3. Corneal Disorders

Q Mention the layers of the cornea

Answer

• **Anatomically:** The cornea consists of the following ***5 layers:***

1. **Epithelium**
2. **Bowman's layer**
3. **Stroma**
4. **Descemet's membrane**
5. **Endothelium**

Q What is the nerve supply of the cornea?

Answer

Nerve Supply of the cornea:

- Is from the 2 long ciliary nerves (of the nasociliary branch of ophthalmic division of trigeminal nerve).

Q What are the types of sensations receptors present

Answer

- The cornea is only sensitive to pain, cold and touch.

Q Why the cornea is transparent?

Answer

• ***The factors responsible for corneal transparency are:***

a) *Anatomical factors*:

1) Non-Keratinized epithelium.
2) The stromal lamellae are regular and parallel.
3) Non-myelinated nerve fibres.
4) Absence of blood vessels and lymphatics.

b) *Physiological Factors*:

1) Dehydrated state of the cornea (endothelial pump)
2) Constant refractive index.

Q Causes of impaired corneal sensation

Answer

1. Herpetic keratitis especially herpes zoster
2. Acute congestive glaucoma
3. Cvernous sinus thrombosis
4. Partial affection of ophthalmic division e.g. superior orbital fissure syndrome

Q Causes of loss of corneal sensation

Answer

1. Neuroparalytic keratitis
2. absolute glaucoma
3. Complete trigeminal nerve damage or the ophthalmic division by trauma or a lesion in any part of its course, or after treatment of trigeminal neuralgia

Q How can you test the corneal sensitivity?

Answer

We can test the corneal sensitivity by a wisp of cotton twisted to a fine pointed thread, applied to the cornea, from the temporal side without touching the lids. Normally, a powerful rapid reflex blinking occurs.

Q Comment on the types and causes of corneal vascularization

Answer

Vascularization of the cornea: *may be superficial or deep*

Superficial vascularization .	**Deep vascularization.**
1-Vessels derived from conj.Vs 2-Vs.are seen crossing the limbus 3-Bright red, well-defined 4-Branch dichotomously 5-The surface of the cornea is irregular (raised by the B.Vs.) 6-Vessels run in the superficial layers of the stroma 7-Occur in: pannus, corneal ulcers, trichiasis & pterygium.	1-From anterior ciliary Vs 2-End abruptly at the limbus 3-Dark red, ill-defined 4-Run parallel 5-Surface is smooth 6-Run in the posterior 2/3 7-Occur in:interstitial keratitis &deep ulcers

Q What are the types of ulcerative keratitis (corneal ulcers).

Answer

- **Classification of Ulcerative Keratitis**

***A.*—Primary Corneal Ulcers**.—These are due to necrosis occurring primarily in the cornea without conjunctivitis. They are classified into two main categories, namely :—

1. *Infective*.—*(a)* Hypopyon ulcer *(b)* Dendritic ulcer *(c)* Fungal (mycotic) ulcer.

***2. Non-Infective*.**—*(a)* Traumatic ulcer *(b)* Exposure keratitis *(c)* Neuroparalytic keratitis. *(d)* Nutritional ulcer *(e)* Keratomalacia. (f) Atheromatous ulcer (g) Mooren's ulcer.

***B.*—Secondary Corneal Ulcers**, *i.e.* ulcers occurring as a complication of acute or chronic conjunctivitis, *e.g.:*—

1. Catarrhal conjunctivitis. 2. Gonococcal conjunctivitis.
3. Diphtheritic conjunctivitis. 4. Phlyctenular conjunctivitis.

5. Trachomatous conjunctivitis.

Q Give the classification of non-ulcerative keratitis

Answer

Classification of Non-UIcerative Keratitis

***A.*—Superficial Keratitis :**

1. Superficial punctate keratitis.
2. Pannus, *e.g.* trachomatous, phlyctenular and leprotic.

B. —Interstitial (Parenchymatous) Keratitis :

1. Interstitial keratitis. 2. Disciform keratitis.

***C.*—Deep Keratitis (Keratitis profunda).**

Q Mention the causes of corneal ulceration

Answer

► **According to etiology, *corneal ulcers may be classified into:***

1. ***Infective ulcers****: e.g.(a)* Hypopyon ulcer, *(b)* Dendritic ulcer, *(c)* Mycotic ulcer.
2. ***Non-Infective ulcers*** *e.g. (a)* Traumatic ulcer. *(b)* Exposure keratitis. *(c)* Neuroparalytic keratitis. *(d)* Nutritional ulcer, *(e)* Keratomalacia. (f) Atheromatous ulcer. (g) Mooren's ulcer.

Q What are the organisms that can invade the intact corneal epithelium

Answer: gonococci, diphtheria, Listeria species

Q Enumerate causes of central corneal ulcers

Answer

• ***Causes of Central Keratitis***

1. Hypopyon ulcer. 2. Gonococcal ulcer.
3. Herpetic keratitis. 4. Neuroparalytic keratitis.
5. Nutritional ulcer, *(e.g.* atheromatous ulcer and keratomalacia).

Q Enumerate causes of peripheral corneal ulcers

Answer

• ***Causes of Marginal Keratitis***

1. Catarrhal ulcer. 2. Trachomatous ulcer. 3. Phlyctenular ulcer.
4. Marginal infiltrates. 5. Mooren's ulcer.

Q What are symptoms of a simple corneal ulcer

Answer

Symptoms of corneal ulcer:

1- Lacrimation. [From reflex irritation of the nerve endings].

2- **Blepharospasm** [Spasm of orbicularis oculi].

3- **Photophobia.**

4- **Diminution of vision:** due to loss of transparency of the cornea (oedema, cellular infiltration, irregularity of the surface).

5- **Pain**, Pricking sensation**.**

Q What are signs of a simple corneal ulcer

Answer

Signs of a simple corneal ulcer:

1 Oedema and redness of lids + Blepharospasm.
2- Lacrimation
3- Ciliary injection (marked), slight conjunctival injection,
4- Loss of corneal lustre,
5- The ulcer stains green with fluorescein.
6- Variable degree of iridocyclitis ± hypopyon.

Q How can you diagnose a corneal ulcer?

Answer: Diagnosis of a corneal ulcer is done by:

1. History
2. Clinical picture: pain, photophobia, lacrimation, diminution of vision, blepharospasm
3. Slitlamp examination
4. Fluorescein stain test: → green defect

Q Mention the complications of non-perforated corneal ulcer

Answer

Cmplications of non-perforating corneal ulcers:

a) Early Complications:
b) Late complications

(a) Early Complications:

1) Iridocyclitis ± hypopyon: due to diffusion of toxins through the cornea to the AC.

2) 2ry Glaucoma:
- **Early:** due to plasmoid aqueous or hypopyon.
- **Late:** due to PAS with angle block.

3) Descematocele (Keratocele).

Cause: Bulging of DM due to destruction of the whole thickness of the cornea except DM which can withstand the IOP.

Clinically:
transparent bulging vesicle from the cornea.

Fate:
- Rupture with perforation.
- Healing with cicatrization.

NB: ***Descematocele can't occur in:***
1) Children, due to thin DM.
2) Typical hypopyon ulcer, due to destruction of DM by the posterior abscess.

(b) Late complications:

1. **Corneal opacities**
2. **Defective corneal scarring**
3. **corneal vascularization**

1. Corneal opacities:

a-Nebula: Faint superficial corneal opacity; the iris is partially seen through it.
b-Macula: Corneal opacity of medium density; the iris is seen with difficulty through it.
c-Leucoma non adherent: Dense deep-white opacity; the iris cannot be seen through it.

2. Defective corneal scarring:

1) Corneal facet: depressed area in the cornea. Due to insufficient scarring in which the epithelium is not raised to its original level.
2) Keratectasia: (Ex-ulcero) the scar is weak & bulges in front of IOP.
3) Pseudo-pterygium: adherent fold of the conjunctiva to the base of the ulcer (in peripheral ulcers with severe conjunctivitis).

N.B. ***Corneal scaring leads to visual disturbances by several mechanisms:***

1) Corneal astigmatism: due irregular surface.
2) Squint: if the corneal scar occurs during the 1st 6 months after birth and is unilateral.
3) Nystagmus: if the corneal scar occurs during the 1st 6 months after birth and is bilateral.
4) Xerosis: due to dryness of the cornea.
5) Atheromatous ulcer: due to poor vitality in old dense corneal scars.

3. Corneal vascularization: late and may be:

- Superficial (from conjunctival vessels.)
- Deep (from episcleral vessels.)

Q What are the complications of perforated corneal ulcer

Answer

Complications of perforating corneal ulcers:

• Mechanism of perforation:
Sudden increase in IOP on the weak floor of a deep ulcer (straining, blepharospasm,squeezing the eye, virulent organism).

• Effects of corneal ulcer perforation:

1- Immediate good effects:

- Diminution of pain.
- Antibodies in fresh aqueous.
- Lowering of I.O.P. with better corneal nourishment, extension of the ulcer ceases and cicatrization proceeds rapidly. \

2- Bad effects: see sequelae (complications).

► ***Complications of perforation:*** *depend on the* ***site and size*** *of the perforation*:

1. **Peripheral perforation may be:**

    a. Small perforation
    b. Large perforation

2. **Central perforation:**
    a. Small perforation
    b. Large perforation

1) <u>Peripheral perforation</u>:

a) Small peripheral perforation: → Ant. Synechiae.
b) Large peripheral perforation: → *Iris prolapse* ***which leads to:***
i- Leucoma adherent.
ii- Corneal (anterior) staphyloma (partial or total).

2) <u>Central perforation</u>:
a) Small central perforation: →
i-Leucoma non-adherent
ii-Corneal fistula
iii-Anterior polar cataract
b) Large central perforation:→
i-Intra-ocular infections:
- Suppurative iridocyclitis
- Endophthalmitis
ii-Intra-ocular hemorrhage:
- Vitreous, retinal, or choroidal hemorrhage
- Expulsive hemorrhage
iii-Subluxation, anterior dislocation or extrusion of the lens.

1-<u>Anterior synechia</u>: The iris closes the perforation and becomes adherent to the back of the cornea. This adhesion may be drawn into a fine band when the anterior chamber (AC) reforms

2-<u>Leucoma adherent</u>:
A scar in the cornea in which the iris is incarcerated.It may be total or partial.

3-<u>Anterior staphyloma</u>:
Is an ecstatic scar in the cornea in which the iris is incarcerated. It may be total or partial. Because all or a big part of the angle of the AC is closed, secondary glaucoma is common.

4-<u>Corneal fistula</u>:
- Occurs in small central ulcers as here the iris cannot close the perforation.
- The perforation will be closed by a plug of fibrin which may become dislodged and reformed and so on.
- The corneal epithelium might move to line the perforation thus a fistula lined by epithelium will form.

a- ***Diagnosis of corneal fistula***:
- ***The diagnosis of fistula depends on:***
    1- Soft tension.
    2- Absent A. Chamber.
    3- Fluorescein test and slit lamp.: A drop of Fluorescein is instilled into the eye and gentle pressure is applied to the globe; a green colored fluid (fluorescein). is seen coming out of the fistula when examined by the slit lamp with the cobalt-blue filter.

b- ***Dangers of corneal fistula:***
1-Introduction of infection.
2-secondary glaucoma due to angle closure by PAS(peripheral anterior synechia).

3-Bad effects of prolonged hypotony: Edema of the retina, macula & optic nerve head .
4-Lens opacities.

c- ***Fate of corneal fistula:***

Temporary healing (due to closure by exudate) &reopening(by secondary glaucoma).

5-<u>Anterior polar cataract</u>: Following small central perforation,the lens comes into contact with the perforation and the toxins from the ulcer irritate the subcapsular epithelium which proliferates (fibrous metaplasia) and an anterior polar cataract is formed.

6-<u>SubIuxation of the lens</u>: is due to sudden drop in IOP.

7-Antcrior dislocation of the lens: occurs if the ulcer is very large.The lens may become extruded and the vitreous prolapses.

8-<u>Intra-ocuIar haemorrhage</u>: results from sudden drop in IOP. The he may be localized to the retina or choroid or it may be severe pushing all the intra-ocular contents infront of it(expulsive hge).

9-<u>Intra-ocuIar infection:</u> purulent irido- cyclitis, endophthalmitis, panophthalmitis.

Q What is the general ttt of a corneal ulcer

Answer

► ***The treatment of corneal ulcers include:***

1) **Aetiological** Treatment
2) Treatment of the **ulcer itself.**
3) Treatment of **complications.**

(I) <u>Aetiological Treatment</u>:

a. Removal of the predisposing factors
b. Treatment of the source of infection e.g. conjunctivitis

(II) <u>Treatment of the ulcer itself:</u>

A. <u>*Local*</u> Treatment
1. Non-specific measures
2. Specific treatment
B. <u>*General*</u> Treatment

A. <u>Local Treatment</u>

1. <u>*Non-specific measures*</u>: e.g.
- Frequent eye wash if there is discharge.
- Worm compresses :decrease pain due to the counter-irritant effect and dilate the blood vessels thus toxins are removed and WBCs and antibodies increased.
- Protection of the eye: eye patch (if there is no discharge) or, dark Glasses (if there is discharge).
- Therapeutic (Bandage) soft contact lens for resistant corneal ulcers.

2-<u>*Specific treatment*</u>*: which may be:***

a. Medical treatment
b. Surgical treatement

a. <u>*Medical treatment*</u>

<u>General Guidelines</u>

(1) Identification of the organism: Ideally, a smear is taken to diagnose the causative organism, stained by Gram and Giemsa stain and culture and sensitivity test is carried out but treatment must be started immediately.

(2) Start with a broad-spectrum topical antibiotics as drops hourly during the day and ointment at bedtime, fluoroquinolone (Ciprofloxacin or ofloxacin). Then, modify the dose of eye drops according to the condition of the eye.
(3) In severe (vision-threatening) cases → use ***fortified eye drops***: tobramycin or gentamycin (15 mg/ml) every hour, alternating with fortified cefazolin (50 mg/ml) or vancomycin (25 mg/ml) every hour.
(4) Consider subconjunctival injection of antibiotics (e.g. gentamicin 20 – 40 mg and cefazolin 100 mg), in severe cases with AC reaction and hypopyon or if no improvement with fortified eye drops.

- **Causes of failure of antibiotic treatment:**
  a) Incorrect diagnosis: due to unrecognized infection with HSV, Fungi, acanthameba or atypical mycobacteria.
  b) Incorrect treatment. Due to inappropriate choice of Abs.
  c) Drug toxicity. e.g. aminoglycosides may cause conjunctival and corneal epithelial changes and delay healing.

(5) Mydriatic-Cycloplegic drugs: Atropine or cyclopentolate. To prevent the formation of post. Synechiae & reduce pain from ciliary spasm (they have no effect on the ulcer itself, they just treat the accompanying iritis).
(6) Antiviral or antifungal treatment for viral (e.g. herpetic keratitis), or fungal ulcers respectively.

b. Surgical Treatment of Corneal ulcer

1- Cauterization
2- Paracentesis.
3- Conjuncttival flap
4- Tarsorrhaphy.
5- Theapentic keratopasty

1. Cauterization

➢ **Indications:-**

- Indicated if the ulcer progresses despite the previons therapeutic measures.

➢ **Principle:-**
it includes removal of the necrotic maternal by a spatula or a small curette followed by cauterization of the ulcer (floor and edges) by cryocautery or **chemical** agents **(e.g. *Pure carbolic acid)*:**:

2. Paracentessis

Indications:-
Progressive ulcer despite the previous measures, with:-
- Impending perforation
- Large thick hypopyon with 2ry glaucoma.
- In Descematocele

Value:-
1- To slowly evacuate the anterior chamber ➢ lowering IOP to avoid the complications of sudden perforation.
2- To bring fresh aquons with antibodies.
3- better nourishment for the cornea and dilate the paralimbal lymphatics

3. Conjunctival Flap
-To assist healing of the ulcer.

4. Tarsorrhaphy

Indications:-

Indicated in neuroparalytic keratitis, exposure keratopathy with proptosis or facial palsy.

5. Therapeutic kerato plasty

(III) Treatment of complications:

1-Iritis:
It is secondary to the ulcer and is treated by increasing the frequency of atropine.
2-Secondary glaucoma (acute inflammatory 2ry glaucoma): continue the use of antibiotics & atropine, give acetazolamide. Paracentesis may be considered (never use miotics)
3-Perforation:
This depends on the site and size of perforation:

a) ***If the ulcer is small*** or in the pupillary area,no fear from prolapse of the iris give the usual treatment + bilateral bandage+ rest in bed+ avoid straining. Closure of small perforation without iris prolapse with cyanoacrylate glue to save the eye may be done.

b) ***If prolapse of the iris occurs***: Give the same treatment and do not reposit it (carries infection in),and do not excise it as it assists healing and its excision may lead to fistula.
4-DescematoceIe:
Give the usual treatment + rest in bed + bilateral bandage + acetazolamide to lower the IOP, if there is danger of perforation; do paracentesis.
5-Corneal fistula:
Is treated as a perforated ulcer in the early stage. If the fistula is established, healing will be prevented by the epithelium which lines the track and this epithelium must be destroyed by a fine diathermy needle (a trace of the AC must be present to protect the lens) followed by a soft contact lens.
-Conjunctival flap, to cover the fistula.
-Penetrating keratoplasty in resistant cases
6-Anterior synechiae: need no treatment.
7-Large leucoma adherent:
is liable to produce 2ry glaucoma, it is treated by penetrating keratoplasty. If glaucoma occurs,glaucoma surgery is done followed by penetrating keratoplasty.
8-Partial ant.staphyloma:
Glaucoma surgery followed by penetrating keratoplasty.
9-Total ant.staphyloma: is treated by enucleation.
10-Ant.polar cataract:
if small; no treatment. If large; do cataract surgery.
11-ExpuIsive hge: Is treated by enucleation or evisceration.
12-Panophthalmitis: By evisceration.

B. General Treatment: e.g.

1. Rest
2. Sedatives for pain
3. Systemic antibiotics for severe infections
4. Laxatives to avoid straining especially in advanced ulcers with danger of perforation.

Q How can you diagnose a corneal fistula?

Answer

- ***The diagnosis of fistula depends on:***
  1- Soft tension.
  2- Absent A. Chamber.
  3- Fluorescein test and slit lamp.: A drop of Fluorescein is instilled into the eye and gentle pressure is applied to the globe; a green colored fluid (fluorescein). is seen coming out of the fistula when examined by the slit lamp with the cobalt-blue filter.

Q what are the complications of corneal fistula

Answer

Complications of corneal fistula:

1-Introduction of infection.
2-secondary glaucoma due to angle closure by PAS (peripheral anterior synechiae).
3-Bad effects of prolonged hypotony: Edema of the macula & optic nerve head .
4- Lens opacities.

Q Mention the difference between the following conditions: corneal nebula, macula, leucoma, Keratectasia, Keratocele, and anterior staphyloma

Answer

a-Nebula: Faint superficial corneal opacity; the iris is partially seen through it.
b-Macula: Corneal opacity of medium density; the iris is seen with difficulty through it.
c-Leucoma non adherent: Dense deep-white opacity; the iris cannot be seen through it.
d- Leucoma adherent: A scar in the cornea in which the iris is incarcerated.It may be total or partial.
e. Keratectasia: the corneal scar is weak & bulges in front of IOP
f- Keratocele (= Descematocele) → see next Q
f- anterior staphyloma: Is an ecstatic scar in the cornea in which the iris is incarcerated. It may be total or partial. Because all or a big part of the angle of the AC is closed, secondary glaucoma is common.

Q What is the descematocele. Mention its ttt

Answer

Cause: Bulging of DM due to destruction of the whole thickness of the cornea except DM which can withstand the IOP.

Clinically:
transparent bulging vesicle from the cornea.

Fate:
- Rupture with perforation.
- Healing with cicatrization.

NB:
Descematocele can't occur in:
1) Children, due to thin DM.
2) Typical hypopyon ulcer, due to destruction of DM by the posterior abscess

Q Write short notes on the causes, pathogenesis, signs, and treatment of corneal edema?

Answer

<u>Causes:</u>-

I- Elevated IOP :-
1) Angle closure glaucoma
2) Congenital glaucoma.

II- Trauma: -
1) Birth Treume
2) Non surgical Contusion
3) F.B
4) Surgical Traunx

III- Dystrophy:

IV- Endothedial dysfuction 2ry to inflammation.

VII- Epithelial damage

Pathogenesis:-
➢ Corneal deturgcscence (Dehydeation) is achieved with pump function of the corneal endo thelium balances the fluid accumulating effect of intraoular hydrostatic pressure and corneal swlling pressure
➢ Disturbance of this balance or disruption of the limiting membranes of the cornea [epith and endothelium] ➔ corneal edema result in significant visual impairment and painfnt surface break down.
Signs:-

I- Epithelial edema:-
1- Loss of corneal lustre
2- Vesicles and bullae
II- Stromal edema:-
1- Appearance of optical empty spaces.
2- increased corneal thickness
3- Decreased corneal sensations.

• **Treatment: mainly treatment of the cause (+):**

i. Lower IOP
ii. Control of inflammation
iii. Hypertonic agents

Q Describe ulcer serpens (typical hypopyon ulcer): aetiology, clinical picture, DD, and ttt

Answer:

Hypopyon Ulcer
(Ulcus Serpens = Acute Serpiginous ulcer)

• **Definition:** A severe form of corneal ulcer which is usually assosiated with hypopyon. It creeps over the cornea because it has a healing edge and an advanceing edge and has a great tendency to perforate.
• **Aetiology:**

1) Predisposing factors:
(organism + abrasion + poor general resistance).
a)Chronic dacryocystitis provides the pneumococci (50% or more of typical hypopyon ulcer is associated with ch.dacryocystitis).
b)An abrasion is necessary because the organism cannot invade the normal epithelium.The abrasion is produced by a F.B.,a finger nail, a lash...etc.
c)Poor general resistance: It is common in old debilitated people and following fevers.

2) Causative organisms:
a) Pneumococci: cause 80% of cases **(Typical hypopyon ulcer)**
b) Atypical hypopyon ulcer (20 %):
- **Bacteria:** Morax-Axenfeld diplobacilli, other pyogenic organisms.
- **Fungi:** aspergillus fumigatus.
- **Viruses and protozoa** (rare).

• **Clinical picture:**
a) Symptoms: as in corneal ulcer but severer.
b) Signs: as in corneal ulcer but:

1) The ulcer is disc shaped, usually near the center of the cornea, serpiginous (creeps over the cornea) ***and has an advancing and a healing edge:***
- **-Central advancing edge:** crescentic, undermined and densly infilterated.
- **peripheral healing edge:** flat, epithelialized, vascularized and cicatrizing.

2) Deep ulceration, posterior abscess formation and perforation are common.
Perforation occurs commonly because:
a-The ulcer tends to go deep.
b-A posterior abscess commonly forms. This occurs opposite the ulcer just anterior to DM in the form of cellular infiltration which might ulcerate posteriorly (posterior ulcer).This will weaken the cornea and make perforation very common.Also for the same reason descematocele is very rare.

3) Hypopyon: It is a sterile pus which is yellow in color and tends to settle at the bottom of the AC. It has an upper straight level. It originates from the inflamed iris and is composed of polymorphs-f fibrin-Hris pigment (no organism).

N.B: The pneumococcal type is called the ***typical hypopyon ulcer.*** Hypopyon ulcers caused by other organisms are called ***atypical hypopyon ulcers***.

Chacters	Typical hypopyon ulcer	Atypical hypopyon ulcer
-Cause -Site -Character -Descematocele -Perforation	-Caused by pneumococci . -Starts near the center. -Serpiginous . -Never occur. -Perforation is common.	-Caused by other organisms. -Starts anywhere. -Spreads in all directions . -May occur. -Perforation less common.

Q Why the ulcer is serpiginous?

The ulcer is disc shaped, usually near the center of the cornea, serpiginous (creeps over the cornea) ***and has an advancing and a healing edge:***
- **-Central advancing edge:** crescentic, undermined and densly infilterated.
- **peripheral healing edge:** flat, epithelialized, vascularized and cicatrizing.

Q Give an account on aetiology, clinical picture, and ttt of herpes simplex keratitis

Answer

(1) Herpes Simplex Keratitis

• **Introduction**
■ Herpes simplex virus (HSV) is a DNA virus with humans as the only host.

■ HSV is divided into 2 types:
1. ***HSV-1***: causes infection above the waist (face, lips, and eyes)
2. ***HSV-2***: causes infection below the waist (genital herpes)

• **Clinical features**
• HSV infection can be classified into ***3 stages***:

1. Primary ocular infection

■ Blepharoconjunctivitis
■ Keratitis

2. Recurrent keratitis

■ Epithelial
■ Stromal

3. Trophic Keratitis (metaherpetic keratitis)

(1) *Primary ocular infection*

• **Cause:** direct transmission of virus through infected secretions to a non-immune subject.
• **Clinical features**

■ ***Presentation:*** typically occurs in children between the age of 6 months and 5 years, with unilateral ocular irritation and redness, and may be associated with generalized symptoms of viral illness. (it is rare during the first 6 months of life because of the protection given by the maternal antibodies).
■ ***Signs:***

1. Mild, self-limited ***blepharoconjunctivitis***.
2. The ***skin lesions*** (vesicles, then crusts, then heal without scarring) involve the lids and periorbital area (Fig. 8).
3. **Keratitis** (fine epithelial punctate keratitis) develops in 50% of cases.

• **Treatment**

Treatment with topical antiviral ointment (e.g. Zovirax ointment) is applied to the eye 5 times/day for 21 days.

(2) *Recurrent Keratitis*

(A) Recurrent epithelial keratitis

• **Cause**

Reactivation of latent virus and invasion of the epithelium.

• **Clinical features**

■ ***Presentation***

With an acute onset of unilateral irritation, redness, photophobia, and blurring of vision.

■ ***Signs***

1. Dendritic ulcer

- Starts with coarse stellate PEK and develops into a branching (dendritic) ulcer
- Fluorescein stains bed of the ulcer and rose Bengal stains its margins
- Corneal sensation is reduced

2. Geographical ulcer starts as a dendritic ulcer and enlarges to assume a geographical shape.

- **Treatment:**

(1) With antiviral drugs

1. **Acycloguanosine** 3% ointment **(Acyclovir, Zovirax),** 5 times/day.

▪ Efficacy ++++
▪ Toxicity +

2. **Trifluorothymidine** 1% drops, every 2 hours.

▪ Efficacy ++++
▪ Toxicity +++

3. **Adenine arabinoside** 3% ointment, 0.1% drops (rarely used nowadays).

▪ Efficacy +++
▪ Toxicity ++

4. **Idoxuridine** 0.5% ointment, 0.1% drops(rarely used nowadays)

▪ Efficacy +++
▪ Toxicity +++

The antiviral drugs used are shown in this table:

No	Drug	Dose	Action	drawbacks
1	Acyclovir (zovirax) as ointment or tablets 3%	5 times/day for 14 dayes & should be tapered gradually.	Acts no virus infected cells only. it acts by inhibiting DNA synthesis (by mutation)	Expensive
2	Trifluorothymidine (Bephen)	5 times/day for 14 days	Act on healthy and infected celles	Has teratogenic effects
3	Ara-A (araposionide) = vidrabine	5 times/day	Less sensitive than acycleoin	Causes marked skin allergy
4	Iduxouridie	Not used nowdays		

(2) Debridement

▪ ***Indications***

1. resistance to antivirals
2. Allergy to antivirals
3. Unavailability of antivirals
4. Non-compliance

(B) Stromal necrotic keratitis

• **Cause**

Direct viral invasion and destruction of the corneal stroma.

•Clinical features

• ***Presentation:*** is with a gradual onset of unilateral pain, redness, and severe visual impairment.

• ***Signs:*** are cheesy and necrotic stroma similar to bacterial or fungal infection. There may be an associated anterior uveitis with KPs underlying the area of active stromal infiltration.

•Complications

1. Vascularization and scarring (common)
2. Perforation (rare)

• **Treatment:** is difficult and frequently unsatisfactory

1. **The first aim** is to heal any associated active epithelial lesions with antiviral drugs and lubricants.

2. **In severe, resistant cases**, the cautious use of topical steroids combined with antiviral and antibiotic cover, may be necessary to relieve symptoms and prevent severe corneal scarring.

(C) Disciform keratitis

• **Cause:** exaggerated hypersensitivity reaction to the virus antigen.

• **Clinical features**

• ***Presentation:*** is with subacute onset of unilateral blurred vision which may be associated with haloes around light.

• ***Signs***

1. Central (occasionally eccentric) zone of stromal and epithelial edema
2. KPs underlying the involved area
3. Small stromal infiltrates with a surrounding ring (Wessely ring) around the lesion.

• **Treatment:** is more satisfactory than stromal necrotic keratitis

1. ***Initially***, topical steroids and antivirals (drops), are given 4 times daily
2. ***As improvement occurs***, antivirals are given 3 times daily, and steroids should be tapered gradually over a period of several weeks.

(3) Trophic Keratitis

Also referred to as **" metaherpetic keratitis"**

• **Cause:** it is not caused by active viral disease alone, but also by persistent defects in the basement membrane, drug toxicity, and elements of denervation

• **Clinical features**: the trophic ulcer can be differentiated from the geographical ulcer on examination in that the latter have linear ' foot-like' extensions branching from its margins which stain with rose Bengal but trophic ulcer have not.
• **Treatment** consists of:

1. Withdrawal of potentially toxic topical drugs
2. Lubricants to promote epithelial healing
3. Topical antibiotics to prevent secondary bacterial infection.

Q What is dendritic ulcer? What is its ttt?

Answer → see before

Q Why the ulcer is dendritic

Answer → see before

Q Enumerate the ocular manifestations of herpes zoster ophthalmicus

Answer

Unilateral skin eruptions affecting half of the face including the eyelids, nose, along the trigeminal nerve branches, postherpetic severe neuralgia, nonspecific corneal ulcer, deep keratitis, scleritis, conjunctivitis, iridocyclitis, 2ry glaucoma, ocular nerve palsies, retrobulbar neuritis.

Q In a table form, differentiate between herpes simplex and HZ keratitis

Differential Diagnosis

	HS	HZ
Cause	Eptheliotropic virus	Neurotropic virus.
Laterality	May be bilateral	Always unilat.
Distribution	No specific distribution	Follows the affected nerves
Pain	No preceding neuralgia	Precedes the eruption
Shape of ulcer		Never typical dendritic
Corneal sensitivity	Diminished (Hyposthesia)	Absent (Anesthesia)
Immunity	Recurrence is common	Solid, no recurrence
Level of corneal affection	Superficial layers of cornea	Deep layers of cornea
Course	Variable	3-4 weeks

Q what is the ulcer without pain and its ttt

NEUROPARALYTIC KERATITIS
(Neurotrophic Keratitis)
(Ulcer without Pain)

• The term **"neuroparalytic** keratitis" is a **misnomer** because the keratitis develops as a sequel to a trigeminal nerve lesion and lesions of sensory nerves do not lead to paralysis.

• **Its appropriate term** is neurotrophic keratitis.

• **Aetiology:**

Neurotrophic keratitis develops in some cases in which the trigeminal nerve is paralysed or if the Gasserian (5^{th} C.N.) ganglion is divided for the radical treatment of trigeminal neuralgia.

- It may, also, occur if the ***5th C.N. ganglion is destroyed by invasive:***

1) Intracranial tumors.
2) Gummatous basal meningitis
3) Fracture base of the skull.
4) As a trigeminal nerve surgery complication.
5) Herpes simplex or zoster.
6) Aneurysms & cerebro-vascular accidents.
7) Post irradiation therapy.

• **Pathogenesis**:

1- Loss of trophic impulses conducted by the nerve fibres.

2- Abrasions are unnoticed due to loss of sensation so pathogenic organisms have free play.

3- Irritative changes in or about the degenerating nerves leading to antidromic impulses and axon reflexes, may result in loss of certain mediators as acetyl choline, proteins or perhaps substances that are similar to epidermal growth factor. Interfering with cell metabolism and repair.

4- Corneal dryness due to:

a) Absence of blinking reflex.
b) Absence of reflex lacrimation.
c) Lagophthalmos if 7th C.N. is affected as well.

• **Clinical picture:**

1) **Irritative stage:** At first upper lid vessels vasodilatation followed by vaso dilatation of deep perilimbal vessels followed by oedema of upper lid then conjunctiva, later corneal oedema. Conjunctival hyperaemia. No pain due to anaesthesia. No lacrimation, photophobia, blepharospasm.
2) **Corneal involvement:** leads to diminution of vision and marked ciliary injection.

a) Haziness and loss of lustre (corneal oedema).

b) Vesicles formation followed by: epithelial exfoliation, which starts at the centre then the interpalpebral zone (conjunctiva and corneal **epithelial**

loss, stained by rose bengal) then spread over the whole cornea except a narrow rim at the periphery. **Associated iritis.**

c) Secondary infection of the stroma occurs which appears yellowish followed by ulceration with hypopyon, large perforation may occur. If healing occurs dense scar results. However panophthalmitis may result due to severe secondary infection. The stromal ulceration results from proteoglycan and collagenolytic enzymes.

N.B. Relapses are the rule (ulceration of the old scar).

- **Diagnosis:**

1- The clinical picture.

2- Qualitative assessment is done with a sterile cotton wisp.

- Quantitative assessment of corneal sensation is best performed with an anesthesiometer.
- Compare sensation of cornea with the other eye.

3- Associated manifestations e.g. herpes zoster. However the areas of hyposthesia in herpes simplex is more localized than that seen in other causes.

- **Treatment:**

1 Ordinary treatment of corneal ulcers, but never cauterize + **lid taping** at bed time

2. **Bandage is essential, bandage contact lens, lubricants,** artificial tears, **antibiotics.**

N.B. Contact lens may be complicated with infection, iritis, hypopyon. **Cycloplegic** is ordered with contact lens to avoid iritis.

3. **Closure of puncti:** to conserve moisture.
4. **Tarsorrhaphy** is the essential line of treatment. Two rectangular areas behind the grey line on either side of midline are denuded from epithelium and sutured by 2 mattress sutures. Sutures are removed after 10 days. Leave the lids adherent together for at least one year. **Tarsorraphy prevent effect of trauma and dryness.**
5. **Topical fibronectin** and **epidermal growth factor.**
6. **If the eye is lost: enucleation is indicated.**

Q What are the types and characters of trachomatous corneal ulcers

Answer

Trachomatous ulcers:

a) **Ulcers related to the pannus:** at the edge or on the surface
b) Ulcers un-related to the pannus: marginal, central & paracentral
c) **Ulcers produced mechanically** by the lashes or PTDs

Q What is arcus senilis and its DD

Answer

ARCUS SENILIS
(Corneal arcus)

It occurs in old people. It is an annular infiltration of the periphery of the corneal stroma with lipoid material. The infiltration at first appears down then up but soon surrounds the whole cornea.

The increasing permeability of limbal B.vs with age allows the low density lipoproteins to pass into the cornea. Hyper cholestrolaemia play a good role.

Symptoms: No symptoms as it never affects vision.

Signs: A ring shaped opacity one mm in breadth, being separated from the limbus by a clear interval (lucid interval of Vogt), which is attributed to the limbal vessels and the termination of Bowman's membrane.

The arcus consists of cholestrol, cholestrol esters, phospholipids and triglycerides. The lipids are found extracellularly in the stroma in hour glass pattern.

Complications: Non, but may lead to marginal corneal degeneration, ectasia, ulceration.

DD:

1) Dry trachomatous pannus: young age ,no clear zone ,usually in upper cornea, Herbert's pits.
2) Pseudogerotoxon: manifestations of spring catarrh, young age, no clear zone.

Treatment:

No treatment in simple arcus senilis.

N.B. Arcus Juvenilis (Juvenile Arcus):

- Occurs in young age (< 30 yrs).

• **Causes:**

- Familial lipidemia.
- Megalocornea.
- Keratoconus.
- Spring catarrh.
- Blue sclera (osteogenesis imperfecta).

Q Mention other senile changes in the eye

Answer

1. Senile ptosis
2. Senile Entropion
3. Senile Ectropion
4. Dermatochalsis
5. Xanthelasma
6. Xerosis
7. Senile cataract
8. Vitreous degeneration
9. Age-related macular degeneration (ARMD)
10. Chorioretinal degerations

Q What is keratomalacia?

Answer

Keratomalacia

(Malnutritional Ulcer)

Affecting badly nourished children, usually occurs in the first year, of life, many of them are syphilitic or suffering from severe starvation, parasitic infection or malnutrition. With vitamin A deficiency. The disease commences by **night blindness,** and may be bilateral.

- **Conjunctiva:** dry with Bitot's spots.
- **Cornea:** The corneal affection start interiorly as punctate keratopathy and spread allover the cornea **giving peau d'orange appearance.** The cornea appears hazy and **insensitive,** and gradually the haziness increases, **yellowish infiltrates** form until finally the whole tissue necroses and may seem to **(melt away)** within few hours, **perforation** occurs with **iris prolapse and panophthalmitis.**
- **No or minimal inflammatory reaction**
  i.e. no ciliary injection... etc.
- The ulceration is aggravated by the associated lagophthalmos.

Treatment:

A. General treatment (is more important): by fluids, plasma protein, vitamin A drops (cod liver oil-10-20 drops/day).

B. Local treatment: as ulcer treatment, vitamin-A- eye ointment and moist warm compresses.

Q What are the most common corneal ulcers in Egypt ?

Answer:

1. traumatic, 2. trachomatous, 3. phlyctenular, 4. ulcers with MP and gonococcal conj., 5. hypopyon ulcer, 6. Malnutrition ulcer

Q What is keratoplasty. Mention indications, contraindications, and complications

Answer

Definition: An operation in which abnormal corneal host tissue is replaced by healthy donor cornea. The graft may be:
1) Full-thickness ***(penetrating keratoplasty)***
2) Partial-thickness ***(lamellar keratoplasty)***

Penetrating Keratoplasty
Indications:

1) Optical K:
to improve vision (e.g. pseudophakic bullous keratopathy, keratoconus, corneal scarring, dystrophies & degenerations).

2) Tectonic (structural)k:
to restore or preserve corneal integrity in eyes with severe structural changes, such as stromal thining (descematocele, pterygium).

3) Therapeutic K:
removal of infected corneal tissue in eyes unresponsive to antimicrobial therapy.

4) Cosmetic K:

to improve the appearance of the eye.

Donor tissue:

Age: young patients are preferred (infants →floppy graft→ high astigmatism, old age →low endothelial count)

-The donor tissue should ideally be removed within 24 hs of death.

-Recent advances in storage media that contain vitamins, amino acids and antioxidants have increased the viability of corneal grafts for more than 2 weeks.

Contraindications:

1) Infectious diseases of the CNS (Jacob disease, systemic sclerosing panencephalitis).
2) Certain systemic infections (hepatitis, AIDS,syphilis, septicemia).
3) Leukemia and disseminated lymphoma.
4) Intrinsic eye disease (malignancy or active inflammations) Leukemia and disseminated lymphoma.).
5) Previous intraocular surgery.

Complications:

1) Early: wound leak, flat AC, iris prolapse, uveitis, increased IOP.
2) Late: astigmatism, recurrence of initial disease, glaucoma &graft rejection.

Graft Failure (Rejection):

Early: from the 1st day (due to defective donor endothelium).
Late: due to immunological graft rejection, usually within 1 year.

Lamellar keratoplasty

Partial thickness excision of the corneal epithelium & stroma so that the endothelium & pan of the deep stroma are left behind.

Indications:

1) Opacification of the superficial 1/3 of the corneal stroma.
2) Marginal corneal thining or infiltration (recurrent pterygium, limbal tumours).

Advantages:

less risk of rejection, less astigmatism, increased availability of graft material since endothelial quality is irrelevant.