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Review Article

CONCEPT OF *SHUSHKAKSHIPAKA* (DRY EYE SYNDROME) IN AYURVEDA WITH MODERN COUNTERPART: A REVIEW

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ABSTRACT

Eyes are the windows to the soul. Ayurvedic classical texts have mentioned about eye and its diseases since *Vedic* and *Samhita kala*, thus showing the importance of eye and ways to protect it. That is why eye care has been one of the priorities since the ages. In Ayurveda clinical features related to Dry eye are generally seen in *Suskaksipaka*.

Over use of Video Display Terminals (V.D.T) like Computer, Mobiles, and Television & faulty lifestyle are some of the causes. Among these disorders, many are related to *Indriya* (Sensory Organ) especially *Caksurindriya* (Eye). In metro city, more than 50% cases from ophthalmic OPD are of Dry Eye Syndrome. Dry Eye Syndrome is a common ophthalmic condition, in its real sense is a problem for eye care practitioners due to its variegated etiologies, multi factorial physiopathology, lack of gold standard for diagnostic procedure. No curative treatment modalities are available and the palliative measures are inadequate too. It presents a great challenge to physicians. That's why its prevalence is very high. It is 5% to 35% worldwide, while in India it is 29.25%. Dry Eye Syndrome also known as Kerato-Conjunctivitis Sicca (KCS) is considered a disease of the modern era. Dry eye syndrome, one of the most frequently encountered ocular morbidities, a growing public health problem and one of the most common conditions seen by the eye care practitioners. Tear secretion provides continuous moisture and lubrication on the ocular surface and provides oxygen to the corneal- epithelium. When the normal tear production is compromised, it leads to dry eyes.

INTRODUCTION

Śālākya Tantra is one of eight branches of Āyurveda which has evolved a long time ago. It deals with the etiology, diagnosis, prognosis, prevention and treatment of diseases that are located above the *Ūrdhwa jatrugata* i.e., eyes, nose, head and ears. In this branch the prime importance is given to *Netra*[1], as it is considered as *Pradhāna* among all *Indriyas.*

Dry eye syndrome (DES) is a multifactorial disorder of the tears and ocular surface[2] have clinical features like irritation, foreign body sensation, feeling of dryness, itching, non specific ocular discomfort, chronic sore eyes and photophobia etc[3]. For treatment the lubricating drops like- Carboxy methyl cellulose 0.5%, used which reduces the effects of Dry Eye, but its preservatives are harmful to eye. So long term use is not possible and effects of artificial drops are temporary. Also other treatment modalities or surgery are not accessible and not so effective. Therefore these limitations make us think about effective and alternative treatment for dry eye. This opens the door to other systems of medicine including *Āyurveda* to suggest and contribute alternative treatment modalities to check the suffering from dry eye.

Śuskāksipāka

One of the classification given by *Ācārya Suśruta* for eye diseases is "Diseases affecting all parts of eye ball i.e. *Sarvagata Netraroga*; includes disease *Śuskāksipāka*- a very similar to Ocular surface

disease i.e, Dry Eye Syndrome in modern opthalmology. *Śuskāksipāka* described as a *Vātaja* disease in *Suśruta Saṁhitā*[4]. *Vātapitta* vitiated condition by *Vāgbhaṭa*[5] and *Śārañgadhara* considered this as *Vātaraktaja*[6]. By analyzing these different opinions it is clear that vitiation of *Vāta*, *Pitta,* and *Rakta* play the major role in the disease pathology which makes the eye dry by reducing tear secretion or by changing the quality of tears, (Tear film defect).

The *Śuskāksipāka* mentioned in *Suśruta Saṁhitā* is seem to be its early stage but description of *Ācārya Vāgbhaṭa* is that of well established advanced disease state with preponderance of *Pāka* - inflammation and it is mainly due to vitiated *Vāta* and *Pitta dosas*. The disease is characterised by features such as *Kuṇita vartma* (inability to close lids), *Dāruṇa* and *Ruksa vartma* (hard, rough lids), *Avila daṛeṣana* (blurring of vision) and *Dāruṇa Pratibodhana* (difficulty in opening the lids)[7]. *Ācārya Vaghbhaṭa* mentioned additional features such as *Gharsa* (foreign body sensation), *Toda* (pricking pain), *Bheda* (tearing pain), *Upadeha* (stickiness of lids), *Rūksatwa* and *Dāruṇatwa* of *Vartma* and *aksi* (hardness and roughness of the eyelids and eye), *Śīteksā* (desire for cold), *Śūla* (Pain) and *Pāka* (Inflammation)[8]. The signs and symptoms of *Śuskāksipāka* frame a picture of dry eye syndrome in modern science.

Dry Eye Syndrome

Etymology: Dry eye is a synonym of Kerato-conjuctivitis sicca. The term Kerato conjunctivitis sicca is made up of four words- 'Kerato', 'conjunctivus', 'itis' and 'sicca'.

- The word 'Kerato' is derived from the Greek word 'Keras' which means the cornea.
- The word conjunctivus is derived from the Latin word 'Conjungo', which means, to join, owing to the fact that this membrane joins eyeball to the lids.
- The word 'itis' is derived from the Greek word 'ites' used as an adjectival suffix denoting inflammation of the part indicated by the word stem to which it is attached.
- The word 'sicca' is derived from the Latin word 'Siccus' which means dry or dryness.[9,10]

Definition: According to AAO 2013 defined dry eye as:

"Dry eye is a common ocular condition that has a high impact on the quality of life of afflicted individuals owing to discomfort or visual disability. Although the symptoms improve with treatment, the condition is usually not curable. Dry eye can be a cause of visual disability and may compromise results of corneal, cataract and refractive surgery.

Epidemiology: The prevalence of dry eye has not been determined accurately due the lack of a single definition of the condition as well as variability of criteria included in several studies. However the large number of studies carried out in various countries estimate the prevalence of dry eye disease to be between 5-34 percent. The Baever Dam study demonstrated as incidence of dry eye of 13.3 percent that significantly correlated with patient age.[11] Dry eye is apparently higher in women (57.2%) than man (51%) in India. Being a heterogeneous group of conditions with multifactorial etiologies, prevalence vary with the many subcategories of the diseases. Prevalence of dry eye increased with age, sex, current smoking history, ophthalmic surgeries, contact lens wear and coexisting ocular conditions like meibomian gland dysfunction, terygium, blepharitis, and conjunctival diseases. Dry eye disease is more frequently found in patients with arthritis, thyroid dysfunction and poor general health.

Risk Factors for Dry Eye Syndrome

Dry eye is one of the most frequent ophthalmologic conditions which can be produced by hundreds of causes. Diseases causing dry eye are almost always chronic, progressive and produce mild or moderate manifestations but in severe cases it causes discomfort and severe low vision. Some risk factors which are responsible for dry eye Syndrome are as follows:

- **Physiological conditions**- Slowing of the blink rate increases the blink interval and increases the period of evaporative loss between each blink.[12]
- **Anatomical Conditions**- Height of the palpebral is wider in upgaze than down gaze.[13] Evaporative loss per eye increases with increasing palpebral width and is therefore, increased in upgaze.[14]
- **Sex Hormones**- Low levels of androgens and high levels of estrogens are risk factors for dry eye. Androgens promote lacrimal and meibomian glands function.[15] Female sex and Post-menopausal estrogen therapy are important risk factors for dry eye.[16]
- **Systemic drugs**- Some systemic medicines have a collateral exocrinic hyposecretory effect. Among them are antidepressants, anxiolytics, sleeping pills, antiparkinsonian, diuretics, vascular antihypertensive, anticholinergic, antihistaminic and antiarrhythmic; besides some preservatives used in ocular medications can cause the disease.
- **Aging**- It is associated with physiological changes that may predispose dry eye, including decrease in tear volume and flow, increased osmolarity,[17] decreased tear film stability and alteration in the composition of the meibomian lipids.[18]

- **Environmental factors**- Evaporative water loss from the eye is increased in conditions of low relative humidity, occurring either as part of natural variation at different geographic locations or in special circumstances created by air conditioning, air travel or other artificial environments.[19] Similarly, tear evaporation is increased by exposure to high wind velocity.
- **Occupational factors**- It may cause a slow blink rate, representing a risk for dry eye in those working with video display terminals.[20]

Pathogenesis

Dry eye is recognized as a disturbance of the Lacrimal Functional Unit (LFU), an integrated system comprising the lacrimal glands, ocular surface (cornea, conjunctiva and meibomian glands) and lids, and the sensory and motor nerves that connect them.[21] The core mechanisms of dry eye are driven by tear hyperosmolarity and tear film instability. The major causes of tear hyperosmolarity are reduced aqueous tear flow, resulting from lacrimal failure, and/or increased evaporation from the tear film. Increased evaporative loss is favored by environmental conditions of low humidity and high air flow and may be caused clinically, in particular, by meibomian gland dysfunction (MGD), which leads to an unstable tear film lipid layer. Reduced aqueous tear flow is due to impaired delivery of lacrimal fluid into conjunctival sac. Tear delivery may be obstructed by cicatricial conjunctival scarring or reduced by a loss of sensory reflex drive to the lacrimal gland from the ocular surface. Eventually, the chronic surface damage of dry eye leads to a fall in corneal sensitivity and a reduction of reflex tear secretion (Fig. 1 & 2).[22]

Classification based on etiopathogenesis

Aqueous Tear Deficient Dry Eye[23]

It occurs due to either decreased secretion of tears or inability of the secreted tears to reach the ocular surface. This is subdivided into two categories:

- Sjogren's syndrome tear deficiency (SSTD)
- Non-Sjogren's tear deficiency(NSTD)

The former (NSTD) has definite association with autoimmune systemic diseases, where as the latter (SSTD) has no relationship with it.

- **Sjogren's tear deficiency:** Decreased tear secretion occurs in Sjögren's syndrome which is a chronic inflammatory disorder characterized by lymphocytic infiltration of exocrine glands, especially the lacrimal and salivary glands. The condition may be primary or secondary.
- The primary Sjögren's syndrome is characterized by dry eyes with dry mouth, a positive focus score on minor salivary gland biopsy and positive serum autoantibodies like antinuclear antibody (ANA), rheumatoid factor or Sjögren's syndrome specific autoantibodies like anti-Ro (SS-A) and anti La (anti- SS-B).
- Secondary Sjögren's syndrome is characterized by the presence of systemic connective tissue disorder like rheumatoid arthritis, systemic lupus erythematosus and scleroderma along with the features of primary Sjögren's syndrome.
- **Non-Sjogren's tear deficiency:** This condition is not associated with any clinical manifestations or systemic features of autoimmune disease. The largest category of patients who have dry eye syndrome comprises those with acquired primary lacrimal gland deficiency and idiopathic kerato conjunctivitis sicca syndrome.

Evaporative dry eye: It may be intrinsic, where the regulation of evaporative loss from the tear film is directly affected and extrinsic evaporative dry eye embraces those etiologies that increase evaporation by their pathological effects on the ocular surface.

Intrinsic causes

- **Meibomian gland dysfunction:** It is the most common cause of evaporative dry eye. Its multiple causes and associations include dermatoses, such as acne rosacea, seborrheic dermatitis and atopic dermatitis.[24] MGD can be primary or secondary, simple or cicatricial.
- **Disorders of lid aperture and lid/globe congruity or dynamics:** Endocrine exophthalmos and specifically increased palpebral fissure width is associated with ocular drying and tear hyperosmolarity.[25] Increasing palpebral fissure width correlates with increased tear film evaporation.[26]
- **Low blink rate:** Drying of the ocular surface may be caused by a reduced blink rate, which lengthens the period during which the ocular surface is exposed to water loss before the next blink.[27]

Extrinsic Causes

- **Ocular surface disorders**: Disease of the exposed ocular surface may lead to imperfect surface wetting early tear film breakup, tear hyperosmolarity, and dry eye.
- **Vitamin A deficiency**: Vitamin A is essential for the development of goblet cells in mucous membrane and the expression of glycocalyx mucins. Vitamin A deficiency can cause lacrimal acinar damage and therefore some patients with xerophthalmia may have a lacrimal, aqueous tear deficient dry eye.[28]

- **Topical drugs and preservatives**: Many components of eye drop formulations can induce a toxic response from the ocular surface. Of these, the most common offenders are preservatives, such as benzalkonium chloride (BAC), which causes surface epithelial cell damage and punctate epithelial keratitis, which interferes with surface wet ability.[29]
- **Contact lens wear**: Poor lens wet ability, could be a basis for a higher evaporative loss during lens wear and was attributed to potential changes in tear film lipid composition, rather than to a loss of meibomian gland oil delivery.[30]
- **Allergic conjunctivitis**: The general mechanism leading to disease is that exposure to antigen leads to degranulation of Ig-E primed mast cells, with the release of inflammatory cytokines. Surface epithelial cell death occurs, affecting conjunctival and corneal epithelium (punctate kerato-conjuctivitis). Surface damage and the release of inflammatory mediator's leads to allergic symptoms and to reflex stimulation of the normal lacrimal gland.[31]

Classification based on causative mechanism

- **Tear hyperosmolarity:** Tear hyperosmolarity arises as a result of water evaporation from the exposed ocular surface, in situations of a low aqueous tear flow, or as a result of excessive evaporation and as a combination of these events.[32]
- **Tear film instability:** Tear film instability may be due to disturbance of ocular surface mucins (xerophthalmia) and due to allergic eye disease.[33]

Classification based on symptoms

Triple classification

- Symptoms when overexposed- Mild dry Eye syndrome
- Symptoms+reversible signs (eg. Epithelial erosions)- Moderate dry eye syndrome
- Symptoms+permanent signs (eg. Corneal ulcers)- Severe dry eye syndrome
- Symptoms+permanent signs and subnormal vision- Disabling (dry eye syndrome)

Symptoms

- Foreign body sensation
- Burning or itching
- Fluctuating vision
- Grittiness or irritation
- Sore or tired eyes
- Feeling of dryness
- Ocular discharge
- Photophobia
- Contact lens intolerance
- Watering or excessive tearing

These symptoms characteristically worsen during the day. A history of exacerbation by reading, computer use or in windy environment is often elicited.[34]

Signs:

- External observation of the face for acne rosacea.
- **Lids and lashes-** Examined for lagophthalmos, infrequent blinking, floppy eyelids, lid retraction, ectropian, entropion, notching, trichiasis and distichiasis.
- **Lid margins:** Observations of meibomian gland architecture and openings, presence of blepharitis, telengectasia and position and size of lacrimal puncta. Enlargement of lacrimal gland and function of 5th & 7th cranial nerves has to be checked.
- **Conjunctiva:** It may show mild redness and keratinization.
- **Tear film:** Marginal tear meniscus, presence of foam or debris suggestive of meibomian gland dysfunction. In the dry eye syndrome, lipid contaminated mucin accumulates in the tear film as particles and debris that move with each blink.
- **Cornea:** Punctate epithelial erosions in the interpalpebral and inferior cornea.
- **Mucus filaments:** stain with Rose Bengal.
- **Mucus Plaques:** Composed of mucus epithelial cells.

Classification based on severity

For a classification of dry eye on the basis of severity, the Delphi Panel Report[35] was adopted and modified as a third component of the DEWS (Table 1).

Table 1 Classification based on severity

Dry eye severity level	1	2	3	4
Discomfort, Severity and frequency	Mild and/or episodic; occurs 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	Annoying and/or	Annoying, chronic	Constant and/or

	mild fatigue	activity limiting episode	and/or constant, limiting activity	possibly disabling
Conjunctival injection	None to mild	None to mild	Mild to moderate	Marked
Conjuctival staining	None to mild	Variable	Moderate to marked	Marked
Corneal staining	None to mild	Variable	Marked central	Severe punctate erosions
Corneal/ tear signs mucus	None to mild	Mild debris, decreased meniscus	Filamentary keratitis, mucus clumping, increased tear debris	Filamentary keratitis, mucus clumping, increased tear debris, ulceration
Lid/Meibomian glands	MGD variably present	MGD variably present	Frequently present	Trichiasis, keratinization, symblepharon
TFBUT (sec)	Variable	≤10	≤5	Immediate
Schirmer's Score (mm/5min)	Variable	≤10	≤5	≤2

Diagnosis

Objective tests: Quantitative tests

Schirmer's test: It is an invasive test in which a strip of filter paper is placed in lower conjunctival cul-de-sac and measurement of wetting length is done over a certain period of time. There are two commonly used variants of Schirmer's test.[36]

Schirmer's' I: Measures the total tear production including both basal and reflex tears. Jones modifications of this test can measures only basal secretion with the aid of an anesthetic agent.

Schirmer's II: Schirmer's II test is performed by irritating the nasal mucosa with cotton – tipped applicator prior to measuring tear production, which is mainly used for measuring the reflex tear secretion of main lacrimal gland.

Schirmer's III: This test is obsolete now as in this measurement was done with the subject staring at sun. A value of less than 5mm wetting in 5 minutes is considered abnormal for both tests. A 1- minute (with anesthesia) and 2-minutes Schirmer's test has been suggested, with cut-offs of 6mm and 10mm (99% confidence interval), respectively. It has been found to lack accuracy and reproducibility.

Qualitative Tests

Tear Film break-up time: The tear break-up time (TFBUT) is defined as the time interval between a complete blink and the first appearance of a dry spot in the tear film after preservative free fluorescein administration. A TFBUT of less than 10 seconds suggests tear film instability, and less than 5 seconds suggests definite dry eye.

Factors, which reduce the reliability/reproducibility of this test include:

- Volume of fluorescein administered
- Preservatives, such as benzalkonium chloride shorten the TFBUT
- Superficial punctate keratopathy

There are non-fluorescein (noninvasive) measurements of TFBUT that employ reflective devices with a grid projected onto the corneal surface. These values are slightly higher that the invasive technique.[37]

Treatment [38]

The underlying causative processes of dry eye generally not reversible and management is therefore structured around the control of the symptoms and the prevention of surface damage. DEWS have produced guidelines based on earlier International Taskforce guideline for Dry eye, in which suggested treatment options depend on the level of severity of disease graded from 1 to 4. The DEWS guidelines can also be applied in a graded approach, proceeding to the next level if the preceding measures are inadequate.

Level 1

A) Education and environmental/dietary modifications

- Establishment of realistic expectations and emphasis on the importance of compliance.
- Lifestyle review including the importance of blinking whilst reading, watching television or using a computer screen and the management of contact lens wear.
- Environmental review, e.g. increasing humidity may be possible for some environments.
- Instillation aids for eye drops should be advocated for patients with reduced dexterity.

- Caution the patient that laser refractive surgery can exacerbate dry eye.

B) **Systemic medication review** to exclude contributory effects and eliminate offending agents, discontinuation of toxic/ preserved of topical medication if possible.

C) **Artificial tears substitutes** including gel and ointments**-** Use of preserved drops and gel.

D) **Eye lid therapy**- Basic measures such as warm compresses and lid hygiene for blepharitis; reparative lid surgery (eg. Entropion, ectropion, excessive lid laxity or scleral show) may be considered as an early measure. Nocturnal lagophthlmos can be addressed by taping the lid closed at bedtime, wearing swimming goggles during sleep or in extreme cases by lateral tarsorraphy.

Level 2

A. Non- preserved tear substitutes are categorized as level 2 treatments.
B. Anti inflammatory agents such as topical steroids, oral omega fatty acids and other agents such as topical cyclosporin.
C. Tetracyclins (for meibomitis, rosacea)
D. Punctal plugs
E. Secretagouges e.g. Pilocarpine, cevilamine, rebamipide
F. Moisture chamber spectacles and spectacle side shields

Level 3

A. Serum eye drops Autologous or umblical cord serum
B. Contact lenses
C. Permanent punctal occlusion

Level 4

A. Systemic anti-inflammatory agents.
B. Surgery
   - Eye lid surgery, such as tarsorrhaphy
   - Salivary gland auto transplantation
   - Mucous membrane or amniotic membrane transplantation for corneal complications.

Tear Substitutes: Tear substitutes have a relatively simple formulation that cannot approximate the complex components and structure of the normal tear film. Their delivery is also periodic rather than continuous. In theory, the ideal artificial lubricant should be preservative free, contain potassium, bicarbonate and other electrolytes and have a polymeric system to increase its retention time [39].

Drops and gels

- Cellulose derivatives (e.g. hypromellose, methylcellulose) are appropriate for mild cases.
- Carbomer gels adhere to the ocular surface and so are longer- lasting, but some patients are troubled by slight blurring.
- Other agents include polyvinyl alcohol (PVA), which increases the persistence of the tear film and is useful in mucin deficiency, sodium hyaluronate, povidone, glycerine, propylene glycol, polysorbate and others.
- Diquafosol is a newer agent that works as a topical secretagogue.

Ointments containing petroleum mineral oil can be used at bedtime to supplement daytime drops or gel instillation; marked blurring precludes day time use. Some practitioners do not prescribe these for long-term use.

Eyelid Spray: It is applied to the closed eye and typically contains a liposome-based agent that may stabilize the tear film and reduce evaporation.

Artificial Tear: Inserts emplaced once or twice daily offer extended duration treatment and are preferred by some patients.

Mucolytic agents: Acetylcysteine 5% drop may be useful in patients with corneal filaments and mucous plaques which acetylcysteine dissolve. It may cause stinging or installation. Acetylcysteine is malodorous and has a limited shelf life. Manual debridement of filaments may also be useful.

Non-preserved drops: Preservatives can be a potent source of toxicity, especially after punctal occlusion. Numerous non preserved drops are now available, including some multi-dose products and in general should be used in preference to preservative containing preparations in any more than mild diseases or with installation more than three or four times daily.

Punctal occlusion: Punctal occlusion reduces drainage and thereby preserves natural tears and prolongs the effect of artificial tears. It is of greatest value in patients with moderate to severe KCS who have not responded to frequent installation of topical agents.

Temporary Occlusion can be achieved by inserting collagen plug in to canaliculi; these dissolve over a number of weeks.

Reversible prolonged occlusion can be achieved with silicone or long acting (2-6 months) collagen plugs.

Permanent occlusion should be undertaken only in patients with severe dry eye who have had a

positive response to temporary plugs without epiphora.

Anti- inflammatory agents

Topical steroids generally low intensities preparations such as fluorometholone, are effective supplementary treatment for acute exacerbations.

Omega fatty acids supplements (e.g. Omega-3 fish oil, flex seed oil) can have a dramatic effect on symptoms and may facilitate the reduction of topical medication.

Oral tetracyclines for an extended course, often 3 months at a relatively low dose, may control associate blepharitis, especially meibomitis and reduced tear levels of inflammatory mediators.

Topical cyclosporin (usually 0.05%) reduces T-cell mediated inflammation of lacrimal tissue, resulting in an increase in the number of Goblet cells and reversible of squamous metaplasia of the conjunctiva.

Contact lenses

Although contact lens wear can exacerbate dry eye, particularly due to inflammatory, sensory and evaporative effects, these can be out weighted by the reservoir effect of fluid trapped behind the lens, and they are effective at relieving symptoms from secondary corneal changes. Patients should be cautioned regarding the possibility of bacterial keratitis.

- Low water content hydroxyl ethyl metha acrylate (HEMA) lenses may be successfully fitted to moderately dry eye.
- Silicon rubber lenses that contain no water and readily transmit oxygen are effective in protecting the cornea in extreme tear film deficiency, although deposition of debris on the surface of the lens can blur vision and be problematic. The continued availability of these lenses is in doubt.
- Occlusive gas permeable scleral contact lenses provide a reservoir of saline over the cornea. They can be worn on an extremely dry with exposure.

Optimization of environmental humidity

- **Reduction of room temperature** to minimize evaporation of tears.
- **Room humidifiers** may be tried but are frequently disappointing because much apparatus is incapable of significantly increasing the relative humidity of an average sized room.

Miscellaneous options

- Botulin toxin injection
- Oral cholinergic agonists
- Submandibular gland transplantation
- Serum eye drops

Fig. 1: Etiopathogenesis of Dry Eye Diseases

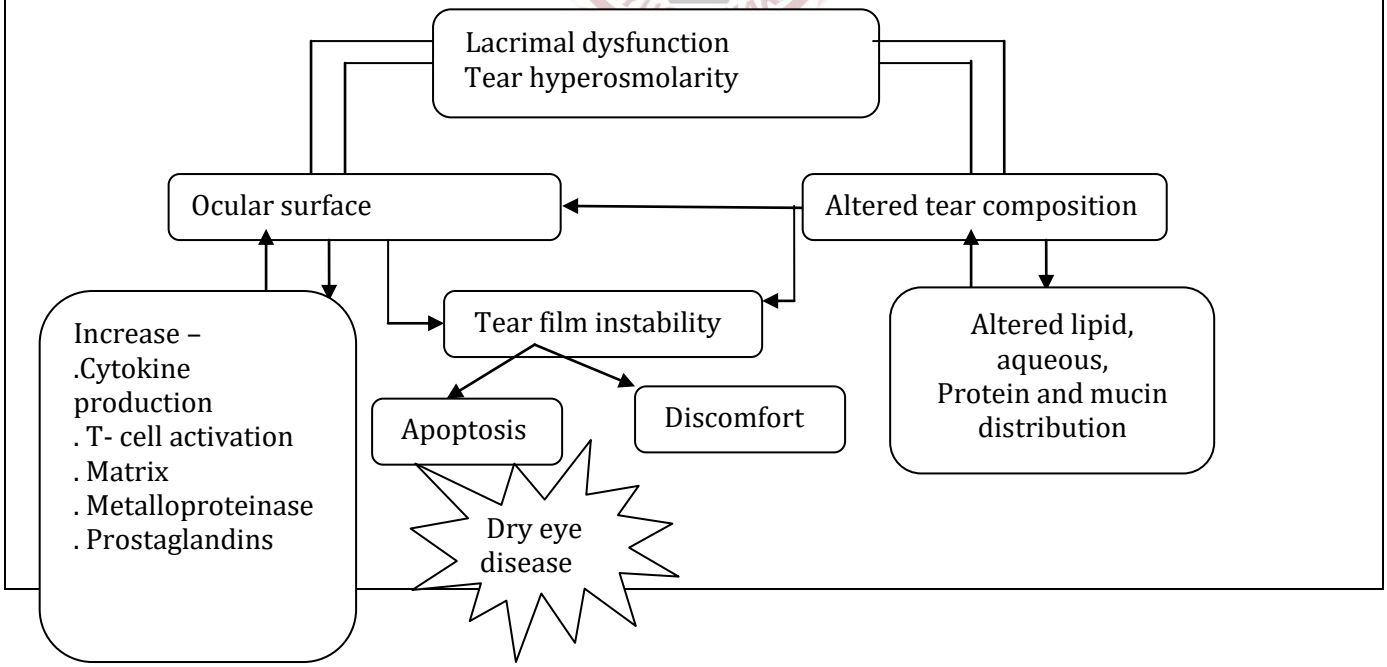
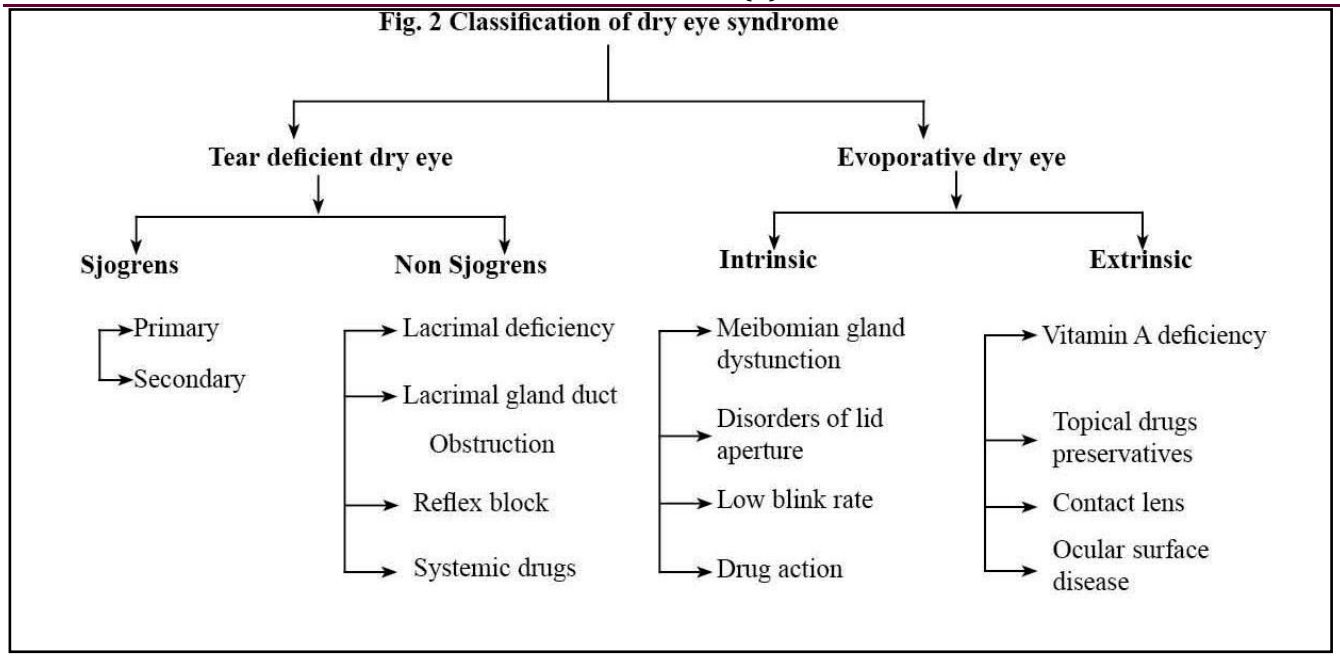


Fig. 2 Classification of dry eye syndrome



DISCUSSION

In *Śuskāksipāka* i.e. Dry Eye Syndrome there is dryness in eyes due to increase in *Vāta-Paitta-dosa prakopa*, vitiated *Vāta* and *Pitta* reach upto *Netra* (*Prasara-Sthānasaṁśraya*) by the *Śira*. At *Netra* they vitiated *Rakta dhātu* to form *Śuskāksipāka*, which results in the scanty *Aśru*, so as to form dry eye.

In Ayurvedic literature various treatment modalities are applicable in treating *Śuskāksipāka* including *Snehpana, Tarpaṇa, Putpāka, Nasya, Añjana* etc. Thus it may reduce the dryness of the eyes, irritation and inflammation occurring in the eyes. In the present scenario we understanding of the disease *Śuskāksipāka* (Dry Eye Syndrome) and planning the treatment protocol accordingly; has proved much effective than the prevailing management modalities.

CONCLUSION

The eye is the main sense organ gifted by God to human beings. An individual who is blind, day and night are the same and this beautiful world is of no use to him even if he possesses a lot of wealth, so sincere efforts should be made by every individual to preserve his/her vision till the last breath of life. In spite of remarkable progress and advances in the field of modern ophthalmology, there are some limitations. *Āyurveda*, the ancient system of medicine gives valuable guide lines not only in treatment aspects but also in preventive line. *Netra Kriyā kalpa* like *Snehpana, Tarpaṇa, Puṭpāka, Nasya, Añjana* is one of the local therapeutic procedure which if promptly used shows objective evidences of excellent responses. The systemic approach to treat the disease *Śuskāksipāka*, (*Sarvagata Vāta-Pitta/Raktaja Netra Roga*) and managing this humeral imbalance, along with local/ topical therapeutically procedures, the condition could be managed well. According to *Āyurveda*, dry eye is not merely an ocular surface disorder, rather this is one of manifestation of the deranged metabolism of body tissues. *Aśru* (tear film) is the by product of *Rasa*, *Meda*, and *Majjā dhātu* and without normalizing/altering them we cannot treat dry eye syndrome optimally.

Thus, as we can conclude that the dry eye syndrome is a condition for which modern medicine has no treatment except for the symptomatic management; the treatment and approach of Ayurvedic system of medicine according to the *Dosas* provided both subjective and objective relief to the patient.

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