Keratoconjunctivitis sicca (dry eye)

Abstract
Keratoconjunctivitis sicca, more commonly known as dry eye, or dry eye disease, is common. It results from improper functioning of the lacrimal functional unit. First-line treatment with tear substitutes, such as eye drops, gels and ointments, have an essential role. However, a newer understanding of the inflammatory aetiology of the disease has resulted in a shift in treatment strategies.

Introduction
Dry eye is an extremely common condition, which affects most people at some stage in their lives. It is also the most common ocular condition that is seen by eye care practitioners.1

In the past, dry eye treatment has always been palliative, in the form of tear substitutes. However, in recent years, numerous studies have suggested that dry eye seems to be caused by inflammation. This concept has led to a shift in its treatment. Nonetheless, conventional treatment still plays a fundamental role.1

Definition
The 2007 International Dry Eye Workshop defined dry eye as follows: “Dry eye is a multifactorial disease of the tears and ocular surface, that results in symptoms of discomfort, visual disturbance, and tear film instability, with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film, and inflammation of the ocular surface”.2

Signs and symptoms
Dry eye is a symptom of dry eye disease. While most people may complain of dry eyes from time to time, dry eye disease is usually associated with chronic symptoms:3-5

- Burning
- A sense of dryness
- Foreign body sensation
- Ocular pain
- Photophobia

- Blurred vision
- Eye fatigue.

Risk factors
The prevalence of dry eye increases with age, with an approximate incidence of 15% in persons over the age of 65 years.3 Dry eye is approximately 50% more common in women, and particularly in postmenopausal women.3,4 Sex hormones are known to play an important role in the homeostasis of the ocular surface, and declining androgen levels in aging men or women, or anti-androgen therapy, increase the likelihood of dry eye.4

Risk factors: medicines and medical procedures4,5
- Hormone replacement therapy
- Antihistamines
- Antidepressants (tricyclic antidepressants and selective serotonin reuptake inhibitors)
- Diuretics
- Beta blockers
- Isotretinoin
- Chemotherapy and radiation therapy
- Laser-assisted in situ keratomileusis (LASIK) eye surgery (especially immediately postoperatively)
- Corneal transplantation.

Risk factors: medical conditions4,5
- Diabetes mellitus
- Thyroid disease
- Sjögren’s syndrome
- Hepatitis C
- Vitamin A deficiency
- Human immunodeficiency virus (HIV) infection
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- Ovarian dysfunction
- Androgen deficiency.

**Risk factors: environmental**
- Air pollution
- Low humidity, high temperature
- Computer use (associated with reduced blinking).

**Other risk factors**
- Wearing contact lenses (50-70% of contact lens users experience discomfort and dryness)
- Diets low in omega-3 fatty acids, or with a high ratio of omega-6 to omega-3 fatty acids.

**Pathophysiology**

The tear film consists of an inner mucous layer, a middle aqueous layer, and an outer lipid layer. Tears provide lubrication, wash away foreign bodies in the eye, and reduce the risk of ocular infections. A healthy tear film relies on the optimal functioning of the lacrimal function unit, which consists of the ocular surface (cornea, conjunctiva, accessory lacrimal glands and meibomian glands), the main lacrimal gland, and the interconnecting innervation. Dysfunction of any component in this unit may lead to dry eye.5,7

Dry eye is classified into two groups:

**Increased evaporative loss**

Increased tear evaporation is most commonly caused by posterior blepharitis, also known as meibomian gland dysfunction. With this condition, the lacrimal glands responsible for producing the lipid layer of tear film, are dysfunctional.5

Other causes of increased tear evaporation include reduced blinking, or structural abnormalities of eyelid position.5

**Decreased tear production**

Reduced aqueous fluid volume causes hyperosmolarity of tear film and the ocular surface, which causes inflammation. Reduced tear production is further classified into Sjörgen’s, and non-Sjörgen’s syndrome.5

**Sjörgen’s syndrome**

Sjörgen’s syndrome is an autoimmune disease that results in dry eye and a dry mouth. It is characterised by exocrine gland dysfunction, in which the salivary and lacrimal glands are implicated. Patients may manifest inflammatory damage to musculoskeletal, gastrointestinal, urogenital and respiratory systems. When associated with an autoimmune disease, such as rheumatoid arthritis or systemic lupus erythematosus, a diagnosis of secondary Sjörgen’s syndrome is given.8

**Non-Sjörgen’s syndrome**

Non-Sjörgen’s dry eye can be caused by either primary, or secondary, lacrimal disease.9

The classification of dry eye is shown in Figure 1.

**Diagnosis**

Currently, there are no standardised criteria for the diagnosis of dry eye.1 In most cases, it is difficult to diagnose dry eye due to inconsistent correlation between reported symptoms and clinical signs, and poor sensitivity and specificity of diagnostic tests. As most dry eye conditions are chronic, monitoring of patients repeatedly reporting symptoms over an extended time period, may allow for more accurate diagnosis of chronic dry eye.10

A number of questionnaires are available in assessing patients with dry eye symptoms. One of these is the Ocular Surface Disease Index (OSDI), which helps to quantify symptoms.1

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**Figure 1: Classification of dry eye**
Patient instructions for the use of eye drops
1. Wash hands with soap and water.
2. Dry thoroughly, with a clean towel.
3. Shake the bottle gently.
4. Remove the eye drop bottle cap, and rest it on a clean tissue.
5. Do not touch the dropper tip.
6. Quickly turn the bottle upside down.
7. Look up at the ceiling with both eyes.
8. Pull the lower lid of one eye down with one hand, while keeping the eye drops in the other hand (resting the thumb on the forehead to keep it steady, if necessary).
9. Place one drop, or a small amount, of ointment, into the lower eyelid, taking care not to touch the eye with the tube or bottle.
10. Wait at least five minutes between putting in each drop.
11. Replace the eye drop cap.
12. If it is necessary to instill eye drops and ointment, use the eye drops first.
13. When it is necessary to instill more than one medicine into the eyes, wait at least five minutes between instilling each preparation.
14. On the bottle, write the date on which the dropper bottle was opened. Eye drops containing preservatives should be discarded within 30 days of opening.
15. Single-use containers should be used on opening, and then discarded.
16. Do not wear contact lenses when instilling eye drops or ointments, unless your doctor instructs you to do so.

Treatment

Tear conservation strategies

Behavioural and environmental modification
- Take breaks while reading, and when working at a computer
- Humidification of the environment
- Lower computer monitor height to decrease lid aperture
  - Moisture chamber eye-wear. These wrap around the eyes, helping to retain moisture, and protect the eyes from irritants.

Surgical or mechanical intervention
Punctal occlusion may be considered in patients with aqueous tear deficiency, when other strategies are unsuccessful. Punctal occlusion may be achieved temporarily with punctual plugs, or permanently, by argon laser canaliculoplasty, thermocautery, or radiofrequency needle.

Tear substitutes
Tear substitutes are considered to be first-line treatment for mild-to-moderate aqueous tear deficiency. Available formulations include drops, gels and ointments. Tear substitutes mostly include cellulose to maintain viscosity, and a spreading agent, e.g. polyethylene glycol, or polyvinyl alcohol, to prevent evaporation.

The more viscous formulations, e.g. gels and ointments, may relieve symptoms for a longer period of time, but may cause blurred vision shortly after application. The general recommendation is to use these formulations before bedtime.

Preservative-free formulations are preferred, as those containing preservatives, particularly benzylkonium chloride, are poorly tolerated, and potentially harmful in moderate-to-severe cases of dry eye. (Table I)

Anti-inflammatory therapy

Ciclosporin
Ciclosporin is an immunosuppressive agent used systemically in organ transplant patients. In 1995, ciclosporin was approved in the United States to treat dry eye in dogs. In 2003, testing in humans led to US Food and Drug Administration approval for the treatment of dry eye. Ciclosporin eye drops (Restasis) are not yet available in South Africa.

The exact mechanism of action of ciclosporin in the treatment of dry eye is not fully understood, but it is thought to act as a partial immunomodulator, and an anti-inflammatory, which stops T-cell activation, thereby preventing T-cells from releasing cytokines which initiate the inflammatory cycle of dry eye.

Corticosteroids
Several studies have found that topical or systemic corticosteroids are effective in the treatment of dry eye. However, patients must be regularly monitored for steroid-related complications, such as glaucoma, infections and subcapsular cataracts.

Conclusion
Dry eye is a common, yet complex, multifactorial condition, that can have a significant impact on patient quality of life and productivity.

Understanding of the disease has changed in recent years, leading to newer treatment strategies for ocular surface inflammation, in addition to traditional tear substitutes.

References
3. Gunus K, Cavanagh DH. The role of inflammation and anti-inflammatory

Book review

This 73-page “easy to read” book was written with the aim of helping people with addictions (PWAs) and their families to cope with the problem of addiction. The motivation arose from the family physician’s more than 20 years personal encounters and experience with those suffering from substance abuse especially in the Mitchelles Plain of Cape Town South Africa. Dr. Rapiti realised that many of his patients with drug and alcohol related problems struggled to understand books and resource materials which had complicated steps to accomplish during rehabilitation and recovery. The fact that most of them were semi-literate having dropped out of school due to their addiction made sense to have a simpler program to help PWAs and their families.

Therefore the 4-step program is designed to help people, who have difficulty in reading, understanding and remembering to get through their rehabilitation and recovery phases capturing the main aspects of the 12-step programs used by Alcoholics Anonymous, Noranon and Narcotics Anonymous etc. Dr. Rapiti’s 4 step program focuses on the importance of spirituality in one’s life; importance of correcting one’s faults; value for family in the recovery process; and sharing gratitude for all that one has. Going through the book, it provides practical approaches on how to run a meeting for PWAs and their families, to explaining what addiction is; and various pledges that have to be adhered to. The book offers guidelines for PWAs and their families and friends on how to cope with addiction. Counselling features as an important strategy to include in the management and how to deal with manipulation by addicts. He concludes with 61 values points to ponder which he refers to as – “Jewels that will change your life”. This is a book that is practical in approach that I recommend for any family physician that manages PWAs and their families. It should be a resource book for both undergraduate and postgraduate family medicine students during their training programmes.

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