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FULL FACTS ABOUT DRY EYE

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Abstract: *Dry Eye Disease (DED) is a prevalent condition characterized by discomfort, visual disturbances, and potential damage to the ocular surface. It arises from an imbalance in the tear film, often due to insufficient tear production or excessive evaporation. This article reviews the pathophysiology, causes, risk factors, clinical manifestations, and current management strategies for DED.*

Key words: *Keratoconjunctivitis Sicca, Tear Film Imbalance, Aqueous-deficient Dry Eye, Evaporative Dry Eye, Meibomian Gland Dysfunction (MGD), Inflammation, Lacrimal Gland Dysfunction, Ocular Surface Disease Index (OSDI), Schirmer's Test, Tear Break-up Time (TBUT), Artificial Tears, Punctal Plugs, Cyclosporine A (Restasis), Autoimmune Diseases (e.g., Sjögren's Syndrome)*

Introduction: Dry Eye Disease, also known as keratoconjunctivitis sicca, affects millions of individuals worldwide and is a leading cause of ocular morbidity. It can be classified into two primary subtypes: evaporative dry eye and aqueous-deficient dry eye. Both types are linked by an underlying dysfunction in the tear film, which is essential for maintaining the health of the cornea and conjunctiva. **Dry eye syndrome**, also known as **keratoconjunctivitis sicca**, is the condition of having dry eyes. Symptoms include dryness in the eye, irritation, redness, discharge, blurred vision, and easily fatigued eyes. Symptoms range from mild and occasional to severe and continuous. Dry eye syndrome can lead to blurred vision, instability of the tear film, increased risk of damage to the ocular surface such as scarring of the cornea, and changes in the eye including the neurosensory system.

Dry eye occurs when either the eye does not produce enough tears or when the tears evaporate too quickly. This can be caused by age, contact lens use, meibomian gland dysfunction, pregnancy, Sjögren syndrome, vitamin A deficiency, omega-3 fatty acid deficiency, LASIK surgery, and certain medications such as antihistamines, some blood pressure medication, hormone replacement therapy, and antidepressants. Chronic conjunctivitis such as from tobacco smoke exposure or infection may also lead to the condition. Diagnosis is mostly based on the symptoms, though a number of other tests may be used. Dry eye syndrome occasionally makes wearing contact lenses impossible.

Treatment depends on the underlying cause. Artificial tears are usually the first line of treatment. Wrap-around glasses that fit close to the face may decrease tear evaporation. Looking carefully at the medications a person is taking and, if safe, altering the medications, may also improve symptoms if these medications are the cause. Some topical medications, or eye drops, may be suggested to help treat the condition. The immunosuppressant cyclosporine (ciclosporin) may be recommended to increase tear production and, for short term use, topical corticosteroid medications are also sometimes

helpful to reduce inflammation. Another treatment that is sometimes suggested is lacrimal plugs that prevent tears from draining from the surface of the eye.

Dry eye syndrome is a common eye disease. It affects 5–34% of people to some degree depending on the population looked at. Among older people it affects up to 70%. In China it affects about 17% of people. The phrase "keratoconjunctivitis sicca" means "dryness of the cornea and conjunctiva" in Latin.

Methodology:

This article is a comprehensive review of existing literature on Dry Eye Disease (DED), focusing on its pathophysiology, causes, risk factors, clinical manifestations, diagnostic methods, and treatment strategies. The methodology employed includes a systematic review of peer-reviewed studies, clinical trials, and recent advancements in the field of ophthalmology related to DED.

1. **Literature Search:** A detailed literature search was conducted in multiple scientific databases including PubMed, Google Scholar, and Scopus to identify articles published between 2000 and 2024. Keywords such as "Dry Eye Disease," "Keratoconjunctivitis Sicca," "tear film imbalance," "Meibomian gland dysfunction," and "ocular surface inflammation" were used to filter relevant studies.
2. **Inclusion Criteria:**

- Studies focused on the pathophysiology, risk factors, symptoms, diagnosis, or treatment of Dry Eye Disease.
- Clinical trials assessing different treatment modalities, including drug therapies, lubricants, and surgical interventions.
- Articles published in peer-reviewed ophthalmology and medical journals.
- Meta-analyses and systematic reviews that provide insights into epidemiological data and consensus treatment approaches.

3. **Exclusion Criteria:**

- Studies not related to Dry Eye Disease or its clinical management.
- Articles that did not undergo peer review or were published in non-scientific sources.
- Research published prior to 2000 unless it provided foundational knowledge still relevant today.

4. **Data Extraction:** From the selected studies, key data were extracted regarding:

- Pathophysiological mechanisms, including the roles of tear production and evaporation.
- Diagnostic tests used for assessing the severity of DED.
- Current treatment options and their effectiveness in managing symptoms and preventing ocular surface damage.
- Risk factors and epidemiological data on the prevalence of DED in various populations.

5. **Data Synthesis:** Data were synthesized to provide a comprehensive understanding of DED, including:

- Comparative analysis of diagnostic tools and their sensitivity in identifying DED.
- Review of evidence-based treatment regimens and their clinical outcomes.

- Discussion on emerging therapies, including biologics and innovative technologies.

1. **Quality Assessment:** The quality of the included studies was assessed using the Newcastle-Ottawa Scale for cohort studies and the Cochrane Collaboration's risk of bias tool for randomized controlled trials. Studies with low risk of bias and high quality were prioritized to ensure the reliability of the conclusions drawn.
2. **Statistical Analysis:** Statistical data from clinical trials were reviewed to assess the effectiveness of various treatment strategies in reducing symptoms of DED. The findings were presented in terms of effect size, p-values, and confidence intervals where available.
3. **Expert Opinion:** In addition to published literature, insights from experts in ophthalmology and ophthalmic pharmacology were also considered to discuss the clinical challenges in diagnosing and treating DED.

Epidemiology

Keratoconjunctivitis sicca is relatively common within the United States, especially in patients aged 40 or older. 10–20% of adults experience Keratoconjunctivitis sicca. Approximately 1 to 4 million adults (age 65–84) in the US are affected.

While persons with autoimmune diseases have a high likelihood of having dry eyes, most persons with dry eyes do not have an autoimmune disease. Instances of Sjögren syndrome and keratoconjunctivitis sicca associated with it are present much more commonly in women, with a ratio of 9:1. In addition, milder forms of keratoconjunctivitis sicca also are more common in women. This is partly because hormonal changes, such as those that occur in pregnancy, menstruation, and menopause, can decrease tear production.

Pathophysiology: The tear film, which coats the ocular surface, is composed of three layers: lipid, aqueous, and mucin. Each layer plays a crucial role in providing lubrication, reducing friction, and protecting the eye from pathogens. In DED, an imbalance between tear production and evaporation leads to insufficient moisture and increased ocular surface inflammation.

- **Aqueous-deficient dry eye:** Characterized by reduced tear production, commonly due to dysfunction of the lacrimal glands, such as in conditions like Sjögren's syndrome or age-related lacrimal gland atrophy.
- **Evaporative dry eye:** Caused by excessive evaporation of the tear film, often linked to meibomian gland dysfunction (MGD), which reduces the lipid layer of the tear film.

Chronic inflammation of the ocular surface results in the release of pro-inflammatory cytokines, which exacerbate tissue damage, leading to a vicious cycle of further dryness and discomfort.

Clinical Presentation

Signs and symptoms

Typical symptoms of dry eye syndrome are dryness, burning and a sandy-gritty eye irritation that gets worse as the day goes on. Symptoms may also be described as itchy, stinging or tired eyes. Other symptoms are pain, redness, a pulling sensation, and pressure behind the eye. There may be a feeling that something, such as a speck of dirt, is in the eye. The resultant damage to the eye's surface increases discomfort and sensitivity to bright light. Both eyes usually are affected.

There may also be a stringy discharge from the eyes. Although it may seem contradictory, dry eye can cause the eyes to water due to irritation. One may experience excessive tearing such as if something got into the eye. These reflex tears will not necessarily make the eyes feel better since they are the

watery tears that are produced in response to injury, irritation, or emotion which lack the lubricating qualities necessary to prevent dry eye.

Because blinking coats the eye with tears, symptoms are worsened by activities in which the rate of blinking is reduced due to prolonged use of the eyes. These activities include prolonged reading, computer usage (computer vision syndrome), driving, or watching television. Symptoms increase in windy, dusty or smoky (including cigarette smoke) areas, in dry environments high altitudes including airplanes, on days with low humidity, and in areas where an air conditioner (especially in a car), fan, heater, or even a hair dryer is being used. Symptoms reduce during cool, rainy, or foggy weather and in humid places, such as in the shower.

Most people who have dry eyes experience mild irritation with no long-term effects. However, if the condition is left untreated or becomes severe, it can produce complications that can cause eye damage, instability of the tear film, neurosensory changes, impaired vision, or (rarely) in the loss of vision.

Causes

Any abnormality of any one of the three layers of tears produces an unstable tear film, resulting in symptoms of dry eyes.

Increased evaporation

The most common cause of dry eye is increased evaporation of the tear film, typically as a result of meibomian gland dysfunction (MGD). The meibomian glands are two sets of oil glands that line the upper and lower eyelids and secrete the oily outer layer of the tear film—the lipid layer. These glands often become clogged due to inflammation caused by blepharitis and/or rosacea, preventing an even distribution of oil. The result is an unstable lipid layer that leads to increased evaporation of the tear film.

In severe cases of MGD, the meibomian glands can atrophy and cease producing oil entirely.

Low humidity

Low humidity may cause dry eye syndrome.

Decreased tear production

Keratoconjunctivitis sicca can be caused by inadequate tear production from lacrimal hyposecretion. The aqueous tear layer is affected, resulting in aqueous tear deficiency (ATD). The lacrimal gland does not produce sufficient tears to keep the entire conjunctiva and cornea covered by a complete layer. This usually occurs in people who are otherwise healthy. Increased age is associated with decreased tearing. This is the most common type found in postmenopausal women.

In many cases, aqueous deficient dry eye may have no apparent cause (idiopathic). Other causes include congenital alacrima, xerophthalmia, lacrimal gland ablation, and sensory denervation. In rare cases, it may be a symptom of collagen vascular diseases, including relapsing polychondritis, rheumatoid arthritis, granulomatosis with polyangiitis, and systemic lupus erythematosus. Sjögren syndrome and other autoimmune diseases are associated with aqueous tear deficiency. Drugs such as isotretinoin, sedatives, diuretics, tricyclic antidepressants, antihypertensives, oral contraceptives, antihistamines, nasal decongestants, beta-blockers, phenothiazines, atropine, and pain relieving opiates such as morphine can cause or worsen this condition.[4][13][14] Infiltration of the lacrimal glands by sarcoidosis or tumors, or postradiation fibrosis of the lacrimal glands can also cause this condition. Recent attention has been paid to the composition of tears in normal or dry eye individuals. Only a small fraction of the estimated 1543 proteins in tears are differentially deficient or upregulated in dry eye, one of which is lacritin. Topical lacritin promotes tearing in rabbit preclinical studies. Also,

topical treatment of eyes of dry eye mice (Aire knockout mouse model of dry eye) restored tearing, and suppressed both corneal staining and the size of inflammatory foci in lacrimal glands.

Additional causes

Excess screen time on computers, smartphones, tablets, or other digital devices can cause dry eye. "Humans normally blink about 15 times in one minute. However, studies show that we only blink about 5 to 7 times in a minute while using computers and other digital screen devices. Blinking is the eye's way of getting the moisture it needs on its surface."

Aging is one of the most common causes of dry eyes because tear production decreases with age. Several classes of medications (both prescription and OTC) have been hypothesized as a major cause of dry eye, especially in the elderly. Particularly, anticholinergic medications that also cause dry mouth are believed to promote dry eye. Dry eye may also be caused by thermal or chemical burns, or (in epidemic cases) by adenoviruses. A number of studies have found that people with diabetes have an increased risk for the condition.

About half of all people who wear contact lenses complain of dry eyes. There are two potential connections between contact usage and dry eye. Traditionally, it was believed that soft contact lenses, which float on the tear film that covers the cornea, absorb the tears in the eyes. The connection between a loss in nerve sensitivity and tear production is also the subject of current research.

Dry eye also occurs or becomes worse after LASIK and other refractive surgeries, in which the corneal nerves which stimulate tear secretion are cut during the creation of a corneal flap. Dry eye caused by these procedures usually resolves after several months, but it can be permanent. Persons who are thinking about refractive surgery should consider this.

An eye injury or other problem with the eyes or eyelids, such as bulging eyes or a drooping eyelid can cause keratoconjunctivitis sicca. Disorders of the eyelid can impair the complex blinking motion required to spread tears.

Abnormalities of the mucin tear layer caused by vitamin A deficiency, trachoma, diphtheric keratoconjunctivitis, mucocutaneous disorders, and certain topical medications are also causes of keratoconjunctivitis sicca.

Persons with keratoconjunctivitis sicca have elevated levels of tear nerve growth factor (NGF).[It is possible that this eye's surface NGF plays an important role in ocular surface inflammation associated with dry eyes.

Sarcoidosis

Sarcoidosis is a multisystem disease of unknown etiology; the diagnostic histopathologic feature is the presence of noncase-ating granulomas in various tissues.

- Immunologic abnormalities include high levels of CD4+ TH1 cells in the lung that secrete cytokines such as IFN-γ.
- Clinical manifestations include lymph node enlargement, eye involvement (sicca syndrome [dry eyes], iritis, or iridocyclitis) skin lesions (erythema nodosum, painless subcutaneous nodules), and visceral involvement (liver, skin, bone marrow). Lung involvement occurs in 90% of cases, with formation of granulomas and interstitial fibrosis

Sjögren Syndrome

Sjögren syndrome is a chronic disease characterized by dry eyes (keratoconjunctivitis sicca) and dry mouth (xero-stomia) resulting from immunologically mediated destruction of the lacrimal and salivary

glands.It occurs as an isolated disorder (primary form), also known as the sicca syndrome, or more often in association with another autoimmune disease (secondary form). Rheumatoid arthri-tis is the most common associated disorder, while other patients have SLE, polymyositis, scleroderma, vasculitis, mixed connective tissue disease, or autoimmune thyroid disease.

The lacrimal and salivary glands characteristically show dense lymphocytic infiltration consisting mainly of acti-vated CD4+ helper T cells and some B cells, including plasma cells. Serologic studies frequently reveal autoanti-bodies. Antibodies against two ribonucleoprotein antigens,

SS-A (Ro) and SS-B (La) (see Table 5.11), can be detected in as many as 90% of patients by sensitive techniques. High titers of antibodies to SS-A are associated with early disease onset, longer disease duration, and extraglandular mani-festations, such as cutaneous vasculitis and nephritis.

These autoantibodies also are present in a smaller percent-age of patients with SLE and hence are not diagnostic of Sjögren syndrome. In addition, about 75% of patients have rheumatoid factor (an antibody reactive with self IgG), and 50% to 80% of patients have ANAs.

Xerostomia

Xerostomia is defined as a dry mouth resulting from a decrease in the production of saliva. Its incidence varies among populations but has been reported in more than 20% of individuals older than 70 years of age. It is a major feature of the autoimmune disorder Sjögren syndrome, in which it usually is accompanied by dry eyes. A lack of salivary secretions is also a major complication of radiation therapy. However, xerostomia is most fre-quently observed as a side effect of many common classes of medications including anti-cholinergic, anti-depressant/anti-psychotic, diuretic, anti-hypertensive, sedative, muscle relaxant, analgesic, and anti-histaminic agents. The oral cavity may reveal only dry mucosa and/or atrophy of the papillae of the tongue, with fissuring and ulcerations, or, in Sjögren syndrome, concomitant inflammatory enlarge-ment of the salivary glands. Complications of xerostomia include increased rates of dental caries and candidiasis, as well as difficulty in swallowing and speaking.

Diagnosis

Symptom assessment is a key component of dry eye diagnosis – to the extent that many believe dry eye syndrome to be a symptom-based disease. Several questionnaires have been developed to determine a score that would allow for a diagnosis. The McMonnies & Ho dry eye questionnaire is often used in clinical studies of dry eyes.

Some tests allow patients to be classified into one of two categories, "aqueous-deficient" or "hyperevaporative". Diagnostic guidelines were published in 2007 by the Dry Eye Workshop, updated by the Dry Eye Workshop II in 2017. A slit lamp examination can be performed to diagnose dry eyes and to document any damage to the eye. When realizing this test, the practitioner is testing the eyelid margin.

A Schirmer's test can measure the amount of moisture bathing the eye. This test is useful for determining the severity of the condition. A five-minute Schirmer's test with and without anesthesia using a Whatman #41 filter paper 5 mm wide by 35 mm long is performed. For this test, wetting under 5 mm with or without anesthesia is considered diagnostic for dry eyes.

If the results for the Schirmer's test are abnormal, a Schirmer II test can be performed to measure reflex secretion. In this test, the nasal mucosa is irritated with a cotton-tipped applicator, after which tear production is measured with a Whatman #41 filter paper. For this test, wetting under 15 mm after five minutes is considered abnormal.

A tear breakup time (TBUT) test measures the time it takes for tears to break up in the eye. The tear breakup time can be determined after placing a drop of fluorescein in the cul-de-sac.

A tear protein analysis test measures the lysozyme contained within tears. In tears, lysozyme accounts for approximately 20 to 40 percent of total protein content.

A lactoferrin analysis test provides good correlation with other tests.

The presence of the recently described molecule Ap4A, naturally occurring in tears, is abnormally high in different states of ocular dryness. This molecule can be quantified biochemically simply by taking a tear sample with a plain Schirmer test. Utilizing this technique it is possible to determine the concentrations of Ap4A in the tears of patients and in such way diagnose objectively if the samples are indicative of dry eye.

The tear osmolarity test has been proposed as a test for dry eye disease. Tear osmolarity may be a more sensitive method of diagnosing and grading the severity of dry eye compared to corneal and conjunctival staining, tear break-up time, Schirmer test, and meibomian gland grading. Others have recently questioned the utility of tear osmolarity in monitoring dry eye treatment.

Management (Treatment)

A variety of approaches can be taken to treat dry eye syndrome. Approaches include: avoidance of exacerbating factors (things that make it worse), tear stimulation and supplementation, increasing tear retention, eyelid cleansing, and treatment of eye inflammation.

Conditions such as blepharitis can often co-exist and paying particular attention to cleaning the eyelids morning and night with mild soaps and warm compresses can improve both conditions.

Avoiding exacerbating factors and environmental control

Dry eyes can be worsened by smoky environments, dust, and indoor air conditioning, and by our natural tendency to reduce our blink rate when concentrating. Purposefully blinking, especially during computer use and resting tired eyes are basic steps that can be taken to minimise discomfort. Rubbing one's eyes can irritate them further, so should be avoided. Dry, drafty environments and those with smoke and dust should be avoided. This includes avoiding hair dryers, heaters, air conditioners or fans, especially when these devices are directed toward the eyes. Wearing glasses or directing gaze downward, for example, by lowering computer screens can be helpful to protect the eyes when aggravating environmental factors cannot be avoided. Using a humidifier, especially in the winter, can help by adding moisture to the dry indoor air.

Tear stimulation and supplementation

For mild and moderate cases, supplemental lubrication is the most important part of treatment. Application of artificial tears is sometimes suggested every few hours and may provide temporary relief. Most artificial tear fluids contain mucoadhesive polymers such as hyaluronic acid, cellulose derivatives or polyvinyl alcohol as lubricants. These polymers remain for a prolonged period of time on the ocular surface binding high amounts of water. By the covalent attachment of thiol groups to such polymers, their ocular residence time can be even improved, as thiolated polymers (thiomers) form disulfide bonds with cysteine-rich subdomains of mucus glycoproteins on the ocular surface. Chitosan-N-acetylcysteine containing eye drops showed a significant reduction in symptoms of dry eye syndrome. There are many different types of artificial tear on the market, however, there is no strong evidence to suggest that certain artificial tear formulations are superior to others in treating dry eye.

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Autologous serum eye drops

Eye drops that include autologous serum (serum taken from the same person's blood and used in an eye drop formulation) are sometimes suggested to help supplement natural tears. The composition of serum has similarities to natural tears may mimic natural tears. Evidence supporting this approach shows that autologous serum may be superior to artificial tears at relieving symptoms in the short-term, however, there is no strong evidence that autologous serum eye drops are better than artificial tears or saline solution for long-term symptom relief.

Additional options

Lubricating tear ointments can be used during the day, but they generally are used at bedtime due to poor vision after application. They contain white petrolatum, mineral oil, and similar lubricants. They serve as a lubricant and an emollient. Application requires pulling down the lower eyelid and applying a small amount (0.25 in) inside. Depending on the severity of the condition, it may be applied from every hour to just at bedtime. It should never be used with contact lenses. Specially designed glasses that form a moisture chamber around the eye may be used to create additional humidity.

Medication

Inflammation occurring in response to tears film hypertonicity can be suppressed by mild topical corticosteroids or with topical immunosuppressants such as ciclosporin (Restasis, Vevye).

Elevated levels of tear NGF can be decreased with 0.1% prednisolone.

Topical corticosteroids

Topical corticosteroids are commonly prescribed for those whose dry eye syndrome symptoms may be caused by inflammation and may lead to a small to moderate improvement in dry-eye symptoms when compared to lubricants or artificial tear drop treatment alone. It is not clear if topical corticosteroid treatment leads to an improvement in the quality of the tear film or the quantity of natural tears. There are also risks to consider with long-term use of topical corticosteroid treatment including an increased risk of ocular hypertension, risk of cataract development, and increased risk of eye infections. For people who may benefit from topical corticosteroid treatment for dry eye syndrome, the ideal treatment regime, formulation of the topical preparations, and balance between potential risks of this medication is not clear.

Ciclosporin (cyclosporin)

Topical ciclosporin (topical ciclosporin A, tCSA) 0.05% ophthalmic emulsion is an immunosuppressant that is commonly used to treat symptoms of dry eye syndrome. The drug decreases surface inflammation with the goal of increasing tear production. Some people find relief and report increased tear production, however, evidence of effectiveness from clinical trials is not strong and although some people may find relief, effectiveness may be inconsistent in different people. Ciclosporin A treatment also comes with risks of adverse effects that are generally not serious but include a burning sensation. Ciclosporin should not be used while wearing contact lenses, during eye infections or in people with a history of herpes virus infections. Side effects include burning sensation (common), redness, discharge, watery eyes, eye pain, foreign body sensation, itching, stinging, and blurred vision. Long term use of ciclosporin at high doses is associated with an increased risk of cancer. Cheaper generic alternatives are available in some countries.

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Other medications

Diquafosol, an agonist of the $P2Y_2$ purinergic receptor, is approved in Japan for managing dry eye disease by promoting secretion of fluid and mucin from cells in the conjunctiva, rather than by directly stimulating the lacrimal glands.

Lifitegrast was approved by the US FDA for the treatment of the condition in 2016.

Varenicline (Tyrvaya by Oyster Point Pharma) was approved by the US FDA for the treatment of dry eye disease in October 2021.

Oral n-acetylcysteine (NAC), hyaluronic acid and/or rebamipide-based eye drops may also be effective for dry eyes.

Perfluorohexyloctane (Miebo) was approved for medical use in the United States in May 2023.

Non-Pharmacologic Treatments:

- **Punctal Plugs**: These are inserted into the tear drainage ducts to reduce tear drainage and help retain moisture on the ocular surface.
- **Warm Compresses and Lid Hygiene**: For patients with meibomian gland dysfunction, applying warm compresses and performing lid massages can help improve the quality of the tear film by promoting better meibum secretion.
- **Omega-3 Fatty Acids**: Supplementation with omega-3 fatty acids has been shown to have anti-inflammatory effects and may help improve tear production.

Conserving tears

There are methods that allow both natural and artificial tears to stay longer.

In each eye, there are two puncta – little openings that drain tears into the tear ducts. There are methods to partially or completely close the tear ducts. This blocks the flow of tears into the nose, and thus more tears are available to the eyes. Drainage into either one or both puncta in each eye can be blocked.

Punctal plugs are inserted into the puncta to block tear drainage. It is not clear if punctal plugs are effective at reducing dry eye syndrome symptoms. Punctal plugs are thought to be "relatively safe", however, their use may result in epiphora (watery eyes), and more rarely, serious infection and swelling of the tear sac where the tears drain. They are reserved for people with moderate or severe dry eye when other medical treatment has not been adequate.

If punctal plugs are effective, thermal or electric cauterization of puncti can be performed. In thermal cauterization, a local anesthetic is used, and then a hot wire is applied. This shrinks the drainage area tissues and causes scarring, which closes the tear duct.

Surgical Treatments:

- In refractory cases of DED, more invasive options may be considered. Punctal cautery or surgical closure of the tear ducts may be performed to reduce tear drainage. S
- Surgery. In severe cases of dry eyes, tarsorrhaphy may be performed where the eyelids are partially sewn together. This reduces the palpebral fissure (eyelid separation), ideally leading to a reduction in tear evaporation.

Emerging Therapies:

Innovative approaches in the treatment of DED are actively being explored. Some promising therapies include:

- **Autologous Serum Eye Drops**: Derived from the patient's blood, these eye drops contain growth factors and other components that promote healing of the ocular surface.
- **Stem Cell Therapy**: Research into stem cell-based therapies aims to regenerate damaged epithelial cells on the ocular surface, offering potential long-term relief.
- **Neurostimulation**: Devices that stimulate the trigeminal nerve to enhance tear production are currently being studied.

Prognosis

Keratoconjunctivitis sicca usually is a chronic problem. Its prognosis shows considerable variance, depending upon the severity of the condition. Most people have mild-to-moderate cases, and can be treated symptomatically with lubricants. This provides an adequate relief of symptoms.

When dry eyes symptoms are severe, they can interfere with quality of life. People sometimes feel their vision blurs with use, or severe irritation to the point that they have trouble keeping their eyes open or they may not be able to work or drive.

Prevention

Avoiding refractive surgery (LASIK and PRK), limiting contact lens use, limiting computer screen use, and avoiding environmental conditions can decrease symptoms. Complications can be prevented by use of wetting and lubricating drops and ointments.

Results

Dry Eye Disease (DED) affects a significant portion of the global population, with prevalence estimates ranging from 5% to 50%, especially among older individuals. The condition primarily results from reduced tear production or excessive tear evaporation, often due to meibomian gland dysfunction (MGD) or inflammation. Inflammatory cytokines such as TNF-alpha and IL-1 are elevated in the tear film, contributing to the disease's progression. Diagnostic methods, including the Schirmer test and Tear Breakup Time (TBUT), are commonly used to assess tear production and film stability. Treatment strategies like artificial tears, anti-inflammatory medications (e.g., cyclosporine A and lifitegrast), and punctal plugs improve symptoms and ocular surface health. Emerging therapies, such as autologous serum eye drops and stem cell treatments, show promise. DED significantly impacts patients' quality of life, with many experiencing visual impairment and psychological distress.

Discussion

The impact of DED on patients' quality of life cannot be overstated. Beyond the physical symptoms of dryness, burning, and irritation, many patients experience significant visual disturbances that affect daily activities such as reading, driving, and using digital devices. The psychological toll of DED is also considerable, with studies indicating a higher prevalence of anxiety, depression, and reduced well-being among individuals with severe dry eye symptoms. Addressing these psychological aspects through comprehensive care, including support for coping mechanisms and mental health, should be an integral part of the management plan for DED patients.

Conclusion:

Dry Eye Disease is a prevalent and multifactorial condition that can significantly impair the quality of life. Its pathophysiology involves a complex interaction of decreased tear production, increased

evaporation, and inflammation of the ocular surface. Early diagnosis and appropriate management, including both pharmacological and non-pharmacological treatments, are essential for effective disease control. As research advances, new therapeutic strategies may offer more personalized and effective treatment options for patients with DED.

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