

ETIOLOGY OF DRY EYE

Introduction

Dry eye disease (DED), also known as dry eye syndrome (DES) or keratoconjunctivitis sicca (KCS), is characterized by ocular irritation and visual disturbance resulting from alterations of the tear film and ocular surface.

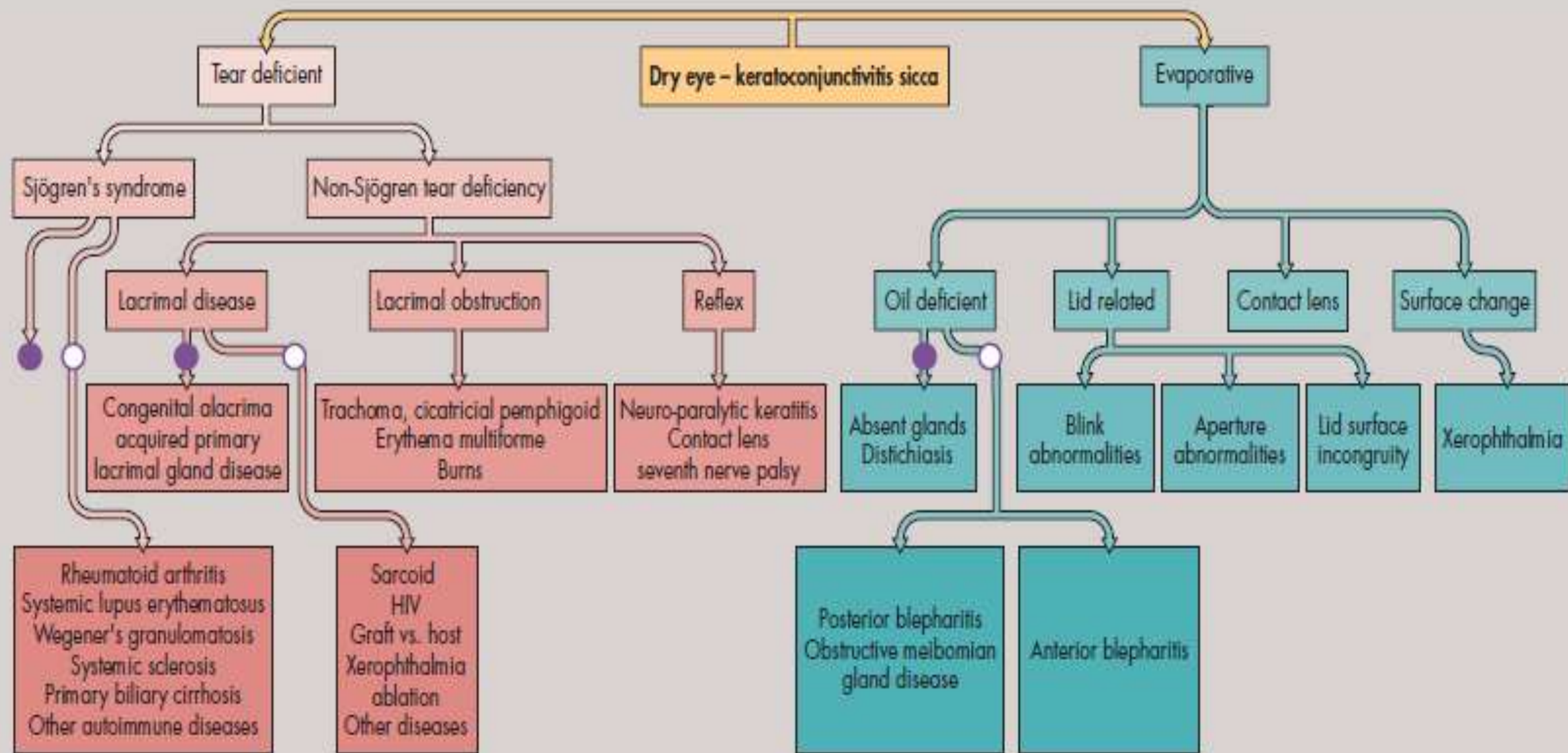
Classification

International Dry Eye Workshop (DEWS)

with a basic division into

- aqueous-deficient
- evaporative types

DRY EYE CLASSIFICATION



● primary ○ secondary

Aqueous-deficient

- **Sjögren syndrome** dry eye
- **Non-Sjögren syndrome** dry eye.

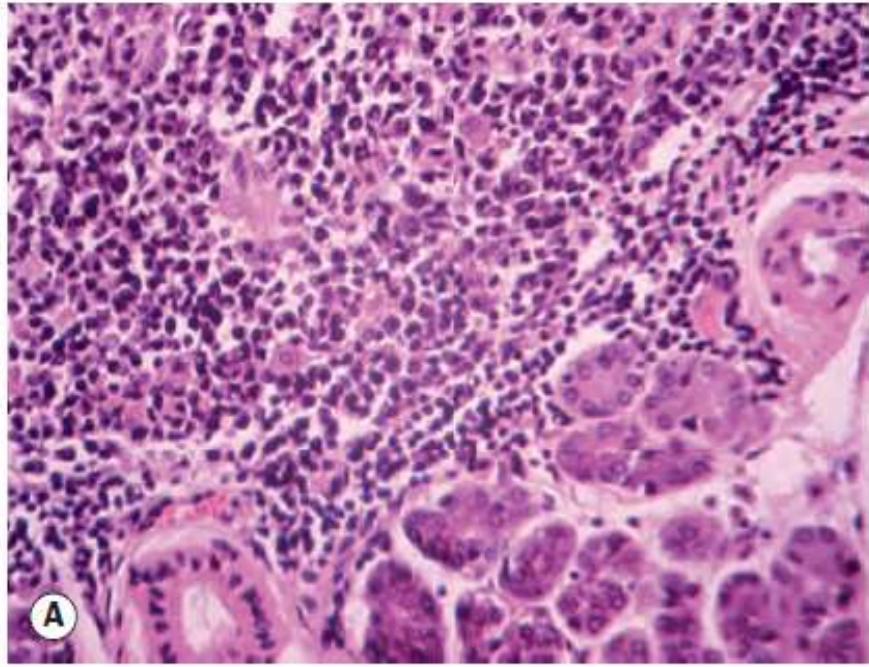
Sjögren syndrome dry eye

autoimmune disorder

lymphocytic inflammation and destruction of lacrimal and salivary glands and other exocrine organs

classic clinical triad

- dry eyes
- dry mouth
- parotid gland enlargement



- Primary
- Secondary: associated with another disease, commonly rheumatoid arthritis or systemic lupus erythematosus.

Revised American-European consensus diagnostic and classification criteria for Sjögren's syndrome

Ocular symptoms—daily dry eye symptoms for more than 3 months, ocular irritation, use of artificial tears more than three times per day.

Oral symptoms—daily dry mouth symptoms for more than 3 months, presence of swollen salivary glands, frequent drinking of liquids to aid in swallowing.

Ocular signs—Schirmer's test I (without anesthetic) ≤ 5 mm in 5 minutes, Rose Bengal score ≥ 4 according to the van Bijsterveld scoring system.

Histopathology—biopsy of minor salivary gland showing inflammation with lymphocytic foci.

Oral signs—reduced salivary flow ≤ 1.5 mL in 5 minutes, parotid sialography

showing salivary duct dilation without obstruction, salivary scintigraphy showing signs of decreased saliva production.

Autoantibodies—presence of anti-Ro(SSA) antibody, presence of anti-La(SSB) antibody.

Non-Sjögren syndrome dry eye.

- Lacrimal deficiency:

primary

secondary

- Lacrimal gland duct obstruction

- Reflex hyposecretion:

sensory (e.g. contact lens wear, diabetes, refractive surgery, neurotrophic keratitis)

motor block (e.g. seventh cranial nerve damage, systemic drugs).

Evaporative dry eye disease

Intrinsic

Meibomian gland deficiency, e.g. posterior blepharitis, rosacea. ○

Disorders of lid aperture, e.g. excessive scleral show, lid retraction, proptosis, facial nerve palsy.

Exposure: Low blink rate, e.g. Parkinson disease, prolonged computer monitor use, reading, watching television.

Drug action, e.g. antihistamines, beta-blockers, antispasmodics, diuretics.



TABLE 4.23.1 Medications Associated With Dry Eye Disease

Mechanism of Action	Class	Medications
Anticholinergic	Antimuscarinics	Tolterodine tartrate (Detrol) Scopolamine
	Antihistamines (sedating compounds are associated with greater dryness)	Chlorpheniramine (Chlor-Trimeton) Diphenhydramine (Benadryl) Promethazine (Phenergan)
	Antiparkinsonian	Benzotropine (Cogentin) Trihexyphenidyl (Artane)
	Antidepressants MAO inhibitors	Amitriptyline (Elavil) Nortriptyline (Pamelor) Imipramines (Tofranil) Doxepin (Sinequan) Phenelzine
	Antipsychotics	Chlorpromazine (Thorazine) Thioridazine (Mellaril) Fluphenazine (Prolixin)
	Antimanics Antiarrhythmics	Lithium Disopyramide (Norpace) Mexiletine (Mexitil)
Antiadrenergic	Alpha-agonists	Clonidine (Catapres) Methyldopa (Aldomet)
	Beta-blockers	Propranolol (Inderal) Metoprolol (Lopressor)
Diuretic	Thiazide	Hydrochlorothiazide
Other	Nonsteroidal anti-inflammatory drugs	Ibuprofen (Advil) Naproxen (Naprosyn, Aleve)
	Cannabinoids	Marijuana

- **Extrinsic**

Vitamin A deficiency.

Topical drugs including the effect of preservatives.

Contact lens wear.

Ocular surface disease such as allergic conjunctivitis.