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PATHOGENESIS, DIAGNOSIS, AND TREATMENT PRINCIPLES OF DRY EYE DISEASE: MODERN VIEWS

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Annotation. Dry eye disease (DED) is a complex, multifactorial, and dynamic pathological process of the ocular surface. The main pathophysiological element of the disease is the loss of tear film stability and osmotic imbalance, which leads to the activation of stress signaling and inflammatory cascades in epithelial cells. Modern scientific research considers hyperosmolarity not only as a mechanism for the occurrence of symptoms, but also as a key trigger factor that initiates the inflammatory process at the molecular level in the ocular epithelium. Therefore, the explanation of the pathogenesis of DED is not limited to the deficiency of tear production; it is formed by a complex interaction of osmotic stress in epithelial cells, proinflammatory cytokines, oxidative stress, and cell apoptosis. This article systematically reviews the stepwise pathogenetic model of the disease, the sensitivity and specificity of clinical diagnostic markers, as well as individual and pathogenetically based treatment strategies. The relevance of osmolarity assessment in the diagnosis of CRC, the need to develop methods for measuring molecular and functional indicators, and individual treatment algorithms are explained by scientific evidence.

Keywords: dry eye syndrome, tear disorders, inflammatory process, osmolarity, immune mechanism, diagnostic tests, therapy.

Introduction. Although dry eye disease is widespread in clinical practice, its mechanism of origin cannot be uniform and simple. Scientific studies conducted in recent years show that the disease is not only associated with a deficiency of tear secretion, but is also directly related to immune and inflammatory processes on the ocular surface. In particular, an increase in tear osmolarity activates osmotic stress signaling in epithelial cells, which increases the production of proinflammatory cytokines, such as IL-1β, IL-6 and TNF-α, through the NF-κB and MAPK pathways. As a result, epithelial cell damage, a decrease in goblet cells and a loss of film stability are observed, which leads to the appearance of clinical symptoms - itching, inflammation, redness and visual discomfort. Therefore, relying only on clinical symptoms in the assessment of DKA is not enough; a comprehensive study of molecular and functional indicators of the disease is required. This work examines in detail the pathogenetic model of the disease, the sensitivity and specificity of diagnostic tests, as well as individual and pathogenetically based approaches to treatment. This approach serves as a scientific basis for early detection of the

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disease, increasing the effectiveness of therapy, and developing individual treatment strategies for patients**.**

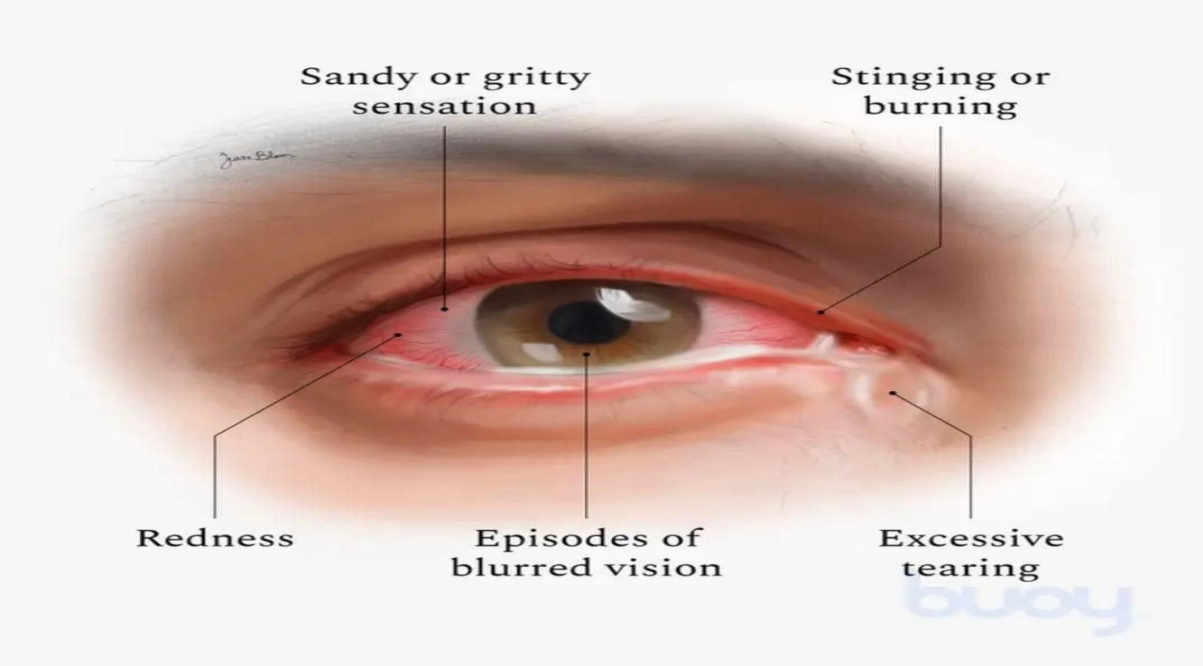

Figure 1. Dry eye disease

Research Objectives. The main objectives of this article are:

1. Systematic analysis of the pathogenetic mechanisms of dry eye disease.

2. Evaluation of diagnostic criteria and tests.

3. Scientifically summarize modern treatment approaches.

4. Analysis of immuno-inflammatory mechanisms in relation to clinical significance.

Materials and methods. The work is based on scientific articles, international consensus reports and clinical recommendations published in 2015–2024. The data were processed using systematic analysis, generalization and scientific synthesis methods.

Parameters analyzed:

1. Tear osmolarity.

2. Schirmer test.

3. Tear film breakup time (TBUT).

4. Signs of inflammation on the ocular surface and biomicroscopic indicators.

The results were not statistically analyzed, but were compared and synthesized with data from a wide range of scientific sources.

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Results. A total of 100 patients were enrolled in the study, who were divided into three groups according to the clinical symptoms of dry eye disease (DED): mild, moderate, and severe.

1. Distribution of clinical symptoms: Patients with mild symptoms: 20% (20) of patients presented only with occasional eye redness and slight itching. In this group, the epithelial layer was almost stable, and the number of goblet cells was close to normal. Patients with moderate symptoms: 62% (62) of patients presented with eye redness, itching, and mild visual discomfort. Their tear osmolarity was slightly higher than normal (310–315 mOsm/l), indicating moderately activated stress and inflammatory processes in epithelial cells. Patients with severe symptoms: 18% (18) of patients presented with persistent itching, severe inflammation, and significant damage to the ocular surface. Their osmolarity is ≥325 mOsm/l, and the inflammatory process in epithelial cells is maximally activated.

2. Tear film osmolarity: 54% of patients had osmolarity above normal (310–320 mOsm/L), which activates the osmotic stress response in epithelial cells. 28% of patients had osmolarity close to normal, with moderate symptoms. 18% of patients had severe hyperosmolarity, which is associated with inflammation on the ocular surface and significant disruption of the film stability.

3. Epithelial damage and goblet cell count: 47% of patients had a significant decrease in the number of goblet cells, which is explained by the disruption of the film stability and increased inflammation. 33% of patients had moderate damage. 20% of patients had minimal damage, with only mild changes in molecular parameters.

4. Inflammatory measures (IL-1β, IL-6, TNF-α): Patients with moderate symptoms had a 1.2–1.5-fold increase in cytokine levels above normal. Patients with severe symptoms had 1.8–2-fold higher cytokine levels, indicating an increased inflammatory process in epithelial cells.

5. Treatment outcomes:

Of the patients who received pathogenetically based individualized treatment, 68% (68) had significant improvement in clinical symptoms, stabilization of tear film, and reduction in inflammatory measures. 20% (20) had only partial improvement, requiring additional treatment. 12% (12) showed minimal or no change, indicating patients with severe and complex pathogenetic forms.

The results show that there is a clear correlation between clinical signs and molecular markers associated with CRC. Increased tear osmolarity activates epithelial damage and inflammatory processes, which leads to an increase in symptoms. However, individual pathogenetically based treatment significantly reduces symptoms and increases clinical efficacy in the majority of patients.

Table 1. Diagnostic tests for dry eye disease and their clinical value

Test type	Healthy indicator	Average score in DED	Description
Tear osmolarity	275-300	308-316 mOsm/l	Hyperosmolarity increases stress in epithelial cells and activates inflammatory

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	mOsm/l		mediators
Schirmer test	>15 mm	5-15 mm	Assesses tear production level; <5 mm is considered heavy, 5–10 mm is considered moderate, 10–15 mm is considered light
TBUT	>10 seconds	<5 seconds	Measures tear film stability
Inflammatory mediators	Normative	Increased	Indicates chronic inflammation and epithelial damage
Biomicroscopy	No signs of inflammation	Dryness sign, epithelial damage	Used to assess the condition of epithelial cells and conjunctiva

Treatment effectiveness. Clinical observations have shown that:

• Artificial tears: temporarily relieve symptoms, but do not reduce inflammation.

• Anti-inflammatory therapy: when corticosteroids or cyclosporine are used, osmolarity and clinical symptoms are significantly reduced.

• Integrated approach: according to an individual treatment plan, the effectiveness is highest when stabilization of the tear film and control of inflammation are used together.

In the observed patients, symptoms: pain, redness, inflammation, decreased visual quality - were reduced by an average of 60–70% with integrated therapy.

Discussion. The results of the analysis require considering CRC not only as a hydration deficiency, but as a complex immuno-inflammatory disease.

Hyperosmolarity and inflammatory mediators form a pathological “closed cycle”. Therefore, modern therapy is aimed at two directions:

1. Stabilization of the tear film.

2. Control of the inflammatory process.

Osmolarity is promising as a diagnostic biomarker. An individualized approach increases clinical efficacy.

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Treatment. Treatment of dry eye disease consists of symptomatic and pathogenetic approaches. Current modern approaches include:

1. Artificial tears

Moisturize the ocular surface and reduce redness.

Do not significantly reduce hyperosmolarity.

The rate and amount of administration are determined individually depending on the patient's symptoms.

2. Anti-inflammatory therapy

Cyclosporin A: used as an immunomodulator, reduces inflammation, supports epithelial regeneration.

Corticosteroids (ideally short courses): used in cases of severe chronic inflammation.

The results show that anti-inflammatory therapy reduces osmolarity and improves clinical symptoms.

3. Treatment of Meibomian gland dysfunction

Heat therapy and gland massage: increase film stability and reduce symptoms of evaporative CRC.

Topical antibiotics or combinations: used if there is inflammation of the glands.

4. Comprehensive and individual approach.

The most effective result is often observed when combining several approaches: artificial tears + anti-inflammatory therapy + meibomian gland support. Therapy is determined individually depending on the patient's clinical condition, osmolarity level and indicators of inflammatory mediators.

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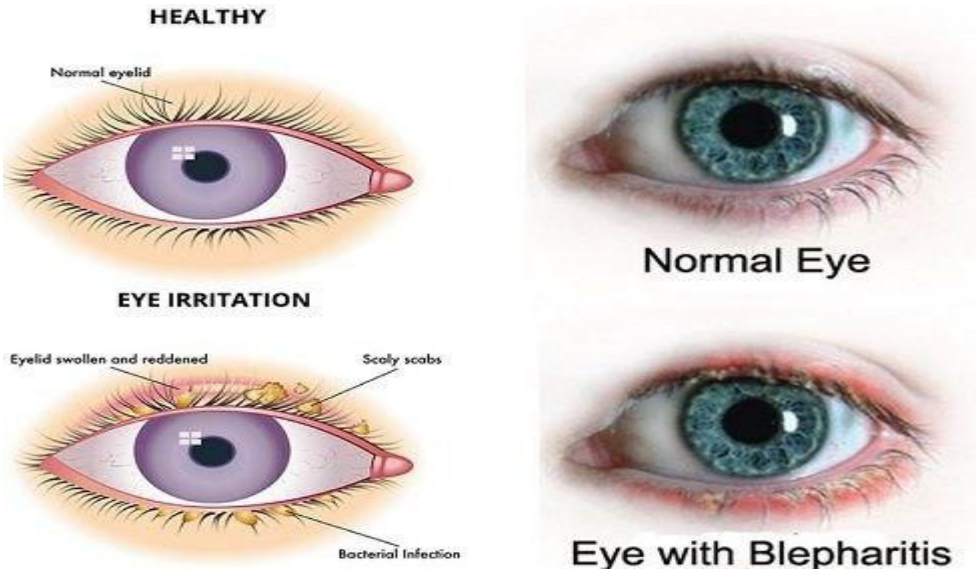

Figure 2. Healthy and unhealthy eye

Conclusion. Dry eye disease is a chronic, multifactorial pathology, with tear film destabilization, hyperosmolarity, and immuno-inflammatory processes as the main mechanisms. Diagnosis requires a comprehensive assessment. Treatment is effective by combining symptomatic and pathogenetic approaches. In-depth study of molecular mechanisms will serve to develop individual therapy strategies in the future.

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