Punctal plugs**: sometimes inserted by ophthalmologists into drainage punctae to preserve tear volume. Useful in aqueous deficient dry eye. Usually an outpatient eye clinic procedure.

**Pharmacotherapy: Artificial tears, anti-inflammatory drops and systemic treatment**

- **Artificial tears**: Evidence from studies of aqueous-deficient dry eye provides a basis for rational selection of artificial lubricants. Key factors in the selection of artificial tear include the role of preservatives, the role of viscosity, and more recently, the supplementation of oil (lipid) to the tear film. Topical treatments include Hypromellose, Carmellose, Carbomer, Hyaluronate, Castor oil and lubricating eye ointment. Ultimately the choice depends on which is more comfortable and efficacious for the patient. Many of these preparations are available over the counter. Treatment failures can occur if patients do not use ocular lubrication often enough, and should be encouraged to use non-preserved drops as often as the need arises, perhaps many times a day.
- **Preservative-free drops**: ocular surface toxicity has received increasing attention over the past decade. Even with indisputable in vitro evidence of preservative-induced toxicity it is unknown how frequently a preserved tear drop can be used without damaging corneal epithelium. Conventional advice has been that preserved artificial tears can be used from four to six times daily. Newer artificial tear drops are more often supplied with low dose or no preservative.
- **Topical steroid**: anti-inflammatory properties are useful in the short term [13], but no study supports the long-term use of topical steroid in management of dry eye. The risk of steroid induced glaucoma has been suggested to be as
high as 60% [14]. This treatment would be initiated by the Ophthalmologist or GPSI.

- In more severe dry eye there may be a role for topical cyclosporine and tacrolimus ointment initiated by the Ophthalmologist.
- Systemic therapy: Tetracycline or Doxycycline may be prescribed for blepharitis or ocular rosacea complicating severe dry eye where their effect is thought to be lipid modulating and anti-inflammatory [15].

**Patient leaflets and website**
http://www.patient.co.uk/health/dry-eyes-leaflet

Understanding dry eye leaflet from RCOphth and RNIB available as PDF to download (http://www.rcophth.ac.uk/page.asp?section=365)

Sjogren’s Syndrome on http://www.arthritis.ca/page.aspx?pid=1002

**Useful Resources**

Recalibrate Dry Eye Management.
http://www.revoptom.com/continuing_education/

**References**


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Thank you to all the authors and peer reviewers for the time they dedicated to developing this resource.

May 2014