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Inflammations of Cornea



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INFLAMMATIONS OF CORNEA

➤ Inflammations of the cornea (keratitis) is characterised by **corneal oedema, cellular infiltration and ciliary congestion.**

Classification :

- Topographical (morphological) classification :
- **Ulcerative keratitis** (corneal ulcer) :



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Corneal ulcer can be further classified variously :

1. Depending on location

- (a) Central corneal ulcer
- (b) Peripheral corneal ulcer

2. Depending on purulence

- (a) **Purulent corneal ulcer or suppurative corneal ulcer** (most bacterial and fungal corneal ulcers are suppurative).
- (b) **Non-purulent corneal ulcers** (most of viral, chlamydial and allergic corneal ulcers are non-suppurative).



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3. Depending upon association of hypopyon

- (a) Simple corneal ulcer (without hypopyon)
- (b) Hypopyon corneal ulcer

4. Depending upon depth of ulcer

- (a) Superficial corneal ulcer
- (b) Deep corneal ulcer
- (c) Corneal ulcer with impending perforation
- (d) Perforated corneal ulcer



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5. Depending upon slough formation

- (a) Non-sloughing corneal ulcer
- (b) Sloughing corneal ulcer

Etiological classification

1. Infective keratitis

- (a) Bacterial
- (b) Viral
- (c) Fungal
- (d) Chlamydial
- (e) Protozoal
- (f) Spirochaetal



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2. Allergic keratitis

- (a) Phlyctenular keratitis
- (b) Vernal keratitis
- (c) Atopic keratitis

3. Trophic keratitis

- (a) Exposure keratitis
- (b) Neuroparalytic keratitis
- (c) Keratomalacia
- (d) Atheromatous ulcer

4. Keratitis associated with diseases of skin and mucous membrane.

5. Keratitis associated with systemic collagen vascular disorders.



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6. Traumatic keratitis, which may be due to mechanical trauma, chemical trauma, thermal burns, radiations

7. Idiopathic keratitis

- (a) Mooren's corneal ulcer
- (b) Superior limbic keratoconjunctivitis
- (c) Superficial punctate keratitis of Thygeson

ULCERATIVE KERATITIS :

- Corneal ulcer may be defined as discontinuation in normal epithelial surface of cornea associated with necrosis of the surrounding corneal tissue.
- Pathologically it is characterised by oedema and cellular infiltration.



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Common types of corneal ulcers :

Infective Keratitis :

1. Bacterial Corneal Ulcer :

- ❖ Infective corneal ulcer may develop :
 - Jeopardised local ocular defence mechanism
 - Local ocular predisposing disease
 - Host immunity is compromised
 - Virulent causative organism



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Etiology

1. Damage to **corneal epithelium**
2. **Infection** of the eroded area
3. Pathogens can invade the intact corneal epithelium and produce ulceration:
 - **Neisseria gonorrhoeae,**
 - **Corynebacterium Diphtheriae**
 - **Neisseria meningitidis.**



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1. **Corneal epithelial damage.** It may occur in following conditions:

- i. **Corneal abrasion** due to small foreign body, misdirected cilia, concretions and trivial trauma in contact lens wearers or otherwise.

- ii. Epithelial drying as in **xerosis and exposure keratitis.**

- iii. **Necrosis** of epithelium as in keratomalacia.



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iv. **Desquamation of epithelial cells** as a result of corneal oedema as in bullous keratopathy.

v. **Epithelial damage** due to trophic changes as in neuroparalytic keratitis.

2. Source of infection include:

i. **Exogenous infection.** Most of the times corneal infection arises from exogenous source like conjunctival sac, lacrimal sac (dacryocystitis), infected foreign bodies, infected vegetative material and water-borne or air-borne infections



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ii. From the ocular tissue. Owing to direct anatomical continuity, diseases of the conjunctiva readily spread to corneal epithelium, those of sclera to stroma, and of the uveal tract to the endothelium of cornea

iii. Endogenous infection. Owing to avascular nature of the cornea, endogenous infections are of rare occurrence.

3. Causative organisms. Common bacteria associated with corneal ulceration are: **Staphylococcus aureus, Pseudomonas pyocyanea, Streptococcus pneumoniae, E. coli, Proteus, Klebsiella, N. gonorrhoea, N. meningitidis and C. diphtheriae.**



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Pathogenesis

1. Once the damaged corneal epithelium is invaded by the offending agents the development of corneal ulcer can be described under four stages :
 - **Stage of progressive infiltration**
 - **Stage of active ulceration**
 - **Stage of regression**
 - **Stage of cicatrization**



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2. Terminal course of corneal ulcer depends on the virulence of the infecting agent, host defence mechanism and treatment received.

3. Forms of ulcer :

- Localised and heal
- Deep penetration to corneal perforations
- Fast spreading to involve whole cornea as sloughing corneal ulcer.



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A) Pathology of localised corneal ulcer :

1. Stage of progressive infiltration :

- Characterised by the infiltration of polymorphonuclear and/or lymphocytes into the epithelium from the peripheral circulation supplemented by similar cells from the underlying stroma if this tissue is also affected.

2. Stage of active ulceration :

- Active ulceration results from necrosis and sloughing of the epithelium, Bowman's membrane and the involved stroma.



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- The walls of active ulcer project owing to swelling of lamellae by the imbibition of fluid and the packing of masses of leukocytes between them.
- During this stage of active ulceration there occurs:
 - ✓ **Hyperaemia of circumcorneal** vessels network results into accumulation of purulent exudates on cornea
 - ✓ **Vascular congestion of iris and ciliary body, iritis** due to absorption of toxins from ulcer. Exudation into AC



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- ✓ Ulceration may further progress to diffuse superficial ulceration leading to **Descemetocele** formation and corneal perforation.

3. Stage of regression :

- Regression is induced by the natural **host defence mechanisms** (humoral antibody production and cellular immune defences) and the treatment which augments the normal host response.
- A line of demarcation develops around the ulcer, which consists of leucocytes that neutralize and eventually phagocytose the offending organisms and necrotic cellular debris.



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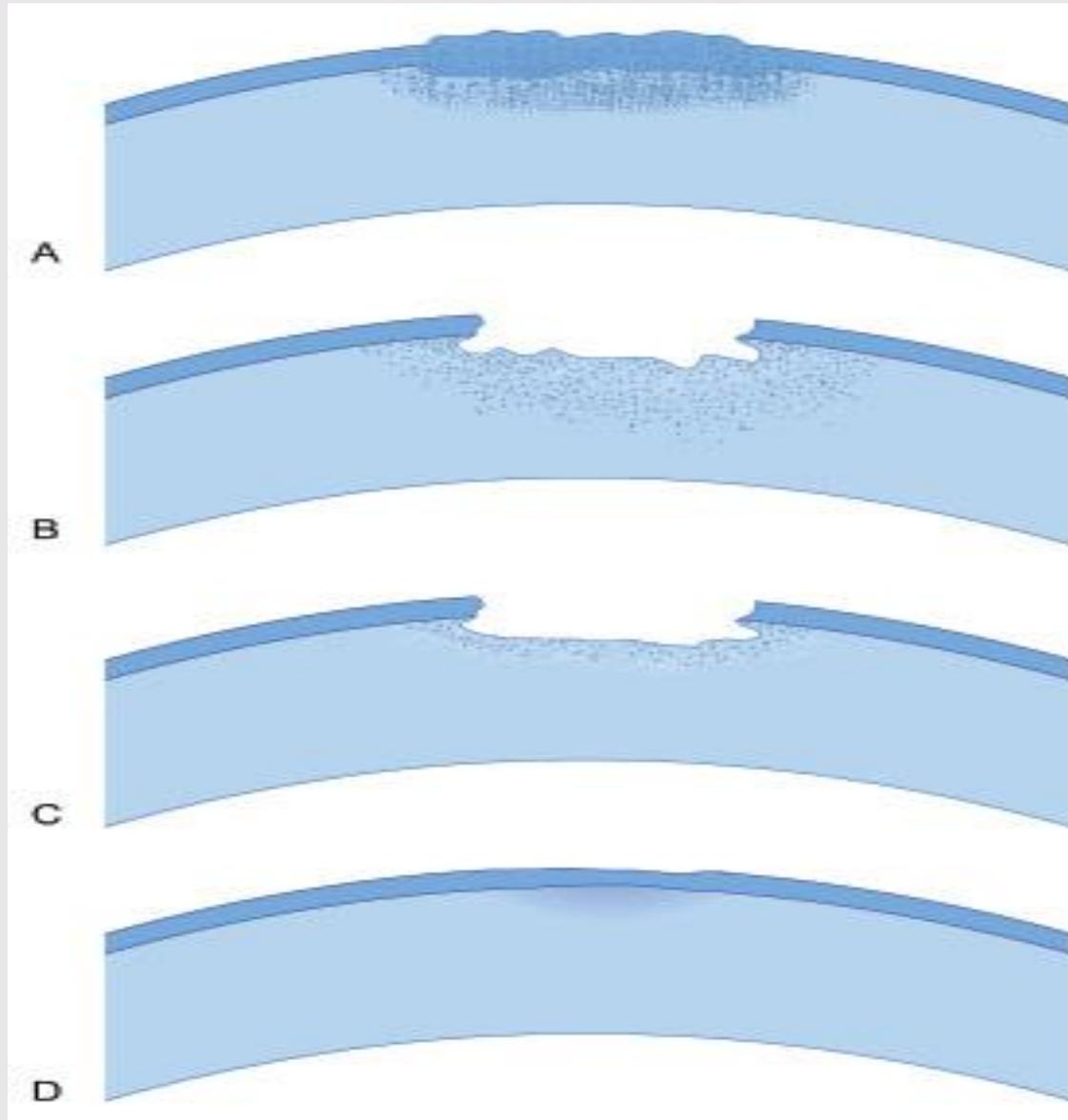
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- The digestion of necrotic material may result in initial enlargement of the ulcer.
- This process may be accompanied by **superficial vascularization** that increases the humoral and cellular immune response. The ulcer now begins to heal and epithelium starts growing over the edges.



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Pathology of corneal ulcer : A, stage of progressive infiltration; B, stage of active ulceration; C, stage of regression; D, stage of cicatrization.



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4. Stage of cicatrization :

- Healing continues by progressive **epithelization** which forms a permanent covering.
- Beneath the epithelium, fibrous tissue is laid down partly by the corneal fibroblasts and partly by the endothelial cells of the new vessels.
- The stroma thus thickens and fills in under the epithelium, pushing the epithelial surface anteriorly.



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- The degree of scarring from healing varies. If the ulcer is very superficial and involves the epithelium only, it heals without leaving any opacity behind.
- When ulcer involves Bowman's membrane and few superficial stromal lamellae, the resultant scar is called a '**nebula**'.



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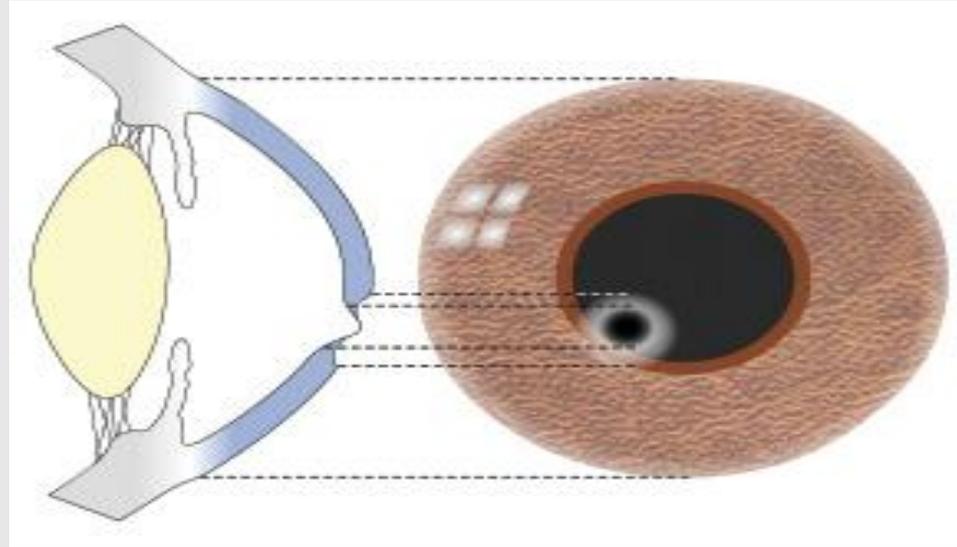
B) Pathology of perforated corneal ulcer:

- **Perforation of corneal ulcer** occurs when the ulcerative process deepens and reaches up to Descemet's membrane.
- This membrane is tough and bulges out as **Descemetocoele.**
- At this stage, any exertion on the part of patient, such as coughing, sneezing, straining for stool etc. will perforate the corneal ulcer.



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Descemetocoele : A, Diagrammatic depiction;
B. Clinical photographs.



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- Immediately after perforation, the aqueous escapes, intraocular pressure falls and the iris-lens diaphragm moves forward.
- The effects of perforation depend upon the position and size of perforation.
- When the perforation is small and opposite to iris tissue, it is usually plugged and healing by cicatrization proceeds rapidly.



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C) Pathology of sloughing corneal ulcer and formation of anterior staphyloma :

- When the infecting agent is highly virulent and/or body resistance is very low, the whole cornea sloughs with the exception of a narrow rim at the margin and total prolapse of iris occurs (fig 6.4).
- The iris becomes **inflamed and exudates block the pupil and cover the iris surface**; thus a false cornea is formed.
- Ultimately these exudates organize and form a thin fibrous layer over which the **conjunctival or corneal epithelium rapidly grows and thus a pseudocornea is formed**.



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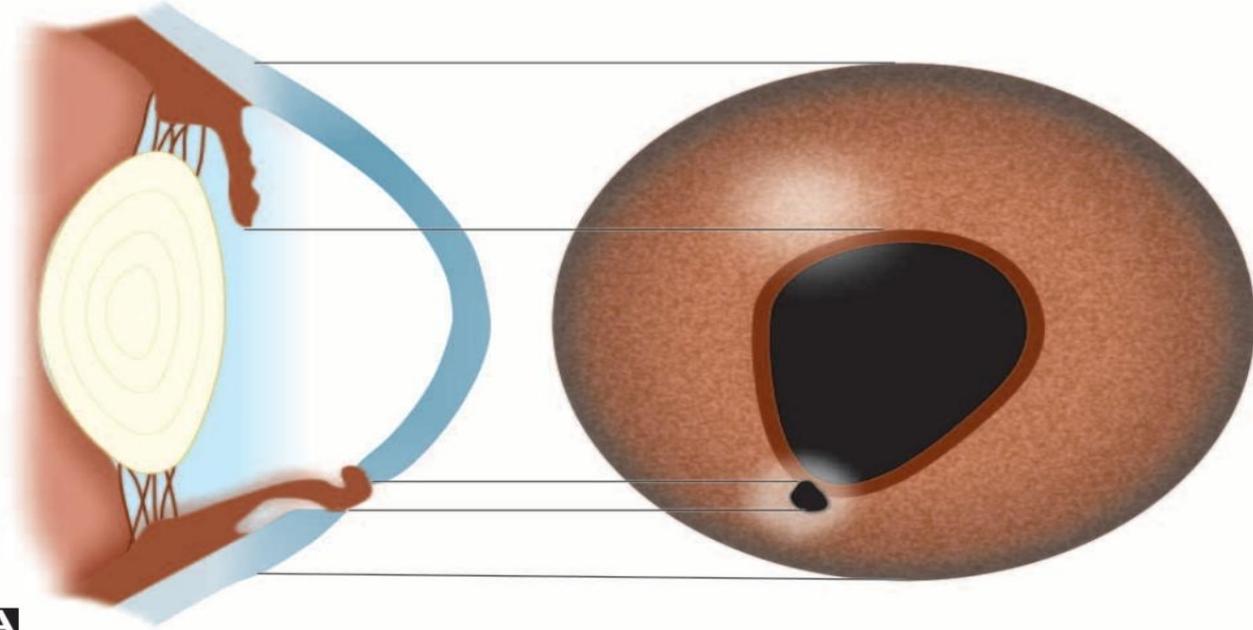
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- Since the pseudocornea is thin and cannot withstand the intraocular pressure, so it usually bulges forward along with the plastered iris tissue.
- This ectatic cicatrix is called **anterior staphyloma** which, depending upon its extent, may be either partial or total.
- The bands of scar tissue on the staphyloma vary in breadth and thickness, producing a lobulated surface often blackened with iris tissue which resembles a bunch of black grapes .



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A



B

Fig. 6.4 *Perforated corneal ulcer with prolapse of iris: A, Diagrammatic depiction; B, Clinical photograph*



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Clinical features

- ❖ In bacterial infections the outcome depends upon the virulence of organism, its toxins and enzymes, and the response of host tissue.

- ❖ Broadly bacterial corneal ulcers may manifest as:
 - Purulent corneal ulcer without hypopyon; or
 - Hypopyon corneal ulcer.



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Symptoms

1. Pain and foreign body sensation
2. Watering
3. Photophobia
4. Blurred vision
5. Redness of eyes

Signs

- Lids swelling
- Blepharospasm
- Chemosed conjunctiva



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➤ Corneal ulcer

1. **Yellowish white area** of ulcer which may be oval or irregular shape
2. Margins of the ulcer are **swollen and over hanging**.
3. Floor of the ulcer is covered by **necrotic material**.
4. **Stromal oedema** is present surrounding the ulcer area.

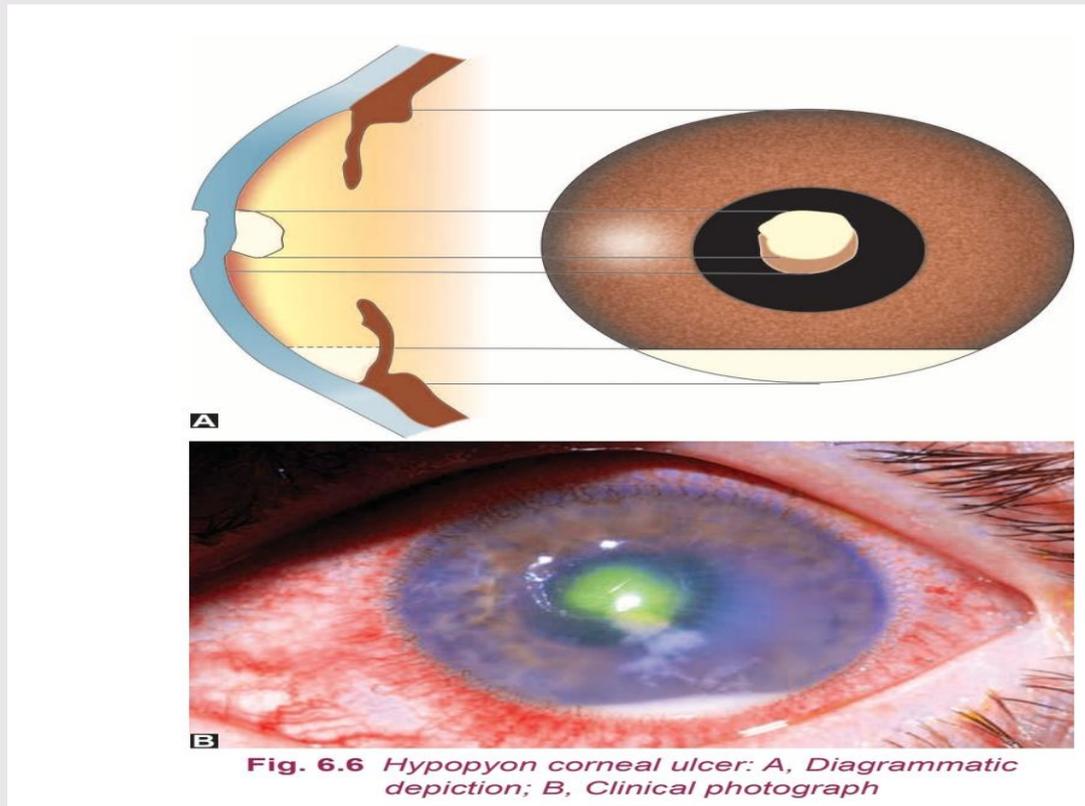


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Characteristic features :

- *Staphylococcal aureus* and *streptococcus pneumoniae* usually produce an oval, yellowish white densely opaque ulcer which is surrounded by relatively clear cornea.





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- Pseudomonas species usually produce an irregular sharp ulcer with thick **greenish mucopurulent exudate, diffuse liquefactive necrosis and semiopaque** (ground glass) surrounding cornea. Such ulcers spread very rapidly and may even perforate within 48 to 72 hours.
- **Enterobacteriae** usually produce a shallow ulcer with greyish white pleomorphic suppuration and diffuse stromal opalescence. The endotoxins produced by these Gram –ve bacilli may produce **ring-shaped corneal infiltrate**.

Bacterial corneal ulcer without hypopyon





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5. **Anterior chamber** may or may not show pus (hypopyon).
6. **Iris** may be slightly muddy in colour.
7. **Pupil** may be small due to associated toxin induced iritis.
8. **Intraocular pressure** may some times be raised



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Hypopyon Corneal Ulcer

Etiopathogenesis :

1.Causative organisms:

- Hypopyon corneal ulcer – **Pneumococcus**
- Corneal ulcer with hypopyon - **staphylococci, streptococci, gonococci, Moraxella.**

2. Source of infection : Chronic dacryocistitis.

3. Factors predisposing to development of hypopyon : Virulence of the infected organism and resistance of the tissues.



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4. Mechanism :

- Corneal ulcer is often associated with some **iritis** owing to diffusion of bacterial toxins.
- When the iritis is severe the outpouring of leucocytes from the vessels is so great that these cells gravitate to the bottom of the anterior chamber to form a **hypopyon**.
- Thus, it is important to note that the hypopyon is sterile since the outpouring of **polymorphonuclear cells** is due to the toxins and not due to actual invasion by bacteria.
- Once the ulcerative process is controlled, the hypopyon is absorbed.



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Clinical features :

Symptoms :

- Are the same as described above for bacterial corneal ulcer. However, it is important to note that during initial stage of **ulcus serpens** there is remarkably little pain. As a result the treatment is often unduly delayed.

Signs : Same as bacterial ulcer

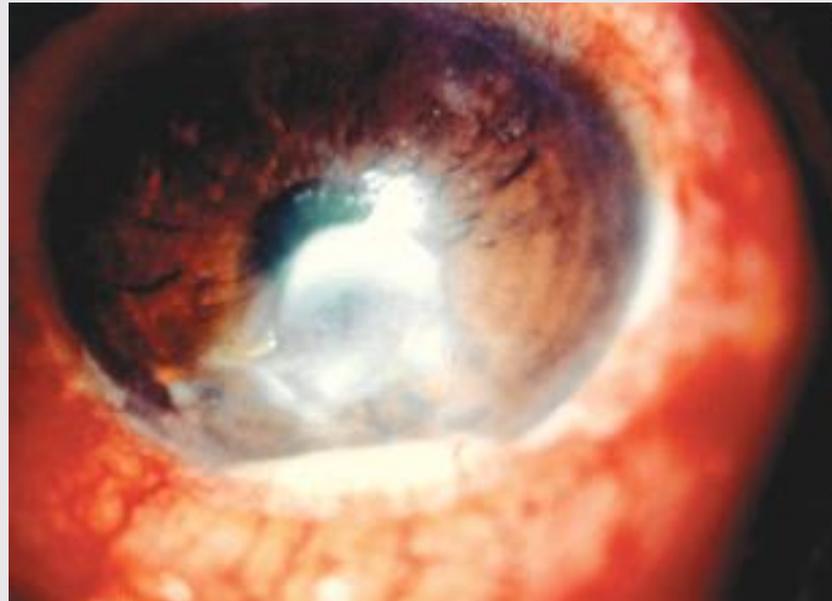
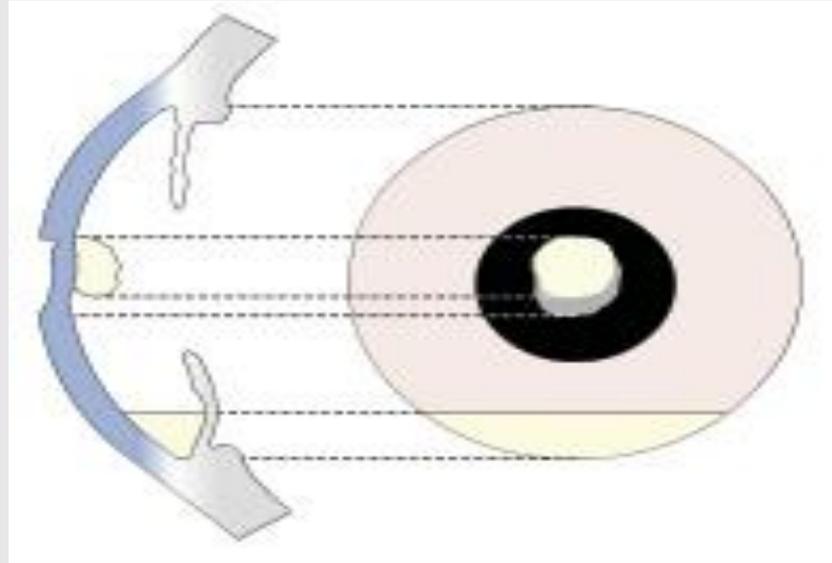
Characteristic features :

- **Greyish white or yellowish dishaped ulcer**
- Ulcer creeps over cornea and **cicatrization**
- **Violent iridocyclitis**
- **Hypopyon** increases in size
- **Rapid spread** of ulcer



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Hypopyon corneal ulcer : A, Diagrammatic depiction; B, Clinical photograph.



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Management

- Secondary glaucoma should be anticipated and treated with 0.5% **timolol maleate**, B.I.D. eye drops and **oral acetazolamide**.
- Source of infection, i.e., chronic dacryocystitis if detected, should be treated by **dacryocystectomy**.



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Complications :

- Toxic iridocyclitis
- Secondary glaucoma
- Descemetocoele
- Perforation of corneal ulcer :
 - ✓ Prolapse of iris
 - ✓ Subluxation or anterior dislocation of lens
 - ✓ Anterior capsular cataract
 - ✓ Corneal fistula
 - ✓ Purulent uveitis , endophthalmitis or even panophthalmitis
 - ✓ Intraocular haemorrhage
- Corneal scar



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Management of cases of corneal ulcer:

The management includes :

1. Clinical evaluation :

- History taking
- General physical examination
- Ocular examination
- ✓ Diffuse light examination
- ✓ Regurgitation test
- ✓ Biomicroscopic examination



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2.Laboratory examination :

- ❖ **Routine laboratory investigations** : Haemoglobin, TLC, DLC, ESR, blood sugar, complete urine and stool examination.
- ❖ **Microbial investigations:**
 - ✓ **Gram and Giemsa** stained smears for possible identification of infecting organisms.
 - ✓ 10 per cent KOH wet preparation for identification of **fungal hyphae**.
 - ✓ **Calcofluor white (CFW) stain** preparation is viewed under fluorescence microscope for fungal filaments, the walls of which appear bright apple green.
 - ✓ Culture on **Sabouraud's dextrose agar** medium for **fungi**.
 - ✓ Culture on blood agar medium for aerobic organisms



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3. Treatment :

I. Treatment of uncomplicated corneal ulcer :

1. Specific treatment of cause :

A) Topical antibiotics:

- Initial therapy
- Fortified cefazoline - 5%
- Fortified tobramycin-1.3%
- Fortified vancomycin – 5%



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- **Frequency of instillation** : The chosen two drugs should be instilled alternatively as follows
 1. Every 5 minutes for 30 minutes
 2. Every 15 minutes for 2 hours
 3. 1 hourly round the clock for first 48 hours
 4. 2 hourly during day and 4 hours at night
 5. 4-6 hours till healing process
- **Subsequent therapy** : There is no need to change initial antibiotics if response is good.



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B) Systemic antibiotics :

- Cephalosporine and aminoglycoside or oral ciprofloxacin (750 mg twice daily) may be given in fulminating cases with perforation and when sclera is also involved.

2. Non-specific treatment :

(a) **Cycloplegic drugs.** Preferably 1 percent atropine eye ointment or drops should be used to reduce pain from ciliary spasm and to prevent the formation of posterior synechiae from secondary iridocyclitis.



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(b) **Systemic analgesics and anti-inflammatory** drugs such as paracetamol and ibuprofen relieve the pain and decrease oedema.

(c) **Vitamins** (A, B-complex and C) help in early healing of ulcer.

3. Physical and general measures :

a) Hot **fomentation**. Local application of heat gives comfort, reduces pain and causes vasodilatation.

b) **Dark goggles** may be used to prevent photophobia.

(c) Rest, good diet and fresh air may have a soothing effect



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II. Treatment of non-healing corneal ulcer :

If the ulcer progresses despite the above therapy the following additional measures should be taken:

1. Removal of any known cause of non-healing ulcer.

A thorough search for any already missed cause not allowing healing should be made and when found, such factors should be eliminated.

Common causes of non-healing ulcers are as under:

i. Local causes. Associated raised intraocular pressure, concretions, misdirected cilia, impacted foreign body, dacryocystitis, inadequate therapy, wrong diagnosis, lagophthalmos and excessive vascularization of ulcer.



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ii. Systemic causes: Diabetes mellitus, severe anaemia, malnutrition, chronic debilitating diseases and patients on systemic steroids.

2. Mechanical debridement of ulcer to remove necrosed material by scraping floor of the ulcer with a spatula under local anaesthesia may hasten the healing.

3. Cauterisation of the ulcer may also be considered in non-responding cases. Cauterisation may be performed with pure carbolic acid or 10-20 percent trichloroacetic acid.

4. Bandage soft contact lens may also help in healing.

5. Peritomy, i.e., severing of perilimbal conjunctival vessels may be performed when excessive corneal vascularization is hindering healing.



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III. Treatment of impending perforation :

1. **No strain.** The patient should be advised to avoid sneezing, coughing and straining during stool etc.
2. **Pressure bandage** should be applied to give some external support.
3. **Lowering of intraocular pressure** by simultaneous use of **acetazolamide 250 mg QID orally**, **intravenous mannitol (20%) drip stat**, oral glycerol twice a day, **0.5% timolol eyedrops** twice a day, and even **paracentesis** with slow evacuation of aqueous from the anterior chamber may be performed if required.
4. Tissue adhesive glue such as **cynoacrylate** is helpful in preventing perforation.



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5. **Conjunctival flap.** The cornea may be covered completely or partly by a conjunctival flap to give support to the weak tissue.

6. **Bandage soft contact lens** may also be used.

7. **Penetrating therapeutic keratoplasty** (tectonic graft) may be undertaken in suitable cases, when available.

IV. Treatment of perforated corneal ulcer :

Use of bandage soft contact lens or therapeutic keratoplasty should be undertaken. Best is an urgent therapeutic keratoplasty.



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Marginal Catarrhal Ulcer

- **Superficial ulcers** situated near the limbs frequently seen in old people



- Marginal corneal ulcer in a patient with acute conjunctivitis.



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Etiology:

➤ Hypersensitivity reaction to staphylococcal toxins associated with –**Staphylococcal blepharoconjunctivitis** ,**Marcella and haemophilus** are known to cause such ulcers .

Clinical features :

- Ocular irritation
- photophobia,
- pain,
- watering
- Ulcer is shallow slightly unfiltered when associated with Staphylococcal conjunctivitis
- Vacularization and recurrences are common



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Treatment

- **Topical corticosteroid** drops and antibiotic treatment
- Treatment of blepharitis and chronic conjunctivitis to prevent recurrences



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Mycotic corneal ulcer

❖ **Suppurative corneal ulcer** caused by fungi

Etiology:

Fungi :

1. **Filamentous fungi** : Asperagillus, Fusarium, penicillium, Cephalosporium
 2. **Yeast** : Candida and cryptococcus
 3. **Dimorphic fungi** : Histoplasma,blastomyces
- ❖ Fungi mostly associated with corneal ulcers include Asperagillus, Fusarium and Candida



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Fungal corneal ulcer.



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Clinical features :

Signs :

- Greyish white corneal ulcer
- Pigmented ulcer (brownish)
- Delicate feathery finger like extensions
- Sterile immune ring – yellow line of demarcation
- Multiple, small satellite lesions
- Big hypopyon
- Endothelial plaque
- Perforation and corneal vascularization

Symptoms : Similar to bacterial ulcer



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Diagnosis :

- ❖ Typical clinical manifestations including history
- ❖ Laboratory investigations : Sample collection
 - Corneal scraping
 - Anterior chamber paracentesis
 - Corneal biopsy
 - Confocal microscopic examination of cornea
 - PCR



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Treatment

A. Specific treatment:

1. Topical Antifungal drugs :

❖ Topical antifungal eye drops: Natamycin , Amphotericin B and either Fluconazole or miconazole.

❖ Nystatin eye ointment

2. Intracranial, intracamrral or intrastormal administration of Voricanazole

3. Systemic antifungal drugs

A. Specific treatment

B. Nonspecific treatment

C. Therapeutic penetrating keratoplasty



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VIRAL CORNEAL ULCERS :

- Incidence of viral corneal ulcers has become much greater especially because of the **role of antibiotics in eliminating the pathogenic bacterial flora.**
- Most of the viruses tend to affect the **epithelium of both the conjunctiva and cornea**, hence the typical viral lesions constitute the viral keratoconjunctivitis.



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HERPES SIMPLEX KERATITIS :

Ocular infections with herpes simplex virus (HSV) are extremely common and constitute herpetic keratoconjunctivitis and iritis.

Etiology :

Herpes simplex virus (HSV) :

- It is a DNA virus
- Its only natural host is man.
- HSV is **epitheliotropic** but may become **neurotropic**.



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Mode of Infection :

- **HSV-1 infection** : It is acquired by kissing or coming in close contact with a patient suffering from herpes labialis.
- **HSV-II infection**: It is transmitted to eyes of neonates through infected genitalia of the mother.

[A] Primary ocular herpes :

- Primary infection (first attack) involves a **nonimmune person**. It typically occurs in children between 6 months and 5 years of age and in teenagers.



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Clinical features :

1. **Skin lesions.** Vesicular lesions may occur involving skin of lids, periorbital region and the lid margin (vesicular blepharitis).
 2. **Acute follicular conjunctivitis** with regional lymphadenitis is the usual and sometimes the only manifestation of the primary infection.
 3. **Keratitis.** Cornea is involved in about 50 percent of the cases. The **keratitis** can occur as a coarse **punctate or diffuse branching epithelial keratitis** that does not usually involve the stroma.
- Primary infection is usually self-limiting but the virus travels up to the **trigeminal ganglion** and establishes the latent infection.



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[B] Recurrent ocular herpes :

➤ The virus which lies dormant in the trigeminal ganglion, periodically reactivates and causes recurrent infection.

➤ **Predisposing stress stimuli** which trigger an attack of herpetic keratitis include:

- Fever such as malaria, flu
- Exposure to ultraviolet rays,
- General ill- health, emotional or physical exhaustion,
- Mild trauma, menstrual stress,
- Administration of topical or systemic steroids and immunosuppressive agents

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1. Epithelial keratitis

Clinical features :

➤ Epithelial (dendritic or geographic) keratitis is associated with **active virus replication**.

➤ **Symptoms** : Mild–moderate discomfort, redness, photophobia, watering and blurred vision.

Signs in approximately chronological order:

➤ **Swollen opaque epithelial cells** arranged in a coarse punctate or stellate (Fig. 6.12A) pattern.



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- **Central desquamation** results in a linear-branching (dendritic) ulcer (Fig. 6.12B), most frequent located centrally; the branches of the ulcer have characteristic terminal buds and its bed stains well with fluorescein.

- The virus-laden cells at the margin of the ulcer stain with **rose Bengal** (Fig. 6.12C), and this may help distinction from alternative diagnoses, particularly an atypical recurrent corneal abrasion.
 - Corneal sensation is reduced.

 - Inadvertent topical steroid treatment may promote progressive enlargement of the ulcer to a geographical or '**amoeboid**' configuration (Fig. 6.12D).

 - Mild associated **subepithelial haze** is typical.



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- Anterior chamber activity may be present, but is usually mild.
- **Follicular conjunctivitis** may be associated; topical antivirals can also cause this.
- **Vesicular eyelid lesions** may coincide with epithelial ulceration.
- Elevated IOP is not uncommon (tonometry should be performed on the unaffected eye first; a disposable prism should be used, or a re-usable tonometer prism carefully disinfected after use).



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- Following healing, there may be **persistent punctate epithelial erosions and irregular epithelium** (Fig. 6.12E) which settle spontaneously and should not be mistaken for persistent active infection.

- A **whorled epithelial appearance** commonly results from assiduous, especially prolonged, topical antiviral instillation.

- **Mild subepithelial haze** (Fig. 6.12F) may persist for weeks after the epithelium heals; in some cases mild scarring may develop, which tends to become more evident after each recurrence and may eventually substantially threaten vision.



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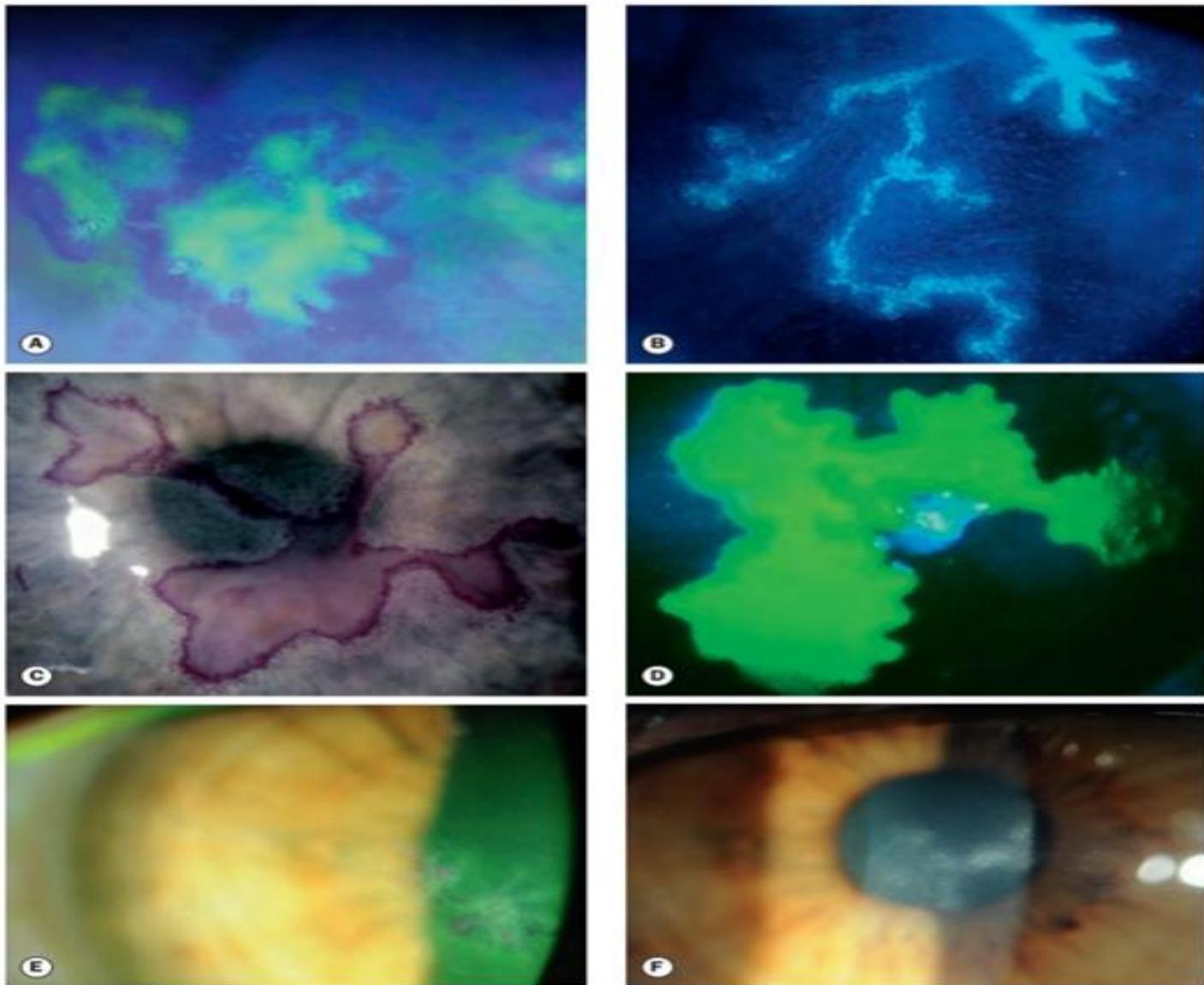


Fig. 6.12 Epithelial herpes simplex keratitis. (A) Stellate lesions; (B) bed of a dendritic ulcer stained with fluorescein; (C) margins of a dendritic ulcer stained with rose Bengal; (D) geographic ulcer; (E) persistent epithelial changes following resolution of active infection; (F) residual subepithelial haze



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- Investigation is generally unnecessary as the diagnosis is principally clinical, but pre-treatment scrapings can be sent in viral transport medium for culture.
- **PCR and immunocytochemistry** are also available.
- **Giemsa staining** shows multinucleated giant cells.
- HSV serological titres rise only on primary infection, but can be used to confirm previous viral exposure, usually in cases of stromal disease when the diagnosis is in doubt.



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❖ Differential diagnosis of dendritic ulceration includes herpes zoster keratitis, healing corneal abrasion , acanthamoeba keratitis, epithelial rejection in a corneal graft, tyrosinaemia type 2, the epithelial effects of soft contact lenses, and toxic keratopathy secondary to topical medication.



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2. Stromal keratitis

Disciform keratitis :

- The aetiopathogenesis of disciform keratitis (endotheliitis) is controversial.
- It may be the result of active HSV infection of keratocytes or endothelium, or a hypersensitivity reaction to viral antigen in the cornea.

Clinical features :

Symptoms :

- Blurred vision of gradual onset, which may be associated with haloes around lights.



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- Discomfort and redness are common, but tend to be milder than in purely epithelial disease.
- A clear past history of epithelial ulceration is not always present, and the possibility of a mimicking infection such as acanthamoeba or fungal keratitis should be borne in mind.
- A central zone of stromal oedema, often with overlying epithelial oedema (Fig. 6.13A); occasionally the lesion is eccentric.



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- Large (granulomatous) **keratic precipitates** underlying the oedema (Fig. 6.13B).
- **Folds in Descemet membrane** in severe cases.
- A surrounding (Wessely) **immune ring** of deep stromal haze (Fig. 6.13C) signifies deposition of viral antigen and host antibody complexes.
- The IOP may be elevated.
- Reduced corneal sensation; this may aid in distinguishing other forms of infection.



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- Healed lesions often have a faint ring of **stromal or subepithelial opacification** and thinning.
- Consecutive episodes may be associated with gradually worsening subepithelial and/or stromal scarring and superficial or deep vascularization (Fig. 6.13D).
- Mid-stromal scarring from disciform keratitis is a cause of interstitial keratitis.



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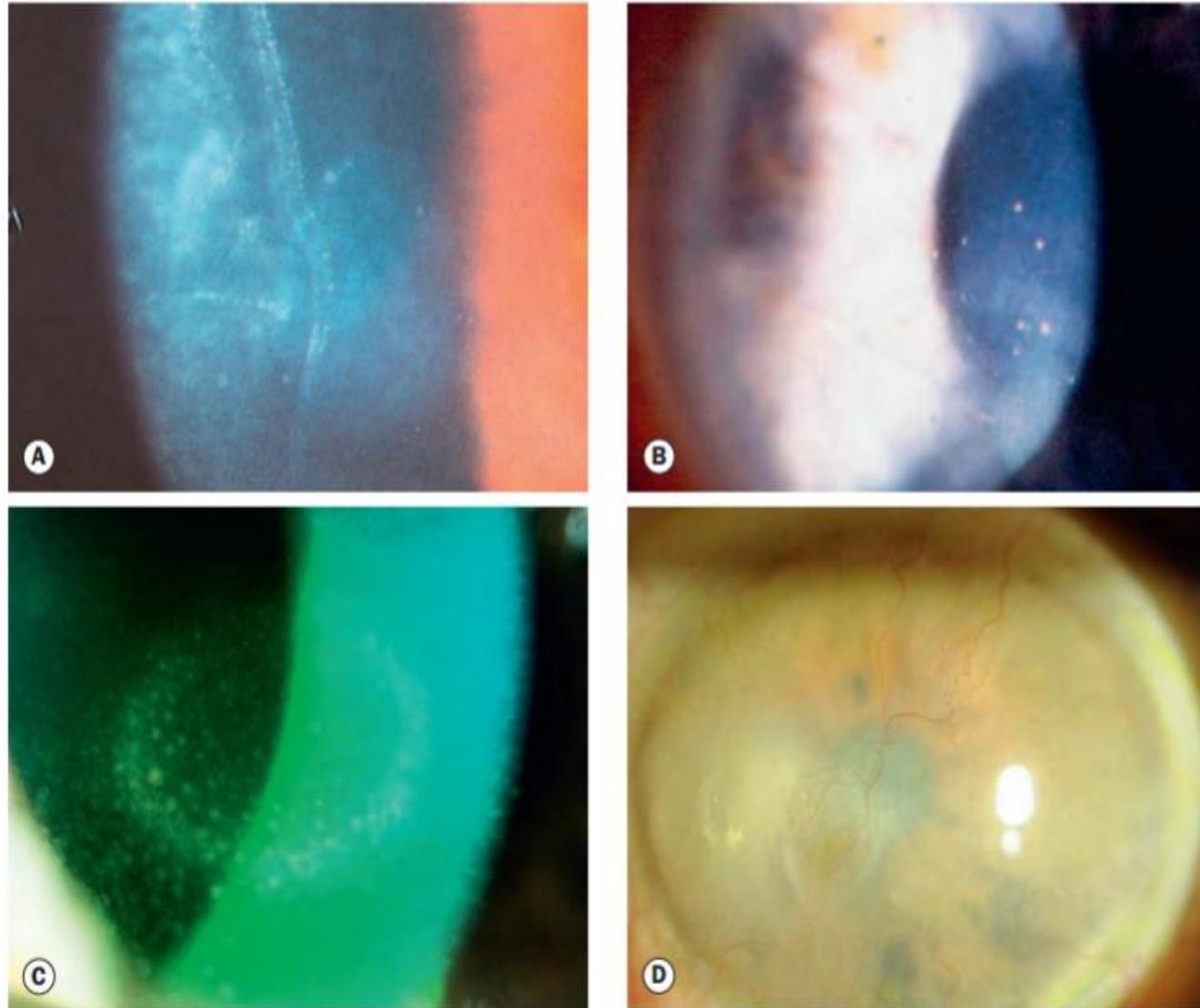


Fig. 6.13 Disciform herpes simplex keratitis. (A) Epithelial and stromal oedema, with Descemet membrane folds; (B) underlying keratic precipitates; (C) Wessely ring; (D) scarring from recurrent disease



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- Treatment consists of **diluted steroid eye drops** instilled 4-5 times a day with an antiviral cover (aciclovir 3%) twice a day.
- **Steroids** should be tapered over a period of several weeks.
- When disciformkeratitis is present with an infected epithelial ulcer, antiviral drugs should be started 5-7 days before the steroids



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Necrotizing stromal keratitis

- This rare condition is thought to result from active viral replication within the stroma, though immune-mediated inflammation is likely to play a significant role.
- It may be difficult to distinguish from severe **disciform keratitis** and there may be a spectrum of disease, including overlap with **neurotrophic keratopathy**.
- As with disciform keratitis, a similar clinical picture may be caused by other infections.



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- **Stromal necrosis** and melting, often with profound **interstitial opacification** (Fig. 6.14).
- **Anterior uveitis with keratic precipitates** underlying the area of active stromal infiltration.
- An **epithelial defect** may be present.
- Progression to scarring, **vascularization and lipid deposition** is common.



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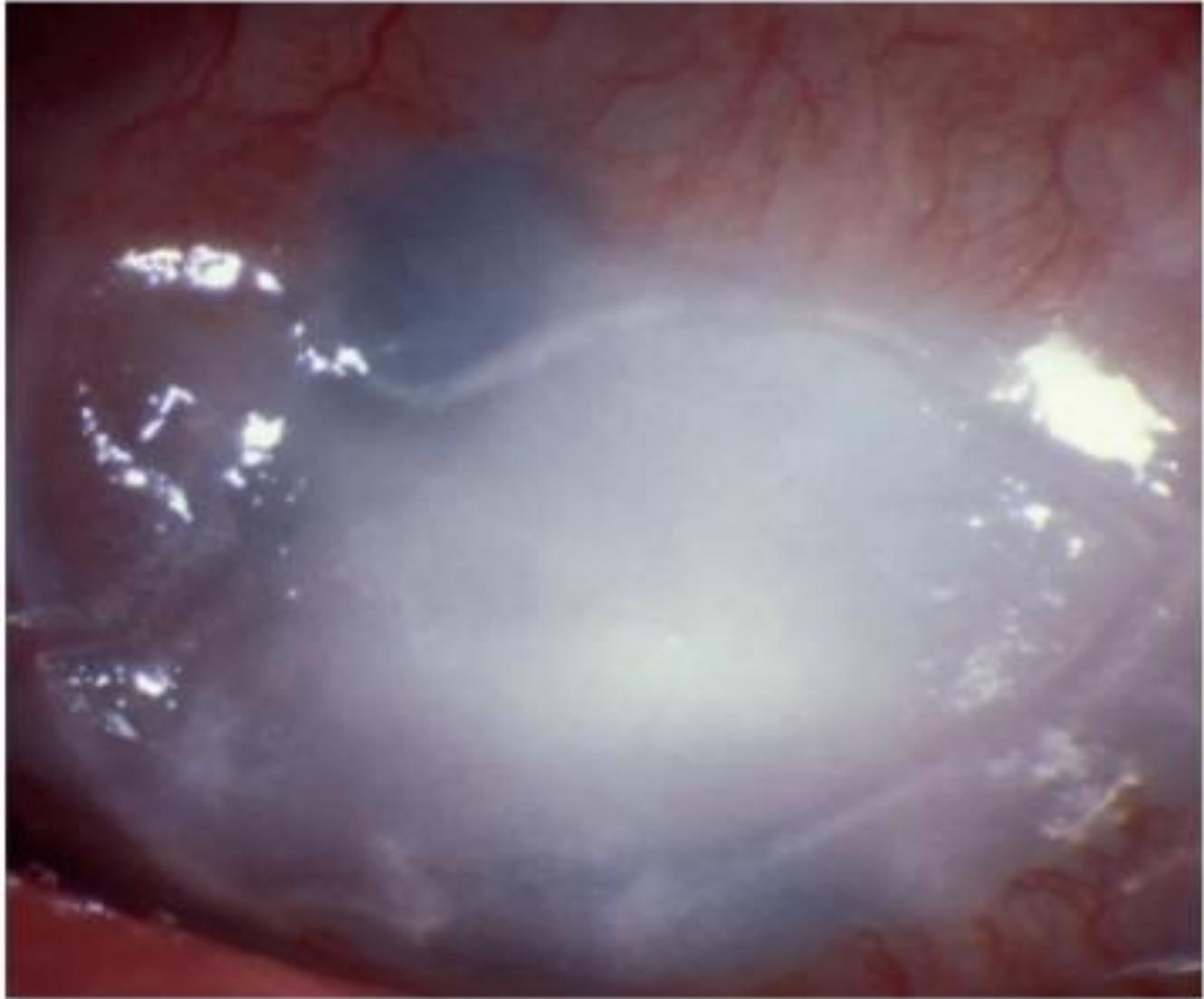


Fig. 6.14 Necrotizing stromal herpes simplex keratitis

(Courtesy of S Tuft)



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Treatment :

- Similar to that of aggressive disciform keratitis, but oral antiviral supplementation, initially at the upper end of the dose range, is commonly used.
- The restoration of epithelial integrity is critical.



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3. Metaherpetic keratitis

- Metaherpetic keratitis (Epithelial sterile trophic ulceration) is not an active viral disease, but is a mechanical healing problem (similar to recurrent traumatic erosions) which occurs at the site of a previous **herpetic ulcer**.
- Clinically it presents as an **indolent linear or ovoid epithelial defect**.
- Treatment is aimed at promoting healing by use of **lubricants (artificial tears), bandage soft contact lens and lid closure (tarsorrhaphy)**.



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HERPES ZOSTER OPHTHALMICUS

- Herpes zoster ophthalmicus is an acute infection of Gasserian ganglion of the fifth cranial nerve by the varicella-zoster virus (VZV).
- It constitutes approximately 10 percent of all cases of herpes zoster.

Etiology :

- ❖ Varicella -zoster virus is a DNA virus and produces acidophilic intranuclear inclusion bodies.
- ❖ It is neurotropic in nature.



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PATHOGENESIS :

- The infection is contracted in childhood, which manifests as chickenpox and the child develops immunity.
- The virus then remains dormant in the sensory ganglion of trigeminal nerve.
- It is thought that, usually in elderly people (can occur at any age) with depressed **cellular immunity**, the virus reactivates, replicates and travels down along one or more of the branches of the **ophthalmic division of the fifth nerve**.



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Risk of ocular involvement :

- The **Hutchinson sign** describes involvement of the skin supplied by the external nasal nerve, a branch of the **nasociliary nerve** supplying the tip, side and root of the nose (Fig. 6.17A).
- The sign correlates strongly with ocular involvement, but there is no apparent correlation between the severity of **the nasal rash** and that of ocular complications.





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➤ **Age.** HZO occurs most frequently in the sixth and seventh decades. In the elderly, signs and symptoms tend to be more severe and of longer duration.

➤ **AIDS patients** tend to have more severe disease, and shingles can be an early indicator of human immunodeficiency virus (HIV) infection.

Clinical features :

- In herpes zoster ophthalmicus, **frontal nerve** is more frequently affected than the lacrimal and nasociliary nerves.
- About 50 percent cases of herpes zoster ophthalmicus get ocular complications.



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- The **Hutchinson's rule**, which implies that ocular involvement is frequent if the side or tip of nose presents vesicles (cutaneous involvement of nasociliary nerve), is useful but not infallible.
- Lesions of herpes zoster are strictly limited to one side of the midline of head.

Clinical phases of H. zoster ophthalmicus are :

- i. **Acute**, which may totally resolve.
- ii. **Chronic**, which may persist for years.
- iii. **Relapsing**, where the acute or chronic lesions reappear sometimes years later.



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❖ **Clinical features** of herpes zoster ophthalmicus include general features, **cutaneous lesions and ocular lesions**.

Acute phase lesions :

General features :

- A prodromal phase precedes the appearance of the rash. It lasts 3–5 days and is characterized by **tiredness, fever, malaise and headache**.
- Symptoms involving the affected dermatome vary from a **superficial itching, tingling or burning sensation to a severe boring or lancing pain** that is either constant or intermittent.
- Older patients with early **severe pain** and a larger area of involvement are at particular risk of **post herpetic neuralgia**



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Skin lesions :

- **Painful erythematous** areas with a maculopapular rash develop (Fig. 6.17B), and may be confused with cellulitis or contact dermatitis.
- Within 24 hours, groups of vesicles (Fig. 6.17C) appear and these become confluent over 2–4 days.
- Although the rash itself does not affect the lower eyelid in HZO, boggy oedema of the upper and lower lids is common (see Fig. 6.17B) and often spreads to the contralateral side of the face.



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- The vesicles often pass through a **pustular phase** before they crust (Fig. 6.17D) and dry after 2–3 weeks.
- Large, **deep haemorrhagic lesions** are more common in **immunodeficient patients** (Fig. 6.17E).
- The lesions heal to leave residual skin destruction and **depigmented scars** (Fig. 6.17F).



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Fig. 6.17 Herpes zoster ophthalmicus. (A) Hutchinson sign – rash involving the side of the nose; (B) early erythema and oedema; (C) vesicular stage; (D) mixed vesicular and pustular rash beginning to show crusting – note the boggy oedema affecting the medial part of both upper lids; (E) severe rash in a patient with AIDS; (F) typical healed appearance with mild to moderate scarring and depigmentation



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Acute eye disease :

- Acute epithelial keratitis develops in over 50% of patients within 2 days of the onset of the **rash** and usually resolves spontaneously within a few days.
- It is characterized by **dendritic lesions** that are smaller and finer than herpes simplex dendrites, and have tapered ends without terminal bulbs (Figs 6.18A and B).



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- The lesions stain better with rose Bengal than with fluorescein. Treatment, if required, is with a **topical antiviral**.
- **Conjunctivitis** (follicular and/or papillary) is common; it often occurs in conjunction with lid margin vesicles.
- Treatment is not required in the absence of corneal disease, though some practitioners give topical antibiotic and/or antiviral prophylaxis.



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- Episcleritis occurs at the onset of the rash and usually resolves spontaneously.
- A mild non-steroidal anti inflammatory may be used if necessary.
- Scleritis and sclerokeratitis are uncommon but may develop at the end of the first week.
- Treatment of indolent lesions is with oral flurbiprofen 100 mg three times daily. Oral steroids with antiviral cover may be required for severe involvement.



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- **Nummular keratitis** usually develops at the site of epithelial lesions about 10 days after the onset of the rash.
- It is characterized by fine **granular subepithelial deposits** surrounded by a halo of stromal haze (Fig. 6.18C).
- The lesions fade in response to topical steroids but recur if treatment is discontinued prematurely.



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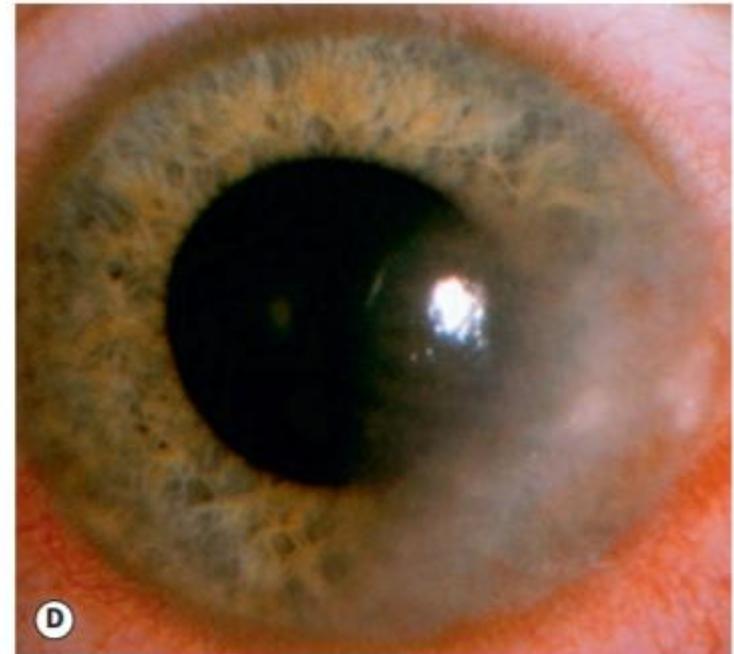
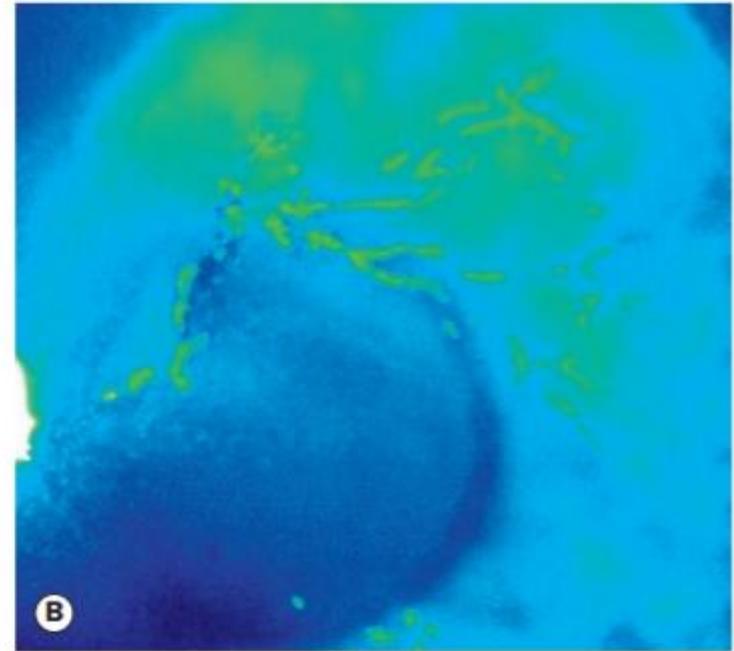
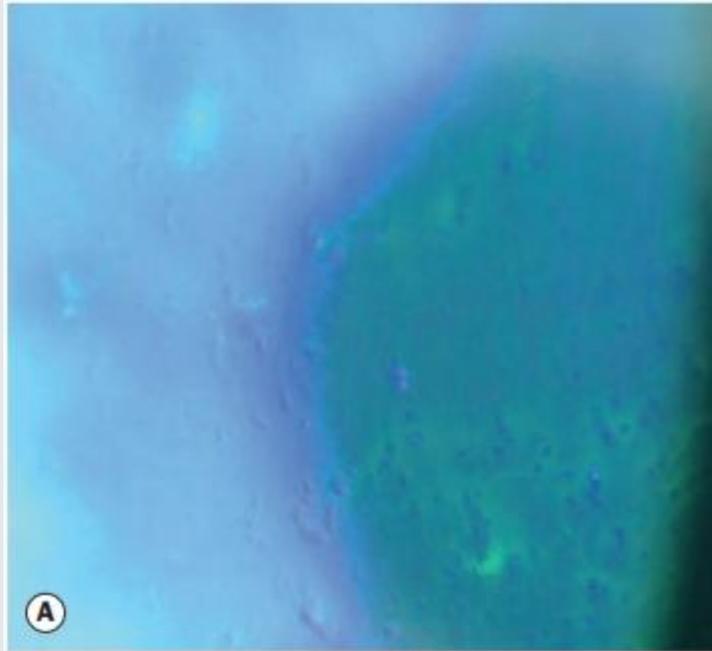


Fig. 6.18 Acute lesions in herpes zoster ophthalmicus. (A) and (B) Dendritic epithelial lesions with tapered ends; (C) nummular keratitis; (D) stromal keratitis

(Courtesy of J Krachmer, M Mannis and E Holland, from Cornea, Elsevier 2005 - fig. B; C Barry - fig. D)



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- **Anterior uveitis** affects at least a third of patients and can be associated with **sectoral iris ischaemia** and **atrophy** (Figs 6.19B and C).
- **Posterior uveitis** . Progressive retinal necrosis is an aggressive retinitis usually occurring in immunodeficient individuals.
- **Acute retinal necrosis** can also be caused by VZV.
- Posterior segment examination should always be performed in patients with HZO; **retinal vasculitis** has also been reported.



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- **IOP should be monitored** as elevation is common, including steroid-induced.
- **Prostaglandin** derivatives should be avoided if treatment is necessary.
- Neurological complications may require **intravenous antivirals and systemic steroids**.
- **Cranial nerve palsies** affecting the third (most common), fourth and sixth nerves usually recover within 6 months.
- **Optic neuritis** is rare.
- CNS manifestations are rare but include **encephalitis, cranial arteritis, and Guillain–Barré syndrome**.



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- **Stromal (interstitial) keratitis** (Fig. 6.18D) develops in about 5% 3 weeks after the onset of the rash; significant scarring can occur (Fig. 6.19A).
- **Disciform keratitis** (immune-mediated endotheliitis) is less common than with herpes simplex infection but may lead to corneal decompensation. Treatment is with topical steroids.



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Chronic eye disease :

- **Neurotrophic keratopathy** similar to that seen in HSV infection develops in up to about 50%, but is usually relatively mild and settles over several months.
- Prolonged severe disease occurs in a minority (see also separate topic).
- Scleritis may become chronic and lead to **patchy scleral atrophy** (Figs 6.19D and E).



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- **Mucous plaque keratitis** develops in about 5%, most commonly between the third and sixth months. It is characterized by elevated **mucous plaques** staining with rose Bengal (Fig. 6.19F).
- Treatment involves a combination of **topical steroid and acetylcysteine**.
- Untreated, plaques resolve after a few months, leaving a faint diffuse **corneal haze**.
- Lipid degeneration may develop in eyes with persistent **severe nummular** or **disciform keratitis** (see Fig. 6.19A).
- Lipid-filled **granulomata** similar to those resulting from chronic irritation may develop in the tarsal conjunctiva, and may progress to erosive calcified concretions.



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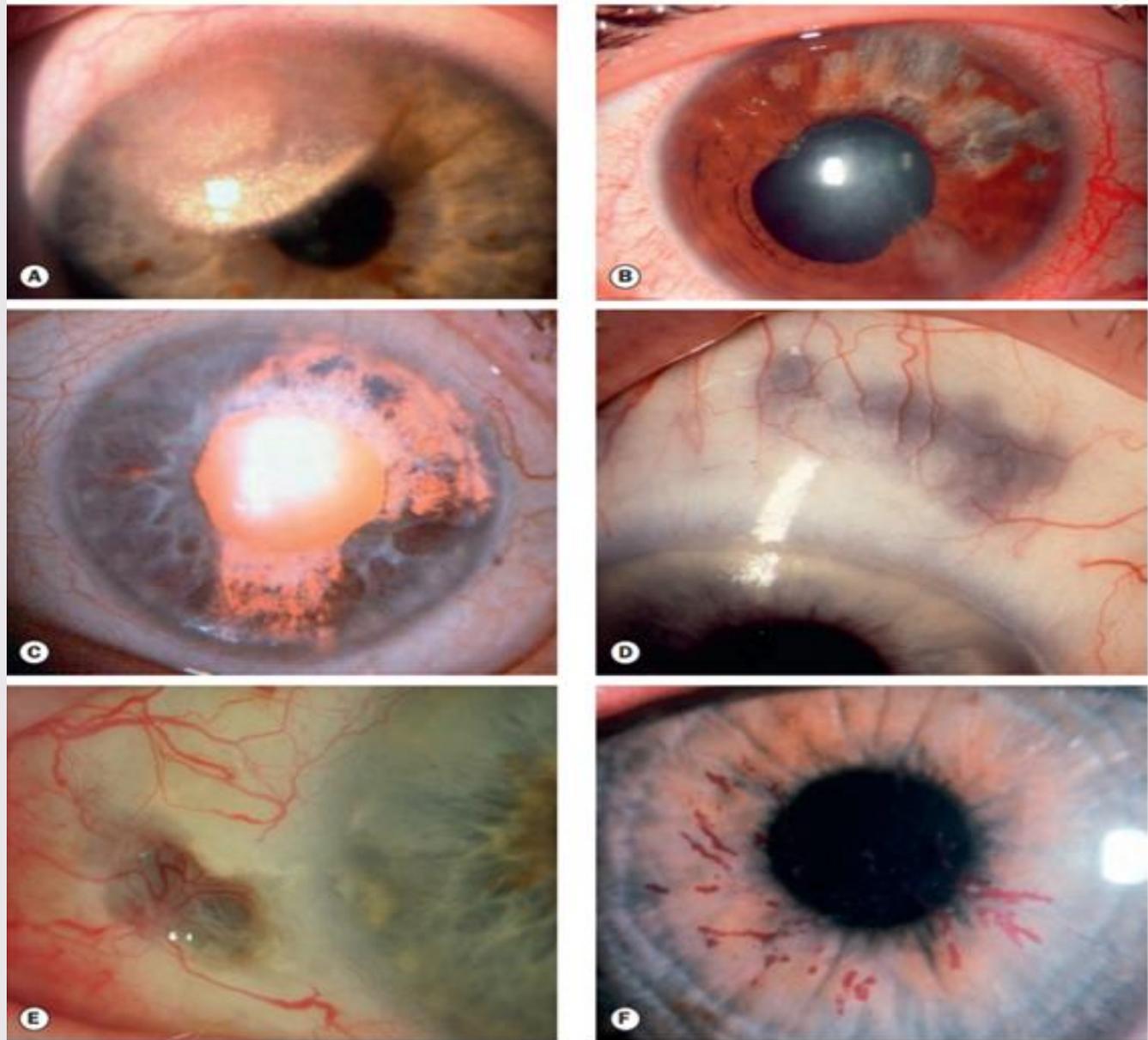


Fig. 6.19 Chronic lesions in herpes zoster ophthalmicus. (A) Scarring following stromal keratitis, with crystalline lipid degeneration; (B) iris atrophy in a typical sectoral pattern; (C) more severe sectoral iris atrophy on transillumination; (D) and (E) scleral atrophy; (F) mucous plaque keratitis



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- **Subconjunctival scarring** may occur.
- Eyelid scarring may result in **ptosis, cicatricial entropion and occasionally ectropion, trichiasis, lid notching and madarosis.**

Relapsing eye disease :

- In the relapsing phase lesions may reappear years after an acute episode, which may have been forgotten; eyelid scarring may be the only diagnostic clue.
- Reactivation of **keratitis, episcleritis, scleritis or iritis** can occur.



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Treatment

1. Systemic therapy :

- Oral antibiotic drugs – **Acyclovir** and **Valaciclovir**
- Analgesics – combination of **mephenamic acid** and **paracetamol**
- Systemic steroids
- Cimetidine
- Amitriptyline



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2. Local therapy for skin lesions :

- Antibiotic – corticosteroid skin ointment or lotions
- No calamine lotion

3. Local therapy for ocular lesions :

- Topical steroids
- Cycloplegics
- Topical acyclovir
- Topical antibiotics
- 0.5% timolol
- Lubricating artificial tear drops
- Bandage soft contact lens



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4. Surgical treatment :

- Lateral tarsorrhaphy
- Amniotic membrane transplantation
- Tissue adhesive with bandage contact lens
- Keratoplasty



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PROTOZOAL KERATITIS

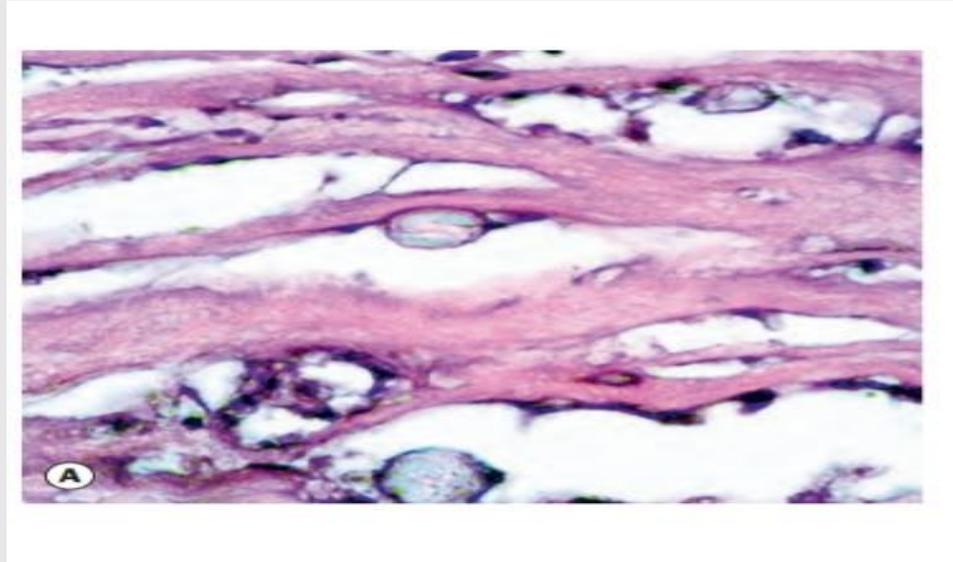
ACANTHAMOEBA KERATITIS :

- Acanthamoeba keratitis has recently gained importance because of its increasing incidence, difficulty in diagnosis and unsatisfactory treatment.
- Acanthamoeba spp. are ubiquitous free-living protozoa commonly found in soil, fresh or brackish water and the upper respiratory tract.
- The cystic form (Fig. 6.24A) is highly resilient. Under appropriate environmental conditions, the cysts turn into trophozoites, with tissue penetration and destruction.



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➤ In developed countries acanthamoeba keratitis is most frequently associated with contact lens wear, especially if tap water is used for rinsing.

Diagnosis :

Early misdiagnosis as herpes simplex keratitis is relatively common, and with more advanced signs the possibility of fungal keratitis should be remembered.



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Symptoms : Blurred vision and discomfort; pain is often severe and characteristically disproportionate to the clinical signs.

Signs :

- In early disease the epithelial surface is irregular and greyish (Fig. 6.24B).
- Epithelial **pseudodendrites** resembling herpetic lesions may form.
- **Limbitis** with diffuse or focal anterior stromal infiltrates (Fig. 6.24C).



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- Characteristic **perineural infiltrates** (radial keratoneuritis) are seen during the first few weeks and are virtually pathognomonic (Fig. 6.24D).
- Gradual enlargement and coalescence of infiltrates to form a ring abscess (Figs 6.25A and B) is typical.
- **Scleritis** may develop and is generally reactive rather than an extension of infection.
- Slowly progressive **stromal opacification and vascularization**.
- Corneal melting may occur at any stage when there is stromal disease. The melt often develops at the periphery of the area of infiltrate (Fig. 6.25C).



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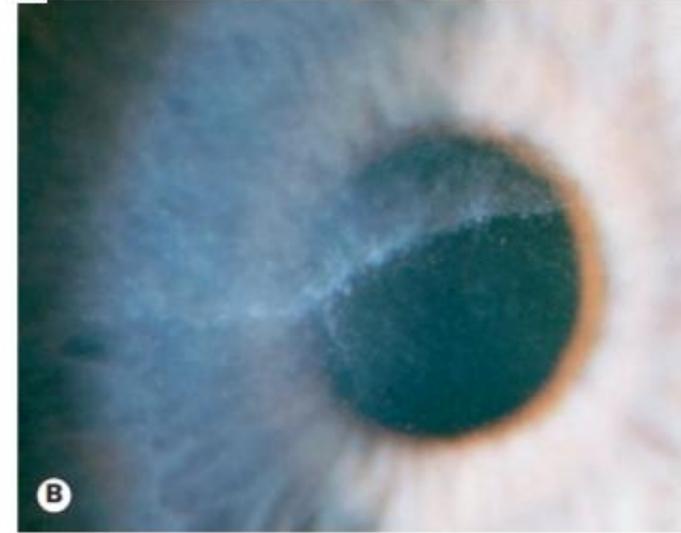
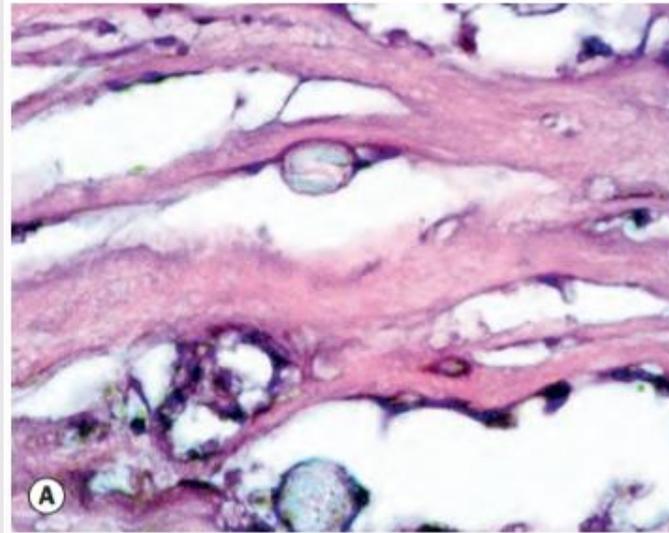


Fig. 6.24 Initial signs of acanthamoeba keratitis. (A) Cysts in a corneal biopsy; (B) greyish early epithelial involvement; (C) focal anterior stromal infiltrates; (D) radial perineuritis

(Courtesy of J Harry - fig. A)



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Investigations :

- **Staining of corneal scrapings** using periodic acid–Schiff or calcofluor white (a fluorescent dye with an affinity for amoebic cysts and fungi).
- Gram and Giemsa stains may also demonstrate cysts.
- Culture. Non-nutrient agar seeded with dead *E. coli*, which trophozoites consume.
- Other investigations include **immunohistochemistry, PCR and in vivo confocal microscopy.**
- Corneal biopsy may be necessary for diagnosis.



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Treatment :

It is usually unsatisfactory.

1. **Non-specific treatment**
2. **Specific medical treatment** includes (a) propamidine isethionate (Brolene) drops; (b) Neomycin drops; (c) Polyhexamethylene biguanide (d) chlorhexidine; (e) other drugs that may be useful are paromomycin and various topical and oral miconazole
3. **Penetrating keratoplasty** is frequently required in non-responsive cases.



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TROPHIC CORNEAL ULCERS

- ❖ Trophic corneal ulcers develop due to disturbance in metabolic activity of **epithelial cells**.
- ❖ This group includes:

(1) Neuroparalytic keratitis

(2) Exposure keratitis.



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NEUROPARALYTIC KERATITIS :

❖ **Neuroparalytic keratitis** occurs due to **paralysis of the sensory nerve supply of the cornea.**

Causes :

I. Congenital

1. Familial dysautonomia (Riley-Day syndrome)
2. Congenital insensitivity to Pain.
3. Anhidrotic ectodermal dysplasia.



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II. Acquired :

1. Following alcohol-block or electrocoagulation of **Gasserian ganglion** or section of the sensory root of **trigeminal nerve for trigeminal neuralgia.**
2. A **neoplasm** pressing on Gasserian ganglion.
3. **Gasserian ganglion destruction** due to acute infection in herpes zoster ophthalmicus.
4. **Acute infection of Gasserian ganglion** by herpes simplex virus.
5. **Syphilitic (luetetic) neuropathy.**
6. Involvement of **corneal nerves** in leprosy.
7. **Injury** to Gasserian ganglion.



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Pathogenesis :

- Exact pathogenesis is not clear; presumably, the disturbances in the **antidromic corneal reflex** occur due to fifth nerve paralysis.
- As a consequence metabolic activity of corneal epithelium is disturbed, leading to accumulation of metabolites; which in turn cause **oedema and exfoliation of epithelial cells** followed by ulceration.
- Corneal changes can occur in the presence of a normal blink reflex and normal lacrimal secretions.



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Fig. 5.12. in a patient with advanced acanthamoeba keratitis.

Ring infiltrate (A)



ring abscess (B)





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Clinical features :

1. Characteristic features are no pain, no lacrimation, and complete loss of corneal sensations.
2. Ciliary congestion is marked.
3. Corneal sheen is dull.
4. Initial corneal changes are in the form of **punctate epithelial erosions** in the inter-palpebral area followed by ulceration due to exfoliation of corneal epithelium.
5. Relapses are very common, even the healed scar quickly breaks down again.



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Treatment :

1. Initial treatment with **antibiotic and atropine eye ointment** with patching is tried. Healing is usually very slow.

✓ Recently described treatment modality include **topical nerve growth factor drops and amniotic membrane transplantation**.

2. If, however, relapses occur, it is best to perform **lateral tarsorrhaphy** which should be kept for at least one year.

✓ Along with it prolonged use of **artificial tears** is also recommended.



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EXPOSURE KERATITIS :

- ❖ Normally cornea is covered by eyelids during sleep and is constantly kept moist by **blinking movements** during awaking.
- ❖ When eyes are covered insufficiently by the lids and there **is loss of protective mechanism** of blinking the condition of exposure keratopathy (keratitis lagophthalmos) develops.



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Causes :

1. **Extreme proptosis** due to any cause will allow inadequate closure of lids.
2. **Bell's palsy** or any other cause of facial palsy.
3. **Ectropion** of severe degree .
4. **Symblepharon** causing lagophthalmos.
5. **Deep coma** associated with inadequate closure of lids.
6. Physiological **lagophthalmos**.



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Pathogenesis :

➤ Due to exposure the corneal epithelium dries up followed by **dessication**. After the epithelium is cast off, invasion by infective organisms may occur.

Clinical features :

- ❖ Initial dessication occurs in the **interpalpebral area** leading to **fine punctate epithelial keratitis** which is followed by **necrosis, frank ulceration and vascularization**.
- ❖ **Bacterial superinfection** may cause **deep suppurative ulceration** which may even perforate.



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Treatment :

1. Prophylaxis. Once lagophthalmos is diagnosed following measures should be taken to prevent exposure keratitis.

- Frequent instillation of **artificial tear eyedrops**.
- Instillation of ointment and closure of lids by a **tape or bandage during sleep**.
- **Soft bandage contact lens** with frequent instillation of artificial tears is required in cases of moderate exposure.

2. Treatment of corneal ulcer is on the general lines.

3. Tarsorrhaphy is invariably required when it is not possible to treat the cause or when recovery of the cause (e.g., facial palsy) is not anticipated.



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KERATITIS ASSOCIATED WITH DISEASES OF SKIN AND MUCOUS MEMBRANE :

ROSACEA KERATITIS :

➤ Corneal ulceration is seen in about 10 percent cases of **acne rosacea**, which is primarily a disease of the **sebaceous glands of the skin**.

Clinical features :

1. The condition typically occurs in elderly women in the form of facial eruptions presenting as **butterfly configuration, predominantly involving the malar and nasal area of face**.
2. Ocular lesions include chronic **blepharoconjunctivitis and keratitis**. Rosacea keratitis occurs as yellowish white marginal infiltrates, and small ulcers across the cornea and almost always become heavily vascularised.



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Treatment :

1. Local treatment. Rosacea keratitis responds to topical steroids, but recurrences are very common.

2. Systemic treatment. The essential and most effective treatment of rosacea keratitis is a long course of systemic **tetracycline** (250 mg QID × 3 weeks, TDS × 3 weeks, BID × 3 weeks, and once a day for 3 months).



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CORNEAL ULCER ASSOCIATED WITH SYSTEMIC COLLAGEN VASCULAR DISEASES :

- ❖ Peripheral corneal ulceration and/or melting of corneal tissue is not infrequent occurrence in patients suffering from systemic diseases such as **rheumatoid arthritis, systemic lupus erythematosus, polyarteritis nodosa and Wegener's granulomatosis.**
- ❖ Such corneal ulcers are usually indolent and difficult to treat.
- ❖ Systemic treatment of the primary disease may be beneficial.



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IDIOPATHIC CORNEAL ULCERS

MOOREN'S ULCER :

➤ The Mooren's ulcer (chronic serpiginous or rodent ulcer) is a severe **inflammatory peripheral ulcerative keratitis**.

Etiology :

1. It is an **idiopathic** degenerative condition.
2. It may be due to an **ischaemic necrosis** resulting from vasculitis of limbal vessels.
3. It may be due to the effects of enzyme **collagenase and proteoglyconase** produced from conjunctiva.
4. Most probably it is **an autoimmune disease**



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Clinical picture :

Two clinical varieties of Mooren's ulcer have been recognised.

1. Benign form which is usually unilateral, affects the elderly people and is characterised by a relative slow progress.

2. Virulent type also called the progressive form is bilateral, more often occurs in younger patients. The ulcer is rapidly progressive with a high incidence of scleral involvement.

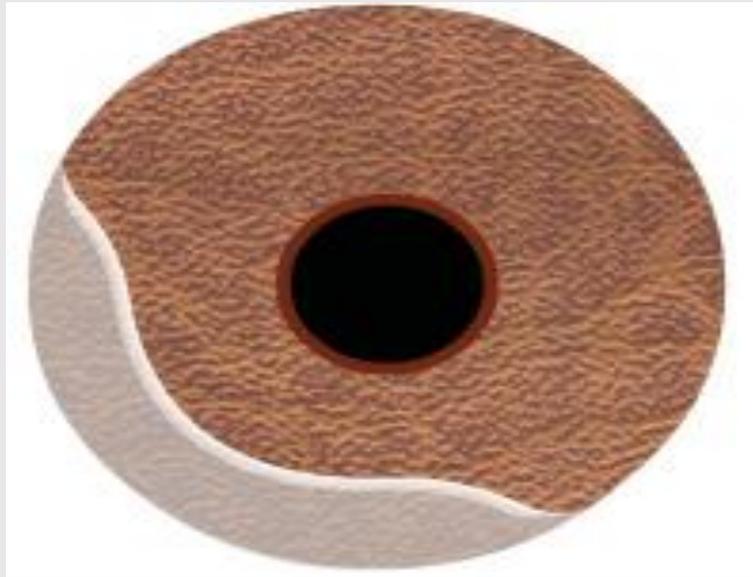
Symptoms: These include severe pain, photophobia, lacrimation and defective vision.

Signs : Features of Mooren's ulcer are shown in Fig. 5.13.



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Mooren's ulcer : A, diagrammatic depiction;
B, clinical photograph.



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- It is a superficial ulcer which starts at the corneal margin as patches of grey infiltrates which coalesce to form a shallow furrow over the whole cornea.
- The ulcer undermines the epithelium and superficial stromal lamellae at the advancing border, forming a characteristic whitish overhanging edge.
- Base of the ulcer soon becomes vascularized. The spread may be selflimiting or progressive.
- The ulcer rarely perforates and the sclera remains uninvolved.



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Treatment :

- 1. Topical corticosteroids** instilled every 2-3 hours are tried as initial therapy with limited success.
- 2. Immunosuppressive therapy** with systemic steroids may be of help. Immunosuppression with **cyclosporin** or other cytotoxic agents may be quite useful in virulent type of disease.
- 3. Soft contact lenses** have also been used with some relief in pain.
- 4. Lamellar or full thickness corneal grafts** often melt or vascularize.



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