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Research Article

The Effect of Eyelid Skin Laxity on Dry Eye Disease

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Abstract

Objective: Eyelid skin laxity is an inevitable consequence of aging, and dry eye disease has a high incidence in the middle-aged and elderly population. This study aims to explore the mechanism by which eyelid laxity affects meibomian gland function and dry eye symptoms, evaluate the impact of eyelid laxity on tear film stability, tear secretion volume and inflammatory response, and provide new clinical evidence for the diagnosis and intervention of dry eye disease.

Methods: A total of 200 subjects in our hospital from January 2023 to December 2024 were selected and divided into a control group of 100 subjects without eyelid laxity symptoms and an experimental group of 100 subjects with different degrees of eyelid laxity. The experimental group was further subdivided into mild, moderate and severe laxity subgroups. Correlation analysis and multiple regression analysis were performed by measuring indicators including Tear Film Break Up Time (TBUT), Schirmer I test (SIt), Tear Meniscus Height (TMH), tear osmolarity and the level of inflammatory marker IL-6 in tears.

Results: SPSS 22.0 was used for data analysis of the samples. Measurement data were expressed as mean ± standard deviation (x±s), and one-way analysis of variance was used for inter-group comparison. The conclusions were as follows: with the aggravation of eyelid laxity, the objective indicators reflecting tear film stability, tear secretion volume, tear osmolarity and local inflammatory state all showed a consistent worsening trend. The specific results were as follows:

TBUT decreased significantly with the aggravation of eyelid laxity: the mild laxity group was (7.87±0.12) seconds, the moderate group was (5.96±0.35) seconds, and the severe group decreased to (3.95±0.47) seconds, all significantly lower than that of the control group [(9.72±0.69) seconds, P<0.01].

The basic tear secretion volume of the control group was (12.0±1.1) mm, while those of the mild, moderate and severe laxity groups were (8.1±0.9) mm, (5.3±1.2) mm and (4.8±0.8) mm, respectively (P<0.01).

The tear osmolarity of the control group was (298.45±7.98) mOsm/L, while those of the mild, moderate and severe laxity groups were (308.12±7.94) mOsm/L, (315.21±4.98) mOsm/L and (323.33±6.09) mOsm/L, respectively, with a significant increase in osmolarity (P<0.01).

The IL-6 level of the control group was (3.98±2.01) pg/ml, while those of the mild, moderate and severe laxity groups were (11.78±3.70) pg/ml, (25.17±4.08) pg/ml and (38.63±7.45) pg/ml, respectively. The IL-6 level increased significantly with the aggravation of eyelid laxity, and the difference between the control group and the severe laxity group was significant (P<0.01).

Conclusion: Eyelid laxity has a significant impact on tear film stability, tear secretion volume and inflammatory level, and may be a potential factor for the aggravation of dry eye symptoms. The significant changes in tear film break-up time and tear secretion volume are closely related to eyelid laxity. Early screening and intervention for patients with eyelid laxity may help prevent the deterioration of dry eye symptoms.

Keywords: Eyelid Laxity; Meibomian Gland Function; Onset of Dry Eye Disease; Treatment.

INTRODUCTION

In the field of ophthalmology, the main functions of the eyelids are to protect the eyeball, maintain the normal movement of the eyelids and the normal drainage of tears. Any structural abnormality of the eyelids may affect these functions and increase the risk of ocular diseases. The skin around the eyes is the thinnest skin of the whole body and one of the parts where aging manifestations appear first on the entire face. With the increase of age, the eyelid tissue gradually loses its elasticity, and the relaxation of the eyelid skin and muscles (or blepharoptosis) occurs, which is a ptosis phenomenon caused by the relaxation of eyelid tissue, including bone collapse, muscle atrophy, and decreased elasticity of subcutaneous tissue. Skin aging is common in the middle-aged and elderly, which may be caused by the degradation of skin elastic fibers and the loss of skin collagen.

The meibomian glands are a group of glands in the eyelids responsible for secreting the lipid layer. Their secretion, meibum, plays a crucial role in the formation and stability of the tear film, mainly by providing the lipid components of the tear film to prevent the evaporation of water on the ocular surface and maintain ocular moisture and balance. Meibomian Gland Dysfunction (MGD) is a chronic, diffuse meibomian gland lesion whose pathological basis is mainly the obstruction of the terminal duct of the meibomian gland and/or the qualitative or quantitative change of meibum secretion [1]. Clinically, it can cause tear film abnormalities, irritative symptoms such as ocular redness and pain, and ocular surface inflammatory reactions. In severe cases, it can lead to ocular surface damage and affect visual function, being a common type of dry eye disease. The normal operation of meibomian gland function depends on the anatomical structure and dynamic activity of the eyelids. The contraction of the orbicularis oculi muscle, especially the Riolan muscle,

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exerts pressure on the meibomian glands, which helps to expel meibum from the glands to the ocular surface [2]. The relaxation of eyelid skin will cover the lid margin and the orifices of meibomian glands, thereby affecting the expulsion of meibum, which may significantly impair the normal physiological function of the meibomian glands and then lead to the atrophy of the meibomian glands, thus exacerbating dry eye symptoms. The relaxation of eyelid skin not only changes the normal tension of the eyelids but also may cause the compression or obstruction of the meibomian gland ducts, resulting in the obstruction of meibum secretion. Eyelid laxity may also alter the contact time between the eyelids and the cornea, leading to an imbalance in tear secretion and excretion, accelerating tear evaporation, and thus aggravating the symptoms of dry eye disease. The importance of meibomian gland function and eyelid laxity (especially that caused by aging) for ocular health is receiving increasing attention. The complex relationship between eyelid laxity and meibomian gland dysfunction reveals its potential importance in the pathogenesis of dry eye disease.

Dry eye disease is a multifactorial, symptomatic chronic ocular surface disease caused by abnormal tear quality, quantity or ocular surface dynamics, which is an imbalance of the ocular surface tear film and/or ocular surface microcirculation [3]. One of its core pathological features is the incompleteness of the tear film lipid layer, hyperosmolarity, ocular surface inflammation and damage, as well as abnormal neural sensation. A normal tear film consists of three layers: the inner mucin layer, the middle aqueous layer and the outer lipid layer. Dry eye disease is characterized by tear film instability and tear hyperosmolarity. The stability of the tear film is closely related to the stability of the lipid layer. Meibum is secreted by the meibomian glands. When the composition or distribution of meibum is abnormal, the lipid layer of the tear film is damaged, and tear evaporation is accelerated, which can cause dry eye disease. Studies have shown that about 60-80% of dry eye disease cases are associated with Meibomian Gland Dysfunction (MGD). The main manifestations of MGD include lipid layer deficiency, meibomian gland duct obstruction and inflammatory reactions, all of which may be aggravated by eyelid laxity.

The relaxation of eyelid skin can lead to the accumulation of dirt such as sebum and lipid plugs in the skin folds, thus affecting eyelid hygiene and potentially inducing inflammation of the meibomian gland orifices. In the long term, meibomian gland obstruction, decreased secretory capacity and long-term structural loss of the glands may occur [4]. The changes in physical mechanical pressure caused by eyelid laxity may induce a chronic inflammatory response of the meibomian glands, which in turn causes degenerative changes in the gland structure. Eyelid laxity may affect the integrity and stability of the tear film by altering the physical environment and functional state of the meibomian glands. The proposal of this hypothesis provides a new perspective for the study of the pathogenesis of dry eye disease.

At present, it is recognized that in some cases, pathological states lead to symptoms but with clinically significant signs, or signs without symptoms, or dry eye symptoms and signs but not dry eye disease. It is equally important to recognize these problems from a therapeutic perspective, because it may suggest that multi-modal and multi-angle treatment methods are needed. This study intends to further reveal the complex pathological mechanism of dry eye disease by exploring the effects of eyelid laxity on a variety of key indicators of dry eye disease, and provide new strategies for early intervention and treatment.

SUBJECTS AND METHODS

Subjects

The study subjects were 200 eligible individuals in our hospital, including 100 individuals with different degrees of eyelid laxity in the experimental group and 100 healthy individuals without eyelid laxity symptoms in the control group. The experimental group was divided into three subgroups according to the mild, moderate and severe degrees of eyelid laxity (mild: upper eyelid skin only touching the eyelashes, 35 cases; moderate: upper eyelid skin hanging over the eyelashes, 35 cases; severe: upper eyelid skin hanging over the eyeball, 30 cases). The age of the subjects ranged from 50 to 80 years old, and none of them had received eyelid surgery or other medical interventions that might interfere with the eyelid structure (Figure 1).

The reason for selecting this age group is that eyelid laxity mostly occurs in the middle-aged and elderly population, and the incidence of eyelid skin laxity increases with age. Selecting the middle-aged and elderly as the main research subjects can more effectively explore the impact of eyelid laxity on meibomian gland function and dry eye disease. To exclude other factors that might interfere with meibomian gland function, individuals with systemic diseases that affect tear film function or cause ocular dry symptoms such as diabetes, rheumatoid arthritis and Sjögren's syndrome were excluded [5].

All selected subjects had no recent ocular infection or severe ocular allergy history to ensure that the impact of eyelid laxity on meibomian gland function in the study subjects was independent of other pathological factors. All study subjects underwent a comprehensive ophthalmic examination before enrollment to ensure no severe ocular lesions, especially excluding diseases that might affect meibomian gland function such as blepharitis or other inflammations of the eyelid area.

In the age design of the experimental group, the average age was (65.2±8.40) years old. In more detailed settings, the average age of patients with mild eyelid laxity was 62.1 years old, that of patients with moderate laxity was 68.5 years old, and that of patients with severe laxity was 73.6 years old. The gender ratio of all individuals in the experimental group was about 40% male and 60% female, which was consistent with the incidence distribution of age and eyelid laxity. The individuals in the control group were basically matched with the experimental group in terms of age and gender ratio to ensure the consistency of basic demographic characteristics between the two groups (Table 1).

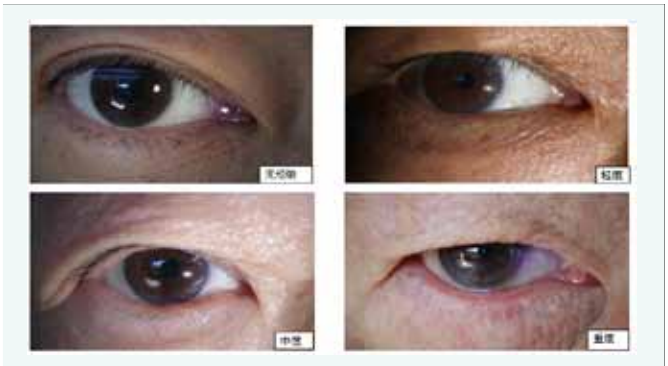


Figure1: Degree of skin laxity

Table 1: Comparison of general data [a]P<0.05

	Sample	Gender		Age (years)
		Male	Female	
Control group	100	40	60	64.15±7.35[a]
Experimental group	100	40	60	65.20±8.40

Information on living habits and ocular health history of all study subjects was recorded, including daily eye use time, long-term contact lens wear, and frequent exposure to polluted environments [6].

These factors may play a role in the pathogenesis of dry eye disease and meibomian gland dysfunction, and their inclusion in background information helps to control these potential confounding variables in data analysis. About 30% of the individuals in the experimental group had the habit of long-term contact lens wear, while the proportion in the control group was only 10%, indicating that living habits may have an additional impact on ocular health, which will be controlled in the subsequent statistical analysis.

METHODS

The sample data included 200 subjects, among whom 100 individuals with eyelid laxity in the experimental group were further divided into three groups: mild (35 cases), moderate (35 cases) and severe (30 cases) according to the degree of laxity; another 100 healthy individuals without eyelid laxity were in the control group. Through a standardized screening process, the study excluded individuals with other ocular diseases, ocular surgery history or systemic diseases (such as diabetes, rheumatic diseases) as confounding factors to ensure the independent impact of eyelid laxity on meibomian gland function.

Slit lamp microscope and imaging technology were used to record and analyze the degree of eyelid laxity to ensure the consistency and accuracy of laxity grading. Non-contact infrared imaging equipment was used to observe the structure and secretory state of the meibomian glands, and key indicators such as tear film break-up time (TBUT) and tear secretion volume (SIt) were measured to help judge whether eyelid laxity poses a threat to tear film function [7].

The tear film break-up time was measured by a non-contact infrared camera to detect the stability of the tear film under a simulated natural environment. The Schirmer test was used to quantify the tear secretion volume to supplement the evaluation of tear film function. In the Schirmer test, a graduated tear detection filter paper (5mm×35mm) was placed at the outer 1/3 of the conjunctival sac of the tested eye. The patient closed the eyes for 5 minutes and then the filter paper was taken out, and the wet length on the filter paper was used to indicate the tear secretion status. To ensure that the results of the Schirmer test were not affected by environmental factors, all tests were carried out in a constant temperature and humidity laboratory environment. Tear osmolarity was measured at the temporal side to evaluate the osmotic balance of the tear film. The detection of inflammatory marker levels was performed by biochemical analysis of collected tear samples, mainly detecting the concentration of interleukin (IL-6). A high IL-6 response indicates an enhanced local inflammatory response, which may exacerbate meibomian gland dysfunction and promote the progression of dry eye disease [8].

All sample collection processes followed aseptic operation specifications and were immediately stored in a low-temperature environment to ensure the stability of samples and the accuracy of analysis results.

Observation Indicators

Tear film break-up time (TBUT), basic tear secretion volume, tear osmolarity and the level of inflammatory marker (IL-6) in tears were used as the main observation indicators.

For the collection of subjective symptoms, the OSDI questionnaire was used in the study to score the severity of dry eye symptoms of the subjects (0-100) and record their subjective feelings.

Statistical Analysis

SPSS 22.0 was used for the sample data of tear film break-up time (TBUT), basic tear secretion volume, tear osmolarity and inflammatory marker IL-6 in tears. Measurement data were expressed as mean ± standard deviation (x±s), and one-way analysis of variance was used for inter-group comparison. To control the influence of other variables, analysis of covariance (ANCOVA) was used to exclude confounding factors such as age and gender, making the significance analysis of the difference in tear secretion volume between the experimental group and the control group more accurate.

RESULTS

Basic Information of the Study Population

There were 100 cases in both the experimental group and the control group. The experimental group was divided into mild, moderate and severe groups according to the degree of eyelid laxity, with the age ranging from 50 to 80 years old. The average age of the experimental group and the control group was consistent, 65 and 66 years old respectively. The average age of the mild laxity group (35 cases) in the experimental group was 62 years old, that of the moderate laxity group (35 cases) was 68 years old, and that of the severe laxity group (30 cases) was 73 years old. In terms of gender ratio, males accounted for 40% and females 60% in the experimental group; the gender ratio in the control group was similar, also 40% males and 60% females (Table 2).

In terms of living habits, about 30% of the subjects in the experimental group and 10% in the control group had the habit of long-term contact lens wear. More than 20% of the individuals in the experimental group reported prolonged use of electronic screens for more than 3 hours a day, while the proportion in the control group was slightly lower (15%). 12% of the individuals in the experimental group were exposed to air pollutants such as dust and chemicals in the working environment all year round, while only 5% in the control group.

Regarding the background information of ocular health history, about 40% of the subjects in the experimental group had reported ocular

Table 2: Comparison of general data [a]P<0.05

	Sample	Gender		Age(year)
		Male	Female	
Control group	100	40	60	64.15±7.35[a]
Experimental group	100	40	60	65.20±8.40

dryness or discomfort symptoms, while the proportion in the control group was 15%. In the basic ophthalmic examination before enrollment, the degree of eyelid laxity of all individuals in the experimental group was professionally evaluated. Mild, moderate and severe laxity showed different degrees of ocular discomfort and the risk of decreased tear film function; the individuals in the control group had no eyelid laxity symptoms and no obvious ocular problems were reported.

The differences in tear film break-up time (TBUT) and tear secretion volume in patients with mild laxity in the experimental group further supported the hypothesis of the potential impact of eyelid laxity on meibomian gland function. There were also significant differences in tear osmolarity and inflammatory level between the experimental group and the control group, indicating that the tear film osmotic balance of individuals in the experimental group was affected to varying degrees. Among the inflammatory level indicators, the interleukin (IL-6) level in the experimental group also showed significant differences between mild, moderate, severe patients and the control group.

The Effect of Eyelid Skin Laxity on Tear Film Break-Up Time

Tear film break-up time (TBUT) was one of the main indicators. In the experimental group, the TBUT of patients with mild laxity was (7.87±0.12) seconds, moderate (5.96±0.35) seconds and severe (3.95±0.47) seconds, while the mean value of the control group was (9.72±0.69) seconds. TBUT reflects the stability of the tear film, and a lower TBUT usually indicates a thin or unstable lipid layer of the tear film, which may aggravate dry eye symptoms (Table 3).

To explore the evolution of eyelid laxity degree in long-term follow-up, repeated measurement data of subjects in the experimental group at 6 and 12 months were included. The TBUT of patients with mild laxity decreased by an average of (0.58±0.01) seconds after 6 months and (1.55±0.52) seconds after 12 months, and those of the moderate laxity group were (3.25±0.55) seconds and (4.12±0.58) seconds respectively. A comprehensive analysis of these data combined with physiological data such as TBUT and tear secretion can more fully reflect the correlation between eyelid laxity and tear film stability and subjective discomfort [9, 10].

The Effect of Eyelid Skin Laxity on Tear Secretion Volume

The basic tear secretion volume, another key indicator, was measured by the Schirmer I test. In the detected data, the tear secretion volume of the control group was (12.0±1.1) mm, while those of patients with mild laxity, moderate laxity and severe laxity were (8.1±0.9) mm, (5.3±1.2) mm and (4.8±0.8) mm respectively (Table 4). These data can effectively indicate that the tear secretion volume shows a decreasing trend with the increase of the degree of eyelid laxity, thus revealing the potential correlation between the decrease of tear secretion and eyelid laxity.

The Effect of Eyelid Skin Laxity on Tear Osmolarity

Tear osmolarity can reflect whether the osmotic balance of the tear film is disturbed. Experimental data showed that the mean tear osmolarity of the control group was (298.45±7.98) mOsm/L, while that of the experimental group was (313.45±6.12) mOsm/L (Table 5). The increase of osmolarity means the accelerated evaporation of tear water,

Table 3: Tear secretion volume [a]P<0.01

		Sample	Tear Film Break Up Time TBUT（s）
Control group		100	9.72±0.69
Experimental group	Mild	35	7.87±0.12
	Moderate	35	5.96±0.35
	Severe	30	3.95±0.47
		100	6.01±0.55[a]

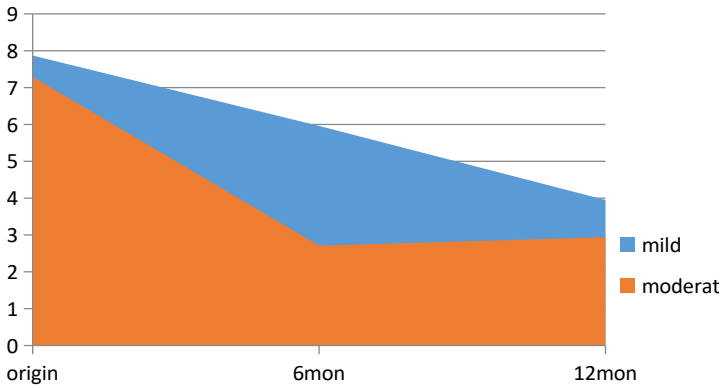


Table 4: Comparison of tear secretion volume [a]P<0.01

		Sample	Schirmer I test SIt(mm)
Control group		100	12.0±1.1
Experimental group	Mild	35	8.1±0.9
	Moderate	35	5.3±1.2
	Severe	30	4.8±0.8
		100	6.2±0.6[a]

Table 5: Comparison of tear osmolarity [a]P<0.01

		Sample	Tear Osmolarity(mOsm/L)
Control group		100	298.45±7.98
Experimental group	Mild	35	308.12±7.94
	Moderate	35	315.21±4.98
	Severe	30	323.33±6.09
	Total	100	313.45±6.12[a]

and the increased osmolarity directly exacerbates the dry state of the ocular surface, which is closely related to the pathological process of dry eye disease. The increase of tear osmolarity may not only be the result of reduced tear water but also stem from the weak protective layer caused by the reduced lipid secretion of meibomian glands.

The Effect of Eyelid Skin Laxity on Tear IL-6

Changes in inflammatory marker levels were also confirmed in the experimental data. Interleukin (IL-6), a typical inflammatory marker, showed a gradual increasing trend in patients with different degrees of eyelid laxity. The mean IL-6 level of the control group was (3.98±2.01) pg/mL, while that of the experimental group reached (28.01±4.55) pg/mL. The increase of IL-6 concentration reflects that eyelid laxity may further affect the normal function of meibomian glands by inducing local inflammatory reactions (Table 6).

In terms of subjective symptom scores, the experimental data also revealed the direct impact of eyelid laxity on dry eye symptoms. The average subjective dry eye score of the control group was 11 points, while that of the mild laxity group was 20 points, the moderate laxity group 26 points, and the severe laxity group as high as 33 points (Table 7). This subjective score can help the study analyze the direct impact of eyelid laxity on dry eye symptoms. Combined with objective indicators such as TBUT and tear secretion volume, it can evaluate the role of eyelid laxity in the pathological development of dry eye disease from multiple angles and provide data support for revealing the correlation between eyelid laxity and patients' symptom experience.

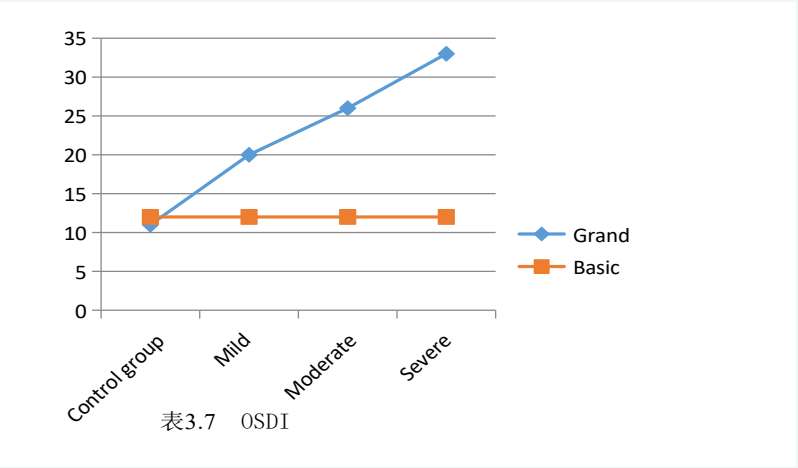
DISCUSSION

This study intends to explore the effect of eyelid skin laxity on dry eye disease by analyzing its impact on the ocular surface and meibomian gland function, so as to find preventive or therapeutic methods for dry eye disease. Through this study, it was found that with the aggravation of eyelid skin laxity, indicators such as tear film break-up time (TBUT), basic tear secretion volume, tear osmolarity and inflammatory marker IL-6 in tears all changed significantly, suggesting that eyelid skin laxity may aggravate dry eye disease.

In the etiology of dry eye disease, Sheppard and Farrand K F [11,12], have repeatedly mentioned that age is an important risk factor for dry eye disease. With the increase of age, the eyelid skin gradually shows aging and laxity. The upper eyelid skin is thinner than other facial skin parts, and the upper eyelid skin and muscles perform functional movements for a long time, so they are more prone to laxity than the lower eyelid. At present, the problem of population aging in China is becoming increasingly serious. By 2020, the elderly population over 65 years old reached 191 million, accounting for 13.5% [13]. With the increase of age, various physiological or pathological changes of the body are inevitable, including the decline of the function of various tissues and organs [14]. Aging is a dynamic process, which is particularly obvious in the periorbital region and its related structures. The aging of the upper eyelid is specifically manifested as blepharoptosis, increased wrinkles and brow ptosis [15]. The uneven resorption of the periorbital bone changes the position of the bone surface [16], so the attachment position of periorbital tissues (including the orbital septum, fat, muscle, ligament,

Table 6: Comparison of tear osmolarity [a]P<0.01

		Sample	Tear Osmolarity(mOsm/L)
Control group		100	298.45±7.98
Experimental group	Mild	35	308.12±7.94
	Moderate	35	315.21±4.98
	Severe	30	323.33±6.09
	Total	100	313.45±6.12[a]



etc.) on the periosteum also moves accordingly, leading to the loss of their mechanical function [17]. The change of stress may lead to incomplete eyelid closure and malposition.

The Effect of Eyelid Laxity on Various Indicators of Dry Eye Disease

This study aims to explore the correlation between the degree of eyelid laxity and key indicators of dry eye disease. The results showed that with the aggravation of the degree of eyelid laxity, the objective indicators reflecting tear film stability, tear secretion volume, tear osmolarity and local inflammatory state all showed a consistent worsening trend, with the specific analysis as follows.

TBUT is the core indicator for evaluating tear film stability, defined as the time required for the first dry spot to appear on the tear film surface after a complete blink. In this study, the gold standard method was adopted, that is, measurement in a natural state by a non-contact infrared camera. It is generally considered that TBUT ≥ 10 seconds is normal, and < 10 seconds indicates tear film instability.

The results of this study showed that the TBUT of patients in the experimental group decreased significantly with the aggravation of eyelid laxity: the mild laxity group was (7.87±0.12) seconds, the moderate group was (5.96±0.35) seconds, and the severe group decreased to (3.95±0.47) seconds, all significantly lower than that of the control group [(9.72±0.69) seconds]. This indicates that the more severe the eyelid laxity, the worse the tear film stability. This result is consistent with the research conclusions of Jiang Pengfei, Li Dongdong et al., that is, TBUT is positively correlated with dry eye symptoms. However, it is worth noting that the study of Lv Juling pointed out that TBUT is easily affected by various factors such as environment, occupation, eye hygiene and palpebral fissure size. Although this study focused on the degree of eyelid skin laxity, potential confounding factors such as palpebral fissure size were not included in the observation indicators, so the accuracy of the conclusion needs to be further verified by subsequent studies.

The basic tear secretion volume measures the secretory function of the lacrimal gland and accessory lacrimal glands in the unstimulated state. In this study, the average tear secretion volume of the control group was (12.0±1.1) mm. In contrast, the secretion volume of patients with eyelid laxity decreased significantly: the mild group was (8.1±0.9) mm, the moderate group was (5.3±1.2) mm, and the severe group decreased to (4.8±0.8) mm. This trend clearly indicates that the increase of the degree of eyelid laxity is associated with the decline of tear secretion function. However, this study has certain limitations. Although obvious pathological conditions such as dacryoadenitis have been excluded, the size, morphology and duct function state of the lacrimal gland were not evaluated by imaging examination, which may be a potential factor affecting the measurement of tear secretion volume.

In this experiment, the tear osmolarity level increased gradually with the aggravation of eyelid laxity: the control group was (298.45±7.98) mOsm/L, and the mild, moderate and severe laxity groups were (308.12±7.94) mOsm/L, (315.21±4.98) mOsm/L and (323.33±6.09) mOsm/L respectively. This trend verified the positive effect of eyelid laxity on tear osmolarity. However, the study of Baenninger PB [18], pointed out that tear osmolarity has high variability in dry eye patients

and has a weak correlation with other diagnostic parameters. In addition, this study only measured the temporal tear osmolarity without detecting the nasal side [19], which may lead to deviations in the results and needs to be confirmed by more comprehensive measurements.

The detection in this study showed that the IL-6 level presented a significant increasing trend from (3.98±2.01) pg/mL in the control group to (38.63±7.45) pg/mL in the severe laxity group, suggesting that eyelid laxity may induce or aggravate local inflammation through a certain mechanism. This finding is consistent with the mainstream view that "inflammation is the core mechanism of dry eye pathogenesis" [20]. From the perspective of pathophysiology, the destruction of the ocular surface microenvironment (such as increased osmolarity) will activate the immune pathway and release inflammatory factors (such as IL-6), which in turn damage the ocular surface epithelial barrier [21]. Inflammatory factors such as IL-6 can further amplify the inflammatory response, forming a vicious circle and leading to the continuous deterioration of the ocular surface microenvironment [22]. This provides theoretical support for the phenomenon that the IL-6 level increases with the aggravation of eyelid laxity in this study. Jiang Yingxing et al., also confirmed that a variety of inflammatory factors such as IL-1β, IL-4, IL-6, TNF-α and INF-γ are involved in the pathological process in the tears of dry eye patients. Therefore, inflammatory factors reflect the degree of local inflammatory response and its relationship with eyelid laxity.

Subjective dry eye score directly reflects the symptom experience and quality of life of the subjects. In this study, the OSDI score of the control group was 11 points, and the score increased gradually to 33 points with the degree of eyelid laxity from mild, moderate to severe. This trend indicates that the more severe the eyelid laxity, the stronger the ocular discomfort felt by the patients. Although the P value (0.39) of this indicator did not reach statistical significance and could not confirm the difference statistically, considering the common phenomenon of "dissociation between symptoms and signs" in clinical dry eye disease and the increasing trend of scores, the OSDI score can still be used as an important reference for evaluating patients' subjective feelings, suggesting that clinicians should pay attention to patients' subjective symptoms.

Possible Mechanisms of the Effect of Eyelid Laxity on Dry Eye Disease

Eyelid laxity refers to a pathological state in which the eyelid skin, muscles and connective tissue show decreased elasticity and support due to aging, chronic inflammation or mechanical stimulation. Its impact on ocular surface function involves multiple links, with the possible mechanisms as follows:

(1).Changes in eyelid anatomical structure and obstruction of meibomian gland secretion channels: The eyelid skin and orbicularis oculi muscle are lax and ptotic, especially in the lateral upper eyelid and lower eyelid. The lax tissue is prone to form folds when the eyelids are closed, compressing the orifices of meibomian gland ducts. The meibomian gland ducts open behind the gray line of the lid margin. Under normal circumstances, the contraction of the orbicularis oculi muscle during blinking can exert pressure on the meibomian glands, promoting lipid secretion. The ptosis caused by eyelid laxity will weaken this mechanical compression effect, leading to insufficient lipid secretion and further affecting the stability of the tear film [23].

When the eyelids are lax, the force distribution of the orbicularis oculi muscle is uneven, the contractility of some areas is weakened, and the mechanical compression of the lax tissue narrows or deforms the duct orifices, leading to poor lipid discharge. Long-term stasis can cause hyperplasia of duct epithelial cells and keratinization of the lumen, further aggravating the obstruction and forming a vicious circle. In addition, lax eyelids may be accompanied by abnormal lid margin position (such as entropion or ectropion), which changes the anatomical relationship between the meibomian gland ducts and the lid margin and affects the normal discharge path of lipids.

(2).Ocular surface microenvironment disorder and abnormal lipid components of meibomian glands: Patients with eyelid laxity often have incomplete eyelid closure (lagophthalmos) or decreased blinking frequency, leading to accelerated tear evaporation and decreased tear film stability on the ocular surface. The lipid layer in the tear film is mainly secreted by the meibomian glands, whose function is to reduce water evaporation and maintain the integrity of the tear film structure. When the eyelid closure is incomplete, the exposed area of the ocular surface increases, and the lipid layer is easily oxidized or denatured by external environmental stimuli (such as dryness and dust), leading to decreased lipid fluidity and increased viscosity. At the same time, long-term dryness of the ocular surface can activate the inflammatory pathway and promote the release of inflammatory factors (such as IL-6 and TNF-α), which can directly damage the acinar cells of the meibomian glands and inhibit the activity of lipid synthetases (such as lipase), leading to the imbalance of lipid component ratio (such as increased content of cholesterol esters and wax esters and decreased content of triglycerides), further reducing the quality of the lipid layer and aggravating ocular surface dryness and meibomian gland dysfunction.

Eyelid laxity also affects the efficiency of the blinking process. Under normal circumstances, blinking not only helps to clean the ocular surface but also releases the lipid layer by compressing the meibomian glands to maintain the stability of the tear film. The blinking frequency and amplitude of patients with eyelid laxity may decrease, which makes the distribution of the tear film on the ocular surface uneven and leads to increased tear evaporation. Studies have shown that the tear film break-up time of patients with eyelid laxity is significantly shortened, which suggests that the function of the tear film in maintaining moisture is affected and dry eye symptoms are more obvious.

(3).Cascade reaction of chronic inflammation and meibomian gland atrophy: Eyelid laxity is often accompanied by blepharitis, manifested as lid margin congestion, scales or ulcer formation. The lax eyelid skin is prone to accumulate sebum, sweat and external pollutants, providing an environment for the breeding of bacteria (such as staphylococcus) and inducing chronic infectious inflammation. Bacterial metabolites (such as lipase) can decompose meibomian gland lipids and produce irritating free fatty acids, which further stimulate the lid margin and meibomian gland tissue and induce aseptic inflammatory reactions. Long-term chronic inflammation can lead to fibrosis of meibomian gland acini, apoptosis of acinar cells, decrease in the number of acini, and ultimately cause meibomian gland atrophy. In addition, the inflammation-mediated oxidative stress response can damage the epithelial cells of meibomian gland ducts, leading to duct stenosis or atresia and aggravating secretory dysfunction. Clinical experimental data show that the level of inflammatory markers such as interleukin (IL-6) in the tears of patients with eyelid laxity is increased, which further confirms that there is a close relationship between eyelid laxity and the inflammatory mechanism of dry eye disease [24]. Studies have shown that the density and length of meibomian glands in patients with eyelid laxity are significantly lower than those in the normal population, and the degree of inflammation is positively correlated with the degree of meibomian gland atrophy.

When the meibomian glands are stimulated by inflammation, their tissue structure may undergo degenerative changes, and the number of functional secretory cells may decrease, leading to insufficient secretion, thus reducing the thickness of the lipid layer of the tear film. The presence of inflammatory reaction not only destroys the normal secretory environment of the meibomian glands but also accelerates the damage process of the tear film, making the pathological relationship between eyelid laxity and dry eye disease closer.

The presence of inflammation may not only lead to the degeneration

of gland function but also cause apoptosis and degenerative changes of meibomian gland cells, further reducing the lipid secretion capacity of the meibomian glands. This process will produce a vicious circle, that is, the inflammatory reaction caused by eyelid laxity affects the meibomian gland function, and the decline of meibomian gland function in turn exacerbates the inflammatory reaction, thus accelerating the development of dry eye disease. Eyelid laxity may also affect the tightness of eyelid closure, leading to ocular surface exposure during sleep, and thus aggravating dry eye symptoms. Ocular surface exposure at night will increase the evaporation of water from the cornea and conjunctiva, leading to more severe ocular dryness and discomfort when waking up in the morning. With the aggravation of eyelid laxity, the incomplete eyelid closure becomes more obvious, leading to the decline of the protective ability of the ocular surface during sleep, further weakening the protective function of the tear film and making the eyes in a dry state for a long time [25].

(4).Mechanical traction and changes in meibomian gland morphological structure: Long-term eyelid laxity can exert persistent mechanical traction on the tarsal plate, leading to deformation of the tarsal plate morphology (such as thinning of the tarsal plate and increased curvature). The tarsal plate is the attachment scaffold of the meibomian glands, and its morphological changes can directly affect the arrangement direction of acini and the course of ducts. When the tarsal plate bends due to traction, the meibomian gland ducts may twist or fold, hindering lipid transport; at the same time, the acini may undergo morphological changes such as acinar cavity enlargement or atrophy under traction stress, affecting lipid synthesis and storage. Histopathological studies have shown that eyelid laxity changes the position and tension of the meibomian glands, leading to deformation or compression of the meibomian gland ducts, thus hindering the normal meibum secretion process [26]. The arrangement of collagen fibers in the tarsal plate of patients with eyelid laxity is disordered, and elastic fibers are broken, leading to the decline of tarsal plate support and further aggravating the structural damage of the meibomian glands.

Meibomian gland photography (1: normal gland, 2-4: mild, moderate and severe gland atrophy respectively) (Figure 1).

With the increase of age, the anatomical structure of eyelid tissue undergoes degenerative changes, the skin gradually loses elasticity, the skin and supporting structures become lax, adipose tissue atrophies, the orbicularis oculi muscle relaxes, etc., forming the so-called eyelid laxity. The horizontal elasticity and tension of the eyelids are both reduced, and the force in all directions is unbalanced, which may lead to eyelid malposition and other manifestations. The anatomical structure and tension of the eyelids are crucial for the normal operation of meibomian gland function. Eyelid laxity will lead to significant changes in these structures, thus adversely affecting the function of the meibomian glands.

Obstructed meibum secretion will directly affect the lipid layer of the tear film, making tears more likely to evaporate, leading to ocular surface dryness and further exacerbating dry eye symptoms.

Eyelid laxity not only affects the physical position of the meibomian glands but also causes changes in their dynamic function. Under normal circumstances, the blinking action will regularly secrete lipids by mechanically compressing the meibomian gland ducts, thus maintaining the stability of the tear film. After eyelid laxity, the compression force of blinking decreases, leading to insufficient lipid secretion and threatening the stability of the tear film [27]. Eyelid laxity will prolong the contact time between the eyelids and the cornea and increase the instability of the tear film. Studies have shown that the tear film break-up time of patients with eyelid laxity is significantly shorter than that of the control group, which suggests that the lipid layer of the tear film is thinner or uneven, thus accelerating tear evaporation and the occurrence of dry eye symptoms.

In addition to physical effects, eyelid laxity may also induce local inflammatory reactions, thus causing destructive effects on the structure and function of the meibomian glands. Chronic inflammation of the meibomian glands may be aggravated by eyelid laxity. The inflammatory reaction will lead to swelling of the tissue around the meibomian gland ducts, further compressing the ducts and increasing the resistance of lipid secretion [28].

The Relationship between Eyelid Laxity and Dry Eye Disease

Eyelid laxity changes the normal position and function of the meibomian glands, thus affecting the formation and stability of the lipid layer of the tear film. The meibomian glands are oil glands in the eyelids, and their secreted lipid layer is an important part of the tear film, which helps to prevent tear evaporation and keep the ocular surface moist. With the aggravation of eyelid laxity, the position of the meibomian glands changes, which may lead to the compression or obstruction of the meibomian gland ducts and hinder meibum secretion. This obstruction of secretion will lead to a thin or uneven lipid layer of the tear film, thus increasing the rate of tear evaporation and leading to ocular surface dryness. Insufficiency of the tear film lipid layer is an important cause of dry eye disease, and eyelid laxity plays a key role in it.

Eyelid laxity may also induce a chronic inflammatory reaction of the eye. Inflammation is not only an important pathological feature of dry eye disease but also further exacerbates ocular dryness by damaging the tear film. Eyelid laxity may cause persistent compression of the meibomian

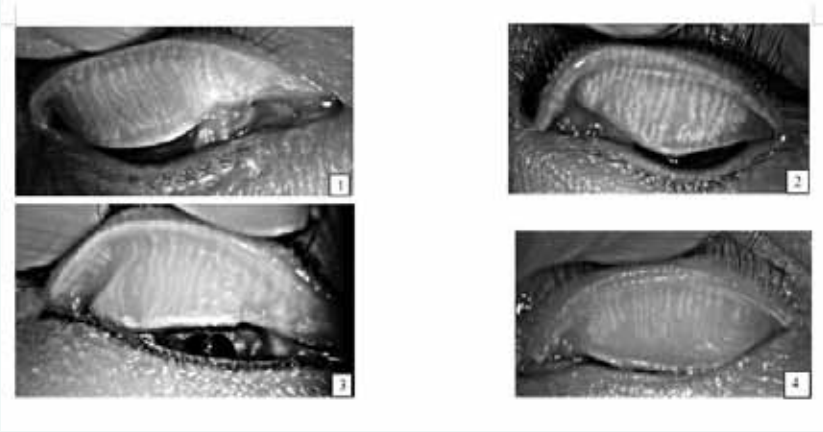


Figure 2: Meibomian gland photography (1: normal gland, 2-4: mild, moderate and severe gland atrophy respectively)

gland ducts, leading to chronic inflammation of the meibomian gland tissue. This inflammatory reaction will further affect the normal secretory function of the meibomian glands. Inflammation will also accelerate the degeneration of the meibomian glands, leading to reduced meibum secretion. The interaction between inflammation and degeneration will form a vicious circle, leading to the gradual aggravation of dry eye symptoms.

The impact of eyelid laxity on dry eye disease is also reflected in the changes of eyelid closure function. At night, the eyelids should be completely closed to protect the ocular surface and prevent tear evaporation. With the aggravation of eyelid laxity, some patients may have incomplete closure or malposition, leading to ocular surface exposure during sleep or obstruction and contamination of the meibomian gland orifices. Exposure will make the cornea and conjunctiva lose the protection of tears at night, leading to more severe ocular dryness and discomfort when waking up in the morning. Incomplete eyelid closure not only impairs the protective function of the tear film but also may lead to damage to corneal epithelial cells, further aggravating the symptoms of dry eye disease. In addition, the accumulation of skin at the lid margin will bring the dust of eyelashes into the ocular surface, which not only causes a sticky feeling in the eyes but also easily induces ocular surface inflammation.

Clinical Significance and Application

As a common phenomenon with aging, eyelid laxity is not only related to cosmetic problems but also affects the normal physiological function of the eyelids. This finding provides a new perspective for the diagnosis, treatment and prevention of dry eye disease [29].

Clinically, dry eye disease is a disease with high incidence and easy recurrence, which seriously affects the quality of life of patients. The study on the evaluation of eyelid laxity and its relationship with dry eye symptoms provides a new basis for clinical intervention. In terms of diagnosis, the detection and evaluation of eyelid laxity can help identify the potential risk population of dry eye disease. By observing whether there is obvious eyelid laxity and ptosis, or performing eyelid tension tests, patients who are more likely to be affected by abnormal tear film function can be screened. Early diagnosis and intervention of eyelid laxity can prevent the further deterioration of meibomian gland function. The stability of the tear film is the key to preventing dry eye disease. Timely meibomian gland function testing for patients with eyelid laxity helps to detect tear film instability problems early and formulate personalized intervention plans [30].

By measuring tear film break-up time (TBUT), tear osmolarity and inflammatory markers such as IL-6 level, combined with the degree of eyelid laxity, the severity of dry eye disease can be more accurately evaluated in clinical diagnosis, providing precise treatment guidance for patients.

In terms of treatment, the relationship between eyelid laxity and dry eye disease has inspired new treatment methods. Traditional dry eye disease treatment mostly uses artificial tears or drugs to directly relieve dry eye symptoms, but the tear film instability and meibomian gland dysfunction caused by eyelid laxity cannot be solved by tear supplementation alone in many patients. Patients with eyelid laxity may need additional physical interventions, such as meibomian gland massage or warm compresses to improve meibomian gland lipid secretion and restore the stability of the tear film lipid layer. The application of eyelid lift surgery or other eyelid correction surgeries in patients with severe eyelid laxity shows the potential to effectively improve tear film stability [31].

Surgical or physical treatment can restore eyelid tension to a certain extent, thus helping the tear film maintain normal distribution and function and alleviating dry eye symptoms. For patients with moderate eyelid laxity, thermotherapy and ocular physical therapy may be an effective non-invasive intervention method, which can improve tear film stability and reduce tear evaporation rate by increasing eyelid circulation and meibomian gland secretion, thus relieving dry eye disease. The clinical application of eyelid laxity in dry eye disease also suggests the importance of prevention. In high-risk groups, regular examination of eyelid health status and monitoring of tear film stability can take measures in the early stage of eyelid laxity to delay the decline of meibomian gland function. Especially for the middle-aged and elderly, proper eyelid care, healthy eye use habits and reasonable ocular care can prevent and reduce the incidence of eyelid laxity. Daily moisturization and nutritional supplementation, avoiding prolonged use of electronic devices or frequent exposure to irritating substances can slow down the aging of eyelid tissue [32]. Promoting the relationship between eyelid laxity and the risk of dry eye disease in clinical ophthalmology can make patients pay more attention to this problem and enhance their awareness of active care to achieve the effect of preventing dry eye disease.s

Study Limitations and Future Research Directions

This study has achieved remarkable results in exploring the effects of eyelid laxity on meibomian gland function and dry eye symptoms, but its sample size is limited, especially the uneven distribution of sample size in subjects of different ages and genders, which may affect the universality and applicability of the study results. Although the study selected a representative middle-aged and elderly population, the manifestations of dry eye disease and eyelid laxity may vary in different age groups and genders. Future studies should consider larger-scale and diverse populations to verify the stability and popularization of the results. Although a number of indicators including tear film break-up time, tear secretion volume, tear osmolarity, IL-6 and other inflammatory markers as well as subjective dry eye scores were used to evaluate the impact of eyelid laxity on tear film function, these indicators may still be insufficient to fully reflect the complex impact of eyelid laxity. Eyelid laxity may further affect meibomian gland function through other unmeasured physiological or biochemical pathways. Future studies can introduce more comprehensive evaluation methods such as imaging analysis of eyelid dynamic structure and lipid metabolism measurement to more fully reveal the multi-level impact of eyelid laxity on ocular health. The detection of inflammatory markers is usually affected by time, environment, individual differences and other factors. In future studies, more accurate and standardized detection procedures should be considered to improve the reliability of data [33].

The degree of eyelid laxity in the experimental group and the control group was mainly graded by routine examinations such as appearance and tear film break-up time, without using more objective imaging or biomechanical analysis tools, which may lead to inconsistency or subjectivity of the grading standards. Future studies can use high-resolution imaging technologies such as artificial intelligence deep learning (ALDL) or confocal microscopy, and at the same time use high-speed cameras to record dynamic videos of blinking to monitor the degree of eyelid closure. It is possible to quantify the degree of eyelid laxity from a more precise structural and functional perspective to improve the accuracy of grading and the rigor of the study.

The experimental design of this study mainly focused on the immediate impact of eyelid laxity on meibomian gland function and dry eye symptoms, but failed to evaluate the long-term dynamic changes of eyelid laxity on these factors. As a progressive process, the impact of eyelid laxity may accumulate over time, so long-term follow-up studies are particularly necessary. Through dynamic observation of eyelid laxity and meibomian gland function data in different time periods, future studies can more accurately reveal the long-term relationship between eyelid laxity and dry eye disease and its disease progression. The impact of eyelid laxity on dry eye disease may be regulated or interfered by other factors such as individual lifestyle, genetic background and environmental factors, but the role of these external variables has not

been deeply explored. These external variables may aggravate or alleviate the impact of eyelid laxity. Future studies can construct a multi-factor model by adding data such as living habits and environmental exposure history to more comprehensively understand the role of eyelid laxity in the pathogenesis of dry eye disease. Especially against the background of rapid population aging, the incidence of eyelid laxity in the elderly population is gradually increasing, and its relationship with dry eye disease is of great significance in public health and clinical practice. Future studies can explore preventive and early intervention measures to help slow down the impact of eyelid laxity on dry eye symptoms.

CONCLUSION

With the aggravation of the degree of eyelid laxity, the objective indicators reflecting tear film stability, tear secretion volume, tear osmolarity and local inflammatory state all showed a consistent worsening trend, and eyelid skin laxity had an adverse effect on dry eye disease.

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