

Corneal neuropathy: An overlooked biomarker for dry eye in type 2 diabetes

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Abstract

The article by Han and associates highlights a frequently neglected consequence of type 2 diabetes mellitus (T2D): Dry eye disease associated with corneal neuropathy. Their findings indicate that patients with T2D exacerbated by dry eye disease exhibit substantial decreases in corneal nerve density, length, and count, accompanied by heightened tortuosity, all of which correlate strongly with clinical severity scores. This study contributes to the expanding literature that regards ocular surface disease not only as a secondary effect of systemic illness but also as a direct indication of neurovascular and metabolic impairment. Clinically, these findings underscore the importance of regularly evaluating dry eye in patients with T2D, extending beyond diabetic retinopathy, and incorporating ocular surface assessments into comprehensive diabetes management. This study demon-

strates that the diabetic eye exhibits both tear film instability and corneal neurodegeneration, thereby expanding our understanding of diabetic eye disease and emphasizing the need for multidisciplinary care to preserve vision and improve quality of life.

Key Words: Type 2 diabetes; Corneal neuropathy; Dry eye syndrome; Ocular surface disease biomarkers; Inflammatory biomarkers; Oxidative stress; Precision medicine

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Core Tip: Dry eye disease in type 2 diabetes is not merely a surface ocular issue; it signifies underlying neurovascular and metabolic impairment. Han *et al*'s paper presents data correlating corneal nerve modifications with the severity of dry eye disease in patients with type 2 diabetes. Together with expanding findings on inflammatory biomarkers, immunological cross-talk, and global research trends, this body of evidence highlights the necessity to include ocular surface assessment into diabetes therapy. Future advancements will depend on precision medicine approaches, biomarker-directed screening, and medicines aimed at neuroinflammation and oxidative stress to maintain eyesight and enhance quality of life.

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INTRODUCTION

Diabetes mellitus constitutes a significant global public health issue, with type 2 diabetes (T2D) comprising the vast majority of instances. Nonetheless, a less apparent although equally debilitating manifestation is its impact on the ocular surface, specifically as dry eye disease (DED). Although widely widespread, DED in the context of T2D is typically undervalued by both clinicians and patients, sometimes eclipsed by the fear of sight-threatening diabetic retinopathy.

DIABETIC DRY EYE: MECHANISMS, BIOMARKERS, AND CLINICAL IMPLICATIONS

The current study by Han *et al*[1] offers significant and pertinent findings. Their research exemplifies one of the most comprehensive efforts to assess and link anatomical and functional abnormalities of the cornea in diabetic patients experiencing DED. The microstructural alterations were strongly associated with symptom severity, highlighting the significance of corneal neuropathy as a pathophysiological contributor to ocular surface illness in diabetes, rather than merely a coincidental relationship.

In addition to the research conducted by Han *et al*[1] supplementary studies are appearing that bolster this viewpoint. Elevated levels of inflammatory biomarkers, including lactoferrin (LF), indicative of both systemic and ocular immune activity, have been observed in T2D patients with dry eye syndrome, associating with glycemic regulation and systemic inflammatory condition[2]. Bioinformatics research has demonstrated molecular interactions between T2D and autoimmune disorders as Sjögren's syndrome, so suggesting immunological dysregulation in the same pathways contributing to ocular surface damage[3]. Bibliometric analyses indicate that the domain of diabetes-associated dry eye is swiftly evolving, with oxidative stress, inflammation, and corneal neuropathy frequently identified as important mechanistic themes[4].

Collectively, these findings establish a persuasive argument that DED in T2D is both clinically significant and mechanistically intricate. For the practicing clinician, this indicates that assessment of the ocular surface must be incorporated into holistic diabetes therapy, extending beyond just retinopathy monitoring. It presents promising opportunities for biomarker identification, mechanistic investigation, and treatment development for the researcher. This article will place the findings of Han *et al*[1] within the broader scientific and clinical landscape, addressing corneal neuropathy, inflammatory biomarkers, molecular interactions with autoimmunity, and global research trends. It will also underscore existing limitations and delineate future paths, stressing the necessity for multidisciplinary collaboration and precision medicine strategies to more effectively tackle this hidden yet significant challenge.

Chronic hyperglycemia promotes metabolic stress, leading to corneal nerve loss, immunological activation, and tear-film instability. Metabolic dysfunction [insulin shortage, high glycated hemoglobin (HbA1c)] induces oxidative stress and mitochondrial damage, whereas immunological dysregulation exacerbates cytokine-mediated inflammation at the ocular surface. The neurodegenerative and inflammatory pathways interact reciprocally, leading to compromised epithelial integrity, diminished reflex tearing, and advancing ocular surface disease. This interconnected network elucidates how diabetes dry eye manifests as a multisystem illness rather than a singular ocular ailment.

INSIGHTS AND IMPLICATIONS

The acknowledgment that T2D predisposes individuals to DED has been established for some time; however, the systematic exploration of the molecular relationship between these illnesses has only just commenced. The research conducted by Han *et al*[1] offers one of the most thorough clinical validations of this correlation. The authors performed a retrospective observational research involving 81 patients with T2D, of which 27 were diagnosed with concomitant DED. They compared these patients to diabetic persons without DED and a control group of healthy subjects. Corneal staining exhibited no significant differences between groups, indicating that functional tear instability occurs prior to evident epithelial breakdown. Corneal confocal microscopy (CCM) demonstrated significant corneal neuropathic alterations: Nerve density, length, and quantity were decreased, tortuosity was elevated.

The authors illustrate that DED in diabetes is not solely attributable to lacrimal hyposecretion or meibomian gland dysfunction, but is closely associated with corneal nerve degeneration. This substantiates the notion of DED in diabetes as a neuro-epithelial disorder, wherein diminished corneal innervation results in decreased reflex tearing, poor epithelial trophism, and ultimately, instability of the tear film. Secondly, the identified relationships suggest the potential for employing CCM-derived measures - specifically nerve density and tortuosity - as objective biomarkers for disease severity and progression.

Corneal nerve changes identified in T2D are aligned with a wider range of diabetic consequences. Peripheral neuropathy, for instance, results from oxidative stress generated by persistent hyperglycemia, mitochondrial dysfunction, and microvascular damage, which all hinder axonal transit and facilitate neurodegeneration. Numerous previous investigations have indicated that CCM parameters are associated with both ocular surface symptoms and systemic neuropathic burden, underscoring their potential as a surrogate marker for widespread neuronal injury. Multivariate identified age, duration of diabetes, lacrimal gland dysfunction, insulin deficiency, and inadequate glycemic control (both fasting glucose and HbA1c) as independent risk variables. This underscores the multifaceted characteristics of diabetic ocular surface illness, wherein metabolic, secretory, and neurodegenerative processes intersect.

BIOMARKERS AND INFLAMMATION

Han *et al*[1] firmly established that structural corneal neuropathy is a characteristic hallmark of DED in T2D. However, neuropathy is not the sole pathogenic contributor. Both systemic and ocular inflammation significantly influences the microenvironment of the ocular surface. Recent research has prominently focused on identifying biomarkers that indicate this inflammatory condition and can function as diagnostic or prognostic instruments.

Alhalwani *et al*[2] have progressed this domain by investigating plasma LF in individuals with T2D and DED. LF is an iron-binding glycoprotein produced by exocrine glands and neutrophils, with significant antibacterial and anti-inflammatory effects. Decreased LF levels in tears have historically been correlated with DED; however, Alhalwani *et al*[2] broadened this notion by examining systemic plasma LF concentrations and their association with inflammatory status in T2D patients with DED.

Their findings indicated that individuals with T2D-DED demonstrated markedly increased plasma LF levels relative to healthy controls, with favorable connections to glycated hemoglobin and inflammatory markers, including C-reactive protein (CRP). Furthermore, the LF-to-neutrophil ratio, an innovative metric developed in the study, had a substantial correlation with illness severity. From a molecular standpoint, the increase of LF in T2D-DED patients may signify a compensatory anti-inflammatory reaction, indicative of persistent immune activation within the diabetic environment. Hyperglycemia triggers oxidative stress, enhances neutrophil activation, and compromises epithelial tight junctions, resulting in persistent ocular surface inflammation.

The ramifications for clinical practice are substantial. Conventional blood indicators, including CRP, neutrophil-to-lymphocyte ratio, as well as LF and LF-to-neutrophil ratio, may enhance traditional ocular assessments in identifying people predisposed to DED. This is especially pertinent in high-traffic diabetic clinics, where ocular surface assessments are frequently overlooked. Implementing systemic biomarker screening may facilitate earlier referrals to ophthalmology, permitting therapies prior to the onset of irreparable corneal nerve injury.

Topical LF supplementation has demonstrated potential in experimental models of ocular surface illness, where it provides antioxidant and anti-inflammatory benefits while maintaining the tear film. Considering the systemic increase noted in T2D patients with DED, further research is required to ascertain if exogenous LF supplementation can reinstate local ocular homeostasis or if systemic dysregulation constrains its protective efficacy.

In addition to LF, several tear-based inflammatory biomarkers have been implicated in diabetic dry eye. Elevated concentrations of interleukin (IL)-1 β , IL-6, IL-8, and tumor necrosis factor α have been consistently reported in tears of diabetic patients with DED, reflecting nuclear factor kappa B-mediated immune activation[5]. Similarly, matrix metalloproteinase-9 (MMP-9), a degradative enzyme linked to epithelial barrier disruption, is significantly increased in diabetic DED and is already available as a point-of-care diagnostic tool[6]. Tear epidermal growth factor has been shown to possess diagnostic utility in diabetic patients with dry eye, correlating with disease severity[7]. From a therapeutic perspective, the significance of these biomarkers resides not only in their mechanistic importance but also in their capacity to inform targeted therapy. Simplifying this panel to clinically actionable markers - such as MMP-9, LF, or specific cytokines - may facilitate tailored therapy strategies that correspond with the metabolic or inflammatory profile of each patient.

MOLECULAR INTERACTIONS IN AUTOIMMUNITY

Neuropathy and inflammation are pivotal in the pathophysiology of diabetic dry eye; however, recent bioinformatics advancements indicate that these processes extend beyond diabetes, intersecting with wider immune-mediated pathways. This concept was adeptly demonstrated by Wang *et al*[3], who utilized systems biology methodologies to discern shared molecular processes between T2D and Sjögren's syndrome[3]. Sjögren's syndrome is an autoimmune condition marked by exocrine gland impairment, resulting in xerostomia and xerophthalmia, rendering it a pertinent comparator for diabetes-related dry eye.

Wang *et al*[3] discovered 16 "crosstalk genes" that exhibited differential expression in both T2D and Sjögren syndrome by the analysis of numerous gene expression datasets. Notably, genes including IL-11RA, IL-15, and Coiled-Coil-Helix-Coiled-Coil-Helix Domain Containing 10 were identified as linked to immune cell infiltration and inflammatory signaling. Pathway analysis indicated enrichment in immune-related biological processes, such as cytokine signaling and oxidative stress pathways, implying that dysregulated immune responses constitute a common link between metabolic and autoimmune ocular surface diseases. Notably, predictive models that include these crosstalk genes exhibited high classification accuracy in differentiating disease samples from control samples, highlighting their diagnostic potential.

Hyperglycemia-induced oxidative stress and metabolic dysfunction foster a persistent inflammatory milieu, whereas in Sjögren's syndrome, autoreactive lymphocytes attack the lacrimal and salivary glands. In both scenarios, immune-mediated injury impairs glandular output and destabilizes the ocular surface.

This molecular interplay has numerous clinical implications. Initially, it suggests that patients with T2D and severe DED may be especially vulnerable to concurrent autoimmune characteristics, requiring thorough assessment for associated SS or similar conditions. Secondly, it emphasizes immune-modulatory medicines as a prospective advancement in the management of diabetes dry eye. Current treatments mostly emphasize tear substitutes and anti-inflammatory eye drops; however, targeting specific cytokine pathways, such as IL-15 or Janus kinase-signal transducer and activator of transcription inhibition, may present novel therapeutic options. Third, it emphasizes the significance of multidisciplinary collaboration among endocrinology, rheumatology, and ophthalmology in the management of individuals with intricate ocular surface disease.

LIMITATIONS

A further constraint resides in the intricacy of symptom evaluation. In diabetes, corneal hypoesthesia and neuropathic pain can disrupt the correlation between signs and symptoms, occasionally resulting in a paradox where structural damage seems extensive while subjective complaints are subdued, or conversely, where symptoms are pronounced but objective assessments appear relatively benign. The divergence between patient-reported outcomes and structural biomarkers hampers diagnosis and monitoring. In the absence of composite tools designed for the diabetic population that combine neurosensory status with tear and epithelial function, doctors may misinterpret the severity and course of the disease.

Imaging techniques, although potent, often pose challenges. *In vivo* ocular confocal imaging has become essential for visualizing subbasal nerve changes; yet, standardization remains inadequate. Diverse groups employ distinct techniques for picture capture, field selection, and nerve tracing, resulting in inconsistent outcomes among investigations. The sampling location of images, whether central or near the whorl region, the method of analysis, whether human or automated, and the criteria established for distinguishing normal from aberrant significantly affect the results. This variability restricts the capacity of CCM to function as a universal diagnostic or predictive instrument in clinical practice.

The function of biomarkers, while encouraging, remains inadequately defined. The evidence that plasma LF and its ratio to neutrophils link with HbA1c and inflammatory burden is intriguing[2]; nevertheless, validation in larger cohorts and prospective designs is still required. Currently, the majority of biomarker data are correlational rather than predictive, and their effectiveness in assessing therapy response remains largely unverified. Additionally, systemic biomarkers may be confounded by comorbidities prevalent in diabetes, whereas tear-based indicators are susceptible to technical heterogeneity during collection. In the absence of longitudinal and multi-analyte validation, it is premature to deem these biomarkers suitable for incorporation into standard clinical screening.

The identification of molecular commonalities between T2D and Sjögren's syndrome underscores compelling immunological pathways; nevertheless, experimental validation is absent[3]. The relationship between the immunological profiles revealed in bioinformatics research and their direct role in mediating corneal nerve injury and lacrimal gland dysfunction *in vivo* remains ambiguous, as does their potential usefulness as indicators of systemic dysregulation. This ambiguity complicates the formulation of targeted therapy, since patients with primarily metabolic-ischemic neuropathy may exhibit divergent responses to immune-modulatory treatments in contrast to those with para-autoimmune characteristics.

The inadequate incorporation of ocular surface evaluations into diabetes management protocols is equally troubling. Contemporary management continues to prioritize retinopathy, nephropathy, and foot assessments, whereas the ocular surface is afforded minimal systematic scrutiny.

The reproducibility of CCM is an acknowledged problem. Interobserver heterogeneity in nerve selection, imaging field selection, and analytical software may influence quantitative measurements, whereas normative thresholds differ among institutions. Concise automated algorithms and defined acquisition techniques are essential to enhance consistency and promote wider clinical implementation.

Although other potential strategies are emerging - including neurotrophic medicines like topical insulin or nerve growth factor analogues, as well as antioxidants and LF supplements - substantial randomized trials in diabetes dry eye

remain limited. The majority of research are exploratory in nature, characterized by small sample sizes and insufficient follow-up. This results in depending on generic dry eye treatments, which may insufficiently tackle the neuropathic and metabolic aspects of the condition.

Although significant advancements have been made, research on diabetes dry eye is still constrained by inadequate study designs, methodological variability, deficiencies in mechanistic validation, and insufficient acknowledgment in clinical practice. Overcoming these problems necessitates synchronized, multicenter initiatives that standardize diagnostic techniques, authenticate biomarkers, and evaluate focused therapies through meticulously structured clinical trials. Only through these efforts can the discipline transition from descriptive correlation to evidence-based incorporation into standard diabetic management.

Although the findings by Han *et al*[1] offer significant clinical insight, their study is limited by its retrospective single-center design and a relatively small sample size. These limitations limit generalizability and underscore the necessity for prospective, multicenter validation to ascertain the diagnostic significance of corneal nerve measurements in diabetic DED.

PROSPECTIVE OUTLOOKS AND ADVANCEMENTS

The future of this discipline will hinge on the integration of structural imaging, molecular biomarkers, and systemic disease monitoring into a cohesive framework for diagnosis and management, addressing the previously mentioned constraints. This integration corresponds with the increasing focus on precision medicine, wherein individual risk profiles inform early identification, targeted intervention, and tailored follow-up.

The enhancement of corneal nerve measurements as surrogate indicators of systemic disease is a highly promising avenue. Corneal confocal microscopy, albeit its variability, possesses the capacity to function as a minimally invasive biomarker for diabetic neuropathy. Automated image analysis techniques are advancing swiftly, and machine learning methods can facilitate the standardization of measurements for nerve density, length, and tortuosity across several centers. With sufficient confirmation, these criteria might be integrated into standard diabetes assessments, enhancing retinal imaging and enabling clinicians to identify early neurodegenerative alterations well in advance of vision-threatening problems.

LF, when integrated with additional markers like MMP-9, CRP, or the neutrophil-to-lymphocyte ratio, may enhance multi-analyte signatures that forecast patients at heightened risk of ocular surface degeneration. Future research should assess if these panels can categorize patients for targeted therapies, similar to how lipid profiles inform cardiovascular prevention.

The recognition of immunological cross-talk between T2D and Sjögren's syndrome emphasizes the necessity for molecularly characterized endotypes[3]. Patients exhibiting a primarily immune-inflammatory profile may respond favorably to therapy aimed at cytokine pathways, whereas individuals with metabolic or oxidative stress-induced conditions may achieve better outcomes with antioxidants or mitochondrial stabilizers. Progress in bioinformatics, alongside extensive biobanking and omics profiling, will be essential in delineating these endotypes and matching patients with suitable therapies.

Utilizing extensive epidemiological datasets and machine learning techniques, researchers are developing predicting algorithms to detect high-risk patients based on demographic, systemic, and ocular factors[4]. The incorporation of these models into diabetic clinics may enable non-specialists to effectively screen for DED risk, facilitating timely referrals to ophthalmology.

Topical neurotrophic drugs, including as nerve growth factor analogues and insulin-based therapy, are under investigation for their potential to rebuild corneal nerves and restore ocular feeling. Oral nutraceuticals possessing antioxidant capabilities may contribute to the mitigation of oxidative stress, a primary factor in neuropathy and epithelial dysfunction. LF supplementation, whether topical or systemic, is a promising option; nonetheless, extensive controlled trials are required to validate its effectiveness. Concurrently, sophisticated drug delivery systems - such as nanocarriers or sustained-release ocular inserts - may enhance the bioavailability of therapeutic drugs while alleviating treatment burdens.

Future advancements will depend on interdisciplinary cooperation among endocrinologists, ophthalmologists, immunologists, and data scientists. By integrating clinical experience with improved imaging, molecular profiling, and computational modeling, the field is set to elevate diabetes dry eye from an underappreciated consequence to a paradigm of integrated, precision-based therapy. Upcoming worldwide screening strategies will likely combine ocular imaging with systemic biomarkers. Integrating CCM-derived nerve metrics with blood or tear inflammatory indicators may facilitate risk-stratified, population-level screening models, thereby enhancing early identification and tailored management in diabetic ocular surface disease.

CONCLUSION

The research conducted by Han *et al*[1] offers substantial evidence that corneal neuropathy is a definitive and quantifiable element of dry eye in individuals with T2D. Their amalgamation of clinical symptom scores, tear function evaluations, and *in vivo* confocal microscopy demonstrates that the ocular surface indicates both local tear film instability and systemic neurovascular impairment. When considered in conjunction with biomarker studies indicating inflammatory signatures like elevated LF[2], bioinformatics analyses revealing immune cross-talk with Sjögren's syndrome[3], and bibliometric assessments validating the global momentum of this research domain[4], a coherent understanding is established.

Diabetic dry eye is a complex condition that connects metabolic failure, immunological dysregulation, and neurodegeneration.

The clinical message holds similar significance. Evidence supports the integration of ocular surface evaluations into comprehensive diabetes management for practicing ophthalmologists and diabetologists. Incorporating the assessment of dry eye symptoms, tear stability measurement, and, when feasible, evaluating corneal nerve integrity should be integrated into standard follow-up procedures, alongside existing protocols for retinopathy and neuropathy.

Simultaneously, prudence is advisable. Present evidence primarily originates from small, cross-sectional research employing diverse approaches. Biomarkers hold potential but necessitate longitudinal confirmation prior to their reliable application in risk categorization or treatment monitoring. Imaging modalities like corneal confocal microscopy require standardized methods and worldwide reference values for broad clinical implementation. Failure to resolve these deficiencies will result in the continued limitation of translation into practical application.

Anticipating future developments, the domain is poised to transition from descriptive research to precision medicine. Progress in artificial intelligence, multi-omic profiling, and regenerative medicines is expected to enhance diagnostic processes and broaden treatment possibilities. Aligning systemic biomarkers with ocular imaging and tailored therapies, diabetic dry eye may exemplify the integration of ophthalmology with comprehensive chronic illness management.

Dry eye in T2D is not simply a localized ocular surface condition but rather a significant indicator of systemic disease. Identifying and addressing it necessitates both attentiveness and creativity. If physicians and researchers adopt this integrated perspective, corneal neuropathy and related tear film instability may transform from mere complications to significant markers that inform the future of multidisciplinary diabetic management.

FOOTNOTES

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