

KERATITIS

Authored by
Mohammed looti

December 1, 2025

RECOMMENDED CITATION

Mohammed looti (2025). *KERATITIS*. Encyclopedia of psychology. Retrieved from <https://encyclopedia.arabpsychology.com/?p=21008>

Introduction and Definition

Keratitis is defined as the inflammation of the **cornea**, the transparent, dome-shaped outer layer of the eye that covers the iris, pupil, and anterior chamber. This crucial structure is essential for refracting light and focusing it onto the retina, making its clarity vital for optimal vision. When the cornea becomes inflamed, its transparency is compromised, leading rapidly to impaired visual acuity, pain, and significant discomfort. Given its avascular nature, the cornea relies heavily on the tear film and aqueous humor for nutrients and oxygen, and this delicate environment makes it highly susceptible to infectious agents and environmental insults. Early identification and rigorous treatment of keratitis are paramount, as delayed intervention can result in severe and potentially irreversible structural damage, culminating in significant vision loss or even blindness.

The etiology of keratitis is diverse, encompassing a wide range of infectious pathogens, including **bacteria** and **viruses**, as well as non-infectious causes such as allergic reactions, trauma, and exposure. This diversity necessitates a comprehensive diagnostic approach to accurately identify the underlying cause, which in turn dictates the specific therapeutic strategy required for effective resolution. The clinical presentation often involves hallmark symptoms such as ocular pain, intense photophobia (sensitivity to light), and the sensation of a foreign body in the eye, all resulting from the inflammatory cascade triggered within the sensitive corneal nerve endings.

Understanding the anatomical layers of the cornea--the epithelium, Bowman's layer, stroma, Descemet's membrane, and endothelium--is key to appreciating the severity of keratitis. Inflammation confined to the superficial epithelial layer is generally less threatening and heals rapidly without scarring. Conversely, inflammation that penetrates the deeper stromal layers often results in permanent structural changes, such as corneal opacification or scarring, which severely interfere with light transmission and necessitate prolonged and aggressive medical management. The potential for rapid progression, especially in cases involving aggressive pathogens like *Pseudomonas aeruginosa*, underscores the urgency of ophthalmic consultation when symptoms first manifest.

Etiology: Causes and Risk Factors

The causes of keratitis are conventionally divided into infectious and non-infectious categories. Among infectious causes, **bacterial keratitis** is perhaps the most common and often the most aggressive form, frequently associated with poor contact lens hygiene, ocular trauma, or underlying chronic ocular surface disease. Common bacterial culprits include *Staphylococcus aureus*, *Streptococcus pneumoniae*, and, notoriously, *Pseudomonas aeruginosa*, the latter being capable of causing corneal perforation within 24 to 48 hours if left untreated. Prompt culturing and immediate initiation of broad-spectrum, fortified topical antibiotics are critical steps in mitigating the destructive potential of these organisms.

Viral keratitis represents another significant etiological group, with the **Herpes Simplex Virus (HSV)** being the primary concern. HSV keratitis can manifest in various forms, ranging from superficial epithelial disease characterized by classic dendritic ulcers to more severe stromal keratitis, which involves deep inflammation and often requires long-term antiviral and sometimes steroid management. Other viral agents, such as Varicella-Zoster Virus (VZV), particularly in the context of herpes zoster ophthalmicus, can also induce significant corneal inflammation, often accompanied by neurotrophic complications that impair corneal sensation and healing capabilities. Furthermore, less common infectious agents, including **fungi** (e.g., *Fusarium*, *Aspergillus*), often following vegetative trauma, and **Acanthamoeba**, a protozoan typically linked to swimming or showering while wearing contact lenses, require highly specialized and prolonged treatment regimens due to their resistance to standard antibiotics.

Non-infectious etiologies include mechanical trauma, chemical exposure, and immunological reactions. **Allergic keratitis**, such as that seen in vernal keratoconjunctivitis, results from chronic immunological hypersensitivity causing inflammation and remodeling of the corneal epithelium. **Neurotrophic keratitis** arises from damage to the trigeminal nerve, which innervates the cornea, leading to decreased sensation and subsequent failure of the normal regenerative and protective reflexes, resulting in persistent epithelial defects. A significant and preventable risk factor common to all forms of keratitis is the misuse of **contact lenses**, including extended wear, improper disinfection, and sleeping in lenses not approved for overnight use. These practices create a hypoxic environment beneath the lens, allowing pathogens to proliferate and adhere to the compromised corneal surface, thereby dramatically increasing the risk of severe infection.

Classification of Keratitis

Keratitis is broadly classified based on the anatomical depth of the inflammation, which carries significant implications for prognosis and treatment. **Superficial keratitis** involves only the corneal epithelium and sometimes Bowman's layer. This type is generally self-limiting or readily treatable and typically heals without leaving residual scarring that impairs vision. Examples include punctate epithelial erosions caused by dry eye or mild exposure, and early stages of viral epithelial disease. The key characteristic of superficial keratitis is the preservation of the corneal stroma's structural integrity.

In contrast, **Deep Keratitis**, or **Stromal Keratitis**, affects the dense collagen layers of the stroma and represents a far more serious threat to vision. When the infection penetrates the stroma, the resultant inflammation and immune response lead to tissue destruction and the formation of infiltrates. The destruction of stromal collagen results in dense, opaque scarring upon resolution. A specific, severe form is **Interstitial Keratitis**, which involves chronic, non-suppurative inflammation deep within the stroma without primary involvement of the epithelium or endothelium. As noted in historical contexts, this form of keratitis may be deep when the infection causing it is carried by the

bloodstream, suggesting a systemic origin, often associated with congenital syphilis, tuberculosis, or Lyme disease, rather than direct external inoculation.

Further classification is based on the specific causative agent. **Ulcerative Keratitis** refers specifically to inflammation accompanied by a significant epithelial defect and underlying stromal necrosis--essentially, a corneal ulcer. These ulcers are typically associated with aggressive infectious processes, particularly bacterial or fungal. Non-ulcerative forms, such as neurotrophic keratitis or certain allergic types, involve inflammation and epithelial breakdown without extensive stromal tissue loss, though the resulting inflammation can still severely compromise visual function. The distinction between these classifications is fundamental for the ophthalmologist, as it directs the necessity for surgical intervention, the intensity of antibiotic or antiviral regimens, and the prognosis regarding long-term visual acuity.

Clinical Presentation and Symptoms

The clinical presentation of keratitis is often acutely symptomatic due to the rich sensory innervation of the cornea. Patients typically present with a triad of highly specific complaints: significant ocular **pain**, often described as sharp or grating; intense **photophobia**, where exposure to light causes severe discomfort and squinting; and the persistent sensation of a **foreign body** in the eye, even when none is present. This constellation of symptoms usually develops rapidly, prompting immediate medical attention. The severity of the pain often correlates directly with the extent of epithelial damage and the depth of the inflammatory process.

In addition to subjective discomfort, objective signs are readily observable during examination. These include marked conjunctival and ciliary **hyperemia** (redness), often most concentrated around the limbus (the border between the cornea and sclera), a pattern known as ciliary flush. There is often excessive tearing (epiphora) and discharge, which can be watery in viral cases, or thick and mucopurulent in severe bacterial infections. Most critically, visual acuity is compromised, ranging from mild blurring to severe vision loss, depending on the location and density of the corneal infiltrate or ulcer. When the central visual axis is involved, even a small lesion can cause profound impairment.

Specific types of keratitis present with unique findings that aid in differential diagnosis. For instance, **Herpes Simplex Keratitis** classically produces a dendritic (branching) epithelial ulceration pattern when stained with fluorescein dye, which is highly pathognomonic. Severe bacterial keratitis may present with a dense, yellowish-white stromal infiltrate, often accompanied by an **hypopyon**--a collection of inflammatory cells settling in the inferior anterior chamber. **Acanthamoeba Keratitis**, notoriously difficult to diagnose early, may initially mimic HSV but progresses to characteristic ring-shaped infiltrates as the protozoa penetrate the stromal layers. The slit-lamp examination is therefore indispensable for visualizing these fine structural changes

and determining the precise extent of corneal involvement.

Diagnosis and Ophthalmic Evaluation

Accurate diagnosis of keratitis relies heavily on a thorough patient history combined with meticulous ophthalmic examination. The history must specifically investigate risk factors such as recent trauma, exposure to contaminated water, history of ophthalmic surgery, current use of topical medications (especially steroids), underlying systemic immunosuppression, and, critically, **contact lens wear** habits, including solution use and duration of wear. Establishing whether the patient has a history of previous corneal ulcers or recurrent ocular herpes is also paramount for guiding initial management.

The cornerstone of diagnosis is the **slit-lamp biomicroscopic examination**. This high-magnification assessment allows the clinician to determine the exact location, size, depth, and morphology of the corneal inflammation. Fluorescein staining is routinely employed to highlight epithelial defects and ulcers, allowing the ophthalmologist to delineate the borders of the lesion and identify specific patterns, such as the dendritic figures characteristic of HSV or the pseudodendrites associated with VZV. Furthermore, the slit lamp enables evaluation of the anterior chamber for inflammatory cells (flare and cells) and the presence of a hypopyon, indicating severe intraocular inflammation.

In cases of suspected infectious keratitis, particularly those that are large, central, rapidly progressing, or unresponsive to initial empiric therapy, laboratory testing is mandatory. This involves carefully obtaining **corneal scrapings** from the base and edges of the ulcer using a sterile spatula or surgical blade. The collected material is then immediately plated onto various culture media (e.g., blood agar for bacteria, Sabouraud agar for fungi) and subjected to microscopic evaluation (Gram stain, Giemsa stain). In cases where atypical pathogens like *Acanthamoeba* or specific viruses are suspected, specialized techniques such as polymerase chain reaction (PCR) or non-nutrient agar plating may be employed to ensure rapid and precise identification of the causative organism, which is essential for initiating targeted, pathogen-specific therapy rather than relying solely on broad-spectrum agents.

Management and Treatment Protocols

The management of keratitis is dictated entirely by its etiology and severity. For presumed infectious keratitis, treatment must be initiated immediately, often before culture results are available, due to the rapid potential for vision loss. This initial phase involves **empiric therapy** using highly concentrated topical medications. For suspected bacterial keratitis, this often means intensive hourly instillation of broad-spectrum topical antibiotics, such as fluoroquinolones, or fortified antibiotics (e.g., fortified vancomycin and tobramycin) in severe, sight-threatening cases.

When the diagnosis confirms **Viral Keratitis**, specific antiviral agents are required. Epithelial HSV keratitis is treated with topical antivirals like ganciclovir gel or trifluridine drops, sometimes supplemented by oral antivirals (e.g., acyclovir, valacyclovir) to prevent recurrence. Management of stromal keratitis is more complex, often requiring the careful introduction of **topical corticosteroids** alongside antiviral coverage. Corticosteroids are used to suppress destructive inflammation, but they carry the risk of exacerbating undetected or poorly controlled infections, particularly fungal or amoebic infections, necessitating extreme caution and close monitoring by the ophthalmologist.

Treatment for less common infectious types is specialized and challenging. **Fungal keratitis** requires weeks to months of intensive topical antifungal agents (e.g., natamycin, amphotericin B), often requiring surgical debridement of the infected tissue. **Acanthamoeba keratitis** demands prolonged therapy with topical biguanides or diamidines. For non-infectious keratitis, the focus shifts to treating the underlying cause: neurotrophic keratitis requires rigorous lubrication, often with autologous serum tears and punctal occlusion, to promote epithelial healing; and allergic keratitis is managed with mast cell stabilizers and antihistamines. Discontinuation of contact lens wear is mandatory for all infectious and most non-infectious cases until complete resolution is achieved.

Complications and Prognosis

Despite advancements in antimicrobial therapy, keratitis remains a leading cause of unilateral visual impairment globally, primarily due to the severe complications it can induce. The most immediate and sight-threatening complication is **corneal perforation**, which occurs when the infectious or inflammatory process completely erodes the corneal stroma, leading to a collapse of the anterior chamber and high risk of endophthalmitis (infection spreading to the interior of the eye). Perforation necessitates emergency surgical intervention, often involving tissue adhesives or therapeutic penetrating keratoplasty.

Long-term complications primarily revolve around the consequences of stromal tissue destruction. Severe inflammation inevitably leads to the deposition of scar tissue, resulting in **corneal scarring** and opacification. If this scarring occurs within the central visual axis, it causes permanent reduction in visual acuity that cannot be corrected with glasses or contact lenses. Other potential sequelae include chronic irregular **astigmatism**, intractable ocular pain, and the development of secondary **glaucoma** resulting from inflammatory damage to the trabecular meshwork, the eye's drainage system.

The prognosis for visual recovery is highly dependent on the causative agent, the initial size and location of the lesion, and the timeliness of the treatment. Keratitis caused by highly virulent organisms, such as *Pseudomonas* or *Acanthamoeba*, carries a guarded prognosis, often resulting

in significant scarring. When visual impairment is profound due to central, dense scarring, the definitive treatment is often **Penetrating Keratoplasty** (PK), or corneal transplantation. PK is a major surgical procedure that replaces the diseased cornea with a healthy donor graft, offering the potential for restored clarity, although it introduces new risks, including graft rejection and secondary infections.

Prevention Strategies

Given the seriousness of keratitis complications, proactive prevention is the most effective strategy, particularly targeting the major risk factor: contact lens misuse. Strict adherence to **contact lens hygiene** protocols is non-negotiable. This includes washing hands thoroughly before handling lenses, using only fresh, sterile solution for rinsing and storing, and never using tap water, which can harbor organisms like *Acanthamoeba*. Lenses should never be worn while swimming or showering, and the prescribed replacement schedule (daily, weekly, or monthly) must be rigorously followed. Furthermore, sleeping in contact lenses, even those approved for extended wear, significantly elevates the risk of infection and should be avoided whenever possible.

Beyond contact lens care, prevention involves protecting the ocular surface from trauma and managing underlying ocular diseases. Individuals working in environments where flying debris is a risk (e.g., construction, grinding, gardening) must wear certified **protective eyewear**. Prompt and effective treatment of chronic conditions that compromise the corneal surface, such as severe **dry eye syndrome** or lid margin diseases like blepharitis, helps maintain the integrity of the epithelial barrier, reducing the entry points for pathogens.

Finally, patient education regarding the importance of recognizing early symptoms is crucial. Any individual experiencing sudden onset of pain, redness, photophobia, or vision decrease, especially if they are a contact lens wearer, must seek immediate ophthalmic care. Delaying treatment by even a few hours can dramatically worsen the prognosis, particularly in aggressive bacterial or fungal infections. Regular eye examinations also allow for early detection and management of predisposing factors, thereby minimizing the incidence and severity of acute keratitis episodes.