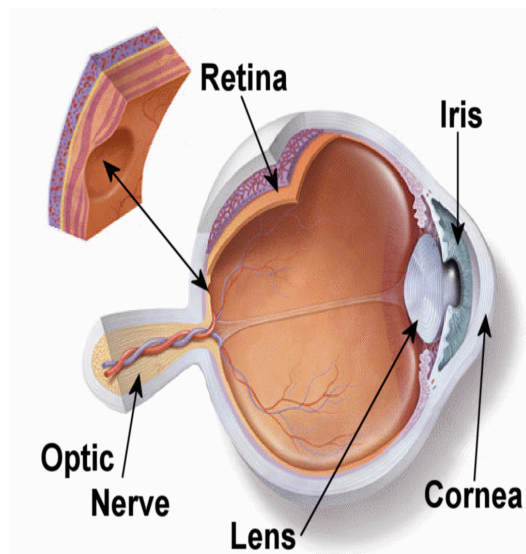


# THE CORNEA

## Introduction

The **cornea** is the transparent front part of the eye that covers the iris, pupil, and anterior chamber, providing most of an eye's optical power. Together with the lens, the cornea refracts light and, as a result, helps the eye to focus. The cornea contributes more to the total refraction than the lens does, but, whereas the curvature of the lens can be adjusted to "tune" the focus, the curvature of the cornea is fixed.

1. **The epithelium** is stratified, squamous and non-keratinized,
2. **Bowman layer** is the acellular superficial layer of the stroma
3. **The stroma** makes up 90% of corneal thickness. It is principally composed of regularly orientated layers of collagen fibrils whose spacing is maintained by proteoglycan ground substance) with interspersed modified fibroblasts (keratocytes).
4. **Descemet membrane** is composed of a fine latticework of collagen fibrils.
5. **The endothelium** consists of a single layer of hexagonal cells that cannot regenerate. It plays a vital role in maintaining corneal deturgescence. The adult cell density is about 2500 cells/mm<sup>2</sup>. The number of cells decreases at about 0.6% per year and neighbouring cells enlarge to fill the space as cells die. At a cell density of about 500 cells/mm<sup>2</sup> corneal oedema develops and corneal transparency is reduced.



## BACTERIAL KERATITIS

Bacterial keratitis is very uncommon in a normal eye and usually only develops when the ocular defences have been compromised. Bacteria that can penetrate an apparently normal corneal epithelium are *N. gonorrhoeae*, *N. meningitides*, *C. diphtheriae* and *H. influenzae*. The virulence of the organism and the anatomic site of the infection determine the pattern of disease. The most common pathogens are:

1. *P. aeruginosa*
2. *S. aureus*.
3. *S. pyogenes*.
4. *S. pneumoniae*

### **Risk factors**

**Contact lens wear**, particularly of soft lenses worn overnight, is the most important risk factor for bacterial keratitis. *Pseudomonas* spp. account for over 60% of cases

**Trauma** such as accidental injury, surgical (refractive surgery) and loose sutures

**Ocular surface disease** such as herpetic keratitis, bullous keratopathy, dry eye

**Other factors** include topical or systemic immunosuppression, diabetes, Vitamin A deficiency and measles.

### **Diagnosis**

**History** with particular attention paid to risk factors mentioned above.

**Presenting symptoms** include pain, photophobia, blurred vision and discharge

- **Signs:** An epithelial defect associated with an infiltrate around the margin and base associated with circumcorneal injection . Severe infiltration associated with enlarging hypopyon. Progressive ulceration may lead to corneal perforation and endophthalmitis.

**Differential diagnosis** : includes fungal keratitis, acanthamoeba keratitis, necrotic stromal herpes simplex keratitis and sterile inflammatory corneal infiltrates associated with contact lens wear.

## Treatment

Bacterial keratitis has the potential to progress rapidly to corneal perforation. Even small axial lesions can cause surface irregularity and scar that can lead to significant loss of vision.

**Topical** therapy can achieve high tissue concentration and initially should involve broad spectrum antibiotics to cover the most common pathogens.

1. **Dual therapy** involves a combination of two fortified antibiotics (an aminoglycoside and a cephalosporin) to cover common Gram-positive and Gram-negative pathogens.
2. **Monotherapy** with a fluoroquinolone (i.e. ciprofloxacin 0.3% or ofloxacin<sup>®</sup> 0.3%) is commercially available

**Oral antibiotics** (ciprofloxacin 750mg twice daily for 7-10 days) is *not usually* necessary.

**Subconjunctival** antibiotics are only indicated if there is poor compliance with topical treatment.

## VIRAL KERATITIS

### Herpes simplex keratitis

Herpetic eye disease is the major cause of unilateral corneal scarring worldwide, and is the most common infectious cause of corneal blindness in developed countries. As many as 60% of corneal ulcers in developing countries may be the result of herpes simplex virus (HSV) and 10 million people worldwide may have herpetic eye disease.

#### *Herpes simplex virus*

HSV is enveloped with a cuboidal capsule and a linear double-stranded DNA genome.

. *HSV-1* primarily causes infection above the waist that may affect the face, lips and eyes.

.*HSV-2* causes venereally acquired infection (genital herpes).

HSV transmission is facilitated in conditions of crowding and poor hygiene.

### *Primary infection*

Primary infection (no previous viral exposure) usually occurs by droplet transmission, or less frequently by direct inoculation. Due to protection bestowed by maternal antibodies, it is uncommon during the first 6 months of life. Most cases are probably subclinical or only cause mild fever, malaise and upper respiratory tract symptoms. Children may develop blepharconjunctivitis which is usually benign and self-limited although corneal microdendrites develop in a minority of cases.

### *Recurrent infection*

**After primary infection** the virus is carried to the sensory ganglion for that dermatome (e.g. trigeminal ganglion) where a latent infection is established.

**Subclinical reactivation** can periodically occur when HSV is shed and patients are contagious. Stimuli such as fever, hormonal change, ultraviolet radiation, trauma, and trigeminal injury may cause a clinical reactivation, when the virus replicates and is transported in the sensory axons to the periphery where there is recurrent disease.

### *Clinical features of epithelial keratitis*

**Presentation** may be at any age with mild discomfort, watering and blurred vision.

### **Sign**

A linear-branching (dendritic) ulcer, most frequently located centrally.

- The ends of the ulcer *have characteristic terminal buds* and the bed of the ulcer stains well with fluorescein
- Corneal sensation is reduced.
- Inadvertent topical steroid treatment may allow progressive enlargement of the ulcer to a *geographical or 'amoeboid'* configuration

### *Treatment of epithelial keratitis*

**Topical** antiviral agents (trifluorothymidine, aciclovir, vidarabine and ganciclovir) are equally effective. The most frequently used drug in Europe is aciclovir 3% ointment administered five times daily. It is relatively non-toxic, even when given for up to 60 days, because it acts preferentially on virus-laden epithelial cells. On this treatment 99% will be resolved by 2 weeks.

**Debridement** may be used for dendritic but not geographic ulcers.

## **Herpes zoster ophthalmicus**

Is a herpes Zoster attack that affect the ophthalmic division of trigeminal nerve

### *Pathogenesis:*

The varicella-zoster virus (VZV) causes chickenpox (varicella) and shingles (herpes zoster). VZV and HSV belong to the same subfamily of the herpes virus group and are morphologically identical but antigenically different. After the initial attack of chickenpox the virus travels in a retrograde manner to the dorsal root and cranial nerve sensory ganglia where it may remain dormant for decades. From there it can reactivate to cause shingles after VZV-specific cellular immunity has faded.

### *Risk of ocular involvement*

**Hutchinson sign** describes involvement of the external nasal nerve, which supplies the side of the tip, and the side and root of the nose

**Age.** HZO occurs most frequently in the sixth and seventh decades

**AIDS** patients tend to have more severe disease. The development of shingles in children or young adults (< 50 years) should prompt a search for immunodeficiency or malignancy.

### **Clinical Features:**

1. **Acute epithelial keratitis** develops in about 50% of patients within 2 days of the onset of the rash and resolves spontaneously within a few days.
  - It is characterized by small, fine, dendritic lesions which, in contrast to herpes simplex dendrites, have tapered ends without terminal bulbs.
  - The lesions stain with fluorescein and rose bengal.
  - Treatment is with a topical antiviral, if appropriate.

**2. Conjunctivitis, Episcleritis, Scleritis, Nummular keratitis, Stromal keratitis, Disciform keratitis, Anterior uveitis**

## **FUNGAL KERATITIS**

Fungi are micro-organisms that have rigid walls and multiple chromosomes containing both DNA and RNA. The main types are:

1. **Filamentous** fungi (*Aspergillus* spp., *Fusarium solani*, and *pscedosporium* spp.)
2. **Yeasts** (*Candida* spp.)

The primary risk factors for infection are trauma (65% of cases in tropical areas) particularly with vegetable matter, chronic ocular surface disease and epithelial defects, diabetes, systemic immunosuppression and hydrophilic contact lenses.

Treatment: Antifungal agents ,topical, subconjunctival and even systemic

## NEUROTROPHIC KERATITIS

Neurotrophic keratopathy occurs when there is loss of the trigeminal innervation to the cornea resulting in partial or complete anaesthesia.

*Causes:*

1. **Acquired.** Damage to the fifth cranial nerve or trigeminal ganglion e.g following surgical ablation for trigeminal neuralgia , or tumour (acoustic neuroma or neurofibroma).
2. **Systemic diseases** such as diabetes and leprosy.
3. **Ocular disease** such as herpes simplex and herpes zoster keratitis,
4. **Congenital** causes include familial dysautonomia (Riley-Day syndrome).

*Treatment*

1. **Topical** lubricants (non-preserved) for associated dry eye or corneal exposure
2. **Protection of the ocular surface**
  - a. **Simple taping** of the lids to provide temporary protection.
  - b. **Botulinum toxin<sup>®</sup> injection** to induce protective ptosis.
  - c. **Tarsorrhaphy** to prevent drying and reduce exposure,
  - d. **Therapeutic silicone contact lenses**

## EXPOSURE KERATITIS

Exposure keratopathy is the result of incomplete lid closure (lagophthalmos) during blinking.

*Causes*

1. **Neuroparalytic**, especially facial nerve palsy which may be idiopathic or the result of surgery for acoustic neuroma or parotid tumour.

**2. Reduced muscle tone** as in coma or parkinsonism

**3. Mechanical** :Eyelid scarring associated with cicatricial pemphigoid, burns (Fig. 9.29b) and trauma.

**4. Abnormality of globe position:** Severe proptosis resulting in lagophthalmos due to thyroid ophthalmopathy or orbital tumour..

: Treatment depends on the severity of exposure and whether recovery is anticipated.

- Artificial tears (non-preserved) during the day and ointment at night.
- Taping the lid closed at night.
- Bandage contact lenses,
- Temporary or permanent tarsorrhaphy

## **CORNEAL ECTASIAS**

### **Keratoconus**

Keratoconus is a progressive disorder in which the cornea assumes a conical shape secondary to stromal thinning and protrusion . The onset is around puberty with slow progression thereafter until the third or fourth decades of life, when it usually arrests, although the ectasia can become stationary at any time. Both eyes are affected, in almost all cases.

*Presentation* is typically during puberty with unilateral impairment of vision due to progressive myopia and astigmatism, which subsequently becomes irregular.

#### *Treatment*

1. **Spectacles** in early cases.
2. **Rigid contact lenses** are required for higher degrees of astigmatism.
3. **Keratoplasty**, penetrating or deep lamellar, is indicated in patients with advanced progressive disease,

## **CORNEAL DEGENERATIONS**

### **Age-related degenerations**

#### *Arcus senilis*

Arcus senilis is the most common peripheral corneal opacity, which frequently occurs without predisposing systemic conditions in elderly individuals.

Occasionally arcus may be associated with familial and non-familial dyslipoproteinaemias. Hyperlipoproteinaemia, most notably type II, is frequently associated with bilateral arcus.

### **Band keratopathy**

Band keratopathy is a common condition characterized by the deposition of calcium salts in Bowman layer, epithelial basement membrane and anterior stroma .

Causes may include chronic uveitis, advancing age ,and chronic renal failure.



