



Integrated Multidisciplinary Management of Pediatric Diabetic Ketoacidosis: Nursing, Emergency Medical Services, Social Work, and Safety Systems Across the Care Continuum

Khaled Lafi Abdulkarim Al-Mutairi ⁽¹⁾, Fahad Bunaydir Shulaywih Almutairi ⁽¹⁾, Mjaed Metaib Atek Almutairi ⁽¹⁾, Fatimah Mohammed Salh Alamri ⁽²⁾, Najmah Abdullah Aldandan ⁽³⁾, Abdulaziz Mohammed Aqdi ⁽⁴⁾, Abdullah Madsus Alanazi ⁽⁵⁾, Bader Abdulhamid Mohammed Alharbi ⁽⁵⁾, Mohammed Faris Alqahtani ⁽⁵⁾, Fahad Abdul Aziz Alsaedi ⁽⁶⁾, Zamzam Ali Sahli ⁽⁷⁾

(1) Saudi Red Crescent Authority, Saudi Arabia,

(2) Erada Mental Health Complex, Ministry of Health, Saudi Arabia,

(3) Health Center in Al-Ahsa City, Ministry of Health, Saudi Arabia,

(4) Jazan Cluster, Ministry of Health, Saudi Arabia,

(5) Al-Ahsa Health Cluster, Ministry of Health, Saudi Arabia,

(6) Al-Ahsa Health Cluster – Maternity and Children's Hospital, Ministry of Health, Saudi Arabia,

(7) Irada Hospital For Mental Health, Ministry of Health, Saudi Arabia

Abstract

Background: Contact lens (CL) use has grown globally, offering cosmetic and therapeutic benefits but introducing risks of ocular complications due to hypoxia, microbial contamination, and tear film disruption. These complications range from mild irritation to vision-threatening infections such as microbial keratitis.

Aim: To review contact lens-related complications, their pathophysiology, and the role of nursing in prevention, early detection, and patient education.

Methods: A comprehensive literature-based analysis was conducted, synthesizing evidence on CL-induced corneal and conjunctival disorders, functional tear-lens interactions, and clinical management strategies.

Results: CL wear alters ocular physiology, reducing oxygen transmission and destabilizing tear film, which predisposes epithelial edema, microcysts, abrasions, and inflammatory conditions. Infectious keratitis, particularly due to *Pseudomonas aeruginosa* and *Acanthamoeba*, remains the most severe complication, often linked to poor hygiene and water exposure. Conjunctival disorders such as allergic conjunctivitis and giant papillary conjunctivitis further compromise lens tolerance. Preventive strategies include patient education on hygiene, avoidance of overnight wear, and prompt discontinuation upon symptom onset. Nursing interventions—structured counseling, triage, and monitoring—are pivotal in reducing morbidity.

Conclusion: CL-related complications are largely preventable through adherence to hygiene protocols, individualized lens selection, and multidisciplinary care. Nurses play a critical role in patient education and early recognition of warning signs, thereby minimizing irreversible ocular damage.

Keywords: Contact lens complications, microbial keratitis, tear film, hypoxia, nursing interventions, patient education

Introduction

Contact lens (CL) use has expanded substantially over recent decades, driven by a broad range of clinical and patient-centered indications, as well as ongoing advances in lens design and material science.[1] Common indications include cosmetic enhancement, therapeutic use in selected ocular surface disorders, correction of refractive error, and the correction or control of myopia progression.[1] Globally, the number of contact lens users is large and continues to rise; estimates commonly cite approximately 140 million users worldwide.[2] This growth is also influenced by the increasing availability of lens types and modalities, enabling many patients to achieve functional vision correction without spectacles and supporting individualized preferences

regarding comfort, aesthetics, and lifestyle.[3] Despite these advantages, contact lenses remain a foreign body interface placed directly on the ocular surface, and their presence can alter the delicate balance of the corneal and conjunctival microenvironment.[4] The lens material, wearing schedule, tear film interactions, and user handling practices collectively influence ocular surface physiology, including oxygen transmission, epithelial integrity, and microbial exposure risk. Consequently, contact lens use is associated with a wide spectrum of complications, ranging from mild, reversible irritation to severe, vision-threatening infection.[4] With the rapid expansion of the contact lens market and the increasing diversity of available products, healthcare professionals involved in contact lens prescribing and

follow-up must maintain current knowledge of both benefits and risks, particularly because early recognition and timely intervention can prevent irreversible ocular morbidity.

Clinically, contact lens–related complications may be categorized in several practical ways, including infective versus non-infective etiologies and conjunctival versus corneal involvement.[5] This classification is useful because it guides the urgency of evaluation, informs differential diagnosis, and helps determine whether immediate cessation of lens wear, antimicrobial therapy, or referral to specialist care is required. Importantly, infectious complications—especially microbial keratitis—represent a time-critical subset, as delayed diagnosis and treatment can result in corneal scarring, perforation, and permanent visual impairment.[5] Corneal complications reported in association with contact lens wear include epithelial edema, microcyst formation, mechanical abrasions, superficial punctate keratitis, contact lens–related peripheral ulceration, peripheral corneal staining, sterile corneal infiltrates, corneal neovascularization, microbial keratitis, bacterial keratitis, fungal keratitis, *Acanthamoeba* keratitis, corneal warpage, and endothelial changes.[6] These complications may arise from hypoxia, mechanical trauma, inflammatory responses, toxic reactions to solutions, or direct microbial invasion, underscoring that lens-related pathology is often multifactorial. Conjunctival complications include allergic conjunctivitis, giant papillary conjunctivitis, and superior limbic keratoconjunctivitis, each of which may be linked to chronic mechanical irritation, immune hypersensitivity, or prolonged exposure to deposits and allergens on the lens surface.[6] Additional concerns involve physical damage to the lens itself and lens discoloration, which may compromise visual quality and contribute to ocular irritation or injury.[6] Although the overall incidence of certain complications has declined with improvements in lens materials, manufacturing processes, and hygiene awareness, adverse outcomes remain clinically relevant—particularly when lens care practices are inadequate or when patients continue lens wear despite symptoms.[7] Accordingly, the nursing and broader healthcare role in contact lens safety includes patient education, early symptom recognition, risk mitigation counseling, and clear escalation pathways to ensure timely evaluation and to protect patients from preventable, irreversible ocular changes.[7]

Function

The functional performance of contact lenses is inseparable from their interaction with the tear film and the ocular surface. In physiologic conditions, the corneal tear film is a dynamic, multilayered structure that lubricates the ocular surface, provides nutrients, supports epithelial integrity, and forms a smooth optical interface essential for high-quality vision.[8]

The cornea, as the principal refractive surface of the eye, relies on maintaining transparency, hydration balance, and structural regularity to preserve its optical function.[8] Contact lens wear introduces a synthetic interface onto this finely regulated system. Although lenses are designed to enhance visual function and convenience, their placement over the cornea can disrupt the native tear film, alter oxygen and metabolite exchange, and modify the biomechanics of blinking and tear distribution. These changes influence both the short-term comfort of lens wear and the long-term health of the corneal and conjunctival tissues, thereby shaping the overall efficiency, efficacy, and acceptability of contact lens therapy.[9] A key functional determinant in contact lens wear is the stability of the tear film and its ability to maintain appropriate wetting across the anterior lens surface. Once the lens is inserted, it is not mechanically “clamped” to the cornea; rather, it is retained through complex interactions involving tear film surface tension, viscosity, and lid dynamics.[10] The pre-lens tear film spreads over the lens surface with blinking, aided by tear fluid that wets the material and supports a stable optical surface.[11] In parallel, the post-lens tear film forms between the posterior lens surface and the corneal epithelium, contributing to lens positioning and to the metabolic environment of the cornea. In this setting, the tear film functions not only as a lubricating layer but also as an adhesive medium: cohesive forces among water molecules and adhesive forces between the aqueous component and the lens material help maintain lens centration and stability.[12] Experimental observations have suggested that approximately 11 grams of force may be required to displace a lens under certain conditions, illustrating the measurable mechanical contribution of tear-mediated adhesion.[12]

When the tear film overlying the lens becomes unstable or breaks, additional mechanisms of retention become more prominent. A negative pressure gradient can develop between the lens, the corneal surface, and the intervening tear layer. This pressure behaves like a “collar” at the lens edge, effectively stabilizing the lens against displacement and maintaining its position on the ocular surface. When quantified, this negative pressure has been reported to approximate 29 dynes/cm, emphasizing that tear mechanics provide not only lubrication but also a physical anchoring function.[12] Clinically, these interactions are influenced by tear quantity, tear quality, eyelid anatomy, blink completeness, and lens design (including diameter, base curve, edge configuration, and material wettability). As a result, tear film abnormalities—such as reduced tear volume or accelerated tear breakup—can impair lens stability, reduce comfort, and promote mechanical microtrauma. From an optical standpoint, the tear lens, also referred to as the post-lens tear film interface, plays an essential role in optimizing vision with

contact lenses.[13] The posterior tear layer can fill irregularities on the corneal surface, creating a more uniform refractive plane and enhancing the optical smoothness of the anterior segment.[12] In rigid lens modalities, this phenomenon can be particularly important because the rigid lens maintains its shape, while the tear layer compensates for corneal irregularities, thereby improving refractive outcomes in conditions characterized by irregular astigmatism. The refractive index difference between the cornea (approximately 1.376) and tears (approximately 1.337) is clinically relevant: because these indices are not identical, the tear layer does not perfectly neutralize all surface irregularities, and residual refractive error—especially at higher degrees of astigmatism—may persist.[14] In practical terms, this underscores why lens design, fitting accuracy, and material selection must be individualized to achieve optimal optical performance. Beyond optics and positioning, the tear–lens interface has critical implications for corneal nourishment and metabolic homeostasis. The cornea is avascular and depends on atmospheric oxygen diffusion, tear film oxygen content, and aqueous humor contribution to sustain aerobic metabolism. Contact lenses can interfere with these pathways in several ways, including reducing tear evaporation, altering tear osmolarity, acting as a barrier to oxygen delivery, trapping water and metabolic byproducts, and mechanically interacting with superficial epithelial cells.[15] The degree of physiologic impact varies by lens type, material permeability, thickness, and movement during blinking. Rigid contact lenses typically cover a substantial portion of the cornea—often described as approximately 50% to 80% of the corneal surface—while still allowing a degree of mobility that promotes tear exchange and renewal beneath the lens.[16] This tear exchange is not merely a comfort feature; it is central to meeting corneal oxygen demands and to removing waste products generated by cellular metabolism.

Under static conditions, oxygen availability in the post-lens tear film may be exhausted rapidly. Observations suggest that oxygen in the post-lens tear interface can be depleted within approximately 90 seconds with hard lenses or certain soft lenses, particularly those with lower hydration characteristics.[13] When oxygen availability decreases, corneal metabolism shifts toward less efficient anaerobic pathways, which are markedly less productive than oxygen-dependent processes. This shift is associated with accumulation of lactic acid, impaired deturgescence mechanisms, and ultimately corneal edema and haze that can degrade vision and increase susceptibility to epithelial injury.[13] In clinical practice, these physiologic changes help explain why prolonged wear, overnight use, or reduced blink dynamics can increase the risk of hypoxic complications, particularly in lenses with inadequate oxygen transmissibility. Blinking serves a

protective, compensatory mechanism by generating a “pump” effect at the lens–cornea interface. Lid pressure during blinking can transiently compress the lens, expelling a portion of the post-lens tear film and permitting fresh tear fluid to flow back under the lens as the pressure is released. This cyclic exchange supports oxygen delivery, removes metabolic waste, and helps maintain an even distribution of tears.[16] The efficiency of this pump mechanism depends on several parameters, including the volume of tear fluid behind the lens, blink frequency, and the percentage of fluid exchanged with each blink.[16] In circumstances where blinking is reduced—such as during prolonged screen use, concentration, sedation, or sleep—the protective exchange diminishes. Consequently, the cornea may experience increased hypoxic stress, greater tear film instability, and higher risk of epithelial compromise.

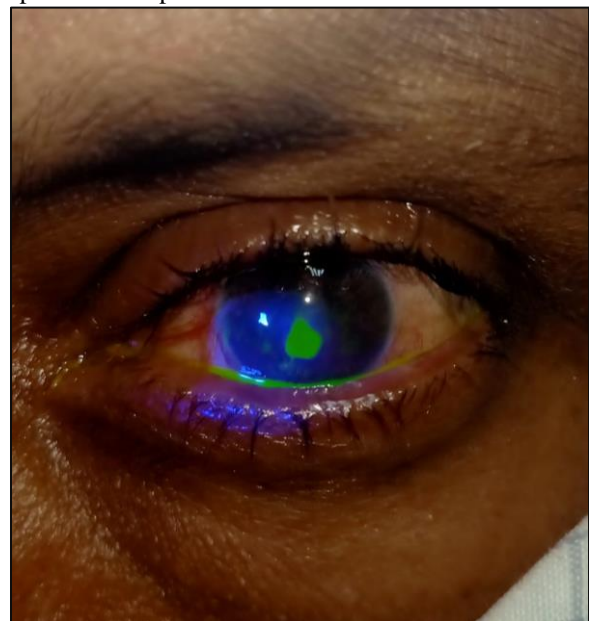


Fig. 1: Digital image of the patient depicting central epithelial defect post overnight contact lens application.

Soft contact lenses introduce unique functional considerations. Their larger diameter and close conformity to the ocular surface lead to more extensive coverage of the cornea and adjacent conjunctiva. The hydrophilic nature of many soft lenses can produce what has been described as an “aqualung effect,” in which the lens retains water and alters the microenvironment at the ocular surface.[17] Soft lenses generally exhibit less tear exchange compared with rigid lenses, and in some descriptions, oxygen delivery under soft lenses may be substantially lower than under rigid modalities, particularly when material oxygen transmissibility is limited.[17] However, the flexibility of soft lenses can allow formation of a thin capillary layer of fluid beneath the lens surface, which may provide some degree of hydration stability. At the same time, alterations in tear film dynamics—such as increased reflex tearing,

changes in blink rate, and impaired evaporation—can influence corneal deturgescence and contribute to edema, particularly when hypotonic tear conditions and metabolic waste retention coexist.[17] These physiologic interactions underscore why material selection (including high-oxygen transmissible silicone hydrogel options), appropriate replacement schedules, and careful evaluation of tear film status are central to preventing complications. Lens edge geometry also influences functional visual performance. The contact lens is enveloped by the tear film, creating a prism-shaped meniscus at the lens periphery. If the lens edge encroaches upon the pupillary zone—such as in low-riding or high-riding lenses—the prismatic effect can generate a secondary focal point on the retina, producing visual phenomena such as ghosting or edge flare.[18] While often described as a nuisance symptom, persistent edge-related optical disturbances may indicate suboptimal lens fit, decentration, or inappropriate diameter/base curve selection and can contribute to patient dissatisfaction and reduced adherence to safe wear practices.

Another major functional domain is lens integrity and surface quality over time. Contact lenses are exposed to a complex mixture of tear film components, including proteins, lipids, mucins, electrolytes, and environmental contaminants. Irregularities in lens–tear interactions can promote deposit accumulation on the lens surface, altering wettability, reducing optical clarity, and increasing frictional forces with blinking.[19] Deposits may also serve as a scaffold for microbial adherence, potentially increasing the risk of inflammatory or infectious complications. Lens spoilage or decreased functional efficiency can occur when deposits and surface degradation accumulate, and some observations suggest that deposit-related changes can compromise lens performance within months, depending on lens type and user practices.[19] Factors that contribute to deposition and spoilage include manufacturing-related surface irregularities, tear film inadequacy, lens porosity, blink irregularities, short tear breakup time, altered tear composition or volume, and changes in tear film pH.[19] Importantly, these contributors often interact: for example, reduced tear volume can accelerate tear film breakup, which increases friction, promotes surface microdamage, and facilitates further deposit accumulation, creating a cycle of discomfort and ocular surface stress. In nursing practice, understanding these functional interactions is not merely theoretical; it provides a physiologic basis for preventive education and early symptom recognition. Patients who report dryness, fluctuating vision, foreign body sensation, halos or ghost images, or discomfort that worsens with prolonged wear may be describing manifestations of tear film instability, poor lens fit, or deposit-related surface changes. Similarly, patients who experience reduced comfort during

digital device use may be encountering decreased blink rate and diminished tear exchange, increasing hypoxic and mechanical stress. By linking symptoms to underlying tear–lens dynamics, nurses and allied health professionals can deliver more targeted guidance, including reinforcement of safe wear schedules, emphasis on appropriate lens hygiene, avoidance of overnight wear when not prescribed, and prompt discontinuation of lenses with early signs of irritation or inflammation.

Overall, the function of contact lenses depends on a delicate equilibrium among tear film biomechanics, optical surface regularity, oxygen delivery, waste removal, and material compatibility. Contact lenses can enhance vision and quality of life, but they also alter corneal physiology and tear film behavior in ways that may predispose to complications if not managed carefully.[9][15] A thorough understanding of tear–lens interactions, lens positioning forces, optical contributions of the tear lens, and metabolic implications for corneal nourishment supports safer prescribing, more effective patient counseling, and earlier identification of adverse changes. When these principles are integrated into clinical education and routine monitoring, the likelihood of preserving ocular surface integrity and preventing irreversible corneal injury is significantly improved.[7]

Issues of Concern

Contact lens (CL) wear, while widely adopted for refractive correction, cosmetic purposes, and therapeutic indications, introduces a distinctive spectrum of clinical concerns because the lens constitutes an exogenous biomaterial placed directly on the ocular surface. This intimate interface between the lens, the tear film, and the corneal and conjunctival epithelium can destabilize normal ocular surface physiology, alter local immunity, and modify oxygen and metabolite exchange. Consequently, CL-related adverse events range from mild, reversible epithelial disturbances to rapidly progressive, vision-threatening infections. The clinical challenge lies not only in the breadth of potential complications but also in the frequent overlap of early symptoms across benign and severe conditions, which may delay appropriate intervention. In practice, the most consequential errors are often rooted in underestimating early warning signs, continuing lens wear despite symptoms, or assuming that discomfort is merely an expected adaptation phase. For this reason, clinicians and nurses involved in patient counseling must communicate that pain, photophobia, redness, discharge, or reduced visual acuity in a contact lens wearer warrants prompt discontinuation of lens use and professional evaluation, given the risk of irreversible corneal scarring and permanent visual impairment. Among the most significant concerns are corneal complications, which are commonly initiated by hypoxia, mechanical microtrauma, chemical toxicity, or microbial

contamination. The cornea's avascular structure renders it particularly vulnerable to any factor that diminishes oxygen delivery or impairs tear exchange beneath the lens. Epithelial edema is often one of the earliest manifestations of hypoxic stress and is typically reversible when lens wear is interrupted and oxygen availability is restored.[15] Pathophysiologically, reduced oxygenation shifts corneal metabolism toward anaerobic pathways, facilitating lactate accumulation and osmotic fluid influx, leading to transient corneal thickening and decreased transparency. Clinically, patients may describe foggy vision, halos, or fluctuating acuity that is most pronounced after prolonged wear or overnight use. Although such edema is frequently reversible, repeated or sustained hypoxia increases the likelihood of downstream sequelae, including inflammatory infiltrates, neovascularization, and longer-term endothelial alterations. Therefore, epithelial edema is concerning not only as a symptom-producing event but also as a sentinel indicator of an oxygen delivery–lens fit mismatch that requires correction.[15]

Microcysts represent another epithelial response to prolonged metabolic compromise and are most often observed with soft lenses or extended wear modalities.[20] These small epithelial inclusions reflect disturbed cell turnover and are often detected as fine dots on slit-lamp examination. Their clinical relevance is twofold. First, they may mimic inherited epithelial dystrophies, complicating diagnostic interpretation when history is incomplete. Second, their presence signifies chronic physiologic stress and suggests that the cornea is repeatedly exposed to conditions that impede normal epithelial metabolism.[20] Importantly, microcysts commonly resolve after lens discontinuation, reinforcing that early identification and modification of lens modality can prevent progression. In practice, transitioning from extended wear to daily wear, or from daily wear to gas-permeable lenses, is frequently advised to reduce chronic hypoxic exposure and improve tear exchange.[20] Mechanical epithelial injury is another major concern in CL wearers and may arise from lens edge interactions, poor lens fit, inappropriate handling techniques, or foreign material trapped under the lens. Corneal abrasions, whether induced by insertion or removal trauma or by a defect or contaminant within the lens, can produce acute pain, tearing, photophobia, and marked conjunctival injection.[21] Because abrasions compromise the epithelial barrier, they substantially increase susceptibility to infectious keratitis. Accordingly, management typically centers on immediate lens removal, epithelial protection, and topical antimicrobial therapy, with an emphasis on avoiding occlusive patching due to the heightened risk of infection under a closed, low-oxygen environment.[22] The clinical priority is to ensure complete epithelial healing before resuming lens wear and to reassess technique, hygiene, and lens integrity to prevent recurrence.[22] The concern is magnified in

contact lens wearers because even a small abrasion may serve as the entry point for aggressive organisms, particularly in settings of poor hygiene or water exposure.

Superficial punctate keratitis (SPK) is similarly common and may reflect either mechanical microtrauma from the lens surface or chemical toxicity associated with lens care systems.[23] Mechanical SPK is often linked to poor fitting lenses, inadequate tear film lubrication, or repeated friction during blinking. In contrast, chemically mediated SPK may result from preservative toxicity, failure to adequately rinse lenses after enzymatic or surfactant cleaners, or incomplete neutralization of hydrogen peroxide disinfectants.[24] The clinical presentation may include burning, stinging, foreign body sensation, and intermittent blur, often exacerbated by prolonged wear or dry environmental conditions. Coexisting ocular surface disorders such as dry eye disease and blepharitis further increase SPK risk by destabilizing the tear film and reducing epithelial resilience.[25] Because SPK is frequently reversible, the principal concern is not irreversibility but mismanagement—particularly if patients continue lens wear or repeatedly expose the ocular surface to the offending chemical agent. Standard care typically includes temporary cessation of lens wear, ocular surface lubrication, and in selected cases topical antibiotics to protect against secondary infection while epithelial integrity is restored.[25] Peripheral corneal staining patterns, particularly the classic “3 and 9 o'clock” staining, represent another clinically important manifestation of tear film disruption and corneal desiccation, most commonly associated with rigid gas-permeable lenses.[26] This staining occurs near the nasal and temporal limbus, reflecting localized drying where the tear film is interrupted or inadequately replenished. Risk factors include suboptimal lens edge design, insufficient edge lift, poor lens mobility, incomplete blinking, mechanical interference from conjunctival elevations such as pingueculae, and increased lens adherence.[26] While the staining itself may be mild, persistent epithelial compromise at the limbus can contribute to discomfort, chronic inflammation, and increased susceptibility to secondary complications. This pattern therefore demands attention to lens fitting parameters and ocular surface optimization rather than symptomatic treatment alone.[26]

Classification	Examples
Corneal infection	Microbial keratitis
Corneal inflammation	Sterile keratitis
Metabolic	Changes could be seen in epithelium, stroma and / or endothelium
Mechanical	Corneal abrasion, corneal erosion, lens binding, warpage/ refractive error changes; superior epithelial arcuate lesion, mucin balls, conjunctival epithelial flaps, ptosis, discomfort
Toxic and allergic	Papillary conjunctivitis, solution-induced corneal staining, incomplete neutralisation of peroxide, limbal stem cell deficiency
Tear film disorders and dry eye	Contact lens-induced dry eye, meibomian gland dysfunction, lid wiper epitheliopathy, lid parallel conjunctival folds, inferior closure stain, three and nine o'clock stain, dellen, dimple veil
Contact lens discomfort	Reduced lens wearing time, discomfort and eventual drop out

Fig. 2: Corneal complications.

Sterile corneal infiltrates are a further concern because they occupy a diagnostic gray zone between benign inflammatory phenomena and the early stages of microbial keratitis.[27] Sterile infiltrates generally reflect an immune-mediated response, often triggered by antigens associated with lens deposits or preservatives in cleaning solutions, leading to leukocyte migration from limbal vasculature into the anterior cornea.[24] These infiltrates may be asymptomatic and discovered incidentally during routine examinations, appearing as small focal opacities within the epithelium, subepithelial layer, or anterior stroma.[24] The principal risk is that clinicians or patients may falsely reassure themselves based on mild symptoms, despite the possibility that an infiltrate represents the earliest stage of infection. In many instances, discontinuation of the offending lens or solution leads to resolution.[28] However, careful assessment is required to exclude microbial causes, particularly when pain, photophobia, discharge, or reduced vision is present. Once the infiltrate resolves, lens wear may resume with modified care regimens, including preservative-free systems when sensitivity is suspected.[28] Peripheral corneal ulceration, sometimes described clinically as marginal keratitis, is characterized by peripheral epithelial and anterior stromal involvement, often with a preserved Bowman layer and a clinical profile that differs from centrally located microbial keratitis.[29] These lesions tend to occur near the limbus and may present as crescentic or oval excavations associated with localized thinning. Their pathogenesis is frequently linked to inflammatory responses to bacterial toxins, particularly staphylococcal products, and may coexist with lid margin disease.[30] Although peripheral ulceration may produce less dramatic symptoms than microbial keratitis, it remains concerning due to the risk of progression, tissue compromise, and diagnostic confusion. Misclassification as noninfectious without appropriate monitoring can allow microbial disease to evolve. Therefore, clinicians often adopt a cautious approach, including temporary discontinuation of lenses, treatment of associated blepharitis, and close reassessment to confirm resolution and exclude infection.

Corneal neovascularization represents a chronic and potentially sight-threatening response to sustained hypoxic stress, typically precipitated by prolonged lens wear, tight-fitting lenses, thicker materials, or insufficient oxygen transmission.[4] In physiologic terms, neovascular growth constitutes an adaptive attempt to increase oxygen delivery to hypoxic tissue; however, the presence of vessels within normally avascular corneal stroma undermines transparency and can facilitate lipid deposition, chronic inflammation, and scarring. A small degree of superficial peripheral vascularization may be observed in specific contexts such as extended wear or

therapeutic lenses; nevertheless, progression beyond limited peripheral involvement, deeper stromal penetration, or encroachment toward the visual axis is clinically concerning and often prompts changes in lens material, fit, and wear schedule.[31] Management emphasizes prevention through high oxygen transmissibility lenses, careful fitting, routine follow-up, and prompt discontinuation of problematic lenses when vascular growth is progressing.[31] Because neovascularization may develop insidiously and remain asymptomatic until advanced, its detection underscores the importance of regular surveillance rather than symptom-driven care. The most serious category of CL-related corneal complications is microbial keratitis, also described as contact lens–induced keratitis (CLIK), which, although not the most frequent complication, carries the highest risk of rapid corneal destruction and permanent vision loss.[4] Microbial keratitis typically begins with epithelial compromise—whether from microtrauma, hypoxia, dryness, or chemical injury—followed by inoculation with pathogens derived from contaminated lenses, solutions, storage cases, or environmental exposures. Major risk factors include poor personal hygiene, inadequate cleaning and disinfection, contaminated lens cases, improper solution use, and comorbid ocular surface disease such as dry eye or blepharitis.[4] Clinically, patients may present with pain, redness, photophobia, discharge, and reduced visual acuity. The urgent concern is that certain pathogens, particularly in CL wearers, can cause fulminant tissue necrosis and stromal melt within short time frames, making delayed treatment a pivotal determinant of outcome.

Acanthamoeba castellanii infection is especially concerning because it is strongly associated with water exposure and is frequently diagnosed late due to nonspecific early findings.[32] Risk factors include using homemade saline, wearing lenses during swimming, exposure to contaminated water, and contact with tub or tap water.[32] A highly suggestive clinical feature is pain that appears disproportionate to early slit-lamp findings, often accompanied by epithelial irregularity and patchy stromal infiltrates.[32] As disease progresses, additional findings such as radial keratoneuritis and characteristic epithelial patterns may appear. Prolonged lens wear, particularly sleeping with lenses, further increases susceptibility by compromising epithelial defenses.[33] The clinical concern is not only the severity of the infection but also the tendency for delayed recognition, leading to prolonged inflammation, scarring, and poor visual outcomes. Prevention is therefore paramount and hinges on strict avoidance of water exposure while wearing lenses, meticulous cleaning and disinfection, and patient education regarding warning symptoms and the need for urgent assessment.[33] *Pseudomonas aeruginosa* keratitis is another major threat in contact lens users

and is widely recognized as a leading cause of bacterial keratitis associated with lens wear.[34] The organism's ability to adhere to lens surfaces and proliferate in contaminated cases and solutions, coupled with its virulence mechanisms, allows it to cause aggressive corneal tissue destruction. Patients often present with pain, redness, and discharge, sometimes appearing severe relative to the initial size of the infiltrate.[34] Clinical signs may include dense stromal infiltrates, ring infiltrates, and hypopyon in advanced cases.[34] The urgency of this condition is driven by its rapid progression and the risk of perforation and profound scarring; hence, immediate discontinuation of lens wear and prompt targeted therapy following clinical evaluation are crucial.

Fungal keratitis, although less common than bacterial infection in many contexts, remains a sight-threatening complication that must be considered in contact lens wearers, particularly when ulcers exhibit atypical features or respond poorly to antibacterial therapy.[35] Classical signs include dense gray-white stromal infiltrates, feathery margins, satellite lesions, endothelial exudate, and hypopyon.[35] Definitive diagnosis relies on corneal scraping with microscopy and culture to identify the causative organism.[35] The incidence of fungal keratitis associated with CL wear has been reported to vary and may be influenced by geographic and environmental factors; commonly implicated organisms include *Aspergillus*, *Fusarium*, and *Candida*. [36] Extended-wear lenses have been associated with increased risk, and trauma with vegetative matter is often cited as a major precipitating factor for fungal infection.[4] Treatment typically depends on topical antifungals, and non-resolving cases may require therapeutic keratoplasty.[4] The clinical concern lies in delayed identification and the possibility of deep stromal involvement that can persist despite therapy, emphasizing the importance of early suspicion and appropriate diagnostic sampling when fungal features are present. Beyond infectious disease, prolonged CL wear can alter corneal shape and biomechanics. Warpage refers to lens-induced corneal contour changes, often manifesting as irregular astigmatism or transient refractive shifts.[37] This phenomenon is particularly associated with chronic hypoxia and long-term use of less permeable rigid lenses, which can mold the corneal surface and distort topographic measurements. Warpage is typically detected via corneal topography and often improves after discontinuation of lens wear, although recovery may be gradual depending on duration and severity.[37] The concern extends beyond symptomatic blur because warpage may complicate refractive assessments, delay accurate prescription updates, and interfere with preoperative planning for refractive surgery or other corneal procedures.

Additional corneal phenomena related to CL use include foreign body tract formation, corneal dellen, vacuoles, mucin ball formation, and dimple veiling. While not uniformly vision-threatening, these

conditions reflect localized mechanical or tear film disturbances and may signal a need to reassess lens fit, wearing schedule, and ocular surface health. Their clinical importance often lies in their role as markers of chronic microtrauma or tear instability that can predispose to more significant pathology if left unaddressed. Corneal endothelial alterations represent another critical area of concern because the endothelium is essential for maintaining corneal deturgescence and transparency. Both short-term and long-term endothelial changes have been attributed to hypoxia, which promotes lactic acid accumulation, elevated carbon dioxide, and reduced pH in the corneal microenvironment.[38] One short-term response described in contact lens wearers is the endothelial bleb phenomenon, which may appear within minutes of wearing thick soft lenses or rigid lenses.[13] This is typically transient and resolves shortly after lens removal or after a brief period of wear, generally without lasting sequelae.[13] In contrast, long-term endothelial polymegathism and pleomorphism reflect chronic stress, presenting as variation in endothelial cell size and shape, respectively.[39] These changes are clinically important because they may be associated with increased vulnerability to corneal decompensation after intraocular surgery, particularly in predisposed individuals. Reducing the risk of these changes often involves favoring daily wear over extended wear and selecting more oxygen-permeable modalities, including RGP lenses rather than older rigid PMMA lenses.[39]

A separate but pervasive issue is contact lens-related discomfort, which has been conceptualized as an adverse sensory experience associated with lens wear that may occur with or without accompanying visual disturbance, reflecting reduced compatibility between the lens and the ocular surface environment.[40] This discomfort is a major driver of reduced wearing time, nonadherence, and permanent discontinuation of CL use. Its etiology is multifactorial and encompasses lens-related variables such as material composition, surface properties, design, fit, wearing schedule, and hygiene practices, as well as environmental variables including humidity, occupational exposures, temperature, and medication effects.[4] Discomfort also frequently intersects with ocular surface disease, particularly dry eye and lid margin dysfunction, where tear film instability amplifies frictional forces between the lid, lens, and corneal epithelium. The principal concern in clinical management is to distinguish benign dryness-related discomfort from early signs of epithelial breakdown or infection, and to implement targeted modifications—whether through changing lens modality, adjusting fit, optimizing lubrication, treating comorbid blepharitis, or improving adherence to cleaning protocols—to prevent progression to more serious pathology.[4] Conjunctival complications represent another important domain of concern, particularly because they commonly reduce lens tolerance and can prompt

patients to adopt unsafe compensatory behaviors such as overusing topical vasoconstrictors or extending lens wear while symptomatic. Allergic conjunctivitis has been associated with thiomersal-containing solutions and may manifest with pain, redness, itching, and burning sensations, sometimes developing after days to months of exposure.[23] Examination often reveals conjunctival hyperemia and papillary responses. The cornerstone of management is avoidance of the offending preservative, accompanied by appropriate anti-inflammatory therapy, including topical steroids administered in tapering regimens when clinically justified.[41] The concern in this context is that ongoing exposure may sustain conjunctival inflammation, compromise the ocular surface, and indirectly increase susceptibility to corneal injury.

Giant papillary conjunctivitis (GPC) is a notable immunologic complication in which lens deposits and accumulated proteins function as allergens or mechanical irritants, stimulating a chronic inflammatory response on the upper tarsal conjunctiva.[23] Risk is influenced by patient susceptibility, wearing schedules, care regimens, lens material, and lens design, and the condition has been reported more commonly with soft lenses.[4] Individuals with atopic predispositions, including asthma, hay fever, or animal allergies, appear more susceptible.[4] Clinically, patients may experience itching, redness, excessive mucus production, photophobia, irritation, and progressive decline in lens tolerance, with characteristic giant “cobblestone” papillae observed on the upper palpebral conjunctiva.[42] Management typically requires discontinuation of lens wear for a sustained period, optimization of cleaning regimens, selection of lenses with reduced deposit propensity, and pharmacologic therapy such as sodium cromoglycate and topical steroids, followed by refitting with newer lenses to minimize recurrence once inflammation has resolved.[43] The primary concern is that persistent GPC may lead to chronic intolerance, reduced adherence, and repeated mechanical trauma, thereby increasing the probability of secondary corneal complications. Superior limbic keratoconjunctivitis (SLK) represents another conjunctival complication and has been described as a hypersensitivity response to thiomersal or preservatives in lens solutions.[44] It is often bilateral but asymmetric and may present with pain, redness, foreign body sensation, and reduced tolerance to lens wear. Examination may reveal superior bulbar conjunctival inflammation and hypertrophy, staining patterns that highlight epithelial compromise, and associated papillary hypertrophy of the superior tarsal conjunctiva.[44] Management generally involves discontinuing contact lenses, implementing frequent lubrication, and, after resolution, refitting with new lenses while recommending non-preserved saline and meticulous cleaning to prevent recurrence.[44] The clinical

concern is that SLK can be persistent and symptomatic and may predispose to corneal epithelial compromise if lens wear continues despite inflammation.

Collectively, these issues underscore that CL-related complications are not isolated events but rather interrelated outcomes arising from disruptions in oxygen delivery, tear film stability, mechanical compatibility, and microbial control. The corneal epithelium serves as the primary barrier against infection and inflammation; once compromised—whether through hypoxia-induced edema, microcyst formation, abrasion, or chemical toxicity—the risk of microbial keratitis escalates substantially.[4] Furthermore, chronic inflammation of the conjunctiva, as seen in allergic conjunctivitis, GPC, and SLK, can destabilize the ocular surface, reduce lens tolerance, and increase the likelihood of poor adherence to safe lens practices, thereby amplifying risk.[23][41][42][44] The overarching clinical concern is therefore the prevention of progression from mild, reversible pathology to irreversible damage through early recognition, timely discontinuation of lens wear, and structured reassessment of lens fit, hygiene behavior, and ocular surface health. Continuous patient education regarding appropriate cleaning regimens, avoidance of water exposure, and strict compliance with prescribed wear schedules is indispensable to minimizing adverse outcomes and safeguarding long-term ocular integrity.[4][33]

Clinical Significance

Contact lens–related disorders represent a clinically important and increasingly prevalent source of ocular morbidity, with consequences that extend beyond localized eye symptoms to include psychological distress, functional limitation, and substantial healthcare expenditure. Because contact lenses function as foreign bodies positioned directly on the ocular surface, they can disrupt the tear film, reduce oxygen transmission, and facilitate microbial contamination, thereby predisposing users to a wide continuum of complications. From a patient-centered perspective, even relatively mild problems—such as discomfort, recurrent redness, or fluctuating vision—may negatively affect quality of life, occupational productivity, and adherence to ongoing refractive correction. More severe complications, particularly infectious keratitis or progressive corneal neovascularization, can lead to corneal scarring, irregular astigmatism, and permanent reduction in visual acuity when diagnosis or treatment is delayed.[4] Consequently, contact lens complications impose both physical and emotional burdens on patients and contribute to a measurable economic impact through emergency visits, specialist consultations, diagnostic testing, pharmacologic treatment, and, in advanced cases, surgical intervention or prolonged follow-up.[4] The clinical significance of these complications is amplified by the fact that many lens-related conditions are preventable.

Inadequate lens hygiene, unsafe wear schedules (especially overnight wear), exposure to nonsterile water sources, and improper cleaning or storage practices are recurring contributors to adverse outcomes. Therefore, comprehensive patient education is not optional; it is central to safe prescribing and should be framed as a core therapeutic intervention. Every patient who is prescribed contact lenses should receive detailed counseling on lens material properties, oxygen permeability considerations, correct fitting and handling techniques, and strict ocular and lens hygiene practices, including appropriate cleaning solutions, storage case maintenance, and replacement intervals.[4] Education should also address early warning signs—pain, photophobia, reduced vision, mucopurulent discharge, or persistent redness—and emphasize that prompt lens discontinuation and urgent clinical evaluation are necessary when these symptoms occur, as delays may permit rapid progression to sight-threatening disease.[4] Equally important is ensuring that patients understand the diversity of lens options currently available and the relative advantages and limitations of each. Patients should be guided through the major lens categories, including daily disposable, planned replacement soft lenses, extended-wear modalities, and rigid gas-permeable designs, with explicit discussion of the clinical “pros and cons” in relation to comfort, oxygen transmission, deposit formation, infection risk, and suitability for the individual’s lifestyle and ocular surface characteristics.[2] This individualized, education-driven approach strengthens adherence, reduces preventable harm, and supports safer long-term contact lens use within a modern preventive eye-care framework.[2][4]

Other Issues

Contact lens use is not only associated with inflammatory or infectious ocular surface disease; it also introduces a separate category of practical, device-related complications that can directly compromise visual performance, comfort, and safety. Over time, contact lenses may become damaged, spoiled, discolored, or lost, and they frequently accumulate deposits that alter their optical quality and surface characteristics.[45] Although these issues may initially appear minor, they can precipitate clinically meaningful symptoms such as blurred vision, foreign-body sensation, and photophobia, and they may indirectly increase the risk of more serious corneal pathology by disrupting the lens–tear film interface and impairing oxygen delivery.[45] Physical damage to the lens is a common and underappreciated complication, occurring as breakage, chipping, tearing, or cracking during routine insertion, removal, or cleaning. Such damage is particularly relevant in rigid lens users, but it may occur with any lens material when handling techniques are improper or when lenses are worn beyond their intended replacement schedule. Beyond mechanical trauma, biochemical degradation

of lens materials over time can reduce structural integrity, making lenses more prone to microfractures and surface irregularities that increase friction on the ocular surface and reduce wearing tolerance.[4] Damaged lenses should not be “tolerated” simply because they remain wearable; even subtle edge defects can contribute to epithelial microtrauma and increase susceptibility to keratitis, necessitating prompt discontinuation and replacement.[4] Lens discoloration represents another clinically relevant issue, most often reflecting exposure to staining agents rather than intrinsic ocular disease. Certain medications and diagnostic dyes may alter lens coloration, including agents such as rifampicin, fluorescein, and phenylephrine, which can stain the lens material and reduce its cosmetic acceptability and optical clarity.[46] While discoloration itself may not always signal danger, it should prompt clinicians to reassess lens hygiene, solution compatibility, and medication exposure, particularly in patients reporting concurrent irritation or visual disturbances.[46]

Lens loss is especially common among pediatric patients, who may have less consistent handling practices and reduced awareness of subtle dislodgement. It has been reported more frequently with rigid lenses than soft lenses and appears more typical with extended-wear regimens compared with daily wear, likely reflecting the combined effects of sleep-related dislodgement and reduced opportunities for routine inspection.[47] From a safety perspective, “lost” lenses require careful confirmation that the lens is not retained in the conjunctival fornices, especially when unilateral discomfort, redness, or persistent foreign-body sensation is present.[47] Deposits on the lens surface are among the most frequent and clinically significant device-related problems. Deposits occur across lens types, including extended wear, daily-wear soft lenses, rigid gas-permeable lenses, and traditional hard lenses, and may arise from tear film constituents, handling practices, cosmetics, topical medications, and environmental pollutants such as dust or fumes.[20] Predisposing factors include inadequate personal hygiene, manufacturing or surface defects, age-related changes in tear composition, material decay, and coexisting ocular surface disorders—particularly blepharitis, meibomitis, lagophthalmos, and dry eye disease.[25] Deposits may be proteinaceous or lipid-laden and can impair gas diffusion, increase surface roughness, and destabilize the tear film, thereby producing blurred or hazy vision, halos, distortion, polyopia, photophobia, optical aberrations, and induced astigmatism. When deposits become clinically significant, replacement rather than repeated cleaning is often the most effective intervention, and routine replacement schedules—such as changing daily-wear lenses at appropriate intervals—remain essential for reducing deposit burden and maintaining ocular health.[48]

Enhancing Healthcare Team Outcomes

Optimizing outcomes in contact lens care depends fundamentally on preventing avoidable complications through careful patient selection, individualized prescribing, and coordinated follow-up. Because contact lenses function as a foreign body on the ocular surface, even modern materials and improved designs cannot entirely eliminate the risks of hypoxia, inflammation, infection, or mechanical trauma. Consequently, avoiding contact lens–related problems is a shared priority for both the patient and the treating surgeon, beginning at the moment a lens is considered as a therapeutic or refractive option. When prescribing contact lenses, the treating surgeon must systematically balance anticipated benefits against the patient’s individualized risk profile and the clinical indication driving lens use.[49] This risk–benefit appraisal should incorporate ocular surface status, history of lens intolerance, occupational or environmental exposure, anticipated adherence to hygiene practices, and the feasibility of timely review if symptoms arise. By aligning lens choice with patient-specific needs and capabilities, clinicians can reduce preventable adverse events and preserve long-term visual function.[49] A high-quality contact lens service is inherently multidisciplinary and relies on well-defined collaboration across professionals who contribute complementary expertise. Typically, the patient undergoes refraction and baseline visual assessment by an optometrist, who establishes refractive parameters and often identifies early ocular surface issues that influence lens suitability. Nursing personnel and allied health staff frequently support patient recruitment, appointment coordination, structured counseling, and continuity of follow-up—functions that are essential to maintaining adherence to recommended wear schedules and hygiene behaviors. The treating surgeon then synthesizes clinical findings and determines the most appropriate lens strategy in accordance with the primary indication and the patient’s risk profile. Contact lens specialists and paramedical staff contribute additional expertise by performing lens fitting, assessing centration and movement, educating the patient in insertion and removal techniques, and reinforcing cleaning and replacement protocols. This integrated workflow supports consistent messaging, early detection of complications, and rapid escalation when warning signs emerge, thereby improving safety and patient satisfaction.[50] In practice, effective interprofessional collaboration reduces fragmented care, limits preventable complications, and promotes durable, patient-centered outcomes.[50]

Nursing, Allied Health, and Interprofessional Team Interventions

Nursing staff and allied health professionals occupy a pivotal position in translating contact lens prescribing into safe daily practice, largely because they interface with patients at multiple time points—initial counseling, troubleshooting visits, and routine

review. Their interventions contribute to appropriate lens selection, improved comfort, and timely recognition of early complications. In many clinical settings, the interprofessional team helps guide decisions regarding lens type by gathering relevant patient information, documenting ocular and systemic history, and clarifying practical factors that influence adherence, such as work schedules, manual dexterity, or access to cleaning supplies. By identifying barriers early, nursing and allied teams can support individualized education and ensure the selected lens modality is realistic for the patient’s lifestyle and capacity to comply.[51] A second essential intervention is early, structured identification of complications. Nursing and allied health professionals are often the first to hear patient reports of redness, pain, photophobia, discharge, blurred vision, dryness, or reduced wearing time. These symptoms should be treated as clinically meaningful until proven otherwise, because they may represent early inflammatory disease or the initial presentation of microbial keratitis. Accordingly, team members should be trained to perform preliminary triage, recognize red flags, and facilitate prompt escalation to the treating clinician when urgent assessment is warranted. In addition, nurses and allied health personnel play a central role in reinforcing evidence-informed behaviors, including appropriate lens handling, avoidance of risky exposures such as water contact, adherence to replacement schedules, and discontinuation of lens wear when symptoms occur. Regular follow-up, coupled with consistent patient counseling, strengthens adherence and reduces the likelihood that mild irritation progresses to more serious corneal pathology.[51]

Nursing, Allied Health, and Interprofessional Team Monitoring

Sustained monitoring is a core determinant of safety in habitual contact lens wearers, particularly because complications can develop gradually and patients may normalize discomfort until significant disease occurs. Nursing staff, allied health professionals, and the broader interprofessional team contribute to ongoing surveillance by ensuring that contact lens users remain engaged in routine review and that any change in symptoms or wearing tolerance is promptly evaluated. Effective monitoring extends beyond documenting visual acuity; it involves tracking patterns of lens wear, replacement behavior, cleaning practices, and recurrent symptoms such as dryness, redness, fluctuating vision, or reduced wearing time. These longitudinal observations can identify early deterioration that may not be apparent in a single clinical encounter and can guide timely adjustments in lens material, fit, solution regimen, or wearing schedule.[52] Monitoring responsibilities also include structured patient education reinforcement at each encounter. Because adherence commonly declines over time, periodic re-education

regarding hygiene, proper storage, and risk avoidance is necessary, especially for high-risk groups such as adolescents, extended-wear users, and patients with ocular surface disease. Team-based monitoring is further strengthened when clinical pathways are standardized—for example, by using consistent symptom checklists, documenting prior complications, and maintaining clear referral thresholds for urgent ophthalmic review when warning signs suggest infection or corneal involvement. When monitoring is performed systematically, complications are more likely to be detected at an earlier and reversible stage, reducing the risk of visual loss and improving patient confidence in contact lens therapy.[52]

Conclusion:

Contact lens wear, while offering significant visual and cosmetic benefits, introduces complex physiologic and microbiologic challenges that can compromise ocular health. The cornea's dependence on oxygen and tear exchange makes it highly vulnerable to hypoxia, mechanical trauma, and microbial invasion when lens care practices are inadequate. Although modern lens materials have improved oxygen permeability, complications such as epithelial edema, microcysts, and microbial keratitis persist, often due to behavioral factors like overnight wear, poor hygiene, and water exposure. Infectious keratitis, particularly from *Pseudomonas* and *Acanthamoeba*, remains the most sight-threatening outcome, underscoring the need for rapid recognition and intervention. Preventive strategies are central to mitigating these risks. Comprehensive patient education on lens handling, cleaning protocols, and early symptom awareness is essential. Nursing and allied health professionals occupy a frontline role in reinforcing these behaviors, performing structured monitoring, and facilitating timely escalation when red flags arise. Interprofessional collaboration further enhances safety by ensuring consistent messaging and individualized lens selection. Ultimately, the clinical priority is to prevent progression from reversible irritation to irreversible corneal damage through proactive counseling, routine surveillance, and adherence to evidence-based care pathways. By integrating these principles, healthcare teams can safeguard visual outcomes and optimize patient satisfaction.

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