The Cornea

Anatomy and physiology

The cornea is a complex structure ,represent the anterior 1/5 of the outer layer of the eye . ball

The normal cornea is transparent , avascular , nutrients are supplied and metabolic .products are removed mainly via the aqueous humor posteriorly and the tears anteriorly

It is the most densely innervated tissue in the body , supplied by the 1st division of the . trigeminal nerve

. The average corneal diameter is 11.5 mm vertically and 12mm horizontally

Average thickness 500 – 550 micrometer centrally and thicker toward the periphery (660 mm)

Functions

Protective role ,protects the internal ocular structures

Refraction …. 2/3 – ¾ of the refractive power of the eye

Layers of the cornea

five layers

1. The epithelium is non keratinized stratified squamous epithelium (5-7)

2..Bowman layer is acellular superficial layer of the stroma formed from collagen fibers

3.The stroma makes up 90% of the corneal thickness . It is arranged in regularly oriented layers of collagen fibrils whose spacing is maintained by proteoglycan ground substance , with interspersed modified fibroblasts (keratocytes) .maintenance of the regular arrangement and spacing of collagen is critical to optical clarity.

The stroma cannot regenerate following damage

4.Descemet membrane is a discrete sheet of distinct collagen fibrils .it serves as modified . basement membrane for the endothelium .It has a regenerative potential

5.The endothelium consist of monolayer of cells .endothelial cells maintain corneal deturgescence throughout life by pumping excess fluid out of the stroma .The cells cannot . regenerate

Promotion of re-epithelialization

in eyes with stromal thinning is important because thinning seldom progresses if the .epithelium is intact

1 Reduction of exposure to toxic medications and preservatives

2..Lubrication with artificial tears and ointments

3. Lid closure is particularly useful in exposure and neurotrophic keratopathies as well as .3
.in eyes with persistent epithelial defects

.Tarsorrhaphy may be necessary in patients with chronic disease

4.Bandage soft contact lenses promote healing because they mechanically protect the .4
regenerating corneal epithelium from the constant rubbing action of the eyelid

; Indications of BSCL

Promotion of healing

. To improve comfort particularly in the presence of large corneal abrasion

. To seal a small perforation

5.Limbal stem cell transplantation ;if there is stem cell deficiency as in chemical burns and .
cicatrizing conjuctivitis

6. Smoking should be discontinued .

Prevention of perforation

:can be with one of the following

. Tissue adhesive glue may be used to limit stromal ulceration and to seal small perforations

Conjunctival flaps which cover the cornea if ulceration is progressive and unresponsive to .
.other measures

Immunosuppressive agents may be useful in certain forms of severe peripheral ulceration
.such as Mooren's ulcer and those associated with systemic connective tissue disorders

Amniotic membrane grafting for persistent epithelial defects

.Corneal grafting may be required to restore corneal transparency

Bacterial keratitis

The pathogens able to produce corneal infection in the presence of an intact epithelium are Neisseria gonorrhoeae, Corynebacterium diphtheriae, Listeria sp. and Haemophilus sp. Other bacteria are capable of producing keratitis only after loss of corneal epithelial integrity :associated with the following predisposing factors

1.Contact lens wear, particularly extended wear soft lenses, is the most common predisposing factor in patients with previously normal eyes. The infection is often caused by

Pseudomonas aeruginosa which requires an epithelial defect for corneal invasion. Such . defects occur in all contact lens wearers at times

2.Ocular surface disease which disrupts defence mechanisms such as post herpetic corneal . disease, bullous keratopathy ,trauma,, corneal exposure and dry eye. The risk of bacterial keratitis is further increased by the presence of chronic dacryocystitis ,chronic blepharitis ,Trachiasis ,entropion as well as the administration of topical or systemic immunosuppressive agents

3.(Keratoconjunctivitis sicca (dry eye)

4. Prolonged use of topical steroids .

CLINICAL FEATURES

Symptoms and signs

Pain usually severe ,photophobia

Purulent discharge

Ciliary injection

Visual impairment ,severe if the visual axis is involved

Hypopyon sometimes (a mass of white cells collected in the anterior chamber)

. A white corneal opacity which can often be seen with the naked eye

MANAGEMENT

A bacterial corneal ulcer is a sight-threatening condition which demands urgent .identification and eradication of the causative organism

1. Hospitalization

2. Corneal scraping for Gram staining and C/S

3.Intensive topical Antibiotics .the initial therapy should be with a combination of a . fortified amino glycoside and cefuroxime orciprofloxacin (dual therapy) or monotherapy .with flouroquinolone & then according to C/S

. Topical Sub conjunctival Systemic

4.Cycloplegics, such as atropine, should be used in all eyes with bacterial keratitis to . prevent the formation of posterior synechiae from secondary anterior uveitis, and to reduce pain from ciliary spasm

5. Steroid therapy is controversial

Causes of failure to respond

The following are the main circumstances in which a bacterial keratitis might not respond to :treatment

1.Wrong diagnosis caused by inappropriate cultures. The most common causes are unrecognized infection with herpes simplex virus, fungi, acanthamoebae and atypical .mycobacterial infection

2..Wrong treatment caused by inappropriate choice of antibiotic

3..Drug toxicity preventing corneal healing .

4. Inadequate control of host response .

Herpes simplex keratitis

PRIMARY OCULAR INFECTION

Primary ocular infection typically occurs in children between the ages of 6 months and 5 years, and may be associated with generalized symptoms of a viral illness. In most cases . primary herpetic infection is self-limited and seldom presents serious problems

Clinical features

Blepharoconjunctivitis is usually benign and self-limited and, in children, it may be the only .manifestation of primary herpetic infection

The skin lesions typically involve the lids and periorbital area. Initially, they consist of vesicles .which rapidly form superficial crusts and then heal without scarring

The conjunctivitis is unilateral, acute, follicular, and associated with a watery discharge

.Pre auricular adenopathy

Fever

Keratitis develops within a few days in about 50% of patients with blepharoconjunctivitis. A fine epithelial punctate keratitis may be a transient finding. A coarse epithelial punctate keratitis may give rise to a variety of epithelial lesions which subsequently progress to dendritic shapes

Dendritic ulceration which may be single or multiple is the hallmark of epithelial herpetic disease

:Other causes of non-herpetic dendritic ulceration include

1. Herpes zoster keratitis . .
2. A healing corneal abrasion. .
3.Soft contact lenses which may rarely give rise to a pseudo dendrite in the mid peripheral .

cornea.
4.Toxic keratopathies usually caused by excessive drop administration .

Treatment

1. Antiviral.

acyclovir eye ointment

2. Debridement .

is an effective way of treating dendritic ulcers, especially when combined with antivirals, .but is not appropriate for geographical ulcers

,this mode of treatment has generally been relegated to resistant cases ,

,non-compliance

allergy to antiviral agents and

.unavailability of antiviral agents

Corneal degenerations

: ARCUS SENILIS

.is the most commonly encountered peripheral corneal opacity

1. Age –related .. all individuals over the age of 80.

2. In young , usually due to familial and non-familial dyslipoproteinaemias.

Unilateral arcus is a rare entity that may be associated with carotid disease or ocular .hypotony

Examination shows bilateral lipid deposition which starts in the superior and inferior perilimbal cornea and then progresses circumferentially to form a band about 1 mm wide , .the sharp peripheral edge is separated from the limbus by a clear zone of cornea

Band keratopathy

a relatively common disorder characterized by the deposition of calcium salts in the .subepithelial space and anterior portion of Bowman's membrane

:The four main causes, in order of frequency, are

,chronic iridocyclitis particularly in children

,idiopathic in the elderly

phthisis bulbi and

. increased serum calcium or phosphorus levels

Examination shows the characteristically interpalpebral distribution of the lesions with a clear space separating the sharp margin of the band from the limbus

Treatment is indicated for visual or cosmetic reasons by
the following methods:
1.Chelation is a simple and effective form of treatment for relatively mild cases. it is .
performed as follows:
(a) The corneal epithelium overlying the opacity is scraped off with a knife
(b) A solution of sodium versenate (0.01 mol/l) is then applied to the denuded cornea with a cotton-tipped bud until all calcium has been removed (Figure 5.44, bottom); this usually .takes about 10 minutes. The eye is then padded until the epithelium has regenerated.
2.Excimer laser keratectomy may be used for cases with more extensive and deeper .involvement

Keratoconus

Keratoconus (conical cornea) is a fairly common, progressive disorder in which the cornea .assumes an irregular conical shape

The hallmark of keratoconus is central or paracentral stromal thinning, apical protrusion and irregular astigmatism. The condition starts at around puberty and progresses slowly thereafter, although it may become stationary at any time

Both eyes are affected in about 85% of cases, although the severity of involvement may be .markedly asymmetrical

The aetiology of keratoconus is obscure

Keratoconus occurs with increased frequency in the following disorders

Systemic disorders include: Down's syndrome, Turner's syndrome, Ehlers-Danlos syndrome, .Marfan's syndrome, atopy, osteogenesis imperfecta and mitral valve prolapse

Ocular associations include: vernal disease, Leber's congenital amaurosis, retinitis pigmentosa, blue sclera, aniridia and ectopia lentis. The wearing of hard contact lenses and .constant eye rubbing have also been proposed as possible predisposing factors

By keratometry keratoconus is classified as mild (<48 D), moderate (48 -54 D) and severe .(>54 D)

CLINICAL FEATURES

Onset is typically between the ages of 10 and 20 years, with impaired vision in one eye caused by progressive astigmatism and myopia. The patient may report the need for frequent changes in spectacle correction or a decreased tolerance to contact lens wear. As a result of the asymmetrical nature of the condition, the fellow eye usually has normal vision with negligible astigmatism. Subsequently, the astigmatism becomes irregular. The course is variable but, as the condition progresses, the amount of astigmatism in the fellow eye also increases

Early signs, which are easy to miss, can be detected by the following methods of examination:

1. Retinoscopy shows an irregular 'scissor' reflex.
2. Keratometry initially shows irregular astigmatism where the principal meridians are no longer 90 D apart and the mires can not be superimposed.
3. Slitlamp biomicroscopy shows very fine, deep, stromal, oblique striae (Vogt's lines) which disappear with external pressure on the globe . Prominent corneal nerves may also be present.

Late signs consist of the following:

1. Progressive central or paracentral corneal thinning, of as much as one-third of the corneal thickness. This is associated with poor visual acuity.
2. Bulging of the lower lid when the patient looks down (Munson's sign; (Figure 5.55, bottom left).
3. Epithelial iron deposits (Fleischer's ring) may surround the base of the cone.
4. Central and paracentral corneal scarring in severe cases.
5. Acute hydrops results from ruptures in Descemet's membrane and acute leakage of fluid into the corneal stroma and epithelium . This causes a sudden drop in visual acuity associated with discomfort and watering. Although the break usually heals within 6-10 weeks and the corneal oedema clears, a variable amount of stromal scarring may develop. Acute episodes are initially treated with hypertonic saline and patching or a soft bandage contact lens. In some severe cases keratoplasty may be necessary

MANAGEMENT

Spectacle correction in very early cases can correct regular astigmatism and very low amounts of irregular astigmatism.

Hard contact lens

Cross-linking

Intracorneal ring (intac)

keratoplasty

Keratoplasty

corneal transplantation or corneal grafting = abnormal host tissue is replaced by healthy donor corneal tissue.

. The graft may be partial thickness (lamellar) or full thickness (penetrating)

; INDICATIONS

1. Optical indications are primarily improvement of visual acuity by replacing opaque corneal tissue with clear donor tissue. Currently, the most common indication is pseudophakic

bullous keratopathy. Other common indications are keratoconus, corneal dystrophies and degenerations, and scarring caused by various types of keratitis and trauma

2.Tectonic indications are restoration or preservation of corneal anatomy in eyes with . severe structural changes such as stromal thinning and descemetoceles

3.Therapeutic indication is removal of inflamed corneal tissue in eyes unresponsive to . conventional antimicrobial or antiviral therapy

4. Cosmetic indication is improvement of the appearance of the eye .

Complications of contact lens wear

ALLERGIC CONJUNCTIVITIS

GIANT PAPILLARYCONTUNCTIVITIS

CORNEAL COMPLICATIONS

Epithelial oedema secondary to hypoxia which is usually reversible

,Corneal vascularization as a response to lens-induced hypoxia

Sterile corneal infiltrates usually small peripheral opacities , asymptomatic and detected during a routine follow-up examination. They usually disappear once contact lens wear has been discontinued

Microbial keratitis

Corneal warping resulting in severe and permanent astigmatism may occur in some eyes as a response to chronic hypoxia

Exposure keratopathy

Exposure keratopathy is caused by improper wetting of the corneal surface by the precorneal tear film because of the inability of the lids to resurface the cornea with each .blink. This occurs despite the presence of normal tear production

Important causes include facial nerve palsy, severe proptosis and scarring of the eyelids. Occasionally, corneal exposure during sleep may occur in normal individuals in the absence .of any of these factors

Examination shows a spectrum of clinical findings which ranges from minimal inferior punctate epitheliopathy to severe ulceration, neovascularization, infection and even perforation

Treatment, if recovery is anticipated, is with frequent use of artificial tears during the day and, at night, instillation of ointment should and taping shut of the eyelids. If the underlying condition is likely to be permanent, lid surgery is usually required

Episcleritis and scleritis

The scleral stroma is composed of collagen bundles of varying size and shape that are not as . uniformly oriented as in the cornea

The anterior episclera consists of a dense, vascular connective tissue which merges with the .superficial sclera stroma and Tenon's capsule

Episcleritis

CLINICAL FEATURES

Episcleritis is a common, benign, self-limiting and frequently recurrent disorder which typically affects young adults. It is seldom associated with a systemic disorder and never .progresses to a true scleritis

.The two clinical types of episcleritis are simple and nodular

.Presentation is with unilateral mild discomfort, tenderness to touch and watering

Examination of simple episcleritis shows sectoral or, rarely, diffuse redness . By contrast, nodular episcleritis is localized to one area of the globe, forming a nodule with surrounding .injection

TREATMENT

Simple episcleritis usually resolves spontaneously within 1-2 weeks although the nodular .type may take longer

Mild cases may need no specific therapy but if discomfort is annoying, topical steroids .and/or topical non-steroidal anti-inflammatory drugs (NSAIDs) may be helpful

In the rare unresponsive recurrent disease, systemic flurbiprofen (100 mg three times daily), taken at the first symptom of recurrence, may be successful in aborting the attack. Systemic indomethacin (50 mg twice daily) may also be used but is not as effective as .flurbiprofen

Scleritis

Scleritis is a granulomatous inflammation of the scleral coat of the eye. It is much less common than episcleritis. The condition covers a spectrum of ocular disease which

extends from trivial self-limiting episodes of inflammation to a necrotizing process that may cause sight-threatening complications such as uveitis, cataract, glaucoma, keratitis, .retinal oedema and optic neuropathy

ASSOCIATED SYSTEMIC DISEASES

:About 45% of patients with scleritis may have one of the following systemic diseases

Rheumatoid arthritis is by far the most frequent

Connective tissue vascular disorders include Wegener's granulomatosis, polyarteritis nodosa . and SLE

Miscellaneous conditions include: relapsing polychondritis, herpes zoster and surgically .induced scleritis

Treatment

.Oral prednisolone 60-120 mg daily for 2-3 days , tapered accordingly, as the pain subsides

Immunosuppressive drugs (cyclophosphamide, azathioprine or cyclosporin) may be . necessary in steroid resistant cases

Combined therapy with pulsed intravenous methyl-prednisolone (500-1000 mg) and cyclophosphamide 500 mg is reserved for the minority of patients who fail to resolve with .oral therapy or those who present with scleral necrosis