REVIEW ARTICLE

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Dry Eye Disease: What Should a Non-Ophthalmologic Clinician Know?

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Dry Eye Disease (DED) is a prevalent and often debilitating condition that affects millions of people worldwide. Although its primary management falls under the purview of ophthalmologist but non-ophthalmologic clinicians- such as primary care physicians, rheumatologists and dermatologists-play a crucial role in identifying, diagnosing, and managing this condition. Understanding dry eye disease, its symptoms, risk factors, and treatment options can enhance patient care and improve outcomes.

The prevalence of DED ranges from 5 to 50% among various populations around the world[1-3]. The prevalence of DED in North India is 32%, with the age group of 21 - 40 years affected most commonly[2]. Computer vision syndrome, smoking, diabetes and contact lens wear have been associated with an increased risk of developing DED.

It has been reported that rheumatoid arthritis (RA) tends to cause keratoconjunctivitis sicca (KCS), scleritis, episcleritis and peripheral corneal ulcers, and Sjögren's syndrome (SS) develops as a complication in 11% to 31% of RA patients[4].

Dry eye was found positive in 16.6% of patients with ocular rosacea. Meibomian glands play an important role in structuring the lipid layer of the tear film. Meibomian gland disorder (MGD), which is present in up to of 92% of patients with rosacea, consequently causes dry eye, mainly of the evaporative type[5]. One of the most common ocular features of systemic sclerosis (SSc) is DED, which has been identified to occur in 37 - 79% of patients. It is due to fibrosis of the conjunctiva and lacrimal gland that leads to a tear deficiency[6].

The prevalence of DED symptoms in patients suffering from depression and anxiety was estimated between 21% to 52% (Ulusoy *et al* 2019)[7]. Apart from the disease process itself, antidepressant, antipsychotic and antianxiety medication use are considered as risk factors for DED due to the potential side-effects on the tear film status.

DED can substantially affect vision and quality-of-life, as symptoms often interfere with daily activities, such as reading, writing, or working on video display monitors. Prevalence rates range from 5% to 50%, but can be as high as 75% among adults over 40 years of age, with women most often affected. Among younger adults ages 18 to 45 years, only 2.7% experience DED[3].

Definition

According to Tear Film and Ocular Surface Society Dry Eye Workshop II (TFOS DEWS II)

"Dry eye is a multifactorial disease of the ocular surface characterised by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play aetiological roles"[8].

Pathophysiology

The ocular surface (cornea, conjunctiva, accessory lacrimal glands), meibomian glands (specific sebaceous glands of the eyelid margin, which produce the outer lipid film of the tear film), the main lacrimal gland, and the innervation between them form a functional unit. Any or all of these structures may be affected in dry eye disease. Many recent studies have stated that dry eye is an inflammatory disease that has many features in common with autoimmune disease. Any stress to the ocular surface (environmental factors, infection, endogenous stress, antigens, genetic factors) is considered as the pathogenetic triggering mechanism. Pro-inflammatory cytokines, chemokines, and matrix metalloproteinases lead to the expansion of autoreactive T helper cells which infiltrate the ocular surface and lacrimal gland .The result is a vicious circle of damage to the ocular surface and inflammation. The pathophysiology has been summarised in Fig. 1 and the immunomodulatory pathways depicted in Fig. 2.

Risk Factors and Aetiology

DED is influenced by a variety of risk factors and underlying causes. Age plays a significant role, as older adults are more prone to DED due to natural declines in tear production. Gender is another critical factor; women, particularly during menopause, experience hormonal changes that can increase their likelihood of developing dry eye symptoms.

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Infrequent blinking, often linked to extended computer use or conditions like Parkinson's disease, reduces tear distribution and leads to dryness. Neurologic conditions such as stroke, Bell's palsy, or trigeminal nerve dysfunction can lead to DED. Inflammatory eye conditions, including uveitis and iritis, as well as infectious keratitis caused by herpes simplex or herpes zoster, can also contribute to dry eye (Table I).

Table I: Ocular and non- ocular causes of dry eye

Systemic Diseases:	**Lids:**
– Sjögren's disease	– Blepharitis
– Rheumatoid arthritis	– Meibomian gland dysfunction
– Systemic lupus erythematosus	– Ectropion
– Diabetes mellitus	– Entropion
Hormonal Changes:	**Meibomian Gland Dysfunction**
– Menopause	**Tear Film Abnormalities**
– Androgen deficiency	
Medications:	**Ocular Surgeries**
– Antihistamines	– LASIK
– Antidepressants	– Cataract surgery
– Diuretics	
– Beta-blockers	
– Isotretinoin	
Vitamin A deficiency	**Contact Lens Use**
Environmental Factors:	**Ocular Surface Disorders**
– Low humidity	**Infections**
– Air conditioning	
– Wind exposure	
– Prolonged screen time	
Neurological Disorders:	
– Parkinson's disease	
– Bell's palsy	
– Stroke	

Nutritional factors and systemic diseases like rheumatoid arthritis, lupus, Sjögren's disease, rosacea, thyroid disorders, and diabetes are associated with an increased risk of developing DED[8]. Various systemic disorders causing dry eye have been summarised in Table II.

As medication is a known risk factor for ocular surface diseases, the Dry Eye Workshop II report on iatrogenic dry eye focused on conventional medications such as antihypertensive, antidepressants, antihistamines, corticosteroids, or nonsteroidal anti-inflammatory drugs.

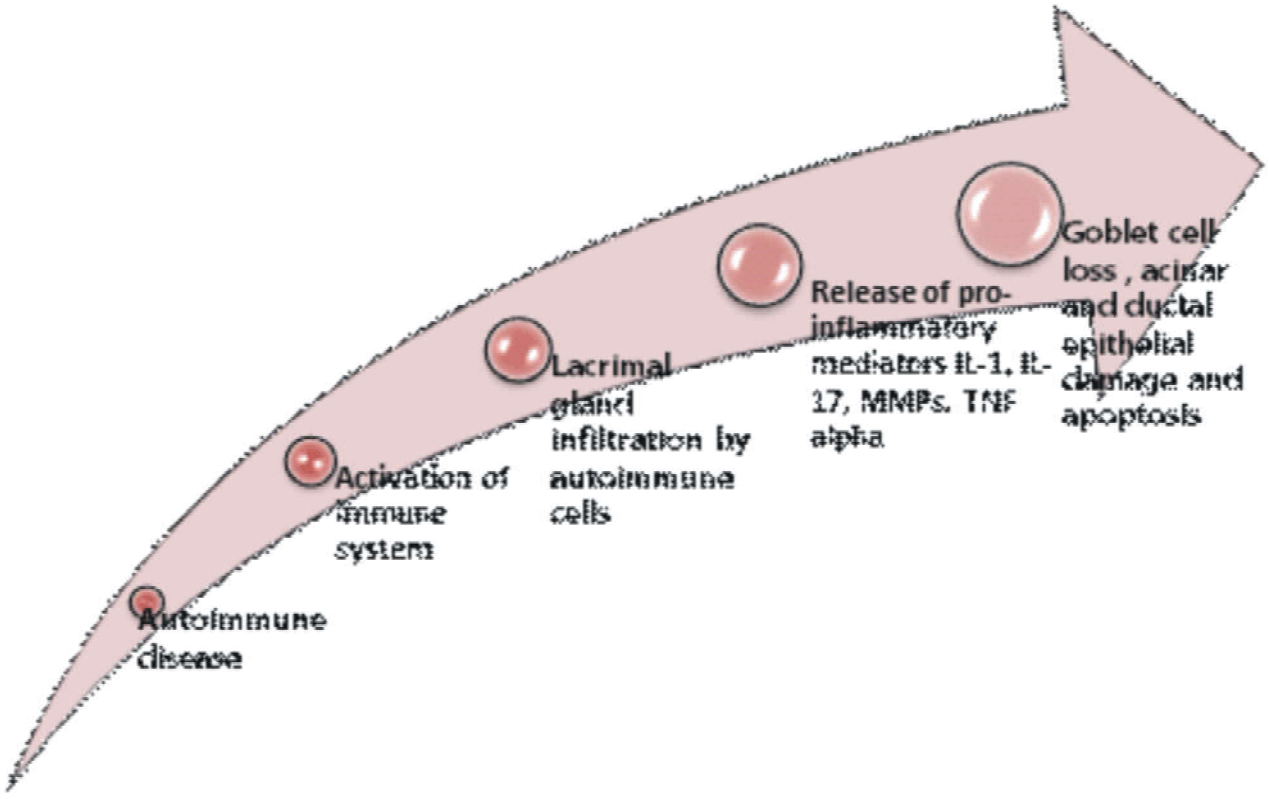


Fig. 1: *Pathophysiology of DED*

Immuno-inflammatory pathway:

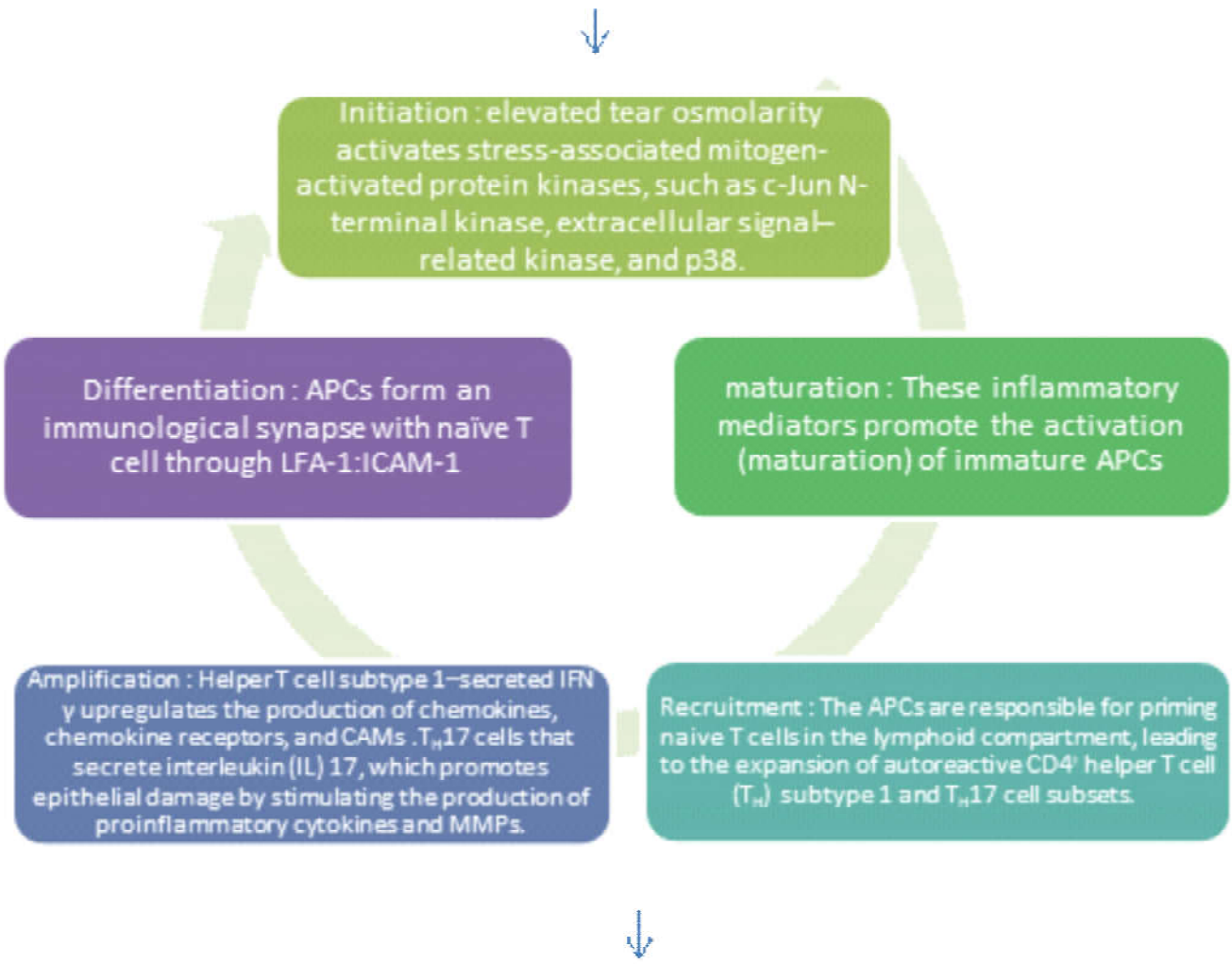


Fig. 2: *Immunoinflammatory pathway. IL: (interleukin), CAMs: (cell adhesion molecules), LFA-1: (lymphocyte function-associated antigen-1), ICAM-1: (intercellular adhesion molecule-1), interferon: (IFN), antigen-presenting cells, MMP: (Matrix metalloproteinase), MPTP: (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine).*

Table II: Systemic diseases associated with DED.

Rheumatologic Disorder	Metabolic Disease	Dermatologic Disease	Psychiatric Disease	Others
Rheumatoid arthritis	Diabetes	Rosacea	Depression	Asthma
Sjögren's disease	Thyroid disorder	Psoriasis	Insomnia	Vitamin A deficiency
Osteoporosis		Skin allergy	Use of antidepressant TCA>SSRI/SNRI	Stevens-Johnson Syndrome
Systemic Sclerosis		Eczema	Parkinson Disease	Migraine
Sarcoidosis	Hypercholesterolemia	Use of isoretinoin		Graft-versus-host disease (GVHD)
	Irritable Bowel Syndrome			

TCA = Tricyclic antidepressant, SSRI = Selective serotonin reuptake inhibitor, SNRI = Serotonin nor-adrenalin reuptake inhibitor.

Some common medications causing dry eye have been summarised in Fig. 3.

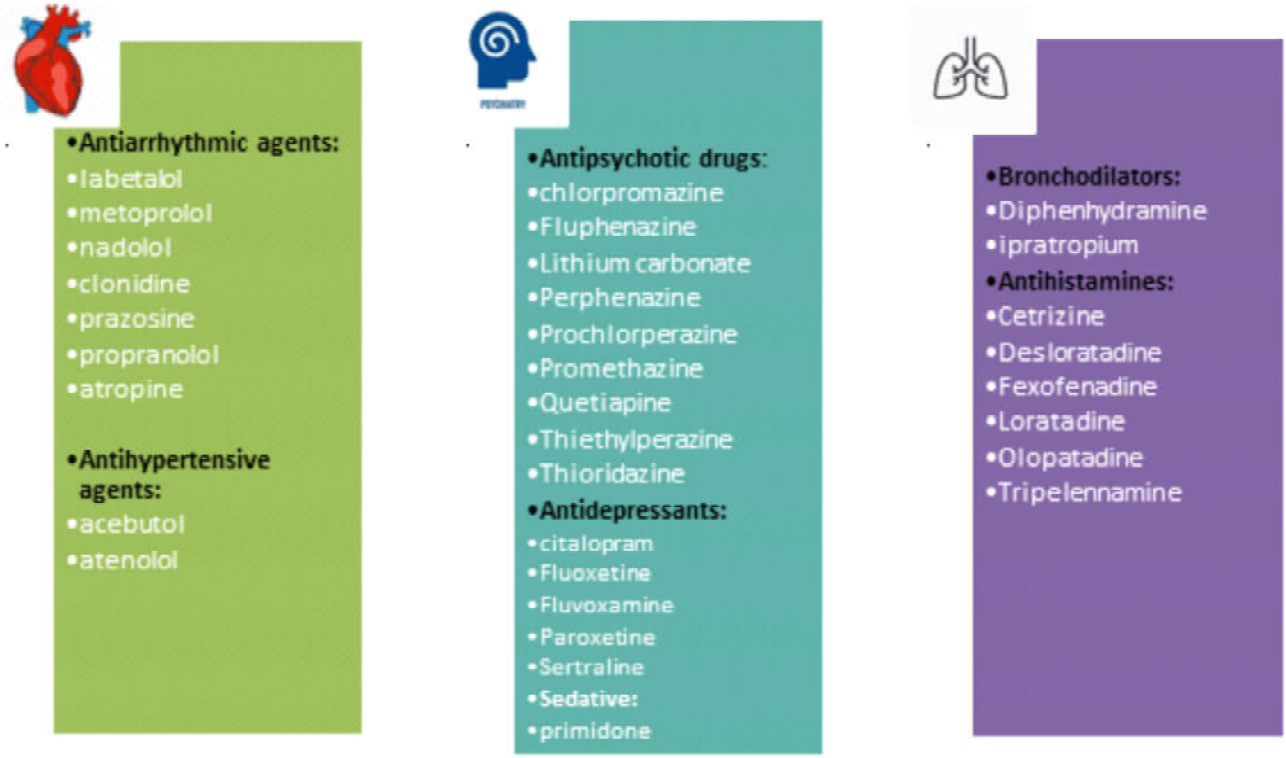


Fig 3: *Some common medications which causes DED*

Classification of DED

National eye institute (NEI)/Industry Workshop classification was useful and durable scheme for over a decade, but it did not reflect newer knowledge on pathophysiological mechanisms, effects on vision, and the utility of an assessment of severity of disease (Fig. 4). So, a classification scheme was presented by the TFOS DEWS based on aetiopathogenesis (Fig. 5).

Due to potential overlap between aqueous deficient and evaporative categories and issues regarding accuracy of placement of some conditions within the DEWS sub-classification zone, a new dry eye classification scheme was made which incorporates triaging elements to provide clarity in diagnosing DED (Fig. 6).

Symptoms

Dry eye disease presents with a range of symptoms (Fig. 7). Individuals often experience persistent dryness and a gritty sensation in their eyes, accompanied by burning or stinging feelings. Redness may also be noticeable. Excessive paradoxical tearing can occur as a reaction to the irritation, leading to watery eyes. This is because the unhealthy tear film and the irritation that comes from it stimulate the brain to produce reflex of tears. It helps in counteracting the irritation. Additionally, blurred vision is common, particularly during prolonged activities like reading or using screens. Some may also notice stringy mucus in or around the eyes. These symptoms can vary in severity and may worsen throughout the day[8].

Diagnosis

Persons with DED symptoms should be referred for a complete ophthalmologic examination. There is no single gold standard sign or symptom for diagnosing DED. Evaluation of symptoms and signs of DED is recommended, as signs may be present without symptoms and *vice-versa*. Understanding the work-up can help non-ophthalmic clinicians identify patients at higher risk and consider appropriate preventive measures or referrals.

Patient's history: A comprehensive history is very essential including risk factors, systemic disease and medication use. Some questionnaires are available for history taking in DED (Table III). Ocular surface disease index (OSDI) or dry eye questionnaire-5 (DEQ-5) are well suited questionnaires for non-ophthalmologists because of ease of use and reliability.

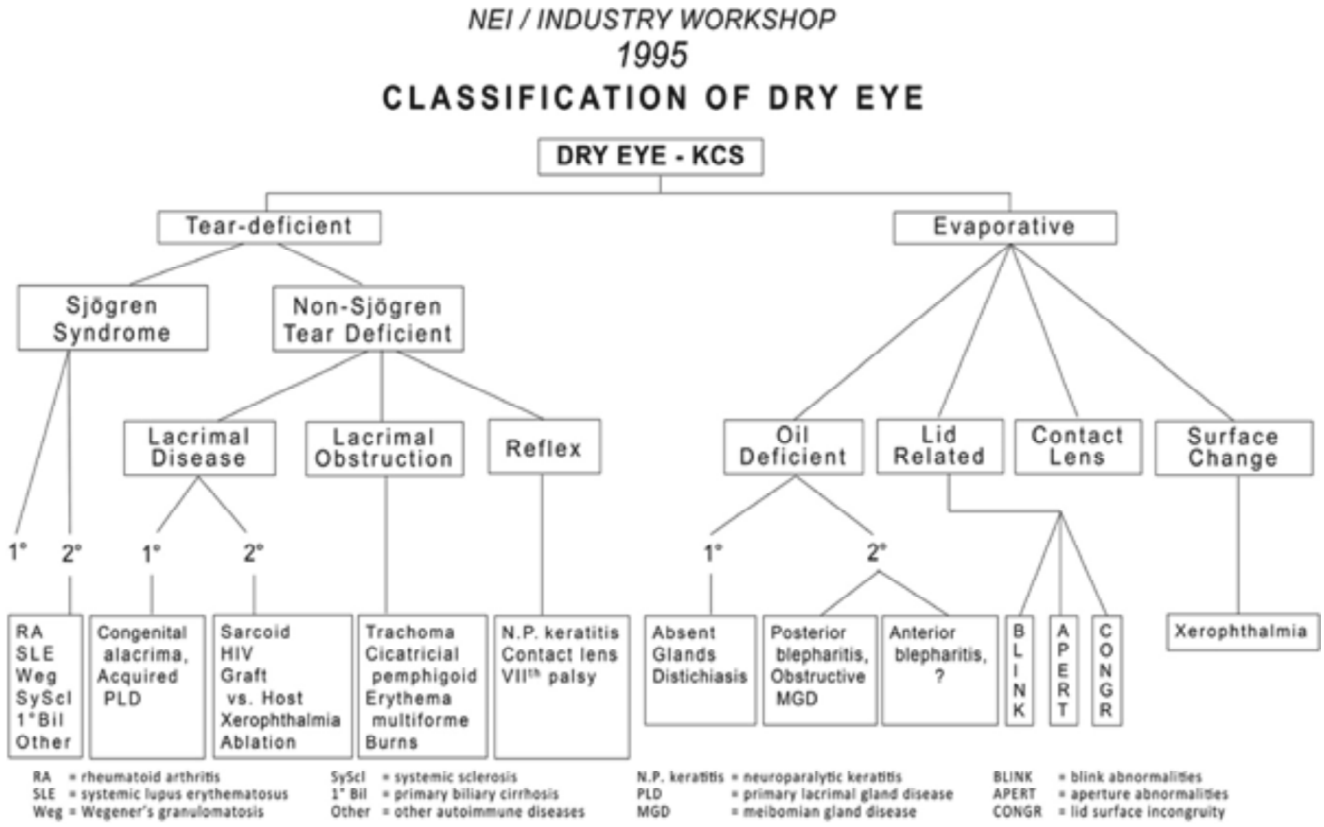


Fig 4: *NEI/Industry Workshop classification[8].*

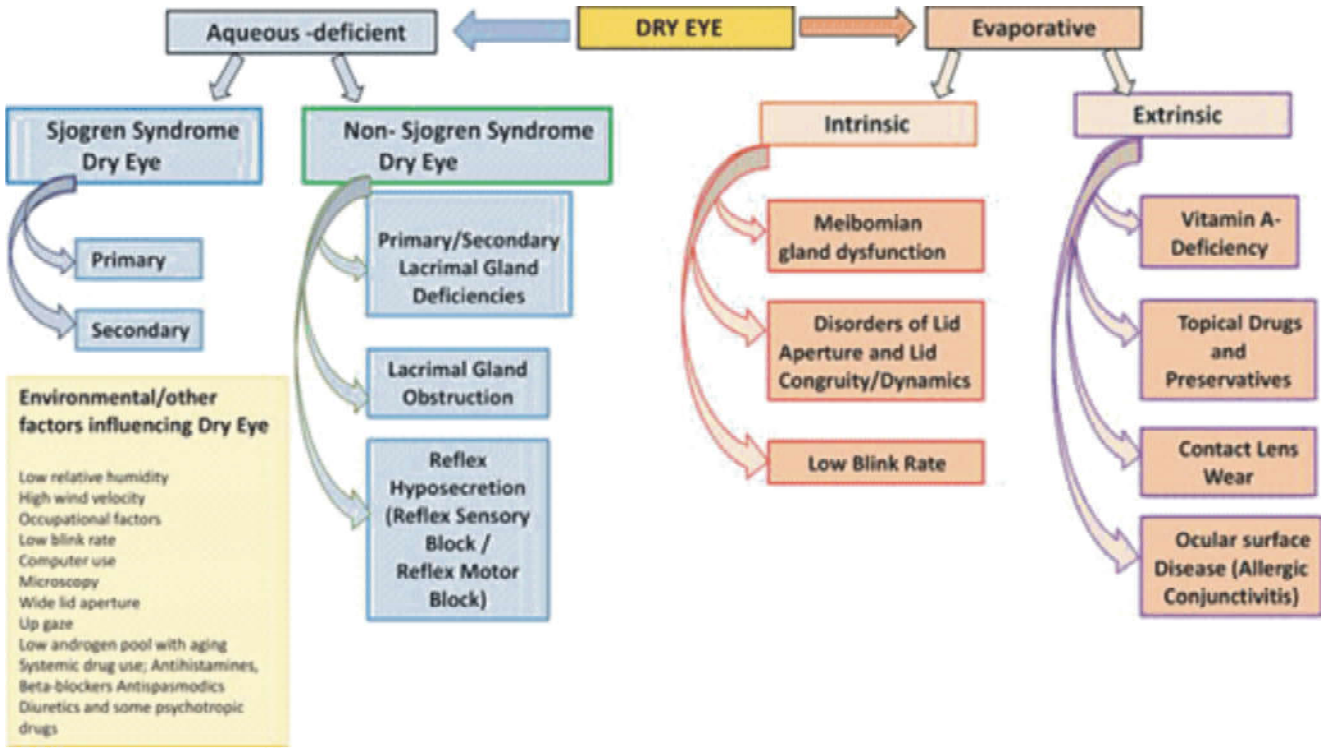


Fig. 5*: The 1995 Classification of dry eye[9].*

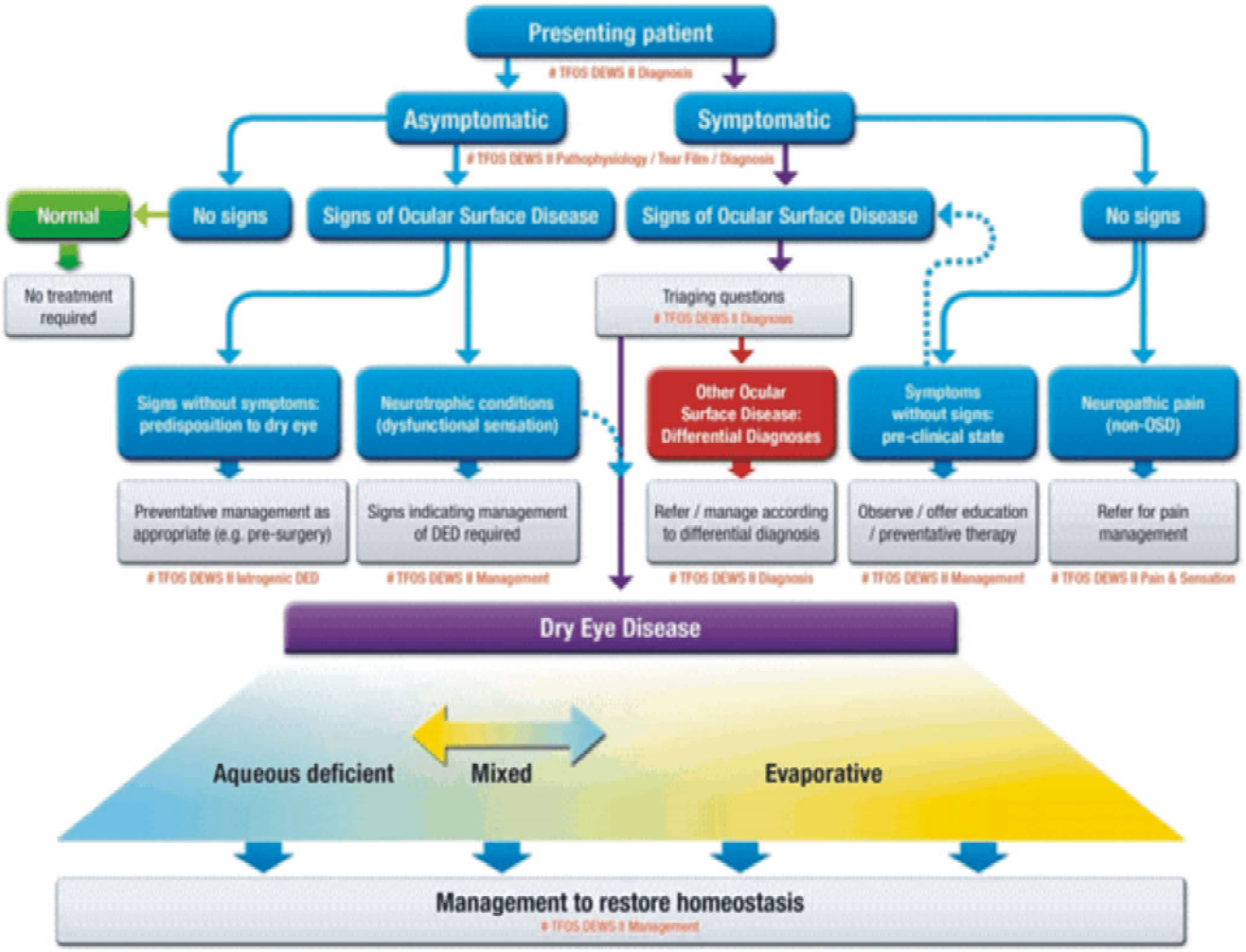


Fig. 6: *Classification of DED which incorporates a clinical decision algorithm[8].*

External examination

Patients can present with a variety of signs and symptoms, especially when DED is associated with autoimmune diseases. Examination of cranial nerves, particularly the trigeminal and facial nerves should be done. The eyelids must also be carefully examined for incomplete closure or malposition, as these can lead to complications like exposure keratopathy. Additionally, erythema of the eyelid margins, abnormal deposits or secretions should be noted, as they can signify conditions such as blepharitis. Furthermore, assessing for trichiasis, ectropion and entropion should be checked properly. Finally, the adnexa should be inspected for enlargement of lacrimal glands, which can suggest lacrimal system disorders. This thorough examination provides critical insights into the health of the ocular surface.

Slit-lamp examination

During a slit lamp examination for DED, several key findings may be observed. These findings have been summarised in Table V.

Diagnostic tests

Diagnostic tests are necessary in order to distinguish

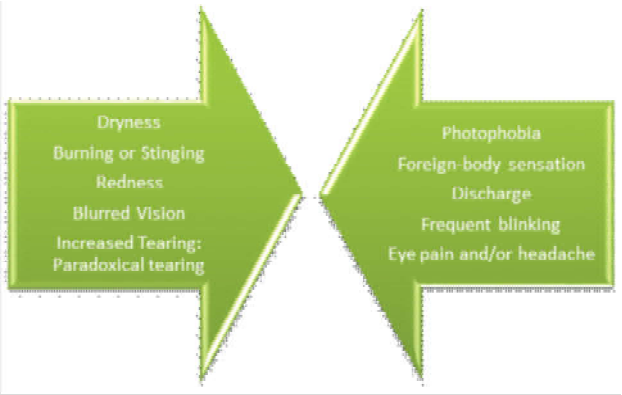


Fig. 7: *Symptoms of DED.*

between dry eye, infections and allergies as patients can present with a similar clinical presentation, but require different treatments. On the other hand, antiallergic or epitheliotoxic antibiotics can worsen the DED. A series of diagnostic modalities are available for the diagnosis of DED and the same have been summarised in Tables VI and VII.

Tear film stability can be assessed by corneal topography, interferometer, and aberrometry.

Table III: Commonly available questionnaires for dry eye assessment.

	OSDI	SPEED	DEQ-5	IDEEL	SANDE	DEEP	NEI-VFQ-25	McMonnies	CLDEQ-8
Items	12 (3 subscales)	8	5	57	2	19	25	14	8
Score Scale	0 - 100	0 - 28	0 - 22	0 - 100	Visual analog scale	0 - 114	0 - 100	0 - 45	0 - 37
Year of development	1997	2005	2009	2003	2007	1998	2001	1986	2009
Description	Symptoms, vision related function, Environment triggers, Quality of life	Frequency and severity of symptoms, assessment of diurnal and 3- month interval changes	Frequency and intensity of symptom within previous month	Symptoms, quality-of-life, treatment satisfaction	Frequency and intensity of symptoms/ discomfort	Frequency of symptoms	Effect of visual impairment on health related quality-of-life	Risk factors, frequency of symptoms, Environment trigger sensitivity	Frequency and intensity of symptoms among contact lens users
Access	Open	Open	Paid	Paid	Paid	Open	Open	Paid	Paid
Validity	Good	Good	Good	Fair	Good	Epidemiologic studies, High specificity	Fair-Good	Weak	Good
Designated Diagnostic cut-off value	Present (Mild 13-22 Moderate 23-32 Severe 33-100)	Absent	Present (>6 - suspected dry eye >12- suspected Sjogrens)	Absent	Absent	Absent	Absent	Present (14.5)	Present (12)
Assessment of Quality-of-Life	Present	Absent	Absent	Present	Absent	Absent	Present	Absent	Moderate
Validated Languages	English, Spanish, Portuguese, Chinese, Farsi, Bahasa, Japanese, Filipino	English Italian	English, Spanish	English, Chinese	English	English	Over 50 languages	English, Chinese	English, Japanese

OSDI: Ocular Surface Disease Index, SPEED: Standard Patient Evaluation of Eye Dryness, DEQ-5: Dry Eye Questionnaire-5, IDEEL: Impact of Dry Eye on Everyday Life, SANDE: Symptom Assessment in Dry Eye, DEEP: Dry eye screening for dry eye epidemiology projects, NEI-VFQ-25: National Eye Institute Visual Function Questionnaire-25, CLDEQ-8: Contact Lens Dry Eye Questionnaire-8.

Table IV: Signs and symptoms of autoimmune diseases associated with DED.

Rheumatoid Arthritis	Sjögren's Disease	Psoriasis	Rosacea	SLE	SSc	SJS
Swollen joints	Dry mouth	Scaly silver plaque with clear border	Facial flushing	Skin rashes	Thick and immobile skin	Fever
Morning stiffness	Dry skin	Nail pitting, thick nails	Facial bumps and pimples	Joint pains	Raynauds phenomenon	Lymphadeno-pathy
Joint pains	Difficulty in opening mouth	Dry skin	Phymatous changes	Headache	Sclerodactyly	Erythematous, Purpuric macule of irregular size and shape
Rheumatoid nodules	Difficulty in swallowing	Joint pains		Muscle pain, etc.	Calcinosis	
Joint deformity of metacarbo-phalangeal joints in late stage					Telangiectasia	

SLE = Systemic lupus erythematous; SJS = Stevens-Johnson syndrome; SSc = Systemic sclerosis.

Table V: Slit lamp examination in DED.

Adnexa:	Conjunctiva	Cornea:
Eyelashes: trichiasis, distichiasis, deposits on eyelashes **Anterior and posterior eyelid margins:** abnormalities of meibomian glands, (e.g., orifice metaplasia, reduced expressibility, atrophy), character of meibomian gland secretions, [e.g., turbid, thickened (tooth-paste sign), foamy, scarring, deficient], keratinisation, scarring **Puncta:** position, patency, position of plugs if present **Tear film:** height of the meniscus, debris, mucus strands, and foam	Inferior fornix and tarsal conjunctiva: e.g., mucous threads, gross scarring, stellate scar (in healed trachoma), erythema, papillary reaction, enlarged follicles, keratinisation, fornix shortening, symblepharon Bulbar conjunctiva: e.g., punctate staining with fluorescein, follicles, Herbert's pit , hyperaemia, localised drying, Bitot's spot, keratinisation Temporal lid parallel conjunctival folds (LIPCOFs): They are the result of increased friction between lid and conjunctiva.	localised inter-palpebral drying, punctate epithelial erosions, superficial punctate staining with Rose Bengal or fluorescein dyes, filamentary keratopathy, epithelial defects, mucous plaques, keratinisation, pannus formation, localised dellen, thinning, infiltrates, ulceration, scarring, neovascularisation, corneal or keratorefractive surgery

Table VI: Slit lamp examination in DED.

Test	Findings
Schirmer I test (without anaesthesia) **basal and trigeminal reflex tear production**	**Wetting of schirmer paper:** • 0 to 5 mm: extremely dry eyes • 5 to 10 mm: moderately dry eyes • 10 to 15 mm: possible dry eyes • Longer than 15 mm: normal tear function The Dry Eye Workshop proposes a Schirmer test I cut-off value of 10 mm for 5 minutes as one of the criteria for diagnosing DED[10].
Schirmer II (with anaesthesia) basal tear production	>10 mm = normal If <10 mm → irritate nasal mucosa: <1 mm = Sjögren's disease >1 mm = non-Sjögren dry eye
Schirmer III (without anaesthesia)	Schirmer test III assesses reflex-stimulated lacrimal secretion after looking at the sun for some time. It has less diagnostic value.
Phenol red impregnated thread test **Yellow to red colour after placing in cul-de-sac for 15 seconds**	<6 mm abnormal
Tear function index = Schirmer II/tear clearance	<96% = suggestive of dry eye <34% = diagnostic of dry eye
Tear Meniscus Assessment	<0.25 mm is suggestive of dry eye
Tear breakup time	Less than 10 seconds is abnormal Grade 1 = 10 sec Grade 2 = 5 - 10 sec Grade 3 = 3 - 5 sec Grade 4 = <3 sec

Tear film integrity

Fluorescein Staining

It stains areas of the corneal and conjunctival epithelia where there is disruption of intercellular junctions to allow the dye to permeate into the tissue. Saline-moistened fluorescein strips are used to stain the tear film. After instilling dye, the ocular surface is examined through a slit lamp microscope using a cobalt blue filter.

Table VIII: Some laboratory tests.

Tear film osmolarity	Matrix Metalloproteinases (MMP) and Lactoferrin	Biopsy
Elevated osmolarity and increased variability of osmolarity of the tears are characteristics of DED. Osmolarity values typically increase with disease severity. Various cutoff values have been reported: 308 mOsm/L = mild-to-moderate disease, whereas 316 mOsm/L is cut-off for more severe disease[6].	MMPs are found in the tears of individuals with dry eyes. Matrix metalloproteinase-9 (MMP-9) levels can be tested using a point-of-care test. MMP-9 levels can be elevated in other inflammatory conditions, such as graft-versus-host disease, Stevens-Johnson syndrome, and following corneal surgery. lactoferrin is secreted by lacrimal glands. So, abnormality in this test is an indicator of lacrimal gland dysfunction.	**Conjunctival biopsy:** Conjunctival sample is taken from lower fornix as maximum goblet cells are present there. Decrease in goblet cell is suggestive of mucin deficiency. **Lacrimal gland and minor salivary gland biopsy:** Lymphocyte and plasma cell infiltration in gland is diagnostic of Sjögren's disease. This is the most specific test for Sjögren's disease diagnosis

Table IX: DEWS dry eye severity grading scheme[9].

Dry eye severity level	1	2	3	4
Discomfort,severity and frequency	Mild and/or episodic; occur under environmental stress	Moderate episodic or chronic, stress or no stress	Severe frequent or constant without stress	Severe and/or disabling and constant
Visual symptoms	None or episodic mild fatigue	Annoying and/or activity limiting episodic	Annoying chronic and/or constant limiting activity	Constant and/or possibly disabling
Conjunctival injection	None to mild	None to mild	+/-	+/++
Conjunctival staining	None to mild	Variable	Moderate to marked	Marked
Corneal staining severity/location	None to mild	Variable	Marked central	Severe punctate erosion

Rose Bengal staining

It may be performed using a saline moistened strip. The saline drop used to moisten the strip should remain in contact with the strip for at least a minute to achieve an adequate concentration of Rose Bengal to stain the ocular surface. Patients should be informed that the drop might irritate the eye. Rose Bengal staining is more intense on the conjunctiva than the cornea. The dye stains ocular surface cells that lack a mucous coating as well as debris in the tear film.

Lissamine green dye

It has a staining profile similar to that of Rose Bengal and may cause less ocular irritation.

Laboratory tests

While the diagnosis of DED is primarily based on clinical evaluation and symptom assessment, certain laboratory tests can provide valuable information regarding tear production, ocular surface health, more accurate diagnosis and help in ruling out other potential conditions which lead to ocular discomfort. A series of relevant laboratory tests available for the diagnosis of DED as in Table VIII.

Some newer laboratory tests

Serum autoantibody measurement

Assessment of autoantibodies (ANA, RF, SS-A, and SS-B) is performed for diagnosis of Sjögren's disease. Of these, SS-A is probably the most sensitive and specific antibody for Sjögren's but alone is not diagnostic since it may be present in other autoimmune disorders. It may be absent in up to a third of Sjögren's disease cases[11].

Anti-centromere antibodies are predominantly observed in limited cutaneous systemic sclerosis, although they may occur in diffuse cutaneous systemic sclerosis. Anti-topoisomerase I antibodies and Anti-RNA polymerase III are predominantly observed in diffuse cutaneous systemic sclerosis. Anti-U3-RNP (fibrillarin) antibodies correlate with increased internal organ involvement, diffuse cutaneous manifestations, interstitial lung disease, pulmonary hypertension and poor prognosis[12].

Tear cytokines and chemokines

The levels of tear cytokines and chemokines are important and reflect the level of epithelial disease. Certain cytokines can highlight a specific disease process, for example, elevation of Th1 and Th17 subclasses of cytokines suggest involvement of particular T lymphocyte differentiation pathways in the disease[13].

Elevation of tear Th2 cytokines suggests a more allergic-based disease. Tear assay for tumour necrosis factor alpha, interferon gamma, IL-1 beta and IL-6 helps in assessing dry eye disease.

Management

The aims for treating DED are to reduce or alleviate signs and symptoms of dry eye, maintain and improve visual function and reduce or prevent structural damage. Patients with dry eye symptoms often have many contributory factors. Tear replacement is frequently unsuccessful when used as the sole treatment if additional causative factors are not concomitantly addressed.

Treatment for dry eye disease involves a step ladder approach, corresponding to disease severity and must take into account the assosciated local and systemic diseases. For patients with irreversible tear deficiency or evaporative disease associated with blephritis or any autoimmune disease – ophthalmologist should educate the patient about the natural history and chronic nature of dry eye disease. Patient education is an important aspect of successful management of DED. Treatment of mild eye disease has been summarised in Table X.

Table X: Management of mild DED.

Mild Dry Eye:	• Education • Environment modifications: Use humidifiers, avoid direct air (fans, cooler, heater, air conditioner), wear sunglasses outdoors • Elimination of offending topical and/or systemic medications • Avoid cigarette smoking • Stay hydrated • Artificial tear substitutes; gels, ointments • Eyelid warm compression and eyelid hygiene • Treatment for contributing ocular factors such as blepharitis or meibomitis – e.g., Systemic Tetracyclines.

Moderate Dry Eye

Preservative free tears are important. The frequency may be increased from 6 - 12 times depending upon the patient's need, occupation, and lifestyles.

In patients with moderate to severe dry eye disease, anti-inlammatory treatment is necessary to break the vicious cycle of surface damage and inflammation. 0.05% topical cyclosporine prevents activation and nuclear translocation of cytoplasmic transcription factors that are required for T-cell activation and inflammatory cytokine production. It also inhibits mitochondrial pathways of apoptosis of lacrimal gland and goblet cells. Topical corticosteroids have been reported to decrease the symptoms of ocular irritation, decrease corneal fluorescein staining, and improve filamentary keratitis. Low-dose topical corticosteroids therapy can be used at infrequent intervals for 2-week to suppress irritation secondary to inflammation. Patients prescribed corticosteroids for dry eye should be monitored closely for adverse effects such as increase in intraocular pressure, corneal melting, and cataract formation. Treatment of moderate DED has been summarised in Table XI.

Severe Dry Eye

In addition to the treatments for mild and moderate dry eye, the following treatments may be considered:

Oral cholinergic agonists like pilocarpine and cevimeline, have been used to treat the symptoms of dry mouth in patients with Sjögren disease. These medications bind to muscarinic receptors, which stimulate secretion of the salivary and sweat glands, and they appear to improve tear production. Oral pilocarpine (5 mg) 4 times daily-causes a significant overall improvement. The most common side effect is excessive sweating. Oral cevimeline (30 mg) 3 times daily, is another cholinergic agonist that has been found to improve ocular irritation symptoms and aqueous tear production. This agent may have fewer adverse systemic side-effects than oral pilocarpine.

Table XI: Management of moderate DED.

Moderate Dry Eye:	• In addition to the treatments for mild dry eye, the following treatments may be considered: • Artificial tears: Use preservative free artificial tears • Anti-inflammatory therapies: • 0.05% topical Cyclosporine (FDA approved) prevents activation and nuclear translocation of cytoplasmic transcription factors that are required for T-cell activation and inflammatory cytokine production. It also inhibits mitochondrial pathways of apoptosis of lacrimal gland and goblet cells. It is used 2 times a day usually for 2 - 4 weeks. • 0.03% Tacrolimus eyedrops • Topical Corticosteroids

Table XII: Management of severe DED.

Severe Dry Eye:	In addition to the treatments for mild and moderate dry eye, the following treatments may be considered: • Oral cholinergic agonists • Oral pilocarpine (5 mg) 4 times daily • Oral cevimeline (30 mg) 3 times daily • Systemic immunosuppressants • Autologous serum drops • Mucolytic agents Topical acetyl-cysteine (10%) • Correction of eyelid abnormalities • Punctal occlusion: It can be temporary and permanent. • Tarsorrhaphy

Systemic immunosuppressants are used for patients with systemic disease such as rheumatoid arthritis, progressive

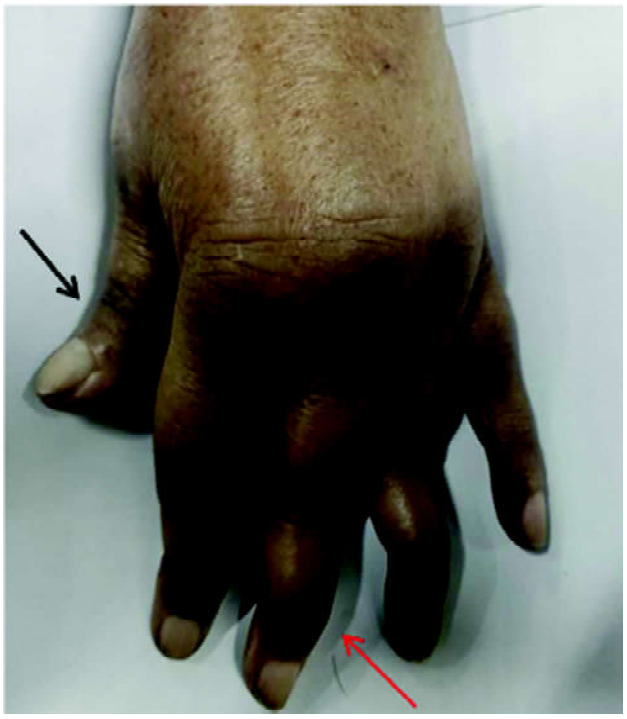


Fig. 8: *Hands in rheumatoid arthritis (red arrow shows swan neck deformity, black arrow shows boutonniere deformity).*

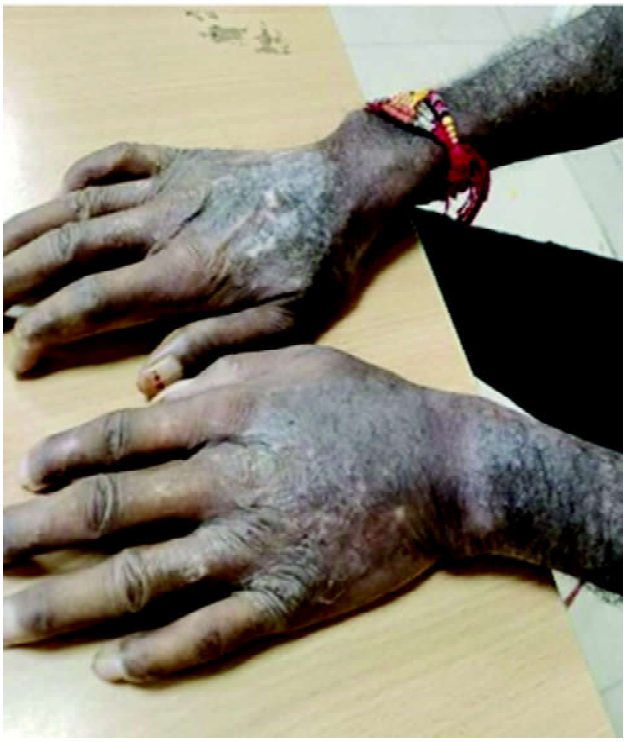


Fig. 9: *Cutaneous systemic sclerosis.*

systemic sclerosis or SLE.

Autologous serum drops have been reported to improve

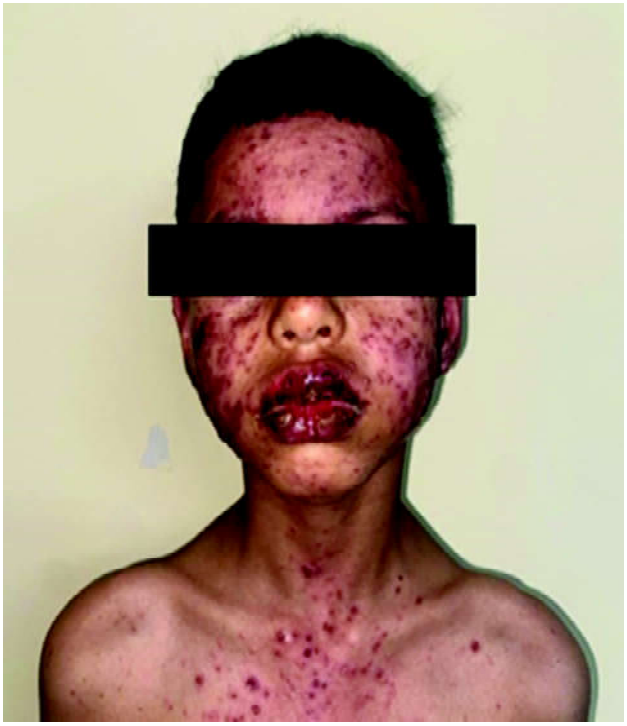


Fig. 10: *Stevens-Johnson syndrome.*

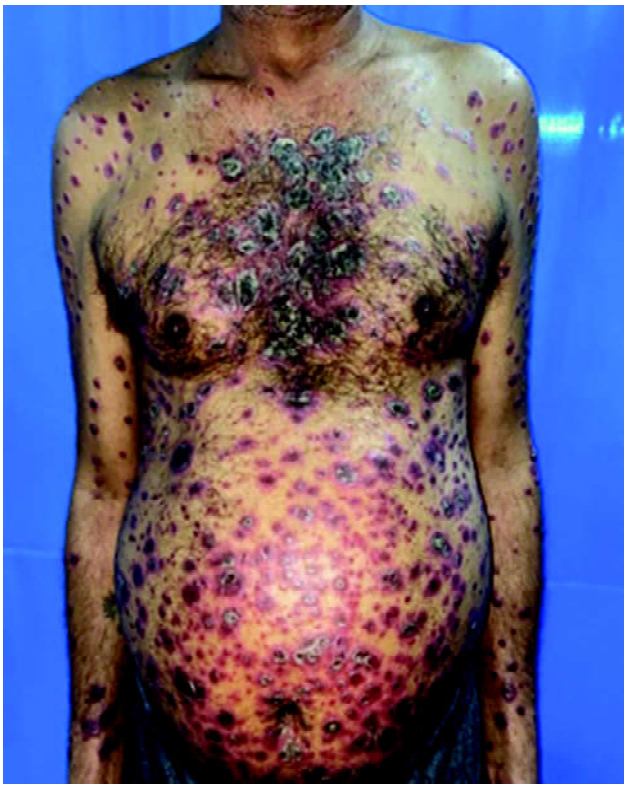


Fig. 11: *Psoriasis vulgaris.*

ocular irritation symptoms as well as conjunctival and corneal dye staining in patients.

Table XIII: Some newer drug for traeatment of DED.

Topical Lifitegrast (Xiidra) 5%:
– Dosage :twice a day with artifical tears
– Mimics intercellular adhesion molecule-1 (ICAM-1), blocking interactions between ICAM-1 and lymphocyte functional associated antigen-1 (LFA-1), thus inhibiting T-cell activation and migration
Rebamipide (2%):
– Rebamipide increases the secretion of both membrane-associated and secreted-type mucins through mucin production in the conjunctival goblet cells and has a good effect on corneal healing
– It has anti-inflammatory action.
– It improves ocular surface epithelial health.
Lacritin:
– It is an ocular specific glycoprotein secreted primarily by acinar cells of lacrimal gland.
– Lacritin levels are significantly decreased in patients with Sjogren's disease as compared to healthy controls.
– Topical lacritin has been found to increase tear secretion, decrease lissamine green staining, and reduce signs of epithelial damage.
Lubricin:
– Mucin-like glycoprotein that is expressed by the normal ocular surface.
– It is an essential part of the ocular surface glycocalyx, preventing epithelial dysfunction and degradation.It decreases friction between cornea, conjunctiva and eyelid .
– It significantly outperformed sodium hyaluronate in ameliorating both signs and symptoms.

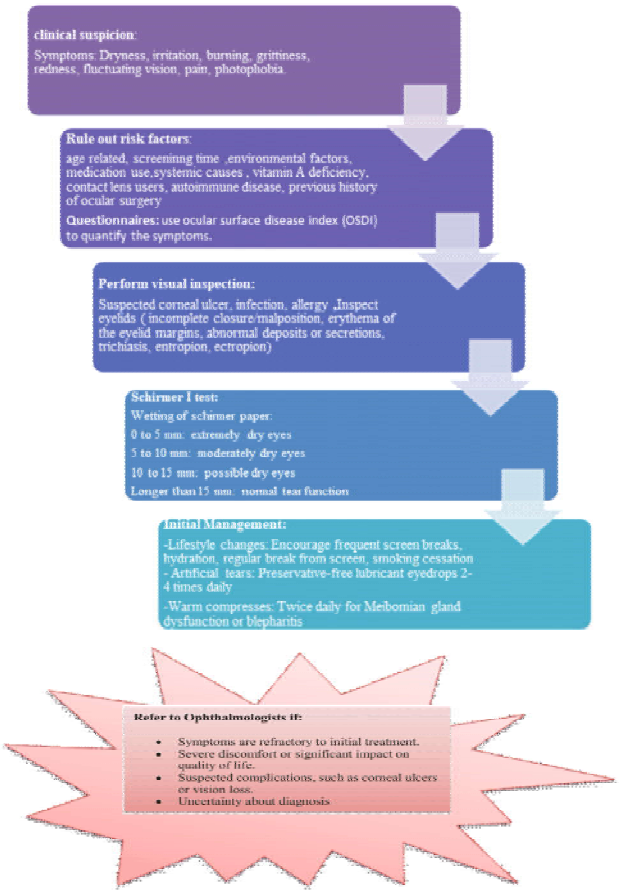
Topical acetylcysteine (10%), a mucolytic agent may be used four times a day to treat filamentary keratitis. Filaments can also be debrided with a cotton-tip applicator, dry cellulose sponge, or with a blunt forceps.

Correction of eyelid abnormalities resulting from blepharitis, trichiasis, or lid malposition, (e.g., lagophthalmos, entropion/ectropion) may be considered prior to permanent punctal occlusion.

Punctal occlusion is considered in dry eye when the medical means of tear substitutes are ineffective or impractical. It can be done surgically with silicone or thermo-labile polymer plugs that are lodged at the punctal orifice. Tarsorrhaphy may be required to decrease tear evaporation.

A collaborative approach to managing DED enhances adherence, addresses underlying causes. Multidisciplinary team involvement of following clinicians can help in createing a patient-centered care environment.

- Rheumatologists: Evaluate and manage autoimmune conditions like Sjögren's disease, SLE, and rheumatoid arthritis.



Flow chart 1: *Approach to the patient with suspected Dry eye disease.*

- Endocrinologists: Manage thyroid eye disease, diabetes or other hormonal conditions which can cause or exacerbate DED.
- Dermatologists: Address rosacea-related ocular surface inflammation and psoriasis.
- Psychiatrist: Many antipsychotic and antidepressant medications cause DED.
- General Physicians: Address systemic diseases like diabetes, rheumatoid arthritis, thyroid dysfunction or medication use contributing to DED.
- Pharmacists: Educate on medication side-effects, (e.g., antihistamines or diuretics) that may worsen dry eye.

Conclusion

Dry Eye Disease is a common and multifaceted condition that requires awareness and understanding from all healthcare providers, not just ophthalmologists. Non-ophthalmic clinicians, by recognising the symptoms, understanding the risk factors, and being knowledgeable about initial management strategies, can significantly

contribute to the effective treatment and management of dry eye disease. A collaborative approach, including timely referrals to specialists will ensure comprehensive care and improved quality-of-life for patients suffering from this condition.

Educating patients about dry eye disease is a critical and essential aspect of management. Non-ophthalmic clinicians should provide information on the chronic nature of the condition, the importance of adhering to treatment regimens, and lifestyle changes that can improve symptoms. Regular follow-up and monitoring of patient progress are essential to adjust treatment plans and ensure optimal outcomes.

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