

Dry Eye Disease - A Holistic Review

Ish Sharma^{1*}

Aparna Sharma²

¹Ayurveda Chair (Ministry of Ayush Government of India) Faculty of Medicine and Health Sciences, University of Mauritius, Mauritius

²Department of Shalaky Tantra, National Institute of Ayurveda, India

Received Date: November 12, 2023; Accepted Date: November 29, 2023; Published Date: December 11, 2023

***Corresponding author:** Prof. Ish Sharma, Ayurveda Chair (Ministry of Ayush Government of India) Faculty of Medicine and Health Sciences, University of Mauritius, Mauritius.

Citation: Ish Sharma and Aparna Sharma. Dry Eye Disease - A Holistic Review. W J Heal Med. 2023;1(3):1011.

Copyright © 2023 Ish Sharma. This is an open access article published under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract

Dry Eye Disease (DED) has emerged as a largely unanswered question, leading to compromised Quality of Life due to impaired ocular vision. Ayurvedic literature mentions it as Shushka Akshi Paka, resulting for vitiated Vata Dosha, with inputs from Pitta however minimal or incidental involvement of Kapha Dosha. DED is a complex multifactorial disease which shows varied symptoms, as eye dryness, photophobia, ocular discomfort, irritation and burning sensation, ultimately the risk to vision. The evaporative water loss causes impairment and loss of tear homeostasis, leading to ocular inflammation. The present review from interpretation of academic and clinical research literature provides various aspects of information on the definition, prevalence, causes, diagnostic tests, and medical management of dry eye disease from the western, and Ayurvedic viewpoints. The aim of this review is to provide awareness among the patients, high-risk groups, health care professionals, and researchers about an overview on Dry Eye Disease; Nasya instillation being the most significant of suggestions.

Introduction

Dry Eye Disease (DED) has emerged as a largely unanswered question, leading to compromised Quality of Life due to impaired ocular vision. Ayurvedic literature mentions it as Shushka Akshi Paka, resulting in vitiating Vata Dosha, with inputs from Pitta, however minimal or incidental involvement of Kapha Dosha. Dry eye disease is a multifactorial disease of the ocular surface, associated with increased osmolarity of tear film, and inflammation of the ocular surface. The core factors responsible for dry eye are tear instability; tear hyperosmolarity, inflammation and ocular surface damage. Dry eye is classified into aqueous deficient and evaporative types. In Aqueous deficient dry eyes, there is reduced lacrimal tear secretion with normal rate of tear evaporation resulting in hyperosmolarity; can be associated with Vata predominant Shushka Akshi Paka. In evaporative dry eyes, there is increased evaporation of tear film with normal lacrimal function; can be associated with Pitta predominant Shushka Akshi Paka [1]. Lowered quantity, and quality of Kapha Dosha is usually present in both the types. Evaporative type is more prevalent, and many patients have both forms.

Diabetes and Dry Eye Disease

There is no confirmatory evidence, approximately 70% of Diabetes Mellitus type 2 experience Dry eye at some point in life [2]. Diabetes Mellitus is also associated with various other ocular diseases such as acute orbital infection, hordeolum, refractive error, chronic inflammation of the lid, neovascular glaucoma, ptosis, diabetic retinopathy, oculomotor nerve palsies and cataract. Prolonged deficiency of tear secretion and dysfunction of tear film are present in diabetic patients [3]. Tear film is jointly produced by meibomian glands, lacrimal gland, and goblet cells of conjunctiva. Nutrition, a sepsis, and lubrication of eye are ensured by tear film. Studies have proven that deficient production and secretion of tears is due to “autonomic neuropathy,” and nerves that supply the lacrimal glands are hampered which causes dry eye in diabetic patients [4]. The situation worsens with rising glycohemoglobin levels [5]. Hyperglycemia can result in insufficient tear production or excessive tear loss, decreased corneal sensitivity, abnormality in blinking and changes in tear film composition (hyperosmolarity). The tear film instability leads to inflammation and damage to the eye. Tear osmolarity increases with severity of diabetic peripheral neuropathy. Hyperosmolarity is due to decrease in aqueous secretion due to microvascular damage to lacrimal glands. Dry Eye Disease in Diabetics links to a shorter tear film span [6].

Diabetes and Meibomian Gland Pathology in DED

Several studies demonstrated that the Diabetics might predispose to Meibomian Gland Dysfunction (MGD) [7], featuring hyposecretion/ hypersecretion or obstruction of Meibomian Gland, the quality/quantity alterations of meibum, subsequently causing wear down of acini [6]. Tear film stability is compromised due to Meibomian gland malfunctioning, and ultimate evaporative dry eye, which compromises the life quality.

Meibomian glands are sebaceous glands situated on the tarsal plates of upper and lower eyelids. A healthy tear film relies on these important glands, which secrete nourishing oils that coat the corneal surface. These oils prevent tears

from evaporating too quickly. The elevated hemoglobin A1C levels can cause meibomian gland deterioration. If MGD isn't treated, glands may atrophy and stop functioning permanently, resulting in severe dry eye symptoms. In addition to diabetes Mellitus, the risk factors for MGD include aging, hormonal changes, some medications, and medical conditions. Ocular surface disease is confluence of multiple factors as the quantity and quality of the tear film, corneal sensitivity, and corneal epithelial health. It causes ocular discomfort, keratopathy, punctate epithelial erosions and non-healing epithelial defects and ulcers. Patients with proliferative diabetic retinopathy and clinically significant macular oedema are predisposed to impaired tear film functions. Diabetes, with passage of time, and / or poor glycemic control causes reduction in the number of goblet cells, and reduction in mucin production thereby leading to tear film instability. Duration of more than ten years has strong association with dry eyes. Patients having poor glycaemia control (HbA1C >8) were having higher degree of dry eye. Superficial punctate keratopathy, trophic ulcers, recurrent corneal erosions, and persistent epithelial defects are frequently associated with DES. The lack of moisture irritates the eye, sending distress signal through nervous system for more lubrication which in turn sends flood of tears to compensate for dryness. The early adequate control is key measure for prevention of dry eye. Pathogenesis is not clear though accumulation of sorbitol may cause damage to the structure of lacrimal gland and dysfunction eventually resulting in decreased tear secretion. Postmenopausal women are at risk due to a decrease in hormonal levels which can lead to loss of anti-inflammatory protection and decreased lacrimal secretion. DES usually affects people older than 50 years of age [8]. Use of contact lens, systemic drug effects, autoimmune diseases, and refractive surgeries are predisposing factors [9]. Dry eyes are prone for bacterial Keratitis and there is an increased risk of complication after laser refractive surgery. Postmenopausal women using hormone replacement therapy especially estrogen is at risk of developing dry eyes. Dry eyes are worsened by low blink rate, wide lid aperture, low androgen levels, high estrogen

level and systemic drug effects [10]. Low relative humidity, air travel, air conditioning, high wind velocity and use of computer screen increases the risks for dry eye [11]. Preservative benzalkonium chloride used in various topical preparations can be toxic in moderate or severe dry eye. The medications that can cause dry eye are tranquilizers, antihistamines, beta blockers, diuretics, and oral contraceptives. Environmental factors like pollution, smoke, indoor heating, deficiency of vitamin A, radiotherapy, refractive eye surgeries, and auto immune diseases like Rheumatoid Arthritis contribute to Dry Eye Disease [12]. Symptoms are irritation, foreign body sensation in eyes, feeling of dryness, itching, and ocular discomfort. The important signs are rapid tear film break time, stinging mucus, conjunctiva is lusterless and mildly congested, conjunctival surface irregularities, MGD, marginal tear meniscus is reduced or absent. Complications of dry eye can cause vision loss, ulcers in cornea and perforation of the cornea.

Pathophysiology of Dry Eye Disease

The principal components in the pathogenesis of DES are tear hyperosmolarity and instability. In the aqueous deficient DED variety, reduced tear production due to damage of lacrimal gland leads to hyperosmolarity. In the evaporative type, deficiency of lipids due to MGD results in evaporation of tear film that leads to the hyperosmolarity of the tear film. Tear hyperosmolarity triggers vicious cycle of inflammation, loss of goblet cells, and damage of the ocular surface. Tear instability and hyperosmolarity activates stress signaling pathways and resident immune cells in ocular surface epithelium and triggers production of the innate inflammatory molecules which initiates vicious cycle resulting in the worsening of the symptoms [13]. Dry environment aging, some medications contribute to the inflammation. The conjunctival epithelium has the second highest density of mucus producing goblet cells next to intestine. The conjunctival goblet cells maintain immunity and homeostasis by producing soluble mucin that helps in the stabilization of the precorneal tear film [14].

Examination of Eye

Examination of lids Eyelid diseases like ectropion, entropion, and facial nerve palsy etc. can upset the integrity of tear film.

Diagnosis of Dry eye includes history-taking, ocular examination, tear film breaks up time, staining, Schirmer test and meibomian gland morphology [15].

The patients with dry eye and xerostomia should be investigated for Sjogren's syndrome, and the status of Diabetes Mellitus.

Treatment

Western medicine Treatment include topical steroids, omega 3 fatty acid, fish oil, flax seed oil, reduction of the room temperature, therapeutic contact lenses, artificial tears, lifestyle changes and treatment of underlying cause. Topical steroids reduce osmolarity. The use of humidifiers, moisture chambers or goggles increases periocular humidity. In severe dry eyes tarsorrhaphy may be necessary.

- **Hydration:** Hydration can maintain moisture in the body.
- **Blinking exercises:** Blinking of the eyes can help in evenly spreading of tears. Blinking helps in the secretion of meibomian glands. The reduced interval between blinks from 6 to 2.6 seconds and incomplete blinking are the typical of dry eyes.
- **Hygiene of eye lid:** Regular cleaning of the eyelids and lashes improve tear quality.
- **Diet:** Diet should be balanced with omega-3 fatty acids, vitamins A, C, and E, and antioxidants. The Omega 3 and 6 are essential fatty acids. They are absorbed in food. They maintain the homeostasis of tear film and reduce cytokines [16].
- **Artificial tears** are the mainstay and the first line of treatment in dry eyes. They provide lubrication and cures dryness and irritation. In mild cases of dry eyes, they are used 4 times a day while in the cases of severe dry eyes they are used 10-12 times per day. They increase the stability of the tear film, reduce ocular surface stress, improve contrast sensitivity and optical quality of ocular surface, and increase the quality of life

in patient. Artificial tear inserts containing hydroxyl propyl cellulose can be used every morning. Artificial tears have varied compositions ranging from polyvinyl alcohol, povidone, cellulose derivatives, and Hyaluronic acid. For MGD aqueous tears contain triglycerides, phospholipids, and castor oil [13].

- **Punctal Plugs:** It is tiny medical device inserted into puncta of eye to block duct and prevent drainage of tears in nasolacrimal duct. It is used in moderate to severe cases of dry eyes.
- **Corticosteroids:** They are used to treat inflammation in the eye. They are recommended for short term use as long term use can cause adverse effects such as cataract and glaucoma.
- **Cyclosporine A:** It is immunomodulatory that prevents the activation of T lymphocytes and decreases the level of inflammatory cytokines in conjunctival epithelium and increases the goblet cell. Cyclosporine A increases tear fluid via the release of parasympathetic neurotransmitters [14].
- **Vitamin A:** It is present naturally in tear film of healthy eyes and is important in the formation of the mucin layer; lubricating layer of tears film. Vitamin A drops protect the eye from free radicals, allergies, inflammations, and toxins. Systemic vitamin A can be used along with topical retinoic acid therapy [17].
- **Eye drops made from your own blood:** These are called autologous blood serum drops. They may be used in severe dry eye symptoms in concentration of 20% to 100 %. The drops contain epitheliotropic growth factors and are anti-inflammatory.
- **Using special contact lenses:** Patients having severe dry eyes can use scleral lenses or bandage lenses.

Tyrvaya is the first nasal spray to treat DED and is approved by the FDA. It is applied B.D on inside of the lower nasal area.

Table 1: Scoring Pattern (Subjective Criteria).

Symptom	Grade 0	Grade 1	Grade 2	Grade 4
<i>Rukshata</i> (Dryness)	No dryness	Occasional	Persistent, does not disturb routine work	Disturb routine work
<i>Gharshan</i> (Gritty sensation)	Nil	Occasional	Persistent, does not disturb routine work	Disturb routine work
<i>Avildarshanam</i> (Blurring of vision)	Nil	Occasional	Persistent, does not disturb routine	Disturb routine work
<i>Kricchronmilana</i> (Difficulty in opening and closing of eye)	Nil	Occasional	Persistent, does not disturb routine	Disturb routine work
<i>Daha</i> (Burning sensation)	Nil	Mild	Persistent, does not disturb routine	Disturb routine work
Intolerance to light	Nil	Occasional	Persistent, does not disturb routine	Disturb routine work

Test	Normal (0)	Mild (1)	Moderate (2)	Severe (3)
Tear Film Break up Time (TBUT)	>10 sec	>8 sec and < 10 sec	>5 sec and < 8sec	< 5 sec
Schirmer 1 st Test	>15 mm	> 8 mm and < 15 mm	> 4 mm and < 8 mm	< 4 mm
Fluorescein Staining	No staining of corneal epithelial surface	Occupies <1/3 of corneal epithelial surface	Occupies >1/3 and <1/2 of corneal epithelial surface	Occupies >1/2 of corneal epithelial surface

In Ayurveda, Dry Eye Disease can be correlated with Shushkakshipaka. It is a Sarvagata vyadhi / disease of ocular surface. The disease is dominated by Vata Dosha as per Sushruta, while it is Vata-Pittaja according to Acharya Vagbhat, and Vataraktaja according to Acharya Sharangdhar. The symptoms of Shushkakshipaka according

to Sushrata are Koonam / narrowing of palpebral aperture, Darunaruksnavartma / hardening of lids, Avildarshanam / Blurring of vision, and Sudarunamyatapatibodhanam / Difficulty in opening and closing of eyelids. Acharya Vagbhat has added more symptoms to above symptoms like Toda / pricking pain, Bheda / tearing pain, Updehavata / mucoid discharge, Vishushkta / dryness, Gharsh /gritty sensation, Sheetecca / liking for cold, Shula / crucifying pain, and Paka / inflammation [18]. Acharya Vagbhat mentioned that disease could acquire chronicity and then termed as Pilla. Panchakarma, and para-Panchakarma procedures described as effective by ancient Ayurveda masters are Tarpana, Putpaka, Seka, Ashchyotana / various types of eye instillations, Anjana / therapeutic collyriums, and Nasya / nasal instillations.

Discussion

The Ayurvedic perspective of the Dry Eye Disease / DED beholds a great scope as the disease does not have effective treatments, and / or cure. Shushkakshipaka mentioned by the Ayurvedic classics can be likened to DED. This chronic condition is mainly caused by Vata Pitta Dosha, with low Agni / deranged metabolism in eyes. Agni / Metabolism enhancing, thus Ama preventing approaches are found effective. Some scholars associate the DED with Vata, and Kapha Dosha due to the western ophthalmology associating the DED with Autoimmunity, which as per Ayurveda is a kind of AmaVata. Both the Vata, and Kapha Dosha being cold in attribute, Agni promotion with Ushna Veerya / hot potency herbs as spices, Ashwagandha / Withania somnifera, Guggulu / Commiphora mukul, Erand / Ricinus communis and so on can be applied as preventive herbs for oral consumption in the high-risk group individuals. Among the procedures, Tarpana, Putpaka, Seka, Ashchyotana / various types of eye instillations, Anjana / therapeutic collyriums, and Nasya / nasal instillations are the most used therapies. Nasya beholds a significant position, as this is one among the five core Panchakarma procedures: the others being more local, and outwardly therapies. As the Vata Kapha vitiation emanates from the gastro-intestinal tract, and compromised tissue Agni / Metabolism, Nasya is

perceived, as well as observed to be the most effective against Dry Eye Disease / Shushkakshipaka. Most patients and the high-risk groups are advised to use Nasya with mustard oil as a daily routine. Three to five drops of mustard oil each nostril, once, or twice a day is a standard protocol. Other Nasya Sneha / fats as cow ghee, various skin friendly oils, Anu Oil, and Shadbindu oil, and so on can also be used. The dose can vary as per various specific factors.

Conclusion

Dry eye can cause corneal ulcer, infections, and blindness. Conventional Tests as Tear film break up time, Schirmer test and ocular staining used for the diagnosis of dry eye have low degree of standardization and some are invasive which can make interpretation challenging. Moreover, there is no permanent cure for dry eyes. Ocular medications can provide short-lived relief. According to Ayurveda it is Vata - Pitta disease and anti-Ama approach, Nasya therapy, and intake of Rasayana / rejuvenators as Ashwagandha can be of immense help. Lifestyle should be improved. The most important message would be to the high-risk groups to initiate self-administered Nasya application as a daily routine well in time, which can be significantly preventive.

References

1. Kaviraj Ambikadatta Shastri. *Susrutha samhita of Maharshi Susruta: edited with Ayurveda Tattva Sandipika hindi commentary, scientific analysis notes. Reprint-edition 2015. Varanasi: Chaukambha Orientalia; part 2 (uttara tantra), p.39.*
2. Seifart U, Stempel I. The dry eye and diabetes mellitus. *Ophthalmologie.* 1994;91(2):235-9.
3. Manaviat MR, Rashidi M, Afkhami-Ardekani M, Shoja MR. Prevalence of dry eye syndrome and diabetic retinopathy in type 2 diabetic patients. *BMC Ophthalmol.* 2008;8:10.
4. Ohashi Y, Ishida R, Kojima T, Goto E, Matsumoto Y, Watanabe K, et al. Abnormal protein profiles in tears with dry eye syndrome. *Am J Ophthalmol.* 2003;136(2):291-9.
5. Schaumberg DA, Dana R, Buring JE, Sullivan DA. Prevalence of dry eye disease among US men: estimates

- from the physicians' health studies. *Arch Ophthalmol.* 2009;127(6):763-8.
6. Moss SE, Klein R, Klein BE. Prevalance of and risk factors for dry eye syndrome. *Arch Ophthalmol.* 2000;118(9):1264-8.
7. Chhadva P, Goldhardt R, Galor A. Meibomian gland disease: the role of gland dysfunction in dry eye disease. *Ophthalmology.* 2017;124(11 Suppl):S20-6.
8. Lin X, Xu B, Zheng Y, Coursey TG, Zhao Y, Li J, et al. Meibomian Gland Dysfunction in Type 2 Diabetic Patients. *J Ophthalmol.* 2017;2017:3047867.
9. Krenzer KL, Dana MR, Ulman MD, Cermak JM, Tolls DB, Evans JE, et al. Effect of androgen deficiency on human meibomian gland and ocular surface. *J Clin Endocrinol Metab.* 2000;85(12):4874-82.
10. Stern ME, Schaumburg CS, Pflugfelder SC. Dry eye as a mucosal autoimmune disease. *Int Rev Immunol.* 2013;32(1):19-41.
11. Pflugfelder SC, Stern ME. Mucosal environmental sensors in pathogenesis of dry eye. *Expert Rev Clin Immunol.* 2014;10(9):1137-40.
12. Tseng SC, Hirst LW, Maurmence AE, Kenyon KR, Sun TT, Green WR. Possible mechanisms for the loss of goblet cells in mucin deficient disorders. *Ophthalmology.* 1984;91(6):545-52.
13. Nichols KK, Foulks GN, Bron AJ, Glasgow BJ, Dogru M, Tsubota K, et al. The international workshop on meibomian gland dysfunction; Executive Summary. *Invest Ophthalmol Vis Sci.* 2011;52(4):1922-9.
14. Barabino S, Rolando M, Camicione P, Ravera G, Zanardi S, Giuffrida S, et al. Systemic linoleic acid and gamma -linoleic acid therapy in DES with an inflammatory component. *Cornea.* 2003;22(2):97-101.
15. Goto E, Shimezaki J, Monden Y, Takano Y, Yagi Y, Shimmura S, et al. Low concentration homogenized castor oil eye drops for noninflammatory obstructive meibomian gland dysfunction. *Ophthalmology.* 2002;109(11):2030-5.
16. Yoshida A, Fujihara T, Nakata K. Cyclosporine A increases tear fluid secretion via release of sensory neurotransmitters and muscaranic pathways in mice. *Exp Eye Res.* 1999;68(5):541-6.
17. Sommer A, Emran N. Topical retinoic acid in the treatment of corneal xerophthalmia. *Am J Ophthalmol.* 1978;86(5):615-7.
18. KR Srikanthamurthy, Vagbhat's Ashtanga Hridayam, Vol.III, Chaukamba Krishnadas Academy, Varanasi, Reprint-sixth edition. 2015. p.140.

Author Contact Information:**Prof Ish Sharma,**

Ayurveda Chair (Ministry of Ayush Government of India),
Faculty of Medicine and Health Sciences,
University of Mauritius, Mauritius.

E-mail: drishsharma[at]gmail[dot]com