KERATITIS

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Anatomy of the Cornea

The cornea is the transparent, avascular anterior-most structure of the eye. It functions as an anterior refractive surface which contributes to nearly 3/4th of the total refractive power of a normal human eye. It corresponds to the lens of a camera. Like the skin, it is the external anatomic barrier between the environment and deeper tissues; it is the most frequently damaged ocular structure from external trauma.

Anatomically the cornea is divided into 5 layers (Figure 1): (1) The epithelium is composed of non-keratinized, stratified squamous cells and accounts for 5% of total corneal thickness. Tight junctions between epithelial cells prevent the penetration of tear film into the corneal stroma and also significantly limits drug penetration into deeper tissues. (2) Bowman’s layer is a thin avascular layer underlying the epithelium. (3) the stroma, an extracellular matrix composed of proteoglycans, principally dermatan sulfate and keratin sulfate, collagen fibrils, fibroblasts (keratocytes), and mucoproteins. The stroma accounts for 90% of the corneal thickness. (4) Descemet’s membrane, which is the basement membrane of the corneal endothelium, and the (5) endothelium, a neuroectoderm derived cell layer composed of hexagonal shaped cells arranged in a honeycomb-like mosaic pattern. The endothelial cells contain a high density of Na+, K+-adenosine triphosphatase (ATPase) pump sites contributing to corneal dehydration and fluid content regulation.

Transparency of the cornea is critical to its’ refractive properties and is dependent upon precise hydration and regular arrangement of stromal keratocytes and collagen fibrils. Any disease state of the cornea leading to disruption of transparency can have deleterious effect on vision.

Keratitis is inflammation of the cornea.

Pathogenesis

The corneal surface is normally protected by a variety of mechanisms including the physical barrier of eyelids to foreign material, regular blink response which sweeps away debris from the tear-lake, and tight junctions between conjunctival and corneal epithelial cells. Immune mediators also protect the corneal surface: conjunctival mast cells, conjunctiva-associated lymphoid tissue (CALT) which is responsible for local antigen processing, immunoactive substances in the tear film (IgA, lysozyme, beta lysine, lactoferrin, and tear-specific albumin), plasma cells, macrophages and T lymphocytes. In the majority of keratitis cases, at least one risk factor that compromises these defense mechanisms can be identified.

In most cases of infectious keratitis, a defect in the epithelium is the initial event predisposing to infection. The inflammatory response leads to cellular infiltration with destruction of the corneal collagen, thinning of the stroma, and in severe cases,
perforation of the cornea with leakage of the aqueous humor and risk of intraocular extension, i.e., endophthalmitis (Figures 2 - 5).

Figure 1. Normal cornea consists of 5 distinct layers. Epithelium (EP), Bowman’s layer (BL), Stroma (ST), Descemet’s membrane (DM), and Endothelial (EN).
Figure 2. Corneal button of a patient with fungal keratitis. Note the fungal elements and inflammatory processes in the cornea.

Figure 3. Fungal corneal ulcer. Stromal infiltration with feathery borders.
Figure 4. 55 year old horsebreeder and contact lens wearer presented with fungal corneal ulcer. Note endothelial plaque on slit beam image.

Figure 5: Fungal gram stain of the same patient as figure 4 with *Phaeoannellonyces werneckii* keratitis; infection resolved with topical Amphotericin.