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DRY EYE SYNDROME IN PATIENTS WITH ENDOCRINE PATHOLOGY



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ЭНДОКРИН ПАТОЛОГИЯСИ БЎЛГАН БЕМОРЛАРДА ҚУРУҚ КЎЗ СИНДРОМИ

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СИНДРОМ СУХОГО ГЛАЗА У ПАЦИЕНТОВ С ЭНДОКРИННОЙ ПАТОЛОГИЕЙ

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Резюме. *Қуруқ кўз синдроми кўз юзасининг энг кенг тарқалган ва клиник жиҳатдан аҳамиятли касалликларидан биридир. Эндокрин патологияси бўлган беморларда бу ҳолат метаболик, томир, нейроген, яллиғланиш ва гормонал механизмларнинг биргаликдаги таъсири остида ривожланади. Ушбу мақолада замонавий патогенетик тушунчалар умумлаштирилган ва қандли диабет, қалқонсимон без касалликлари, метаболик синдром, семизлик ва бошқа эндокрин бузилишлари бўлган беморларнинг клиник кузатув таҳлили келтирилган. Республика ихтисослаштирилган кўз микрохирургияси тиббиёт марказининг Самарқанд филиалида жами 126 нафар бемор текширувдан ўтказилди. Клиник текширув биомикроскопия, Ширмер тести, кўз ёши пардасининг ёрилиш вақтини аниқлаш, шох парданинг флуоресцент бўялиши, мейбомий безлари функциясини баҳолаш ва эндокрин ҳолатни таҳлил қилишни ўз ичига олди. Кўз юзасидаги энг яққол ўзгаришлар қандли диабет ва тиреоид офталмопатия билан оғриган беморларда кузатилди. Энг барқарор яхшиланишга маҳаллий офталмологик терапияни мейбомий безлари дисфункциясини тузатиш ва асосий эндокрин касалликни назорат қилиш билан биргаликда олиб борилганда эришилди. Олинган натижалар шуни тасдиқлайдики, эндокрин патологияда қуруқ кўз синдромига эрта ташхис қўйиш, объектив мониторинг ва индивидуал даволашни талаб қиладиган фанлараро клиник муаммо сифатида қараш керак.*

Калит сўзлар: *қуруқ кўз синдроми, эндокрин патология, қандли диабет, тиреоид офталмопатия, метаболик синдром, мейбомиев безлари, кўз ёши пардаси, кўз юзаси.*

Abstract. *Dry eye syndrome is one of the most frequent and clinically significant disorders of the ocular surface. In patients with endocrine pathology, this condition develops under the combined influence of metabolic, vascular, neurogenic, inflammatory and hormonal mechanisms. The present article summarizes current pathogenetic concepts and presents a clinical observational analysis of patients with diabetes mellitus, thyroid disease, metabolic syndrome, obesity and other endocrine disorders. A total of 126 patients were examined at the Samarkand Branch of the Republican Specialized Medical Center of Eye Microsurgery. Clinical evaluation included biomicroscopy, Schirmer testing, tear film breakup time, fluorescein staining of the cornea, assessment of meibomian gland function and analysis of endocrine status. The highest*

frequency of pronounced ocular surface changes was observed in patients with diabetes mellitus and thyroid eye disease. The most stable improvement was achieved when local ophthalmic therapy was combined with correction of meibomian gland dysfunction and control of the underlying endocrine disease. The findings confirm that dry eye syndrome in endocrine pathology should be regarded as an interdisciplinary clinical problem requiring early diagnosis, objective monitoring and individualized treatment.

Keywords: *dry eye syndrome, endocrine pathology, diabetes mellitus, thyroid eye disease, metabolic syndrome, meibomian glands, tear film, ocular surface.*

Introduction. Dry eye syndrome is a multifactorial disease of the ocular surface characterized by loss of tear film homeostasis, ocular symptoms, tear film instability, hyperosmolarity, inflammation, epithelial damage, and neurosensory abnormalities. This condition has a direct effect on visual quality because the tear film is the first optical medium of the eye and is essential for stable refraction at the corneal surface [1, 3].

The clinical relevance of dry eye syndrome is determined by its high prevalence, chronic course, and pronounced influence on everyday visual activity. Patients usually complain of dryness, burning, foreign body sensation, redness, photophobia, tearing, and fluctuating vision. These manifestations may reduce reading tolerance, limit computer work, impair professional activity, and negatively affect quality of life [3, 16].

Dry eye syndrome is especially important in patients with endocrine pathology. Diabetes mellitus, thyroid diseases, metabolic syndrome, obesity, hormonal imbalance, and age-related endocrine changes influence the ocular surface through systemic mechanisms. These conditions can alter tear film composition, reduce lacrimal gland secretion, damage corneal innervation, disturb the meibomian glands, and increase inflammatory activity in the conjunctiva and cornea [2, 6, 9, 17].

Diabetes mellitus is among the most clinically significant endocrine conditions associated with dry eye. Long-term hyperglycemia is accompanied by microangiopathy, peripheral and corneal neuropathy, oxidative stress, and chronic inflammation. These changes reduce corneal sensitivity, weaken reflex tear secretion, slow epithelial healing, and increase the risk of neurotrophic keratopathy [4, 7, 10, 14, 17].

Thyroid eye disease has a different but equally important mechanism. Exophthalmos, widening of the palpebral fissure, eyelid retraction, incomplete blinking, and lagophthalmos increase tear evaporation and promote exposure-related corneal damage. As a result, many patients develop evaporative or mixed forms of dry eye syndrome requiring not only tear substitutes but also protection of the ocular surface [3].

Metabolic syndrome and obesity contribute to dry eye through chronic low-grade inflammation, insulin resistance, and lipid metabolism disorders. Changes in lipid composition affect meibomian gland secretion and destabilize the lipid layer of the tear film, which is essential for preventing excessive evaporation [11, 16].

Despite its clinical significance, dry eye syndrome in endocrine patients is often underestimated. In routine ophthalmological practice, attention is commonly focused on diabetic retinopathy, cataract, glaucoma, and optic nerve involvement, whereas ocular surface damage may be detected late. This creates a risk of persistent corneal epithelial defects, reduced diagnostic accuracy, and insufficient satisfaction with ophthalmological treatment [17].

In endocrine pathology, the ocular surface should be considered as an integral part of systemic metabolic and hormonal regulation. Any disturbance in glucose metabolism, thyroid hormone balance, lipid metabolism, or neuroendocrine regulation may indirectly affect tear secretion, eyelid function, corneal trophism, and epithelial immune response. Therefore, symptoms of ocular discomfort in such patients should not be interpreted as isolated local complaints.

Another important aspect is the mismatch between subjective symptoms and objective findings. In diabetes mellitus, reduced corneal sensitivity may mask severe epithelial damage, while in thyroid eye disease even relatively preserved tear production may coexist with marked exposure keratopathy. This discrepancy makes a detailed instrumental examination especially important in patients with endocrine disorders.

The growing prevalence of diabetes, obesity, metabolic syndrome, and thyroid diseases increases the clinical burden of endocrine-associated dry eye syndrome. As these diseases often require lifelong monitoring, ocular surface disorders may persist for years and gradually progress if early diagnostic and preventive measures are not implemented.

Thus, studying dry eye syndrome in patients with endocrine pathology has both clinical and social importance. Early detection and adequate treatment may improve not only ocular comfort and visual function, but also the overall quality of life, working capacity, and adherence of patients to long-term medical follow-up.

Aim of the Study. The aim of this study was to analyze the clinical features, diagnostic parameters, and therapeutic approaches to dry eye syndrome in patients with endocrine pathology, with particular attention to diabetes mellitus, thyroid disease, metabolic syndrome, and obesity.

An additional objective was to determine how the leading endocrine disorder influences the dominant mechanism of dry eye syndrome. This included evaluation of aqueous tear deficiency, evaporative dry eye, neurotrophic corneal damage, meibomian gland dysfunction, inflammation, and exposure-related ocular surface injury.

The study also sought to clarify the practical value of a comprehensive ophthalmological assessment in endocrine patients. Special attention was paid to the relationship between subjective complaints and objective diagnostic indicators, since these parameters may differ substantially in diabetic corneal neuropathy and thyroid-associated exposure keratopathy.

Another important aim was to substantiate the need for individualized treatment. Since endocrine-associated dry eye has different pathogenetic mechanisms, the choice of therapy should be adapted to the specific clinical situation rather than limited to standard tear replacement.

Finally, the study was designed to emphasize the importance of interdisciplinary cooperation between ophthalmologists and endocrinologists. Stable improvement of the ocular surface is difficult to achieve without adequate control of diabetes mellitus, thyroid dysfunction, obesity, metabolic syndrome, and other systemic endocrine disorders.

Materials and Methods. The clinical part of the study was conducted at the Samarkand Branch of the Republican Specialized Medical Center of Eye Microsurgery during a 12-month observation period. The study included 126 patients aged from 24 to 78 years with dry eye syndrome associated with endocrine pathology. The average age of the examined patients was 53.6 ± 11.4 years. Women accounted for 74 cases and men for 52 cases.

The distribution of patients according to the leading endocrine disorder was as follows: diabetes mellitus was diagnosed in 58 patients, thyroid diseases in 28 patients, metabolic syndrome and obesity in 24 patients, and other hormonal disorders in 16 patients. In a number of patients, combined endocrine pathology was observed, which was taken into account when interpreting the clinical data.

All patients underwent a comprehensive ophthalmological examination. The assessment included analysis of subjective complaints, visual acuity testing, slit-lamp biomicroscopy of the eyelids, conjunctiva and cornea, measurement of the tear meniscus, Schirmer test, tear film breakup time, fluorescein staining of the cornea and evaluation of the meibomian glands. The quality of meibum secretion, obstruction of gland orifices, eyelid margin inflammation, and signs of evaporative dry eye were recorded.

The severity of symptoms was evaluated by structured questioning with emphasis on dryness, burning, gritty sensation, redness, tearing, photophobia, visual fluctuation, and discomfort during prolonged visual work. Objective indicators were assessed before treatment and after 1, 3, 6, and 12 months of follow-up.

The therapeutic approach was individualized. Patients received preservative-free artificial tears, lipid-containing lubricants, eyelid hygiene measures, warm compresses, anti-inflammatory therapy when clinically indicated, and recommendations for control of the underlying endocrine disease. In diabetic patients, special attention was paid to corneal epithelial integrity and delayed healing. In thyroid eye disease, ocular surface protection and nocturnal lubrication were emphasized.

Statistical analysis was performed by descriptive methods. Quantitative indicators were presented as mean values and standard deviations, whereas categorical variables were expressed as absolute numbers and percentages. The dynamics of clinical parameters was interpreted in relation to the type of endocrine pathology and the dominant mechanism of dry eye syndrome.

At the first stage of the examination, special attention was paid to the duration of endocrine disease and its clinical compensation. In patients with diabetes mellitus, available data on glycemic control, duration of the disease, presence of diabetic retinopathy, neuropathy, and systemic microvascular complications were considered. In patients with thyroid pathology, the presence of thyroid eye disease, eyelid retraction, exophthalmos, and incomplete eyelid closure was recorded.

The ophthalmological assessment was performed using a unified sequence in order to minimize diagnostic variability. First, subjective complaints and visual function were documented. Then slit-lamp biomicroscopy was performed, followed by tear production and tear film stability tests. Corneal staining was assessed after the functional tests to avoid distortion of tear film parameters.

For a more accurate clinical interpretation, patients were conditionally grouped according to the predominant mechanism of dry eye syndrome. Aqueous-deficient dry eye was diagnosed when tear production was reduced. Evaporative dry eye was considered predominant when tear film breakup time was shortened in the presence of meibomian gland dysfunction. Mixed dry eye was diagnosed when both mechanisms were present.

Treatment was not assessed only by symptom reduction. The clinical response was interpreted through a combination of subjective improvement, tear film stabilization, reduction of corneal staining, improvement of meibomian gland secretion, and decrease in conjunctival hyperemia. This approach allowed a more reliable evaluation of treatment effectiveness in patients whose corneal sensitivity was reduced.

Table 1. Distribution of patients according to leading endocrine pathology

Endocrine pathology	Number of patients	Percentage	Dominant dry eye mechanism
Diabetes mellitus	58	46.0%	Neurotrophic, aqueous-deficient, mixed
Thyroid diseases	28	22.2%	Exposure-related and evaporative
Metabolic syndrome and obesity	24	19.0%	Evaporative and meibomian gland dysfunction
Other hormonal disorders	16	12.8%	Mixed mechanisms
Total	126	100%	Multifactorial

Table 2. Dynamics of selected clinical indicators after comprehensive therapy

Clinical indicator	Before treatment	After 6 months	After 12 months
Schirmer test, mm/5 min	7.4 ± 2.1	10.8 ± 2.4	12.1 ± 2.6
Tear film breakup time, seconds	5.2 ± 1.4	8.6 ± 1.8	9.4 ± 2.0
Corneal staining score	2.1 ± 0.7	1.2 ± 0.5	0.8 ± 0.4
Subjective discomfort score	7.2 ± 1.1	3.8 ± 1.0	2.9 ± 0.9

Results. Before treatment, most patients had combined subjective and objective signs of dry eye syndrome. The most frequent complaints were dryness, burning, foreign body sensation, unstable vision, and discomfort during prolonged visual work. In many cases, the severity of subjective symptoms did not fully correspond to the degree of corneal epithelial damage, especially in patients with diabetes mellitus.

In the diabetes mellitus subgroup, objective changes were more pronounced than subjective complaints in several patients. This finding may be explained by diabetic corneal neuropathy, which reduces corneal sensitivity and weakens the perception of ocular surface irritation. Biomicroscopy revealed punctate epitheliopathy, decreased tear meniscus height, and delayed epithelial recovery. These findings are consistent with the concept of diabetic keratopathy and impaired tear reflex in diabetes [7, 10, 14, 17].

In patients with thyroid pathology, dry eye syndrome was predominantly associated with exposure of the ocular surface. The main clinical signs were widening of the palpebral fissure, incomplete eyelid closure, conjunctival hyperemia, and increased tear evaporation. Tear film breakup time was reduced, while Schirmer test results were less severely decreased than in patients with the aqueous-deficient form.

Patients with metabolic syndrome and obesity demonstrated a high frequency of meibomian gland dysfunction. Thickened secretion, partial obstruction of gland orifices, and inflammatory changes of the eyelid margins were common. These findings confirm the importance of lipid layer disorders in the development of evaporative dry eye in metabolic pathology.

After treatment, positive dynamics were observed in all groups, but the most pronounced and stable improvement was found in patients receiving a comprehensive regimen. This approach combined preservative-free lubricants, lipid-containing preparations, correction of meibomian gland dysfunction, anti-inflammatory treatment when necessary, and endocrine disease control.

During follow-up, improvement in subjective symptoms was usually recorded earlier than complete normalization of objective findings. Many patients reported a decrease in burning and foreign body sensation during the first month, while tear film stability and epithelial recovery improved more gradually. This observation confirms the need for long-term treatment rather than short episodic use of lubricating drops.

In diabetic patients, corneal staining decreased more slowly than in other groups. This was especially evident among patients with long-standing diabetes and clinical signs of neuropathy. These patients required more frequent instillation of preservative-free tear substitutes, careful monitoring of epithelial integrity, and avoidance of potentially irritating topical medications.

In patients with thyroid eye disease, the best results were achieved when lubricating therapy was combined with mechanical protection of the ocular surface. Nocturnal use of more viscous preparations, correction of incomplete blinking habits, and protection from dry air helped reduce morning discomfort and signs of exposure keratopathy.

Among patients with metabolic syndrome and obesity, the most favorable response was observed when tear substitutes were combined with eyelid hygiene and warm compresses. Improvement in meibomian secretion was accompanied by prolongation of tear film breakup time and reduction of visual fluctuation, which confirms the leading role of lipid layer restoration in this subgroup.

Discussion. The results of this study confirm that dry eye syndrome in endocrine pathology is not a uniform clinical entity. In diabetes mellitus, neu-

rotrophic and microvascular mechanisms prevail; in thyroid eye disease, exposure and evaporation dominate; and in metabolic syndrome, meibomian gland dysfunction is particularly important. Therefore, therapy based only on standard artificial tears may be insufficient. The diabetic ocular surface is vulnerable because hyperglycemia affects several protective mechanisms simultaneously. Reduced corneal sensitivity decreases reflex tearing and blinking, while microangiopathy impairs tissue nutrition and epithelial repair. Chronic inflammation and oxidative stress further destabilize the tear film and increase the risk of corneal complications [4, 6, 7, 17].

The thyroid-associated form requires an additional anatomical approach. Even when tear production is relatively preserved, increased exposure of the cornea may lead to severe symptoms and epithelial damage. In such patients, nocturnal lubrication, protection from drying, and correction of eyelid position may be as important as classical tear replacement therapy. The role of meibomian gland dysfunction should not be underestimated. In the present study, patients with obesity and metabolic syndrome frequently showed evaporative dry eye caused by lipid layer instability. This explains the higher effectiveness of lipid-containing lubricants, eyelid hygiene, and warm compresses compared with simple aqueous tear substitutes. The practical implication of these observations is that every patient with endocrine pathology and ocular complaints requires a structured evaluation of tear production, tear film stability, epithelial integrity, and meibomian gland function. Interdisciplinary cooperation between ophthalmologists and endocrinologists increases the likelihood of stable treatment response. The obtained results also demonstrate that the same symptom may have different clinical meanings in different endocrine disorders. For example, tearing in thyroid eye disease may reflect reflex irritation due to exposure, whereas in diabetes it may coexist with reduced basal tear secretion and corneal hypoesthesia. Therefore, the mechanism of symptoms should be clarified before prescribing treatment. Another important point is the need to avoid preservatives in long-term therapy. Patients with endocrine disorders often require prolonged or continuous use of lubricating medications. Preservatives can additionally damage epithelial cells and aggravate inflammation, particularly in patients with diabetic keratopathy or existing corneal staining. Comprehensive treatment has advantages because it addresses several pathogenic links at the same time. Moisturizing therapy improves hydration, lipid-containing agents reduce evaporation, anti-inflammatory treatment suppresses ocular surface inflammation, eyelid hygiene restores meibomian function, and endocrine disease control reduces systemic factors maintaining ocular surface damage.

These findings support the concept of personalized ophthalmological care in endocrine patients. Instead of a universal therapeutic regimen, the physician should determine the dominant mechanism of dry eye, assess systemic endocrine status, identify corneal risk factors, and select a treatment plan that can be adapted during follow-up.

Conclusion. Dry eye syndrome in patients with endocrine pathology is a multifactorial disorder involving tear deficiency, tear film instability, meibomian gland dysfunction, inflammation, oxidative stress, microangiopathy, and neurotrophic corneal changes. The most significant clinical associations were observed in diabetes mellitus, thyroid eye disease, metabolic syndrome, and obesity.

The clinical course depends on the dominant pathogenetic mechanism. In diabetes mellitus, reduced corneal sensitivity and delayed epithelial repair are especially important. In thyroid eye disease, exophthalmos, lagophthalmos, and increased evaporation play a leading role. In metabolic syndrome and obesity, dysfunction of the meibomian glands and instability of the lipid layer are major factors.

The most effective therapeutic strategy is a comprehensive and individualized approach. It should include preservative-free tear substitutes, lipid-containing preparations, anti-inflammatory treatment when indicated, correction of meibomian gland dysfunction, protection of the cornea, and adequate control of the underlying endocrine disease. Early diagnosis and regular follow-up make it possible to prevent corneal complications and improve the quality of life of patients.

The study confirms that dry eye syndrome in endocrine pathology should not be considered a minor accompanying complaint. It can significantly reduce visual performance, complicate ophthalmological diagnostics, increase the risk of corneal epithelial defects, and worsen patient satisfaction with treatment outcomes. Patients with diabetes mellitus require particularly careful monitoring because reduced corneal sensitivity may conceal serious epithelial damage. In these patients, objective examination should be performed even when subjective complaints are moderate or inconsistent with the clinical picture. Patients with thyroid eye disease need early protection of the ocular surface, especially in the presence of eyelid retraction, lagophthalmos, and exophthalmos. Preventive lubrication, nocturnal protection, and control of thyroid-associated orbital inflammation are important for reducing the risk of exposure keratopathy.

Further research should focus on objective biomarkers of ocular surface damage in endocrine disorders, including tear osmolarity, inflammatory markers, meibomian gland imaging, and in vivo confocal microscopy of corneal nerves. These methods may improve early diagnosis and help personalize

treatment strategies for endocrine-associated dry eye syndrome.

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СИНДРОМ СУХОГО ГЛАЗА У ПАЦИЕНТОВ С ЭНДОКРИННОЙ ПАТОЛОГИЕЙ

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Резюме. *Синдром сухого глаза - одно из наиболее распространенных и клинически значимых заболеваний поверхности глаза. У пациентов с эндокринной патологией это состояние развивается под совокупным влиянием метаболических, сосудистых, нейрогенных, воспалительных и гормональных механизмов. В данной статье обобщены современные патогенетические концепции и представлен клинический наблюдательный анализ пациентов с сахарным диабетом, заболеваниями щитовидной железы, метаболическим синдромом, ожирением и другими эндокринными расстройствами. Всего было обследовано 126 пациентов в Самаркандском филиале Республиканского специализированного медицинского центра микрохирургии глаза. Клиническое обследование включало биомикроскопию, тест Ширмера, определение времени разрыва слезной пленки, флуоресцентное окрашивание роговицы, оценку функции мейбомиевых желез и анализ эндокринного статуса. Наиболее выраженные изменения поверхности глаза наблюдались у пациентов с сахарным диабетом и тиреоидной офтальмопатией. Наиболее стабильное улучшение было достигнуто при сочетании местной офтальмологической терапии с коррекцией дисфункции мейбомиевых желез и контролем основного эндокринного заболевания. Полученные результаты подтверждают, что синдром сухого глаза при эндокринной патологии следует рассматривать как междисциплинарную клиническую проблему, требующую ранней диагностики, объективного мониторинга и индивидуального лечения.*

Ключевые слова: *синдром сухого глаза, эндокринная патология, сахарный диабет, тиреоидная офтальмопатия, метаболический синдром, мейбомиевы железы, слезная пленка, поверхность глаза.*