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Review Article

Ocular and cutaneous rosacea: early diagnosis and comprehensive approach

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ABSTRACT

Rosacea is a common chronic inflammatory disease, traditionally classified as a purely dermatological disorder. However, ocular involvement (ocular rosacea) occurs in up to 58% of patients and frequently precedes dermal manifestations, underscoring the need for early diagnosis and a transdisciplinary approach. Objective of the study was to analyze the common pathophysiological aspects, clinical criteria for early diagnosis, and current strategies for the comprehensive management of cutaneous and ocular rosacea. Literature review of scientific papers published in biomedical databases, focusing on the immunopathology of the disease, clinical manifestations according to current phenotypes, and topical, systemic, and light-based therapeutic profiles. The pathophysiology of rosacea is driven by a dysregulation of innate immunity (overexpression of LL-37 cathelicidin) and neurovascular dysfunction. Ocular rosacea shares this inflammatory cascade, manifesting primarily as meibomian gland dysfunction (MGD), blefaritis, and evaporative dry eye. Early diagnosis is crucial to prevent severe sequelae such as corneal neovascularization. In the therapeutic field, subantimicrobial-dose tetracyclines (doxycycline 40 mg) and topical 1% ivermectin stand out for their anti-inflammatory efficacy, while intense pulsed light (IPL) emerges as an effective dual alternative to simultaneously treat facial erythema and eyelid alteration. Rosacea is a complex-local pathology that requires transcending the boundary of isolated dermatology. A proper literature review of the evidence shows that cooperative work between dermatology and ophthalmology optimizes symptom control, prevents the progression of ocular damage, and mitigates the negative impact on the patient's quality of life.

Keywords: Ocular rosacea, Cutaneous rosacea, Meibomian glands

INTRODUCTION

Rosacea is a chronic, multi-factorial inflammatory disease that predominantly affects the centrofacial region.[1] Historically, it was classified and managed almost exclusively as a cosmetic dermatosis or a disorder limited to the superficial dermal vasculature.[2] However, contemporary scientific understanding has radically transformed this paradigm, positioning rosacea as a complex neuroimmune disorder with profound systemic implications and an exceedingly high morbidity on the ocular surface.[3] Despite advances in its characterization, the border between purely cutaneous manifestations and ocular involvement has historically fragmented patient care, rendering invisible a clinical correlation that shares the very same molecular inflammatory signature.[4]

The global prevalence of cutaneous rosacea ranges between 5% and 10% in the general population, showing an epidemiological predilection for individuals with fair skin phototypes and between the ages of 30 and 50.[5] Nonetheless, these statistics suffer from a massive underdiagnosis bias when the spectrum of ocular rosacea is analyzed.[6] It is estimated that up to 58% of patients diagnosed with dermatological rosacea present some degree of ocular involvement.[6] The true challenge for the

healthcare system and clinical practice lies in the fact that in approximately 20% of cases, ocular signs and symptoms precede traditional cutaneous manifestations-such as persistent erythema, papules, or pustules by months or even years.[7] This chronological asynchrony usually results in patients wandering through ophthalmological consultations receiving misdiagnoses of chronic conjunctivitis or nonspecific dry eye syndrome, thereby delaying the introduction of effective targeted therapy.[7]

From a molecular perspective, cutaneous and ocular rosacea should not be understood as two independent entities, but rather as the expression of the same pathophysiological dysregulation in different tissues.[8] The common immunological cascade is driven by an aberrant activation of the innate immune system, where the primary event involves the overexpression of Toll-like receptors 2 (TLR2) in both facial keratinocytes and corneal and conjunctival epithelial cells.[8] The activation of TLR2 generates a massive increase in the synthesis and secretion of kallikrein 5 (KLK5), a tissue serine protease responsible for processing the precursor of cathelicidin, resulting in the excessive production of the active pro-inflammatory and angiogenic peptide LL-37.[9]

The LL-37 peptide acts as a potent chemoattractant that perpetuates the inflammatory microvascular cellular infiltrate, but also directly induces the expression of vascular endothelial growth factor (VEGF).[9] In the skin, this clinically translates into erythema and telangiectasia; within the ocular territory, it stimulates corneal neovascularization and inflammation of the eyelid stroma. Added to this immune component is a marked neurovascular dysfunction modulated by the activation of transient receptor potential vanilloid channels (TRPV1 and TRPV4) in afferent nerve fibers.[10] Their stimulation by environmental factors such as ultraviolet radiation, sudden temperature changes, or the presence of commensal microorganisms like Demodex triggers the release of vasoactive neuropeptides responsible for paroxysmal facial and ocular flushing, as well as symptoms of burning and discomfort.[10]

In the ocular architecture, this chronic inflammatory microenvironment directly impacts the homeostasis of the Meibomian glands, which are modified lipid structures located in the tarsal plates.[3] Inflammation mediated by cytokines alters the keratinization process of the glandular duct, inducing obstruction of the terminal orifice and subsequent atrophy of the acinar tissue (Meibomian gland dysfunction or MGD).[3] As the quantity and quality of the secreted meibum are altered, the tear film loses its protective lipid component, accelerating tear water evaporation and establishing a severe evaporative dry eye condition.[4] If this vicious cycle of inflammation, tear instability, and hyperosmolarity is not interrupted early, the patient is exposed to catastrophic corneal complications, including superficial punctate keratitis, ulcerations, stromal scarring, and progressive loss of visual acuity.[6]

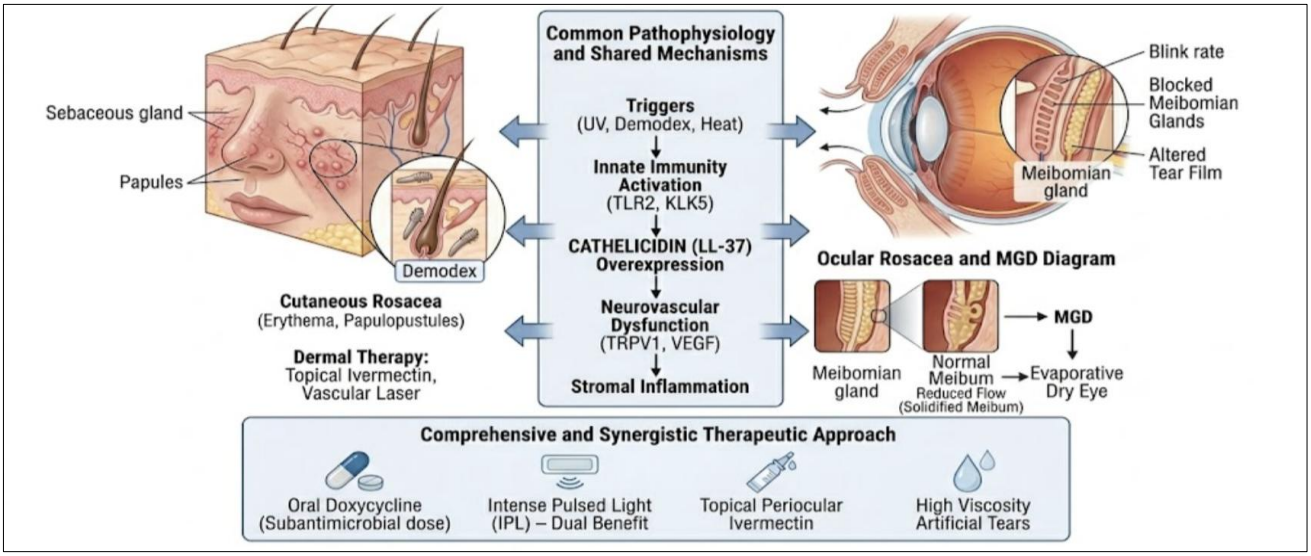

Figure 1: Pathophysiology and comprensive management of ocular and cutaneous rosácea.[3]

METHODS

To carry out this literature review, an exhaustive and detailed search of the scientific literature published in high-impact platforms with recognized methodological rigor within the health sciences field was performed. The biomedical databases consulted included PubMed/ MEDLINE and web of science (WoS), which were strategically complemented by a manual screening of clinical practice guidelines from international societies and several sources of high medical and ophthalmological relevance.

The search strategy was structured by combining health sciences descriptors and Boolean operators, using specific terms in English and Spanish such as: rosacea, ocular

rosacea, meibomian gland dysfunction, blepharitis, dry eye, and intense pulsed light. In order to ensure that the compiled evidence remained fully updated, preference was given to publications from the last ten years, including controlled clinical trials and longitudinal observational studies, as well as narrative reviews and expert consensus articles that addressed the cutaneous-ocular interface of the disease in an integrated manner.

The selection and screening process of the information was executed by applying strict criteria of scientific pertinence and theoretical coherence with the objectives established in this paper. Single-case reports, editorials, or short communications that did not provide reproducible data regarding shared pathophysiological mechanisms or dual therapeutic interventions were excluded. After an initial critical analysis of titles and abstracts, the full texts of potentially eligible manuscripts were retrieved. Ultimately, a total of 35 studies that strictly met the standards of scientific quality and clinical relevance were selected and analyzed in depth to structure the conceptual body, results, and conclusions of this review.

RESULTS

Shared molecular pathophysiology and inflammatory biomarkers

The compiled findings comprehensively demonstrate that cutaneous and ocular rosacea are not independent disease entities, but rather distinct tissue manifestations sharing the exact same immunological and neurovascular signature.[11] Innate immunity dysregulation represents the core of the chronic inflammatory process on both facial skin and the ocular surface. The primary event is mediated by a marked overexpression of Toll-like receptors 2 (TLR2) in epidermal keratinocytes as well as in corneal and conjunctival epithelial cells.[12] Upon aberrant activation by environmental or microbiological triggers, these receptors induce a massive increase in kallikrein 5 (KLK5) secretion, which subsequently cleaves the precursor of cathelicidin to release the biologically active peptide LL-37.[13]

This peptide exerts a highly damaging pleiotropic action: it directly triggers mast cell degranulation and promotes the expression of vascular endothelial growth factor (VEGF), perpetuating both the inflammatory cellular infiltrate and the development of facial telangiectasias and corneal neovascularization.[14] Within the ocular territory, this chronically active immunological cascade destabilizes surface epithelial cellular junctions and alters the lipid and protein homeostasis of tears.[15]

Early diagnosis based on clinical phenotypes

The transition from the classic subtypical classification to a modern clinical phenotype-based approach has allowed timely identification of patients at high risk for ophthalmological complications.[16] The early diagnosis of ocular rosacea represents a latent medical urgency because manifestations in the eyelids and tear film frequently precede obvious skin lesions. Delayed detection favors the development of MGD, characterized by hyperkeratinization of the excretory duct and subsequent solidification of the meibum, blocking the outflow of essential lipids into the tear film.[17] This triggers a vicious cycle of tear instability, severe evaporative dry eye, and ocular surface hyperosmolarity.[15]

Comprehensive, synergistic, and advanced therapeutic approach

The long-term management of rosacea requires a simultaneous transdisciplinary strategy that targets inflammatory pathways while repairing damaged tissue barriers. Subantimicrobial-dose tetracyclines (modified-release doxycycline at 40 mg daily) operate by drastically reducing matrix metalloproteinase (MMP-9) activity and cytokine concentrations in both the skin and the tear microenvironment, restoring normal meibum viscosity without inducing bacterial resistance or altering the digestive microbiome.[18]

At the topical level, 1% ivermectin cream substantially decreases demodex mite burden, lessening perifollicular inflammation in both facial skin and eyelids. Finally, intense pulsed light (IPL) has established itself as the dual physical technology par excellence; its light energy is selectively absorbed by hemoglobin within facial and eyelid margin telangiectasias, resulting in photothermolysis of abnormal vessels, inducing therapeutic heating that liquefies impacted lipids within the Meibomian glands, and decreasing parasitic load through direct thermal stress.[20]

Table 1: Molecular correlation, biomarkers, and shared pathophysiological targets in the cutaneous-ocular interface of rosacea.

Pathophysiological pathway	Mechanisms at the cutaneous level	Mechanisms at the ocular level	Key biomarkers	Clinical impact
Aberrant innate immunity	TLR2 overexpression in keratinocytes; exogenous activation by *Demodex* and UV radiation.[12]	Epithelial activation in cornea and conjunctiva; tear glycocalyx degradation.[15]	TLR2, KLK5, Cathelicidin (LL-37)	Loss of epithelial barrier integrity, neutrophil infiltration

Continued.

Pathophysiological pathway	Mechanisms at the cutaneous level	Mechanisms at the ocular level	Key biomarkers	Clinical impact
Pro-inflammatory cytokine cascade	Dermal accumulation of macrophages and lymphocytes; sustained matrix metalloproteinase secretion.[11]	Eyelid stroma inflammation; degradation of corneal extracellular matrix.[13]	IL-1beta, TNF-alpha, IL-8, MMP-9	Meibomian gland atrophy, corneal ulceration.[14]
Neurovascular dysfunction and angiogenesis	Hypersensitivity of TRPV1/TRPV4 channels; substance P and nitric oxide release.[13]	Stimulation of the corneal nerve plexus; limbal vascular proliferation into stroma.[14]	TRPV1, TRPV4, VEGF, neuropeptides	Persistent erythema, flushing, corneal neovascularization, photophobia

Table 2: Comprehensive clinical spectrum and early screening criteria for cutaneous-ocular phenotypic evaluation.[15]

Anatomical region	Primary clinical signs	Reported symptoms	Early diagnosis tools	Severity/expected sequelae
Centrofacial and periocular	Persistent erythema, telangiectasias, papules, pustules, phymatous changes.[16]	Cutaneous burning, pruritus, paroxysmal local heat (flushing).[13]	Sequential clinical photography, high-resolution dermoscopy	Dermal fibrosis, permanent aesthetic distortion, rhinophyma
Eyelid margin	Anterior and posterior blepharitis, marginal telangiectasias, orifice capping.[17]	Foreign body sensation, eyelid heaviness, pruritus	Slit-lamp examination, infrared meibography.[17]	Recurrent chalazion, madarosis, trichiasis, acinar atrophy
Ocular surface and cornea	Conjunctival hyperemia, superficial punctate keratitis, stromal infiltrates.[15]	Severe photophobia, paradoxical tearing, ocular pain	Tear break-up time (TBUT), fluorescein staining.[16]	Neovascularization, stromal thinning, corneal perforation.[15]

Table 3: Comprehensive management algorithm, synergistic framework, and dual therapeutic efficacy profiles for cutaneous and ocular rosacea.[17]

Treatment modality	Main mechanism of action	Dosage and route of administration	Efficacy in cutaneous manifestations	Efficacy in ocular manifestations
Modified-release doxycycline	Systemic immunomodulator; inhibits MMP-9 cellular degradation and reduces cytokines	40 mg orally once daily (subanti-microbial dose)	Significant reduction of inflammatory papules and pustules	Alleviation of eyelid margin inflammation; improves tear film
Topical 1% ivermectin	Selective antiparasitic against demodex; inhibits cellular signaling pathways	Cream applied once daily to affected facial regions	Rapid clearance of papulo-pustular lesions and facial erythema	Indirect reduction of parasite migration toward the eyelashes
Intense pulsed light (IPL)	Selective photothermolysis of telangiectasias; thermal stimulation of glands	Protocol of 3 to 4 sessions (500-1200 nm spectrum) periocular and facial	Attenuation of persistent central erythema and telangiectasias	Liquefaction of stagnant meibum, increased TBUT, and dry eye relief

DISCUSSION

The phenotypic and molecular correlation between the dermal and ophthalmic manifestations of rosacea demonstrates the urgent need to abandon the fragmented clinical approach that has traditionally prevailed in medical practice. Historically, clinical attention was centered on the clearance of papules and centrofacial erythema, relegating ocular symptomatology to a secondary plane or treating it in isolation as an unrelated disorder of the ocular surface. The findings analyzed in this literature review conclusively demonstrate that the

inflammatory axis driven by the dysregulation of innate immunity (TLR2/KLK5/LL-37) operates simultaneously in both tissues, which renders mutual screening protocols between dermatology and ophthalmology mandatory.[21]

A critical point of debate in current literature involves the asynchrony with which clinical manifestations present. The fact that ocular signs precede cutaneous lesions in up to 20% of cases poses a major diagnostic challenge for ophthalmologists. Faced with cases of refractory marginal blepharitis, recurrent chalazion, or meibomian gland dysfunction in young or middle-aged patients, a meticulous evaluation of the skin in the glabella and malar regions is required to look for subtle telangiectasias or transient erythema.[22] Detecting the disease in this pre-dermatological phase mitigates progression toward severe evaporative dry eye and prevents corneal neovascularization, a sequela that irreversibly compromises visual acuity due to residual stromal opacity.[23]

In the therapeutic arena, current evidence supports a paradigm shift toward systemic immunomodulation and the use of advanced physical technologies.[24] The use of tetracyclines, specifically subantimicrobial-dose doxycycline at 40 mg daily, represents a fundamental pillar with an optimal safety profile.[25] By acting exclusively as an inhibitor of matrix metalloproteinases and reducing the synthesis of pro-inflammatory cytokines, this dosing regimen stabilizes the tear film and liquefies the meibum without the risks associated with inducing bacterial resistance or causing gastrointestinal microbiome dysbiosis, which are typical of conventional antibiotic doses of 100 or 200 mg.[26]

Furthermore, the incorporation of IPL represents the most disruptive technological advance for the synergistic management of this pathology.[27] Reviewed clinical studies agree that IPL offers an irreplaceable dual therapeutic response.[28] By producing selective photothermolysis of the abnormal telangiectasic vessels supplying the eyelid margin and facial skin, it interrupts the continuous flow of inflammatory mediators and cytokines toward the ocular surface.[29] Simultaneously, the heat generated during the procedure transfers thermal energy to the eyelids, facilitating the melting of impacted meibum within the meibomian glands and optimizing its expression.[30] This physical effect, combined with the drastic reduction in demodex parasite burden via thermal stress, breaks the vicious cycle of inflammation and glandular obstruction.[31]

Nevertheless, the implementation of these advanced therapies demands strict protocolization.[32] The use of topical 1% ivermectin, while highly effective in controlling demodex proliferation on facial skin, must be applied with extreme caution in the periocular region to avoid direct conjunctival irritation.[33] This reinforces the notion that long-term treatment success does not rely on a single intervention, but on a combined algorithm that includes systemic immunomodulatory therapy, meticulous eyelid hygiene, preservative-free high-viscosity artificial tears, and scheduled IPL sessions.[34,35]

CONCLUSION

Rosacea is a systemic-local inflammatory entity driven by a common neuroimmune pathway (the TLR2/KLK5/LL-37 axis) that simultaneously compromises facial skin and the ocular surface, necessitating the abandonment of separate, single-specialty approaches. Ocular rosacea frequently precedes cutaneous lesions, meaning that early interdisciplinary screening is crucial to prevent progressive meibomian gland atrophy, severe evaporative dry eye, and irreversible corneal complications like stromal neovascularization. Modern management must utilize synergistic dual therapies, highlighted by subantimicrobial-dose doxycycline (40 mg) for safe systemic immunomodulation, topical 1% ivermectin for demodex control, and IPL to simultaneously resolve facial telangiectasias and Meibomian gland obstruction.

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