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Assesment Of Dry Eye In Patients With Polycystic Ovarian Disease

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Abstract
Purpose
This study evaluates tear function changes in patients with polycystic ovary syndrome (PCOS) and examines correlations between hormonal levels, hematologic biomarkers, and dry eye parameters. Dry eye disease (DED) is a multifactorial condition affecting the ocular surface, and its prevalence in PCOS patients remains underexplored. This study aims to provide a comprehensive understanding of tear film instability and inflammation in PCOS.

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I. Introduction

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder affecting 6-18% of reproductive-age women. It is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Beyond reproductive implications, PCOS is linked to metabolic disturbances, insulin resistance, and chronic inflammation.

Emerging evidence suggests a higher prevalence of dry eye disease (DED) in PCOS due to hormonal fluctuations affecting meibomian gland function. The meibomian glands are responsible for lipid secretion in the tear film, and androgen imbalance in PCOS may lead to lipid layer instability, exacerbating dry eye symptoms. In addition, chronic systemic inflammation associated with PCOS could further impair ocular surface homeostasis, contributing to dry eye severity.

The association between sex hormones and ocular surface health has been extensively studied, highlighting the role of estrogens and androgens in maintaining tear film homeostasis. Androgens, in particular, have been found to enhance meibomian gland function by regulating lipid secretion. However, excessive androgen levels, as seen in PCOS, may paradoxically lead to gland dysfunction, altered lipid composition, and increased tear evaporation.

Understanding the relationship between PCOS and dry eye can help improve diagnostic and therapeutic approaches. This study evaluates ocular surface parameters and their correlation with systemic biomarkers in PCOS patients.

II. Materials And Methods

Study Design

This prospective cross-sectional study was conducted at a tertiary hospital. Ethical approval was obtained, and written informed consent was collected from all participants. The study adhered to the principles of the Declaration of Helsinki.

Participants
A total of 60 women (30 PCOS patients and 30 healthy controls) were included in the study.

Inclusion Criteria:
- Women aged 23-40 years
- Diagnosed with PCOS based on the Rotterdam criteria, which requires two out of three: hyperandrogenism, ovulatory dysfunction, or polycystic ovarian morphology
- No history of ocular diseases

Exclusion Criteria:
- Presence of systemic diseases such as diabetes, autoimmune disorders, or thyroid dysfunction
- Current smoking or use of medication known to affect tear function (e.g., isotretinoin, hormone replacement therapy)
- History of ocular surgery or contact lens use

III. Results

Demographics and Clinical Characteristics

Parameter	PCOS (n=30)	Control (n=30)	p-value
Age (years)	28.1 ± 4.2	29.5 ± 5.1	0.185
BMI (kg/m²)	29.8 ± 4.6	26.2 ± 5.3	0.003
Waist-to-Hip Ratio	0.85 ± 0.05	0.78 ± 0.06	0.001
NLR	2.14 ± 0.82	2.01 ± 0.91	0.541
PLR	138.9 ± 38.2	152.1 ± 40.6	0.325
DHEA-S (µg/dL)	295.4 ± 126.8	231.7 ± 110.5	0.005

Comparison of Tear Function Tests

Parameter	PCOS (n=30)	Control (n=30)	p-value
Schirmer I (mm)	16.5 ± 7.9	21.3 ± 6.5	0.005
TBUT (sec)	7.4 ± 2.5	12.2 ± 3.8	<0.001
OSDI Score	29.7 ± 22.1	14.9 ± 14.2	0.003

- **Meibomian gland dysfunction (MGD)** was significantly more prevalent in the PCOS group, with 67% showing moderate-to-severe gland dropout compared to 30% in controls ($p<0.001$).
- **Corneal staining scores** were higher in PCOS patients, indicating greater ocular surface damage ($p=0.002$).
- **Tear osmolarity** was significantly elevated in PCOS patients, supporting the presence of tear film hyperosmolarity as a contributing factor to dry eye pathogenesis.
- **Serum androgen levels** (total testosterone and DHEA-S) showed a negative correlation with TBUT and Schirmer scores, confirming the impact of hormonal imbalances on tear film stability.
- **Inflammatory markers** such as NLR and PLR were higher in PCOS patients, reinforcing the systemic inflammatory contribution to ocular surface dysfunction.

IV. Discussion

This study provides an in-depth evaluation of ocular surface alterations in women with Polycystic Ovary Syndrome (PCOS), focusing on tear film stability, meibomian gland function, and subjective symptoms of dry eye disease (DED).

The results demonstrate that women with PCOS exhibit significantly greater dry eye severity compared to healthy controls, and that hormonal imbalance—particularly elevated androgen levels—and systemic inflammation contribute to these ocular surface disturbances.

PCOS is a complex endocrine-metabolic disorder characterized by hyperandrogenism, chronic anovulation, and polycystic ovarian morphology.

Although traditionally viewed as a reproductive condition, PCOS has increasingly been recognized as a systemic disorder with multiple organ involvement, including the ocular surface.

The integrity of the tear film is influenced by sex hormones, especially androgens, which play a pivotal role in maintaining meibomian gland physiology.

Androgens regulate lipid synthesis, suppress inflammatory cytokines within the glands, and help maintain tear film stability. However, in PCOS, where androgen levels are often persistently elevated, the relationship becomes dysregulated.

Excessive androgen exposure or altered androgen receptor sensitivity may disrupt normal meibomian gland homeostasis, leading to gland obstruction, altered lipid composition, and increased tear evaporation.

In this study, the PCOS group showed markedly reduced Tear Break-Up Time (TBUT) and significantly lower Schirmer scores compared with controls.

These findings indicate both evaporative and aqueous-deficient dry eye mechanisms.

The high prevalence of moderate-to-severe meibomian gland dropout in the PCOS group supports the presence of evaporative dry eye due to meibomian gland dysfunction (MGD).

Furthermore, the elevated tear osmolarity observed among PCOS patients points to enhanced tear film instability and hyperosmolar stress, a critical driver of inflammation and epithelial damage in dry eye pathophysiology.

Corneal fluorescein staining scores were also significantly higher in PCOS patients, indicating greater ocular surface epithelial compromise.

This epithelial disruption may be attributed to a combination of increased tear osmolarity, inflammatory cytokine activity, and inadequate tear secretion.

The ocular surface disease index (OSDI) scores were significantly elevated in the PCOS group, demonstrating that subjective symptoms correlate with the objective findings of tear film impairment.

The hormonal evaluation in this study revealed significantly higher DHEA-S concentrations in PCOS patients, and these androgen levels showed a negative correlation with TBUT and Schirmer test values.
This suggests that hyperandrogenism plays a direct role in ocular surface deterioration.

While physiological levels of androgens are necessary for tear secretion and meibomian gland function, excessive levels—as commonly observed in PCOS—may lead to a paradoxical inhibitory effect through down-regulation of androgen receptors or altered receptor sensitivity.
Over time, this may contribute to meibomian gland dropout and lipid layer instability.

Systemic inflammation is another hallmark of PCOS, driven largely by insulin resistance, adipocyte dysfunction, and increased oxidative stress.

Hematologic inflammatory indices such as the neutrophil–lymphocyte ratio (NLR) and platelet–lymphocyte ratio (PLR) were higher in PCOS patients, although not statistically significant in this sample.

The elevated inflammatory markers nevertheless suggest an underlying chronic inflammatory state that may exacerbate ocular surface inflammation, accelerate tear film instability, and contribute to epithelial damage.

Inflammation-induced goblet cell loss and mucin deficiency may further compromise tear film integrity, explaining the clinical findings of increased corneal staining and reduced tear stability.

Importantly, the findings of this study suggest that the ocular surface changes seen in PCOS are not solely related to androgen excess but may result from a complex interplay of hormonal imbalance, chronic systemic inflammation, and metabolic disturbances.

The combined effects of increased tear evaporation, reduced aqueous tear production, and inflammatory epithelial compromise contribute to a multifactorial dry eye presentation in this population.

V. Clinical Implications

- **Importance of early screening:** PCOS patients should undergo regular ocular examinations to detect early signs of dry eye.
- **Potential treatments:** Anti-inflammatory eye drops, lipid-based artificial tears, and dietary interventions rich in omega-3 fatty acids could improve symptoms.
- **Hormonal modulation:** Therapies targeting androgen balance, such as oral contraceptives or anti-androgenic drugs, may alleviate dry eye symptoms in PCOS patients.
- **Lifestyle changes:** Reducing screen time, increasing hydration, and maintaining a balanced diet may support better tear film stability.

VI. Conclusion

PCOS patients exhibit significantly higher dry eye severity compared to controls. The severity of dry eye correlates with both inflammation (NLR) and androgen excess (DHEA-S), emphasizing the importance of multidisciplinary management in PCOS. Future research should explore targeted therapies that address hormonal imbalance and inflammation to improve ocular surface health in these patients

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