CORNEA

The cornea forms the anterior 1/6 of the outer coat of the eye. It fits into the sclera as a watch glass.
- Refractive power : + 42 D
- radius of curvature : 7.8 mm
- refractive index : 1.36

Anatomy of the cornea :

Thickness : 0.7 mm in periphery and 0.5 mm in the center
Diameter : 11 mm vertical and 12 mm horizontal

In section, the cornea is formed of the following layers :
1. The epithelium : stratified non keratinized squamous epithelium, 5 to 6 layers.
   - basal layer : columnar
   - intermediate : polygonal
   - superficial : flat
2. Bowman’s membrane : (10-12 microns)
   Homogenous non cellular collagenous membrane. It resists injury but if destroyed it can not regenerate
3. Stroma (substantia propria ) (500 microns)
   - forms 90% of thickness of the cornea
   - collagen bundles arranged in layers: are parallel in the same layer and perpendicular to those of the next layer. Lamellae are compact together.
   - in between, there are keratocytes (fixed corneal cells) and wandering leukocytes

4. Descemet’s membrane :
is an elastic membrane that can regenerate (secretion of the endothelium)

5. **Endothelium**:
   Single layer of flat cells which is continuous with the endothelium of the trabecular meshwork.

The cornea is avascular except for the peripheral 1 mm where the conjunctiva, with its blood vessels, extends and covers the peripheral 1 mm of the cornea.

---

**The limbus** (corneo scleral junction)

Here the following events occur:
- **Corneal epithelium**: becomes continuous with conjunctival epithelium
- **Bowman's membrane**: ends in a rounded border
- **Substantia propria**: becomes continuous with the sclera
- **Descemet membrane**: becomes continuous with the trabecular meshwork
- **Endothelium**: is continuous with that of the angle of the anterior chamber

1. The basal epithelial cells at the limbus contain a reservoir of **stem cells**. These cells act as a barrier that prevents the conjunctival epithelium from invading the cornea and also divide and replace any damaged corneal epithelial cells. Such cells can spread circumferentially or centrally to replace damaged corneal epithelium.
2. It contains the exit channels for the aqueous.
3. The limbus is an important landmark for eye surgeries.
Nutrition: by diffusion from aqueous and tears. The periphery by diffusion from the limbal capillaries:

1. Cornea is avascular except for the peripheral one mm where the conjunctiva overlies the peripheral one mm of the cornea. Anterior ciliary branches form vascular limbal arcade around the cornea. Diffusion of needed elements occurs from these vessels to the periphery of cornea.

2. Oxygen is supplied by diffusion from tear fluid. The direct exposure of the tear film to the atmosphere is essential for the supply of oxygen. Glucose is derived from the aqueous humor.

Nerve supply: ciliary nn (naso-ciliary n.)

Corneal reflex: touching the cornea will result in closure of the lids.

Afferent: 5th (sensation of the cornea) and efferent: 7th (motor to orbicularis oculi)

Corneal transparency:

1. Corneal detergescence (relative dehydration): water content of the cornea is 70% i.e. the cornea is relatively dehydrated in comparison to the surrounding aqueous humor and tears, This is achieved by the endothelial function of sodium pump. Sodium is removed from cornea into the aqueous, taking water with it. Loss of endothelial function will result in corneal edema and loss of transparency (e.g. as occurs in acute congestive glaucoma) ATP is required as an energy source.

2. Intact epithelium and endothelium: prevents the movement of the fluids across them into the corneal substance

3. Corneal lamellae: being of uniform size and compact arrangement minize scattering of light.
   - If this compactness is lost, as in cases of corneal edema, scatter of light will be great and transparency of cornea will be affected.
   - Loss of this unique arrangement of corneal lamellae, as in cases of corneal scars, results in opacification.

4. Absence of blood vessels

5. Absence of keratinization of the epithelium

6. Absence of myelin sheeth in nerves of the cornea

Normally a luster is seen on corneal surface:
due to reflection of light at the interface between the tear film and the epithelium of the cornea. So absence of any of these two layers (tear film and corneal epithelium) will result in loss of luster.

Inflammation of the cornea (keratitis)

Inflammation of cornea could be classified into
Ulcercative keratitis
- Non ulcerative keratitis

Also inflammation could be classified according to inflammatory initiator into:

**A. Toxic agent:**
1. Infective agent
   - Bacterial keratitis
   - Fungal keratitis
   - Viral keratitis: HS and HZ
   - Protozoal keratitis: Acanthamebia
2. Non infective agent
   1. Neurotrophic keratitis (neuroparalytic keratitis): loss of sensation
   2. Exposure keratitis (ulcer with lagophthalmos): dryness
   3. Keratomalacia: vitamin A deficiency
   4. Mooren's ulcer: degenerative condition i.e. of unknown etiology
   5. Trauma to cornea
      - Mechanical: sharp instrument like a finger nail, edge of a paper
      - Chemical: acid or alkali
      - Physical injury: photophthalmia (UV light)

**B. Allergic agent:**
1. Keratitis due to hypersensitivity reactions
   - Marginal ulcers
   - Phlyctenular keratoconjunctivitis
   - Spring catarrh keratitis
2. Autoimmune diseases: rheumatoid arthritis, polyarteritis nodosa

**Corneal abrasion:** means loss of epithelium
(while corneal ulcer means: loss of epithelium + secondary infection)

**Etiology** of corneal abrasion:
- Trauma to the eye (finger nail ....etc), projecting PTCs.

**S.S.:**
- Severe pain (exposed nerve endings)
- Lacrimation, photophobia, blepharospasm
- Loss of luster
- +ve fluorescein stain

**Treatment:**
- Bandage and topical antibiotic drops and oint (guard against secondary infection)
**Microbial corneal ulcer:**
It is a localized necrosis of the epithelium and underlying superficial layers of the stroma.

**Predisposing factors:**
The cornea is protected from external environment by the presence of an intact epithelial layer together with the tear film which has several defense mechanisms (washing away, lysozyme enz.) Gonococci, Diphtheria, Lysteria and Hemophilis can invade intact epithelium, but the rest of organisms can invade the cornea only if epithelium is absent or not healthy.
Factors that facilitate the occurrence of corneal ulcer include:
- abrasion (no epithelial barrier) due to trauma with finger nail, rubbing lash
- unhealthy epithelium:
  - hypoxia e.g. sleeping with daily wear contact lens
  - dry eye e.g. lagophthalmos
  - vitamin A deficiency
  - diminished corneal sensation
- decreased general resistance: very old debilitated persons

Source of organisms:
- lid margin: blepharitis
- conjunctiva: as in conjunctivitis
- lacrimal sac: as in dacryocystitis
- contact lenses not properly handled
- Air borne infection

Organisms: could be bacterial, fungal, viral, protozoal.

Pathology of corneal ulcer:

1. Stage of infiltration: invasion of the cornea with organisms will result in inflammatory reaction:
   - dilatation of the pericorneal B.V, to get leucocytes (ciliary injection)
- infiltration of cornea by polymorphes and keratocytes which becomes transformed into histocytes. Clinically an area of gray colour is present in the cornea.

2. Stage of ulceration:
The battle between the organisms and the defense mechanisms will result in formation of necrotic tissue. The necrotic tissue then casts off resulting in ulcer.

a. Unclean ulcer: at first the ulcer is not clean, its floor is covered by necrotic material and dense infiltration surrounds the ulcer. Clinically the ulcer appears dirty with irregular surface.

b. Clean stage: all the necrotic material is sheded and infiltration becomes less. Clinically the ulcer becomes clean, less opacification, and with sloping edges.

3. Healing stage:
The epithelium heals by migration and mitosis, while substantia propria heals by formation of fibrous tissue which results from activity of keratocytes. As the rate regeneration of epithelium is faster than that of fibrous tissue, epithelium will cover the area while it is still depressed. (facet formation) After sometime, fibrous tissue continues regeneration and the depressed area gradually disappears till the surface becomes smooth. The newly formed fibrous tissue is irregular and of different sizes resulting in opacity formation.

- Bowman’s membrane does not regenerate.

Symptoms:

- Pain: pricky in character. Is due to (1) presence of exposed nerve endings and (2) the associated toxic iritis (with spasm of ciliary and sphincter pupillae muscle)
- **Headache**: fronto temporal headache, is referred pain

- **Photophobia, lacrimation, blepharospasm**: due to exposed nerve endings, disappear with application of local anesthesia.

- **Diminution of vision**: could be due to (1) hazy media, if ulcer is central and (2) if associated iritis is severe (spasm of ciliary muscle..... myopia)

**Signs:**
- **Loss of luster**: corneal luster is due to reflection of light at the interface between the tear film and epithelium. Loss of either of them will lead to loss of the corneal luster (e.g. dry eye, corneal abrasion, corneal ulcer)
- **+ve fluorescein stain**: Stain can get access through the absent epithelium and combines with the ground substance of the stroma giving it greenish discoloration, which becomes more prominent with the use of blue light.
  - The ulcer appears gray with irregular edge in the unclean stage and sloping edge in the clean stage
- **Ciliary injection**
- **Miosis**: due to toxic iritis:
  - Congestion of the radially arranged BVs of the iris
  - Irritation of nerves to sphincter pupillae muscle
- **Diminished visual acuity**:

---

**S & S:**
- Pain (pricky), headache
- Photophobia, lacrimation, blepharospasm

**Loss of lustre**
**+ve fluorescein**

**Ciliary injection**

**Miosis**

---
Complications of corneal ulcer:

A. Complications before perforation:

1. Corneal opacities:
   a. Nebula: is a faint opacity results from healing of ulcer which was superficial. It appears grayish white in colour and the colour of the underlying iris or pupil can be seen partially through it.
   b. Leucoma non adherent: It is a dense opacity in the cornea as the ulcer was deeper. It appears white in colour
   c. Macula: the corneal opacity is intermediate between nebula and leucoma.

   Opacities of the cornea may affect the vision directly or through the resultant astigmatism.

2. Keratoectasia:
   This may occur due to weak scar which bulges under the effect of the intraocular pressure.

3. Facet: depressed area covered by epithelium

4. Iridocyclitis:
   due to diffusion of toxins (not the organisms) The iris may pour exudates into A.C. producing hypopyon (pus in AC) which is sterile i.e. does not contain organisms.
5. Secondary glaucoma:
Secondary to iritis or peripheral anterior synechiae blocking the angle of A.C.

6. Descematocele:
When the ulcer extends deep, reaching the Descemet’s membrane, the later will bulge under the effect of the Intra-ocular pressure. It appears clinically thin and transparent area in the cornea. It may be missed for perforated ulcer.

<table>
<thead>
<tr>
<th>Descematocele</th>
<th>Perforated ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.C.</td>
<td>present</td>
</tr>
<tr>
<td>I.O.P.</td>
<td>normal</td>
</tr>
<tr>
<td>pupil</td>
<td>regular</td>
</tr>
<tr>
<td></td>
<td>Lost</td>
</tr>
<tr>
<td></td>
<td>Soft eye (zero IOP)</td>
</tr>
<tr>
<td></td>
<td>pear shaped</td>
</tr>
</tbody>
</table>

NB Descematocele does not occur in cases of typical hypopyon ulcer due to destruction of Descemet’s membrane in the early stages of disease due to formation of posterior abscess typically seen in cases of hypopyon ulcer.

7. Pseudo pterygium
In cases of resistant corneal ulcers a conjunctival flap is prepared by dissection of nearby conjunctiva and fixing the flap with sutures so that it covers the ulcer. This will help healing as the overlying conjunctiva is full of blood vessels and thus body defense mechanisms can reach the ulcer effectively. Of course this cannot be done in cases of allergic corneal ulcers. In the following days, usually the conjunctiva will recede back to its original position, and may leave behind a triangular band of conjunctiva attached to the base of the ulcer (here two rough surfaces are present)
DD: true pterygium:

<table>
<thead>
<tr>
<th>True pterygium</th>
<th>Pseudo- pterygium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Always nasal or temporal</td>
<td>Any site</td>
</tr>
<tr>
<td>Cannot pass a glass rod underneath</td>
<td>Can pass a glass rod between the limbus and site of ulcer</td>
</tr>
</tbody>
</table>

B. Complications after perforation:

Perforation usually follows sudden rise of I.O.P. It may follow straining, coughing, squeezing the eye......etc. The result of perforation differs according to the site and the size of perforation.

1. Peripheral anterior synechia (PAS)

If the perforation occurs in a small eccentric ulcer, aqueous gets out and anterior chamber is lost, so the iris becomes in contact with the back of the cornea. Since the perforation is too small to allow prolapse of iris, iris becomes in contact to the back of the perforation site and on healing a small opacity is formed with iris attached to its posterior surface. Peripheral anterior synechiae (PAS) may develop in different parts of the angle of the anterior chamber AC is lost for long time.
2. Leucoma adherence:

If perforation is eccentric but bigger in size, allowing part of the iris to prolapse. The exposed iris in the base of the perforation site will develop iritis from exposure to the products of the ulcerative process and the external atmosphere. Iris will end in fibrosis that will fill the perforation site but the iris tissue will be included in it as well. So the end result is opacity (leucoma) to which the iris is adherent.

<table>
<thead>
<tr>
<th></th>
<th>Leucoma adherent</th>
<th>Leucoma non adherent</th>
</tr>
</thead>
<tbody>
<tr>
<td>looking from side</td>
<td>iris is adherent to back of opacity</td>
<td>no iris adhesion to back of opacity</td>
</tr>
<tr>
<td>when light is applied from the side</td>
<td>tent shaped iris one side of iris is illuminated and the second side is not</td>
<td>is of normal shape whole surface of iris is illuminated</td>
</tr>
<tr>
<td>AC</td>
<td>irregular and shallow</td>
<td>regular depth</td>
</tr>
<tr>
<td>Shape of pupil</td>
<td>pear shaped</td>
<td>regular</td>
</tr>
</tbody>
</table>
Partial anterior staphyloma: Sometimes, the scar tissue filling the defect in the cornea, to which the iris is adherent, may give way under the effect of IOP. This is likely to occur specially if the IOP is high. If this occurs, the corneal scar becomes ectatic and the iris is adherent to it.

3. Corneal fistula:

If perforation is small and is central in position (in front of the pupil), aqueous will escape and AC will be lost but iris can not prolapse and occlude the defect. The perforation site will be closed by a plug of fibrin. If the plug is kept in place long enough, fibrin plug will be replaced by fibrous tissue and leucoma non adherent will form.

If fibrin plug falls away as a result of sudden increase of IOP again AC will be lost. Later, another fibrin plug will be formed. Sudden increase of IOP with cough, sneezing, constipation. If this process of plug formation and dislodgment is repeated many times, there will be enough time for epithelium to grow and line the sides of the perforation then changing it into a fistula. Once covered by epithelium, no more fibrin plug can be formed.

Fistula formation means that aqueous keeps going outside the eye and the AC will be lost. Prolonged contact of iris to the back surface of the cornea (more than 5 days) will result in iritis and trabeculitis. This will result in PAS (peripheral anterior synechia)
The diagnosis of corneal fistula:
- AC is lost
- The tension is soft
- River sign: fluorescein 2% is instilled into the conjunctival sac and the patient is examined on the slit lamp. Fluorescein will stain the tear film green, a track of clear aqueous (not stained with fluorescein) will be seen starting from the site of perforation and going down by the effect of gravity.

4. Total anterior staphyloma:

Occurs when the perforation is big in size and central in position. Most of the iris will bulge forward and fill the perforation, exposed to external atmosphere and ulceration process will induce iritis that will end in fibrosis. Iris tissue will be included in this fibrous tissue and the defect will be closed by this fibrous tissue (called pseudo cornea). Since the pathway of the aqueous is disturbed (pupil, AC, angle of AC) secondary glaucoma will eventually occur. Rise of IOP will push the scar tissue forward and bulging will occur irregularly since the density of the scar tissue is not uniform all over the area. The end result will be multiple small areas of bulging scar tissue and in between less bulging areas where scar tissue is more. The iris is amalgamated into the scar tissue,
Other complications of perforated corneal ulcer:

1. Lens:
   - **Anterior polar cataract**: with small central ulcer that perforates, as the AC will be lost and the lens will move forward and come in contact with the perforation. Toxins will affect the anterior pole of the lens resulting in anterior polar cataract.
   - Tear of the suspensory ligament: at time of perforation, sudden decrease of the IOP to zero may result in rupture of part of the suspensory ligament and **subluxation of the lens** occurs. If the whole suspensory ligament was torn, the lens will be **totally dislocated**. Anterior dislocation into the AC may occur, and even may be completely extruded outside the eye if the perforation is big enough to allow escape of the lens outside the eye.

2. Secondary glaucoma
   - PAS
   - Secondary to iritis and its complications
   - Lens induced glaucoma

3. Intra ocular hemorrhage:
   Straining will rise the systemic blood pressure inside the blood vessels of the choroid, at the same time the strain may result in perforation with sudden loss of IOP, so the pressure applied to BVs inside the eye will drop to zero. These two factors may result in rupture of one of the major choroidal blood vessels with occurrence of I.O. hge. This is more likely to occur if the vessel walls were unhealthy as in cases of atherosclerosis.

4. Endophthalmitis:
   Organisms may enter the eye through the perforation site and may result in suppurative inflammation of the interior of the eye.

5. Atrophia bulbi:
   May follow healing of endophthalmitis with formation of fibrous tissue inside the eye.

**In case of children**, visual disturbance of one reason or another may result in:
Squint: if visual affection is only in one eye (or more in one eye), binocular vision will not develop and this will lead to squint.

- Nystagmus: if visual affection occurs in both eyes in the first 6 months of life, nystagmus will occur. Light entry is essential for the development of the macula.

Treatment of corneal ulcer:

1. Removal of the predisposing factors:
   - as removal of FB, rubbing lashes...
   - ttt of dacryocystitis, conjunctivitis, blepharitis

2. Treatment of the ulcer:
   
   | cycloplegics + antimicrobial agent + bandage |
   |
   |

Topical antimicrobial agent:

Infective corneal ulcer is a sight threatening condition demanding urgent identification and eradication of the causative micro organism. Scrapings should be taken from the base and edges of the ulcer. Scraps not only provide material for cultures but also debride necrotic tissue and enhance drug penetration.

- Broad spectrum Antibiotic in the form of eye drops during day and ointment during night. Fortified antibiotic eye drops are prepared and given to patient to ensure high drug concentration.
- We start with broad spectrum antibiotics tell the results of culture are available then type of antimicrobial is modified accordingly.

Cycloplegics:

- atropine sulfate drops or ointment: duration of action of atropine 1 week
  Atropine drops are contraindicated in children for fear of atropine toxicity. Only ointment can be used.
  Also atropine, both drops and oint., is not used in elderly for fear of inducing glaucoma that might occur with dilation of the pupil in elderly with narrow angle. A short acting cycloplegic is safer to use.
- cyclopentolate eye drops: duration of action is only 24 hours
- In resistant cases: subconjunctival injection of mydriacane can be given:
  (the site of subconjunctival injection is touched with a cotton swap soaked in novosine for 15 seconds (local anesthesia then the injection is given. A mixture of atropine and procaine adrenaline is given)

* The cycloplegics have the following values:
- Dilate the pupil and so prevent the formation of posterior synechiae
- abolish pain by preventing the spasm of sphincter pupillae and ciliary muscles which occurs secondary to iritis accompanying the corneal ulcer.
Chapter (5) The cornea

Bandage:
- relieves the pain (exposed nerve endings) and so the reflex photophobia and blepharospasm.
- It helps epithelial healing by protecting it against trauma of the lid that occurs during blinking.
NB. Bandage can not be applied if discharge is present. We need to get rid of the discharge full of infection.

In certain cases, additional measurements can be taken:
- Vitamin “A”: important for healing of epithelium can be given topically (ointment) or systemically if there is systemic deficiency
- Vitamin C”: important for healing of stroma can be given systemically
- Systemic antibiotics: in severely debilitated persons
- Subconjunctival injection of antibiotics in severe cases of ulceration to ensure high concentration of the drug.

The success of therapy is indicated by relief of symptoms, regaining of luster and negative fluorescein stain. This takes 7-10 days.
Failed therapy could be due to
Misdiagnosis
Bad choice of antibiotic
Bad compliance of patient
High resistance of causative agent

Treatment of resistant corneal ulcer:
Most ulcers heal with the previous treatment within 7-10 days. If healing does not occur, the condition is termed resistant corneal ulcer.
1. reculture: the antibiotic must be stopped for 2 days before bacterial culture.
Fungal infection must be considered and fungal culture is very helpful.
2. examination of the **lacrimal sac** (source of infection)

3. **subconjunctival injection of antibiotics**

4. **chemical cautery**: application of carbolic acid to floor of ulcer is an old but still helpful line of treatment when there are no facilities.

   **Method:**
   - *local anaesthetic eye drops*
   - *identify the site of the ulcer by fluorescein stain*
   - *dip a wooden match in carbolic acid and touch the ulcer which will become grayish white in colour.*
   - *Wash any excess chemical with saline*
   - *apply antibiotic ointment and bandage*

5. **Conjunctivoplasty**:
   Cut the conjunctiva at the limbus near the ulcer, mobilize it from the sclera, stretch over the ulcer and fix it. The raw surface of conjunctiva will attach to raw surface of the ulcer carrying to it blood vessels. After healing of the ulcer conjunctiva retracts leaving a pseudo pterygium.

6. **Therapeutic keratoplasty**:
   - Keratoplasty is done to eliminate the organism and necrotic corneal tissue. Therapeutic grafts are done for curing the disease while clarity of the graft is usually not achieved since the keratoplasty is carried out in inflamed eye and all defense mechanisms of the body are active.

**Treatment of complications**:

**Nebula**:
- **Contact lenses**: Being faint opacity, it allows passage of light rays into the eye but may interfere with vision by producing irregular astigmatism. Contact lenses, which cancel the cornea, with its irregularity, from the optical point of view (see errors of refraction)
- **Phototherapeutic keratectomy** (PTK) using eximer laser the superficial layers of cornea are removed.

**Leucoma : "white mass"**
Its effect on vision depends on its size and location.
- Central leucoma is in front of the pupil. Being dense opacity, will not allow light entry. keratoplasty is the line of tt.
- Peripheral leucoma has no effect on vision but could be cosmetically unacceptable. In such cases, Coloured contact lens with transparent center
- Coloured contact lens with black center :If opacity is extensive and the eye is blind for some other causes, coloured contact lens with black center can be applied for cosmetic reason

**Descematocele :**
Adding to the usual treatment of the ulcer, one has to
- lower the IOP and eliminate causes of sudden increase of IOP e.g. cough
- Application of cyanoacrylate (glue) at site of descematocele then a contact lens over the glue.

**Perforated ulcer :**
We add to usual treatment
- therapeutic contact lens if small perforation with no loss of tissues
- cyanoacrylate glue ( adhesive substance ) if small perforation with no loss of tissues
- keratoplasty

**Corneal fistula :**
- Destruction of the epithelium with heat irradiation (chauffage)
- Penetrating keratoplasty

**Total anterior staphyloma :**
Enucleation is considered, being a blind painful eye.

**Endophthalmitis :**
early stages antibiotics and steroids are given in heavy doses.
In established cases, evisceration is considered to relieve the pain.

---

**A. Infective corneal ulcers :**
**Bacterial Keratitis:**
Epithelium is a reliable barrier against the enterance of microorganisms into the cornea. Once the epithelium is unhealthy or absent, organisms can get into the cornea. Few bacteria can invade intact epithelium (see before).

Pathology of bacterial corneal ulcer: (same as discussed before)

Clinical picture: see symptoms and signs discussed before

Complications: see before

Management: of ulcer, of resistant ulcer and of complications (see before)

NB. When corneal ulcer is associated with hypopyon, the condition is called hypopyon ulcer.

**Hypopyon ulcer** (acute serpiginous ulcer)

Hypopyon means pus in the Anterior chamber. Any corneal ulcer accompanied by the presence of hypopyon in the AC could be called hypopyon ulcer.

The causative agent could be bacterial, fungal, ....etc

**Etiology:**

- pneumococci produce hypopyon ulcer with characteristic figures
- other organisms like Morax-Axenfeld diplobacilli, strept., staph., pseudomonus pyocyaneus. fungi ....... lack these characteristic figures

When there is characteristic picture of the ulcer, the condition is called typical hypopyon ulcer. And when these characteristic signs are not present the condition is known as ulcer with hypopyon.

**Typical hypopyon ulcer (pneumococci):**

A disc shaped opacity appears near the center of the cornea, grayish white in colour. It is due to infiltration of the cornea with polymorphes. As the epithelium and superficial stroma fall, an ulcer is formed. The ulcer tends to spread towards the center, having an advanced edge and a healing edge. Also it tends to infiltrate deeply.

**The advancing edge is**

located centrally, undermined, preceded by heavy infiltration

**The healing edge is**

located towards the periphery, covered by epithelium, slopping surrounded by less infiltration, sometimes is vascularised

**Posterior abscess:** commonly found. It appears opposite the ulcer in the deep layers of the cornea, just anterior to Descemet’s membrane. It is nothing but cellular infiltration due to the effect of toxins. It might ulcerate posteriorly as the necrotic area falls into the anterior chamber.

Perforation is a common complication of hypopyon ulcer and Descematocele is very rare. (Early destruction of descemet’s membrane)
Hypopyon:
Here it is a sterile hypopyon i.e. does not contain organisms (if cultured, no growth). It is formed as a result of outpouring of polymorphes and exudates from the inflamed iris and ciliary body. This inflammation is due to the effect of toxins on iris and CB.

S.S.:
like any ulcer + the characteristics points mentioned above.

Complications:
like any ulcer.
N.B. Perforation is very common and Descematocele is very rare.
- ulcer tends to go deep
- posterior abscess

Treatment:
usual ttt + if needed:
- subconjunctival injection of penicillin
- examination of the sac
- chemical cautery: carbolic acid

Viral keratitis:
- Herpes simplex virus
- Herpes zoster virus

**Herpes simplex keratitis**
Herpes simplex virus affects more than 95% of people before the age of 6 years, usually in the form of upper respiratory tract infection. Man is the main reservoir of the virus. It remains dormant in the body. When the resistance of the body diminishes, it becomes active producing vesicles on the mouth or genitals. This is commonly seen after stress, common cold, influenza, fatigue, menses.

There are two types of virus:
- Type 1: face, lips and eyes
- Type 2: genital

Eye manifestation of herpes simplex virus
1. *Congenital and neonatal herpetic infection*:
   Transplacental antibodies protect the baby during the first six months of life against the virus. After that infection may occur which rarely appears in the eye
2. *primary herpetic eye affection*:
   usually in the form of acute follicular conjunctivitis with blepharitis (focal) or skin eruption (see the conjunctiva)
3. *Recurrent herpetic eye affection*:
   Could be in the form of
   - Dendritic ulcer, geographic ulcer, neurotrophic ulcer
   - Disciform keratitis

**A. Dendritic ulcer:**
**Etiology:** dendritic ulcer is due to infection with Herpes simplex virus.

<table>
<thead>
<tr>
<th>Dendritic ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>HS —- dendritic ulcer</td>
</tr>
<tr>
<td>Disciform keratitis</td>
</tr>
<tr>
<td>— epitheliotropic</td>
</tr>
<tr>
<td>— neurotropic</td>
</tr>
<tr>
<td>— recurrence</td>
</tr>
<tr>
<td>— hypothesia</td>
</tr>
</tbody>
</table>

S.S.:
as usual + specific characteristics of DU:
- virus invades the cornea, remains in the epithelium (epitheliotropic) forming vesicles. It follows the nerves. When vesicles rupture minute ulcers appear. Branches originate from these ulcers, they re-branch and finally end in knobs (vesicles)
- as the virus is epitheliotropic, it is very uncommon for the ulcer to perforate
- Tendency to recurrence (virus live in nerve ganglion)
- Hypothesia (healing is followed by decreased sensation)

B. Geographic ulcer:
The use of topical steroid enhances viral replication leading to enlargement of the ulcer from the usual denderitic figure into the widened geographic figure

Treatment of denderitic ulcer:
1. usual treatment + antiviral drugs:
   - Iodo deoxy uridine (IDU) drops: It is an analog of thymidine (a nucleoprotein that enter in the formation of the DNA. Virus is formed of DNA. Thus the drug is incorporated into DNA, it inhibits the growth of the virus. Drops every hour by day and two hourly by night
   - Trifluorothimidine (3FT): drops every hour by day and two hourly by night.
   - Adinine arabinoside Ara A 3% ointment: given 5 times per day
   - Acyclovair eye oint (zovirax) is effective and has least toxicity to the unaffected cells. It has high penetration power into the infected cells only. It is inactive and the products of the virus turn it into active form therefore it has minimal toxic effect. Given as ointment 5 times/ day.
### Chapter (5) The cornea

<table>
<thead>
<tr>
<th>IDU</th>
<th>3FT</th>
<th>Ara A</th>
<th>Acyclovir</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Commercial name</strong></td>
<td><strong>Herplex</strong></td>
<td><strong>Vidrabine oint</strong></td>
<td><strong>Zovirax ointment</strong></td>
</tr>
<tr>
<td><strong>Dose</strong></td>
<td><strong>Drops hourly by</strong></td>
<td><strong>2 hourly by</strong></td>
<td></td>
</tr>
<tr>
<td><strong>day and 2 hourly</strong></td>
<td><strong>by night</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Toxicity</strong></td>
<td><strong>+++</strong></td>
<td><strong>++</strong></td>
<td><strong>+</strong></td>
</tr>
<tr>
<td><strong>Analog of</strong></td>
<td><strong>Thymidine</strong></td>
<td><strong>Pyrimidine</strong></td>
<td><strong>Purine</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Guanosine</strong></td>
</tr>
</tbody>
</table>

2. **Therapeutic keratoplasty**: can be done in resistant cases.

C. **Neurotrophic ulcer**: is due to loss of corneal sensation. See later

D. **Disciform keratitis**:

![Disciform keratitis diagram](image)

It is an allergic reaction to Herpes simplex virus. Virus is in the epithelium but liberates toxins that diffuse deeply into the stroma or the endothelium changing their antigenicity. Body will form antibodies against his own tissues. This antigen-antibody reaction will appear as a disc shaped opacity in the center of the cornea. It looks like opacity but slit lamp examination shows that the cornea is increased in thickness (edematous).

**TTT.** : Topical steroids. This can be accompanied by antiviral therapy to avoid activation of epithelial viral disease.

**Herpes Zoster Keratitis**
It is caused by a virus related to that of chicken pox. Frequently epidemics of the two diseases occur together. The virus is neurotopic and infects the dorsal root ganglia or the ganglia of the cranial nerves. Vesicles affect the skin in the area of distribution of the involved nerve. If the virus affects the gasserian ganglion (the 5th cranial nerve ganglion) the eye and adnexa will be involved. When the nasociliary branch of the trigiminal is affected, the skin of the tip of the nose will show eruption which denotes involvement of intraocular structures as well (e.g. Cornea and Iris and CB)

**Symptoms:**
- Fever and malaise
- Pain and redness precede the appearance of the vesicles.

**Signs:** depending on the branches involved; different clinical pictures may be encountered.
- skin eruption in the form of redness, vesicles, pustules and later crust formation in the distribution of one or more of 5th cranial nerve branches.
- conjunctivitis
- cornea: dendritic lesions - Keratitis profanda - corneal hypotonia after healing
- iridocyclitis

**Treatment:**
- oral acyclovair (800 mg 1 X 5) when given in the early days is effective.
- topical acyclovair eye oint and skin oint are applied to the area

---

**Fungal keratitis:**

Fungi are opportunistic infectious organisms. They rarely affect healthy cornea. Cornea should be weakened for fungi to invade and cause keratitis.
Examples of factors that make the cornea susceptible to fungal keratitis include:

**Predisposing factors**:

1. **Trauma**: Trauma is the most frequent risk factor. Trauma most often occurs outdoors and involves plant matter.

2. **Contact lens wear**: Fungi can grow within the matrix of soft contact lenses. Bad hygiene of contact lenses may lead to corneal infections. e.g.
   - irregular use of cleaning contact lenses solutions
   - over-wear of contact lenses induces hypoxia in the cornea

3. **The use of topical corticosteroids** has been associated with the development and worsening of fungal keratitis. They appear to activate and increase the virulence of fungi. The non-pathogenic fungi are turned into pathogenic after removal of commensal bacteria from the conjunctival sac. Systemic use of corticosteroids may predispose the patient to fungal keratitis by rendering the patient immunosuppressed.

**Clinical picture**:

- there is a positive history of some predisposing factors.
- Symptoms may not present acutely
- On examination:
  - grayish white stromal infiltration.
  - Infiltrate has indistinct margins (feathery edge)
  - Discrete stromal infiltrates separated by clear cornea from the main infiltration could be found (satellites). This is pathognomonic for fungal infection.
  - Sometimes, hypopyon is formed in the AC. It has the characteristic of being feathery and could be raised at certain areas.

**Diagnosis**:

Scarring or even corneal biopsy must be done. Material could be examined pathologically and cultured on suitable media to isolate the fungi.

**Treatment**:
- Antifungal eye drops

---

**Acanthamoeba keratitis (parasitic keratitis)**

Acanthamoebae are free living protozoa, found in soil, fresh or salty water. They exist in both active and cystic forms. The cystic form is highly resistant and able
to survive under hostile environmental conditions including chlorinated swimming pools and hot tubes. Under favorable conditions the cysts turn into trophozoites which produce a variety of enzymes that aid in tissue penetration and destruction.

Acanthamoeba keratitis can occur if there is a minor corneal abrasion "contact lens wearers" specially those who use distilled water for their lens care, or use home made saline.

**Diagnosis:**
Must be suspected in chronic resistant keratitis specially if there is a history of contact lens wear. Characteristically pain is so severe, exceeding the severity of corneal infiltration. Diagnosis depends on scraping or corneal biopsy of the ulcer and pathological examination of the specimen to reveal the presence of the parasite.

### b. Non infective corneal ulcers:
- Neurotrophic keratitis (neuroparalytic keratitis) : loss of sensation
- Exposure keratitis (ulcer with lagophthalmos) : dryness
- Keratomalacia : vitamin A deficiency
- Mooren’s ulcer : degenerative condition i.e. of unknown etiology
- Trauma to cornea
  - Mechanical : sharp instrument like a finger nail, edge of a paper "abrasion"
  - Chemical : acid or alkali "see trauma"
  - Physical injury : photophobia (UV light) "see conjunctiva"

### (1) Neuro-paralytic keratitis (neurotrophic keratitis):
Loss of corneal sensation that occurs following:
- radical treatment of trigeminal neuralgia
- fracture base skull
- basal meningitis
will lead to loss of neurotrophic impulses to the cornea (axoplasmic transport: certain substances are transmitted along nerves to cornea) These substances are important for the function and the structural integrity of the epithelium of the cornea. So the epithelium becomes liable for ulceration.

Clinically, an ulcer is formed in the center of the cornea. Characteristically, there is no pain nor referred photophobia, lacrimation nor blepharospasm.

Signs: Desquamation of epithelium, this is encountered in the center but later may spread to affect the whole surface except a small collar near the limbus. The substantia propria become cloudy and breaks into a large ulcer. Ciliary injection, iritis and even hypopyon may develop but no pain.

Treatment:
usual ttt +
- prolonged bandage
- paramedian tarsorrhaphy

(2) Keratitis with lagophthalmos (Exposure keratitis)
Affects the lower third of the cornea. It has a straight upper border. It affects patients with lagophthalmos. During sleep, the eyeball rolls up "this is known as bell’s phenomenon" so the upper part of the cornea is protected under the upper lid while the lower part remains exposed in the palpebral fissure. As there is minimal tear production during sleep, dryness will affect the epithelium of the lower part of the cornea which becomes devitalized and infection and ulceration may occur.

**Treatment:**
- Prophylactic: ointment by night, lateral tarsorrhaphy
- Curative: usual ttt + ttt of lagophthalmos

(3) Keratomalacia
Affects badly nourished children with acute vitamin A deficiency in the first year of life. Condition is usually bilateral.
- Cornea: becomes dull insensitive with loss of luster. Infiltration occurs and increases until finally the whole tissue melts.
- There is minimal inflammatory response (redness, photophobia, and lacrimation)
- General condition is bad

**ttt:**
- Improvement of the general condition, large doses of vitamin A.
- Usual ttt of corneal ulcer
  - Local vitamin A ointment

**Chapter (5) The cornea**

(4) Mooren’s ulcer: (chronic serpigenous ulcer)

A rare type of unknown etiology. Could be degenerative in origin. It affects the old patients. Usually starts at the periphery of the cornea and progresses centrally and circumferentially. Its central edge is undermined and the bed is vascularised.

**TTT:** very difficult
- Usual lines of ttt
- Conjunctivoplasty
- Keratoplasty

(5) Atheromatous and Calcarious ulcers

Long-standing corneal opacities, like any scar tissue, may suffer secondary degenerative changes after many years. This could be in the form of hyaline or fatty degeneration. Secondary calcification may even occur. This newly deposited material will elevate the overlying epithelium, which will be a subject of repeated trauma by the lid margin as blinking occurs. So it will ulcerate. Ulcer formed on top of fatty degeneration is called atheromatous ulcer while that on top of calcified area is called calcarious ulcer. These ulcers are very resistant to treatment as they occur on top of scarred area so have a badly nourished bed.

**Treatment:** usual lines of treatment
In cases of blind eyes that develop such ulcers enucleation may be encountered to relieve the pain.

c. Allergic keratitis:

Keratitis due to hypersensitivity reactions
- Marginal ulcers
- Phylectenular keratoconjunctivitis
- Spring catarrh keratitis

Autoimmune diseases: rheumatoid artheritis, polyarteritis nodosa

**Marginal ulcers:**
The most common is staphylococcal marginal ulcers. These are small, single or multiple, rounded or oval infiltrate of the periphery of the cornea with clear interval between the infiltrate and the limbus. Later the overlying epithelium ulcerates. It is hypersensitivity reaction of the cornea to staphylococcal toxins. Patient usually suffers from staphylococcal blepharitis or conjunctivitis. Sometimes the condition may appear in association with conjunctivitis caused by Koch-Weeks bacillus or Proteus. Treatment is topical steroids + attacking the original source of organisms.

- **Phylectenular ulcers:**
  * Limbal ulcer (one or multiple that could be a complete ring)
  * Fascicular ulcer

---

**Pannus:** is vascularization and cellular infiltration of the superficial layers of the cornea.

<table>
<thead>
<tr>
<th></th>
<th>Trachomatous pannus</th>
<th>Phylectenular pannus</th>
</tr>
</thead>
<tbody>
<tr>
<td>site</td>
<td>upper part of cornea</td>
<td>any site of cornea</td>
</tr>
<tr>
<td>level</td>
<td>between epith. and B.M.</td>
<td>deep to B.M.</td>
</tr>
<tr>
<td>vessels</td>
<td>divide dichotomously</td>
<td>are straight</td>
</tr>
<tr>
<td>infiltration</td>
<td>ends in a straight line</td>
<td>irregular line</td>
</tr>
</tbody>
</table>

**Degenerative pannus:**
Occurs in degenerated eyes following iridocyclitis, absolute glaucoma. Conditions that leads to persistent corneal edema for a long time, thus the compactness of the layers of the cornea is lost and blood vessels form the limbus can invade the cornea. Thus there is a fibrovascular tissue invading all around the corneal periphery, usually superficially located with destruction of the Bowman’s membrane.
Degenerative conditions of the cornea

Arcus senilis:
It is an annular infiltration of the periphery of the corneal stroma with lipoid material. The infiltration starts up and down but soon surrounds the whole cornea. Infiltration is one mm in width and is separated from the limbus by a clear zone. (Lucid interval of Vogt)

Symptoms: just cosmetic appearance

Arcus juveniles:
same as arcus senilis but affects young persons
Its only significance is that it indicates a high cholesterol level in blood so patient needs medical care.

Keratoconus:
Is a congenital bilateral weakness of the central part of the cornea. Condition is usually apparent at puberty

Symptoms:
* Gradual diminution of vision due to:
  - Simple myopia (early stages)
  - Myopic astigmatism (moderate cases)
  - Opacification of tip of the cone (marked advanced cases)
Signs:

* **Early stages:**
  - Abnormal movement of the red reflex when refraction using the *retinoscope*.
  - Distorted mires of the *keratometer*
  - **Corneal topography**: this is a modern technique of corneal photography. The power (curvature) of different areas of the cornea is measured and result is displayed in a colour coded map. The hot colours (red) indicate high power areas (steep: more curved) and the cold colours (blue) indicate lower power areas (flat: less curved areas)

* **Advanced stages:**
  - The center of the cornea is thin and bulges forwards in the form of a cone. The apex of the cone is below and nasal to the center of the cornea.
  - **Fleischer ring**: a brown ring is sometimes seen at the base of the cone. This is due to haemosidrin deposited in the epithelial cells.
  - The apex of the cornea turns **opaque** is advanced stages

- **Manson's sign**: v shaped lower lid appears when patient looks down.
- **Acute hydrops**: Edema of the conical area of the cornea occurs suddenly so patient suffers from blurrng of vision of sudden onset. This edema occurs when
the endothelium on the back surface of the cornea fails to maintain the cornea dehydrated. If the endothelial cells manage to cover the whole back surface once again and regain dehydration of the cornea, edema will subside and vision will return to its pre-edematous state. If not, keratoplasty will be the only solution to regain useful vision.

**Treatment:**
1. **Glasses** are useful in early stages (only simple myopia or myopia with mild regular astigmatism)
2. **Contact lens:** correct the irregular astigmatism, so are helpful when glasses are of no value
3. **Keratoplasty:**
   - When no contact lens can be fitted or when CL is not tolerated by the patient
   - When there is opacification of the tip of the cone as in advanced cases of the disease or in some cases of persistent edema following acute hydrops.

**Ectatic conditions of the cornea:**
1. Keratoconus
2. Keratectasia
3. Anterior staphyloma

**Keratectasia:**

is protrusion of the cornea (without iris) that may occur as a result of weakness of the cornea following the healing of a corneal ulcer or interstitial keratitis. **TTT:** usually keratoplasty is needed to remove the opacity and restore vision.

**Anterior staphyloma:**

Is an ectatic scar of the cornea in which the iris is incarcerated. It may be:
- Partial: if it involves only a portion of the cornea
- Total: if it involves the whole cornea

It results from a large corneal perforation (ulcer or trauma) with prolapsed iris. The prolapsed iris is first covered with fibrin, which soon is transformed into connective tissue incarcerating the iris. This fibrous tissue is weak and bulges irregularly in front of the I.O.P. forming a bossy surface resembling a punch of grapes (staphyloma = puch of grapes) Glaucoma may occur in partial anterior staphyloma but is always present in total staphyloma. It results from obliteration of the angle of the anterior chamber.

**TTT.**
Partial anterior staphyloma: keratoplasty
Total anterior staphyloma:
Enucleation (blind painful eye) and application of an artificial eye for cosmetic reason.
If patient refuses enucleation, retrobulbar injection of alcohol relieves the pain by destruction of the nerves behind the eye (denaturation of proteins)

### Ciliary and conjunctival injection

<table>
<thead>
<tr>
<th>Conjunctival injection</th>
<th>Ciliary Injection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fire red in colour</td>
<td>Pink in colour</td>
</tr>
<tr>
<td>Maximum in fornix and fades as we go towards the cornea</td>
<td>Maximum around the limbus (0.5 mm zone around the limbus)</td>
</tr>
<tr>
<td>Individual vessels seen</td>
<td>No vessels seen (are deep)</td>
</tr>
<tr>
<td>Vessels move with movement of conj.</td>
<td>Do not move</td>
</tr>
<tr>
<td>Due to dilatation of the conj Bvs</td>
<td>Due to dilatation of branches of the anterior ciliary aa.</td>
</tr>
<tr>
<td>Occurs in: - conjunctivitis</td>
<td>Occurs in: - corneal ulcers - acute iridocyclitis - acute congestive glaucoma</td>
</tr>
</tbody>
</table>

### Superficial vascularization

<table>
<thead>
<tr>
<th>Superficial Vascularization</th>
<th>Deep Vascularization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vessels derived from corneal vessels</td>
<td>From anterior ciliary vessels</td>
</tr>
<tr>
<td>Are seen across the limbus</td>
<td>End abruptly at the limbus</td>
</tr>
<tr>
<td>Branch dichotomously</td>
<td>Run parallel</td>
</tr>
<tr>
<td>SL: vessels run in the superficial layers of the cornea</td>
<td>Vessels run in the posterior 2/3</td>
</tr>
<tr>
<td>Etiology: corneal ulcer, pannus, pterygium, ariboflavinosis</td>
<td>Etiology: - interstitial keratitis - deep corneal ulcers</td>
</tr>
</tbody>
</table>
Keratoplasty (corneal transplantation):
It could be:

* **Lamellar keratoplasty:** the superficial layers of the cornea are transplanted
* **Penetrating keratoplasty:** whole thickness of the cornea is transplanted

**Technique:**
- Using a trephine of a suitable diameter (7-8.5 mm) the diseased part of the cornea is removed.
- A similar disc is cut from a cornea obtained from the eye bank (obtained from a recently dead person, preserved in special fluid for 4 days period, donor blood tested to be certain he is free of infectious diseases)
- The clear healthy disc is now sutured in place

**Indications:**
- **Optical:** to improve vision e.g. Central opacity. Keratoconus
- **Therapeutic:** resistant corneal ulcer, Descematocele, corneal fistula
- **Structural (tectonic):** to restore the integrity of the cornea as cases of recurrent pterygium surgery when a new lamellar keratectomy will leave a very thin bed, in such cases lamellar corneo-scleral graft is applied to restore a cornea of a normal thickness.