



NARRATIVE REVIEW

Krichronmeela and Shushkakshipaka: Ayurvedic Insights into Dry Eye Disease - A Narrative Review

Muhammed Nissam C P¹, Sreeja Sukesan²

¹Assistant Professor, Department of Salakyatanthra, Government Ayurveda College, Kannur

²Professor & HOD, Department of Salakyatanthra, Government Ayurveda College, Tripunithura

*Email: dr.nissam@gmail.com

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Abstract

Dry Eye Disease (DED) is a multifactorial condition of the ocular surface recognized in modern ophthalmology over the past three decades. Interestingly, classical Ayurvedic texts describe conditions-*Krichronmeela* and *Shushkakshipaka*-that exhibit striking similarities to DED. While most contemporary scholars correlate DED primarily with *Shushkakshipaka*, *Krichronmeela* has been considered by only a few authors, and detailed analyses are limited. This narrative review specifically approaches *Krichronmeela*, examining its classical descriptions in *Aṣṭāṅga Hṛdaya* and *Aṣṭāṅga Saṃgraha*, and compares them with *Shushkakshipaka* described in *Suśruta Saṃhita* and *Ashtanga Hrudaya*. By critically analyzing the symptomatology, pathophysiology, and recommended Ayurvedic management, this article highlights the correlation between *Krichronmeela* and the early stages of modern DED, particularly evaporative dry eye associated with Meibomian Gland Dysfunction. Early recognition and intervention at the stage of *Krichronmeela* may prevent progression to *Shushkakshipaka* and improve therapeutic outcomes. The review also identifies potential integrative approaches, bridging classical Ayurvedic wisdom with contemporary ophthalmology.

Introduction

In modern Ophthalmology, the formal history of Dry Eye Disease (DED) began only about three decades ago. In the early to mid-1990s, scientific explorations on the disease peaked, leading to its formal recognition as a disease. As a result, the first official definition was published in 1995 by the NEI/Industry working group¹. With a reported prevalence ranging from 5% to 50%, Dry Eye Disease (DED) significantly impairs quality of life and imposes a substantial economic burden on healthcare systems and society¹.

Interestingly, classical Ayurvedic texts describe two conditions - *Krichronmeela* and *Shushkakshipaka* - that exhibit striking similarity to the clinical features of DED^{2,3}. While most contemporary Ayurvedic scholars correlate *Shushkakshipaka* with DED, a few consider *Krichronmeela* and *Shushkakshipaka* as representing the initial and advanced stages of DED, respectively.²⁻⁴

Krichronmeela is the first *Nethra roga*, enumerated uniquely by Acharya Vagbhata, in both Ashtanga Hridaya and Ashtanga Samgraha.^{5,6} No reference to a disease by this name is found in any other classical works. It is categorized as a *varthmagata roga*, a disease localized to the eyelids. In contrast, *Shushkakshipaka* is explained by both Acharya Vagbhata and Sushruta. It is enumerated as a *Sarvakshigata roga*, a disease affecting multiple ocular structures or the eye as a whole.⁶⁻⁸

This narrative review takes a closer look at the classical descriptions of *Krichronmeela* and relates them to the modern understanding of Dry Eye Disease (DED). By doing so, it seeks to deepen our insight into the condition, identify areas where Ayurveda and modern science intersect, and explore ways to prevent the progression of *Krichronmeela*-a *Vartmagata Roga*-into *Shushkakshipaka*, a *Sarvakshigata Roga*.

Methodology

This review is a descriptive and analytical examination of both classical and contemporary Ayurvedic literature on *Krichronmeela*, followed by a critical analysis of this condition for its similarities with DED.

1. Sources of Data

Relevant Modern literature on DED is collected from

- TFOS DEWS II report
- Ophthalmology Textbooks
- Scientific publications retrieved from databases such as PubMed, Scopus and Google Scholar

Primary Sources for Ayurvedic literature are,

- Classical Ayurvedic textbooks such as Sushruta Samhita, Ashtanga Hridaya, Ashtanga Samgraha, and others
- Commentaries by renowned scholars such as Dalhana, Indu and Arunadatha
- Ayurvedic research publications available through academic portals and online databases

Search Strategy and Keywords

A Literature search was conducted using the following keywords in various combinations:

- Dry Eye Disease, TFOS DEWS II report, Ocular Surface disease, Tear film instability, Ayurvedic Concept of Dry Eye Disease, Shushkakshipaka, Sushkakshipaka, Krichronmeela, Krichronmeela, Ayurvedic Management of Dry Eye.

Inclusion and Exclusion Criteria

- Classical references that describe *Krichronmeela* and *Shushkakshipaka* were included.

- Peer-reviewed Ayurvedic and modern Ophthalmology articles on DED were considered.
- Non-peer-reviewed sources, anecdotal case reports, and commercially oriented websites were excluded.

Method of Analysis

- Extracted the classical references and summarized *lakshana* and *chikitsa* of *Krichronmeela*.
- Comparing these findings with the description of DED based on modern literature.
- Structuring a comparative table to highlight the key points.
- Identifying the possible areas of integration between the classical ayurvedic and modern ophthalmological understanding of the condition.

Review of Literature

- 15 sources including classical Ayurvedic textbooks, Modern textbooks of Ophthalmology and Journal articles published from 01-01-2025 to 01-10-2025 were retrieved and included in this narrative review.

Modern Understanding of Dry Eye Disease (DED)

The refined definition of the disease as proposed by TFOS DEWS II report is "Dry eye is a multifactorial disease of the ocular surface characterized 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 etiological roles¹."

The term ocular surface refers to the anatomical structures of the eye and its adnexa, including the cornea, conjunctiva, eyelids, eyelashes, tear film, and both the main and accessory lacrimal glands as well as meibomian glands. Tear film plays a crucial role in protecting and lubricating the ocular surface and is essential for maintaining the cornea's normal refractive function.

IN DED, various alterations in the tear film and ocular surface are observed, primarily due to disrupted tear film homeostasis triggered by multiple underlying causes. The TFOS DEWS II report identifies three key etiological factors contributing to DED:

1. Tear film hyperosmolarity
2. Ocular surface inflammation and damage
3. Neurosensory abnormalities

Among these, tear film hyperosmolarity is considered a central initiating factor. It triggers stress signals in the ocular cells, leading to the release of inflammatory molecules, which attract more immune cells and a cascade of inflammatory and

cellular responses.⁹ These mechanisms progressively disrupt ocular surface integrity in the form of loss of glycocalyx mucins, death of epithelial cells, and goblet cells loss. This sets off a vicious cycle, making DED a self-perpetuating condition. Importantly, hyperosmolarity is not the only starting point - inflammation (allergy, preservatives, vitamin A deficiency, mucin problems, etc.) can also trigger the same cycle.⁹

Classification Schemes of DED

The TFOS DEWS II report provides an extensive classification system of DED, which is essential to guide diagnosis and thereby improve patient care through appropriate treatment. The classification system is as follows.

As per this classification, there are two primary categories of DED, Aqueous deficient Dry Eye (ADDE) and evaporative Dry Eye (EDE). And the report identifies clearly that both these types of DED are not completely separate disease, instead they lie on a continuum - meaning a person can have a mix of both problems at the same time, in different degrees. The EDE is known to be more common than the ADDE. Sometimes the person may have only one form of the disease, and as the disease progresses, both forms often appear together.¹

EDE occurs when the eyelids or ocular surface cause the tears to evaporate too quickly. Meibomian Gland Dysfunction (MGD) is the main cause of EDE and is considered as the most common cause of DED overall. Other causes include poor blinking, mucin problems or contact lens wear. ADDE occurs

when the lacrimal gland does not work properly. Identifying the predominant type helps to select the main line of therapy, and the all other possible contributing factors should also be considered, while designing the best individualized management plan for each patient.⁹

Clinical presentation and Diagnosis

Ocular discomfort and visual disturbances are the main features of DED. Common symptoms include a foreign body sensation, dryness, itching, and other nonspecific symptoms, such as ocular discomfort and chronically sore eyes that do not respond to previous eye drops.¹ On examination, the tear film may display stringy mucus and particulate matter. The marginal tear strip might be reduced or absent. Frothy secretions can also be seen in the tear film. The conjunctiva often appears lustreless and mildly congested, with possible signs of dryness or keratinization. The cornea may show punctate epithelial erosions, mucus plaques, filaments, and a general loss of lustre. Additionally, signs related to underlying causes, such as posterior blepharitis, may be present.

The diagnosis of DED can be supported by several tests, including Tear film break-up time (TBUT), Schirmer I test and Vital staining of the ocular surface using dyes such as Rose Bengal, fluorescein, or lissamine green, These diagnostic tools help in assessing tear film stability, tear production, and epithelial integrity, which are critical in confirming the presence and severity of DED.¹⁰

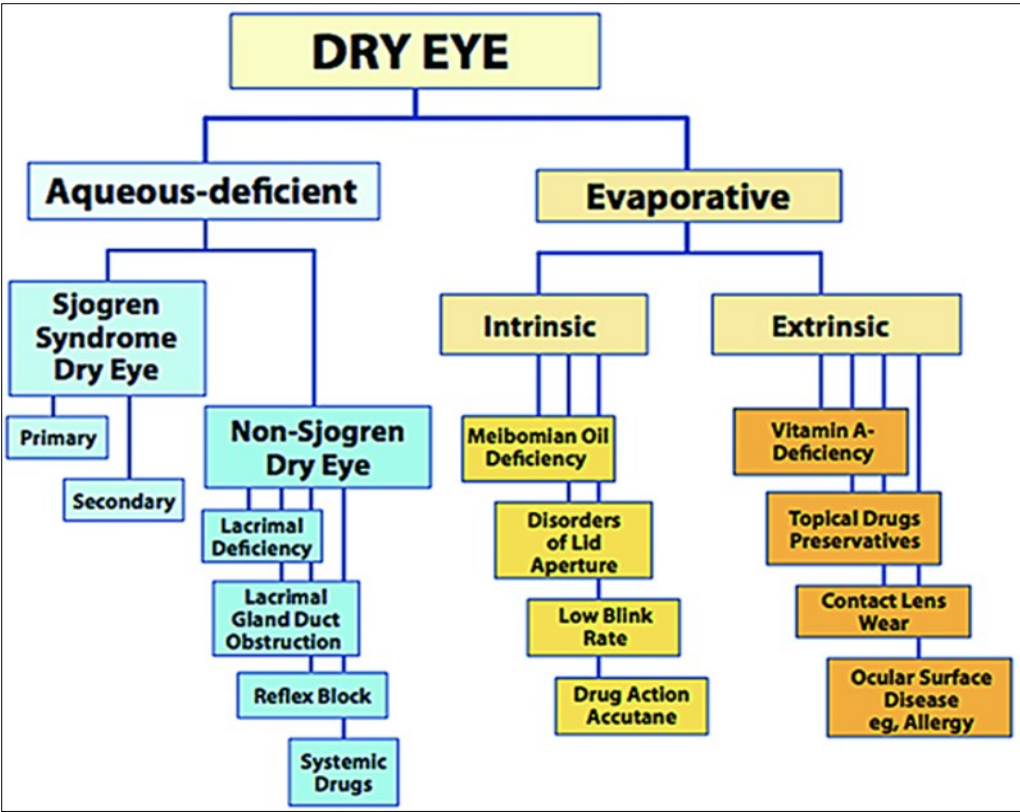


Figure 1: Classification of Dry Eye¹

Treatment

At present, there is no complete cure for DED¹¹. The chronic and episodic nature of the condition necessitates ongoing management rather than one-time treatment. The condition is best addressed through a step-wise management algorithm, beginning with lifestyle and environmental modifications and progressing to ocular lubricants, anti-inflammatory therapies, and advanced interventions such as autologous serum, scleral lenses, and surgical procedures. Procedures such as salivary gland transplantation, amniotic membrane grafting and even psychological approach may be necessary in the management of DED.¹²

Complications of DED

Dry Eye Disease may lead to vision-threatening complications, such as epithelial breakdown, corneal ulceration, melting, and even perforation.

Ayurvedic Concepts of Netra Roga Relevant to DED

Krichronmeela

Krichronmeela or *Krichrabodha* is a *varthmagata roga*, a disease affecting the eyelids. It is a *Vatika roga* that occurs when *Vata* enters the *sira* located in the *Varthma* and causes symptoms such as *varthma sthambha* / difficulty in moving the lids immediately after waking up from sleep, associated with pain. Another important feature of this condition is *pamshupoornabhanethrathva*, i.e., grittiness of the eyes, difficulty to open the eyes and lacrimation. The symptoms are typically relieved by rubbing the eyes.^{13,14}

In Shashilekha Vyaghyana Acharya Indu explains the sloka अस्तत्रैवस्थिते चलो वायुः प्रधानो वर्त्माश्रयाः सिराः प्राप्य सुप्तोत्थितस्य तत्कालत्यक्तनिद्रस्य पुंसो वर्त्मस्तम्भादिकं कुरुते। तस्य च रोगस्य विमर्दनाच्छमः स्यात्। तं रोगविशेषमाचार्या नाम्ना कृच्छ्रोन्मीलमिति वदन्ति. From this is clear that he considers involvement of other doshas other than Vayu in the samprapthi of the disease(चलो वायुः प्रधानो वर्त्माश्रयाः सिराः प्राप्य).¹³ He also explains that the condition occurs immediately after waking up from the bed (सुप्तोत्थितस्य तत्कालत्यक्तनिद्रस्य पुंसो वर्त्मस्तम्भादिकं कुरुते). The condition subsides by rubbing the eyes.¹³

The condition has a good prognosis (*Sadhya Vyadhi*), and the treatment includes *Snehapana* with *Purana ghritha* prepared with *Draksha* as *Kashaya* and *Kalka*, with *Sita* as *anupana*. Other *snigdha prayogas* such as *snigdha nasya*, *snigdha dhooma*, *snigdha anjana*, *tharpana*, and *putapaka* are also mentioned¹⁵. In Ashtanga Sangraha, *Vasthi*, *Svedana* / sudation of eye lids and even *Dahana karma*/ *agni karma* on the eyelid is indicated¹⁵.

A focused literature search identified only a limited number of Ayurvedic research articles discussing Krichronmeela in relation to dry eye disease (DED). All these studies describe Krichronmeela as the initial stage of DED, which subsequently progresses to Shushkakshipaka. The former condition is primarily characterized by the involvement of Vāta doṣa, while the latter involves Pitta doṣa in the Samprāpti. Although most of the identified publications acknowledge the sequential occurrence of Krichronmeela and Shushkakshipaka, they do not critically examine the underlying mechanisms or pathogenesis. These works merely state that Krichronmeela represents the early stage of DED, without providing adequate justification or analytical reasoning

In contrast, the study by Sreeja Sukesan et al. offers a detailed critical analysis, explicating the pathophysiological basis for the progression from Krichronmeela to Shushkakshipaka. This work elaborates the Nidana and samprapthi of dry eye disease. It further elucidates the role of tear film instability, ocular surface inflammation, neurosensory abnormalities and glandular dysfunction from an integrated Ayurvedic and modern perspective, highlighting the importance of early intervention at the Krichronmeela stage to halt progression.²

The study identifies *rookshata* (Dryness) as the root cause of the disease. Etiological factors (*viharaja Nidana* and Environmental factors) cause an increase in rooksha guna of the eye, leading to chaya of Vata in the ocular surface. This in turn leads reduced quality of the tear film affecting the normal movements of the lids, especially Opening the lids (*Unmeelanam*). If untreated or unsuccessfully treated, *Pitha* may involve in the *samprapthi* causing inflammatory changes in other structures such as conjunctiva and cornea, making the disease a *sarvakshigata roga*, *Sushkakshipaka*.²

Shushkakshipaka

Shushkakshipaka is enumerated as a *Sadhya Vyadhi* in the classical texts. A slight difference of opinion exists among Acharyas regarding the *dosha* predominance as well as the clinical features. Acharya Sushruta describes it as a *Vathika* condition, whereas Vagbhata classifies it as *Vata - Paithika* disorder. Slight variations in the clinical features described by the classical texts can also be seen. Slight variations in the clinical features described in the classical texts can also be observed.¹⁶

The clinical features of the condition, as explained by Sushruta include *Koonitha* (interpreted by Dalhana as *Nimeelitha*, meaning 'remains closed'), *Daruna* (explained by Dalhana as *Katina*, i.e., 'hard'), *Rookshata* of varthma (dryness of the eyelids). He also describes *vilokane aviladarshanam*

(interpreted as *ati darshanae aviladarshanam*, which refers to Blurring of vision after prolonged viewing. The term *Sudarunam Yat pratibodhane* is further elaborated by Dalhana as *Pratibodhane unmeshe cha yadatishayena katinam, anye sudarunam kruchronmeelanam*, which indicates marked difficulty in opening the eyes particularly upon awakening.¹⁶

According to Acharya Vagbhata, the condition is characterized by *Gharsha* (grittiness), *toda bheda* and *shoola* (various types of pain), *upadeha* (stickiness), *rooksha* and *darunathva* of *varthma* and *akshi* (dryness and roughness of lids and eyes), *krichronmeelana nimeelanam* (difficulty in both opening and closing the eyes), *vikoonana*, *vishushkathva* (dryness), *sheetecha* (desire for cold), *paka* (inflammatory signs).¹⁷

The management of *Shushkakshipaka* includes *Anjana*, *Snehapana*, *Tarpana*, *Nasya*, and *Parisheka*. *Snehapana* and *Tarpana* are preferably administered using *Jeevaneeya Siddha Ghrita*, while *Nasya* may be performed with *Anutaila*. Acharya Vagbhata also recommends these treatment modalities. He begins therapy with *Snehapana* and *Tarpana* using *Jeevaneeya Ghrita*, and advises *Seka* with lukewarm milk containing a small quantity of *Saindhava*. Additionally, a unique preparation is described for *Anjana*, in which the ashes of hair burned in a *Mallaka Puta* are smeared with *Ghrita* on a mirror or glass surface, collected, mixed again with *Ghrita*, and stored in an iron vessel before application.¹⁸

Most of the contemporary authors correlate DED with *Shushkakshipaka* only.^{19–21} Only a very few authors consider *Krichronmeela* as the early stage and *Shushkakshipaka* as an advanced stage of the disease^{22,23}. A noted limitation across the included literature is the reliance on open-access sources and indexed Ayurvedic journals, leaving gaps due to potential exclusion of subscription-based or more recent publications. Moreover, while symptomatic correlations are robust, comprehensive mechanistic studies remain sparse, underscoring a need for further rigorous research integrating Ayurveda with contemporary ocular science.

Comparative Analysis

From the above table, it is evident that *Krichronmeela*, *Shushkakshipaka* as described by Vagbhata, as well as Sushruta -exhibit notable similarities among themselves and with Dry Eye Disease (DED). Furthermore, a gradual increase in severity can be observed, progressing from *Krichronmeela* to *Shushkakshipaka* described by Sushruta, and then to that described by Vagbhata.

Discussion

In Ayurvedic methodology of clinical evaluation, a disease should be examined in terms of *Panchalakshana Nidana*, which include *Nidana*, *pragroopa*, *lakshana*, *Upashaya* and *Samprapthi*.²⁴ *Nidana* of Either *Krichronmeela* or *Sushkakshipaka* has not explained separately in the classics. Instead one should assume that the *nidanas* explained in the *sarvaroga Nidana* or *Nethra roga samanya Nidana* by Sushruta may lead to these diseases. *Nidanas* such as *Doorekshanata* or looking at distant object for a long time, *Swapna viparyaya* or altered sleep pattern, *Prasakta Samrodana* or continuous weeping, *Kopa* and *Shoka* or excessive anger and grief and *Asatmendriyartha samyoga* especially *athiyoga* and *mithya yoga* of *chakshur indriya* may contribute development of both the abovementioned diseases.²² The above mentioned *Nidana* causes vitiation of *doshas* and *sthanasamshraya* in different parts of the eye, manifestations occur accordingly. For instance, when *doshas* reaches the *Jalavahini sira*, it causes *Jalasrava* and when the *doshas* reaches the *Prathama patala* through the *sira*, it causes visual disturbances. Similarly, when they enter the *Siras* located in the lid, it results in lid diseases including *Krichronmeela*. In this context, *Sira* can be understood as a transport structure - possibly including the channels responsible for conveying the secretions of the Meibomian and lacrimal glands.

Clinical Correlation between *Krichronmeela/Shushkakshipaka* and DED

On careful analysis, the descriptions of *Krichronmeela* by Vagbhata and *Shushkakshipaka* in *Sushruta Samhita* appear to correspond to conditions in which ocular symptoms manifest immediately after waking. In *Krichronmeela*, almost all the

<i>Krichronmeela</i>	<i>Shushkakshipaka</i>	
	Sushruta	Vagbhata
<i>Supthothitasya</i> - symptoms worsen immediately upon waking	<i>Sudarunam Yat pratibodhane</i>	
<i>Varthma stambha</i>	<i>Koonitha (Nimeelitha)</i>	<i>Vikoonana</i>
<i>Vedana</i>		<i>Toda, Bheda, Shoola</i>
<i>Pamsupoornabha nethrathva</i>		<i>Gharsha</i>
<i>Krichronmeelana</i>		<i>Krichronmeelana - Nimeelana</i>
<i>Ashru</i>		
<i>Vimardanath shamam</i>	<i>Daruna, Rooksha, Vilokane avila darshanam</i>	<i>Upadehavath, Rooksha and Daruna - Varthma and Akshi, Vishushkathvam, Sheetecha, Paka</i>

symptoms are subjective and they typically subside following gentle rubbing of the eyes. A comparable phenomenon is observed in inadequate lid seal, a variant of nocturnal lagophthalmos, where the eyelids appear closed but fail to completely exclude air from the ocular surface during sleep, resulting in morning discomfort. Transient incomplete blinking, or partial eyelid closure occurring during upward gaze or prolonged visual tasks, can similarly increase ocular surface exposure and tear evaporation. This clinical pattern closely aligns with the features of evaporative dry eye disease (EDE). Other symptoms of *Krichronmeela* such as *Varthmashabha*, *Vedana*, *Pamshupoornabha nethrathva*, *Krichronmeelana* and *Ashru* shows obvious similarity with symptoms of dry eye⁷.

Most contemporary authors correlate dry eye disease (DED) with *Shushkakshipaka*. *Sushruta* describes *Shushkakshipaka* as a condition that worsens immediately upon waking and after prolonged visual activity. Although primarily classified as a *vatika* disorder, the term *paka* indicates secondary involvement of *pitta* in its pathogenesis. As emphasised by *Sushruta*, *paka* cannot occur without *pitta*, highlighting the inflammatory component of the condition.⁸ In this context, *Sushruta's* description of *Shushkakshipaka* can be interpreted as encompassing clinical features of both early-stage *Krichronmeela* and the more advanced stage described by *Vagbhata*.

Vagbhata's account portrays *Shushkakshipaka* as a more severe condition, with an involvement of *pitta* in the *samprapthi*. In *Krichronmeela*, the patient experiences only difficulty in opening the eyes, but in *Shushkakshipaka*, patient experiences discomfort in both opening and closing the lids, indicating the severity of condition. The clinical presentation includes both subjective and objective symptoms. There are evident signs of inflammation and increased severity, and the recommended treatment-potent therapies such as *Snehapana* -further underscores the intensity of the disorder.

Pathophysiological Parallels

Modern understanding of dry eye disease (DED) attributes its onset to tear film instability and hyperosmolarity, which trigger a cascade of inflammatory events. Ocular discomfort in DED is partly due to hyperosmolarity, which directly stimulates pain pathways. As the tear film thins and breaks up, hyperosmolarity increases, leading to the release of inflammatory mediators such as prostaglandins, cytokines, and neurokinins, all of which contribute to a cascade of inflammatory and cellular responses leading pain. Loss of lubrication between the eyelids and the ocular surface further exacerbates friction-related damage, including reduced tear volume in aqueous-deficient DED, loss of goblet-cell mucin, degradation of the glycocalyx mucin layer, and decreased levels of lubricin, a key boundary lubricant.

Osmolarity is a measure of the concentration of solute particles in a solution, expressed as osmoles per liter (Osm/L). The normal osmolarity of tears is approximately 300 mOsm/L.²⁵ Hyperosmolarity occurs when the water content of the tear film decreases, resulting in a higher concentration of solutes, including organic and inorganic components, which leads to elevated osmolarity. This reduction in water content can be identified as a reduction in *Snigdha* property of the tears, as *Snigdha* is an inherent property of *Jala*.²⁶ Reduction in *Snigdha* causes an aggravation of its opposite quality, *Rookshata*. This increase in *Rooksha guna* causes an increase in *Vata* in the ocular surface or an increase in *Vata* in the ocular surface results in *rookshata* of ocular surface resulting in disturbance of normal ocular milieu. *Rooksha Guna* primarily causes increase in *Vata*, with disturbance in the normal level of *Pitha*, resulting in *Krichronmeela*. Inflammatory and cellular responses, triggered by hyperosmolarity can be identified as the involvement of *Pitha* in the *Samprapthi*, causing *paka lakshana* and more intense symptoms, resulting in *Shushkakshipaka*.

Mechanical complications, including filamentary keratitis, lid-parallel conjunctival folds (LIPCOF - small conjunctival folds seen just above the lower lid margin), and lid-wiper epitheliopathy (LWE - caused by friction between the upper eyelid and the ocular surface due to inadequate lubrication leading to epithelial damage), further contribute to ocular discomfort. Filamentary keratitis causes pain due to traction on corneal nerve endings during blinking, while LIPCOF-related discomfort arises from similar frictional mechanisms. In LWE, pain is associated with hypersensitivity of the lid wiper region and adjacent corneal areas, which normally exhibit mechanical sensitivity comparable to the central cornea in healthy eyes. All these pathological changes align with deranged *Vata and Pitha* in *Samprapthi*. Especially the *rooksha* and *Gharathva* properties caused by *vata* causes friction and traction on the ocular surface. LIPCOF and Keratinization of the conjunctiva also gives the signs of *vata* vitiation to the eye. Pathological changes like filamentary keratitis and other inflammatory changes contribute to the clinical picture of *pitha*.

Ayurvedic Management and its Modern Analogues

The management of *Krichronmeela* primarily involves *shamana* therapy. Classical texts describe the use of medicated *ghrita* prepared with *draksha*, to be taken at night. Both *ghrita* and *draksha* possess *vata-pitta hara* properties.^{27,28} Although *Krichronmeela* is classified as a *vatika vikara*, the inclusion of a *pitta hara* ingredient such as *draksha*, as the key ingredient reveals the involvement of *Pitha* in the *samprapthi*. This fact can be identified by the explanation of *Acharya Indu* on the *samprapthi* of *Krichronmeela*, where he says that, '*Vatadi*

Doshas are responsible for the development of *Krichronmeela*. Moreover, in *soothra sthana*, It is clearly explained that '*na eka dosho vyadhaya*:' which means, diseases cannot be of *eka dosha* origin.²⁷ This is significant because *pitta*-induced *paka* (inflammatory transformation) may lead to disease progression toward *Shushkakshipaka*, a more advanced stage of the condition.

Other therapeutic measures such as *Nasya*, *Dhoomapana*, and *Anjana* are also indicated in the management of *Krichronmeela*. Additionally, *Tarpana*, *Putapaka*, and *Vatahara Vasthi* are specifically mentioned in the *Ashtanga Samgraha*. When the indications of various *Kriyakalpas* are analyzed, *Aschyothana* can be identified as the most appropriate procedure for *Krichronmeela*, since the characteristic symptoms-*ruk* (pain), *toda* (pricking sensation), *kandu* (itching), *gharsha* (foreign body sensation), *ashru* (lacrimation), and *daha* (burning) -correspond closely with those described for *Aschyothana*.²⁹ However, instead of prescribing this relatively mild procedure, the classical texts recommend more potent interventions such as *Nasya*, *Dhoomapana*, *Anjana*, *Tarpana*, *Putapaka*, and *Vasthi*. This approach may be appreciated as a proactive attempt to halt the disease progression and prevent its transition to a more severe stage.

Krichronmeela is mentioned as an indication for *Brimhana Nasya* in the *Nasyavidhi Adhyaya* of *Ashtanga Hridaya*.³⁰ The procedure is advised with *Snigdha-Madhura Dravyas*, or with *Sneha*, *Niryasa*, or *Dhanva Mamsa Rasa* prepared using *Snigdha-Madhura Dravyas*. *Nasya* is considered as a special procedure in *Oordhvajathrugata vikaras*. *Nasya* with *Snigdha-madhura dravyas* can be identified as an effort to control *Vata dosha*. But in *Shushkakshipaka*, *Anutaila* has been indicated for *Nasya*, which is the drug for *Svastha*, to maintain his health. In *Dinacharya Nasya* is explained after *Anjana* and is explained as a measure to expel the *Kaphotklesha*, caused by *Anjana*. *Arunadatha* explains the logic behind the name '*Anutaila*' as अणूनीन्द्रियस्रोतासि प्रविशतीत्यर्थः, indicating its potential to reach even minutes channels of *Indriyas*. And cure the disease.

Interestingly, a comparable concept can be observed in the modern management of Dry Eye Disease (DED), where neuromodulation is employed to stimulate tear production. Neuromodulation utilizes electrical, electromagnetic, chemical, or optogenetic techniques to regulate neural activity and thereby influence lacrimal secretion. In DED, chemical or mechanical stimulation of the nasal mucosa activates the nasolacrimal reflex, enhancing tear production. In this reflex arc, the sensory nerves of the nasal cavity constitute the afferent limb, while parasympathetic fibers supplying the lacrimal functional unit form the efferent limb.³¹ Thus, *Nasya* may be

interpreted as an early form of neuromodulatory therapy aimed at restoring lacrimal function through nasal stimulation-a perspective that merits further exploratory research. *Dhooma pana* is generally indicated to prevent or cure *Vata-Kaphaja* diseases affecting the head and neck region.³² *Ashtanga Samgraha* specifically indicates *Dhoomapana* in the management of ocular pain.³³ It is also said that *Dhooma pana* is beneficial for the *brumhana* and *prasadana* of *Indriyas* and is contra indicated in *Raktha-pithaja* conditions.^{33,34} One must be very cautious while administering *Dhooma* in a patient with *Krichronmeela*, as it may enhance any subtle involvement of *pitha* in the *samprapthi*.

Anjana is another *Upakrama* mentioned in the management of *Krichronmeela*. It is a *Kriyakalpa* indicated only in the *Pakvavastha* of ocular disease and should be performed after *Shodhana* procedures such as *Virechana*.¹³ In *Vatika* conditions, *Snehana Anjana* is recommended, and *Ashtanga Samgraha* specifically mentions preparations using *Vasa* of animals like *Sarpa* in *Vatika Timira*, highlighting the importance of unctuous therapies to counteract *Vata*. In modern ophthalmic practice, similar principles are applied in the management of Dry Eye Disease (DED), where lipid-based ocular formulations-such as emulsions containing mineral oils, phospholipids, and other lipids-are used to replenish the tear film's lipid layer. These formulations reduce evaporation and enhance ocular comfort.¹

Additionally, *Ashtanga Samgraha* mentions *Svedana* with *Ksheera* and even *Dahana Karma* in the management of *Krichronmeela*.³⁵ Interestingly, similar therapeutic concepts are observed in the modern management of Meibomian Gland Dysfunction (MGD). In MGD, several devices have been developed to deliver controlled heat therapy to the eyelids, aiming to improve gland function and alleviate symptoms.³¹

For instance, *Blephasteam*®, an electrically powered goggle-like device that provides moist heat, effectively elevates eyelid temperature, reduces tear evaporation by about 32%, and improves parameters such as visual acuity, tear breakup time (TBUT), and patient comfort-even in cases unresponsive to conventional warm compresses. The *MGDRx EyeBag*®, a reusable flaxseed-filled pouch heated in a microwave, delivers sustained eyelid warming and provides symptomatic relief lasting up to six months with periodic use. Similarly, the *EyeGiene*® mask, which employs disposable warming units, offers comparable symptomatic relief, though with relatively lower heat retention. Collectively, these devices enhance Meibomian gland secretion and overall tear film stability through consistent, controlled thermal therapy.³¹

When *Krichronmeela* fails to respond to the aforementioned measures, *Dahana Karma* is indicated according

to *Ashtanga Samgraha*.³⁵ *Tvak Daha* is advised in the form of small circular burns (*Bindu Akara*) on the eyelid, spaced approximately in the size of a *Mudga* (green gram). *Acharya Indu* elaborates that this procedure should be performed using a pointed heated instrument such as *Shara*.³⁵ This may be interpreted as an attempt to liquefy the obstructed secretions of the Meibomian glands by applying localized heat and pressure. Anatomically, the Meibomian glands-25 to 40 in the upper lid and 20 to 30 in the lower lid-are vertically arranged within the tarsal plate. Thus, the spacing and shape of *Dahana* marks described by the Acharyas could reflect an empirical understanding of the glandular orientation and the targeted delivery of heat.

In modern ophthalmology, physical treatments for MGD are likewise aimed at restoring gland patency and function by relieving ductal obstruction. Common approaches include warm compresses or heating devices to liquefy the secretion, manual gland expression to remove the obstructed material, and intraductal probing to mechanically clear the ducts. The LipiFlow® system (TearScience, USA) represents an advanced form of such therapy, delivering controlled thermal energy (around 42.5 °C) directly to the Meibomian glands while simultaneously applying pulsatile pressure to evacuate the contents-an approach remarkably analogous in principle to *Dahana Karma*.

Conclusion

It is a matter of pride for every Ayurvedic scholar that the features of Dry Eye Disease (DED) were described in Ayurvedic classics centuries ago, long before their recognition in modern ophthalmology, which has developed a detailed understanding of the disease only in the past few decades. Modern literature defines DED as a self-perpetuating condition that may, in advanced stages, even require surgical intervention-thereby classifying it as a *Yāpya Vyādhi* (manageable but not completely curable). In contrast, Ayurveda regards it as a *Sādhyā Vyādhi* (curable) when managed with timely and appropriate interventions.

Among the *Laghutrayees*, *Aṣṭāṅga Samgraha* and *Aṣṭāṅga Hṛdaya*-being relatively later and more clinically oriented compilations-provide detailed and exclusive descriptions of *Krichronmeela*. When these are analyzed alongside *Shushkakshipaka* described in *Suśruta Samhita*, it becomes evident that accurate differentiation of these conditions based on both classical symptomatology and modern clinical correlations is essential. Such diagnostic precision not only refines our understanding of the disease spectrum but also

supports the rational selection of therapeutic strategies for the effective management of DED.

The concept of *Krichronmeela* in Ayurvedic classics shows close similarity with evaporative Dry Eye, particularly that associated with Meibomian Gland Dysfunction. Rather than uniformly categorizing all cases of DED as *Shushkakshipaka*, establishing a differential diagnosis distinguishing *Krichronmeela* from *Shushkakshipaka* is warranted. Such an approach may help prevent disease progression and enable better therapeutic outcomes with minimal intervention.

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