Chapter

1

Embryology and Developmental Anomalies of Conjunctiva

Chapter Outline

EMBRYOLOGY OF CONJUNCTIVA

GENERAL CONSIDERATIONS

Eyelids are formed by reduplication of surface ectoderm above and below the cornea during 2nd month of gestation (Fig. 1.1). The folds enlarge and their margins meet and fuse with each other. The lids cut off a space called conjunctival sac.

DEVELOPMENT OF CONJUNCTIVA

Conjunctiva develops from the ectoderm lining of the lids and covering the globe (Fig. 1.1).

Conjunctival glands develop as growth of the basal cells of upper conjunctival fornix. Fewer glands develop from the lower fornix.

CONGENITAL DISORDERS OF CONJUNCTIVA

A few of the congenital disorders of conjunctiva worth mentioning include:

- Conjunctival choristoma
- Congenital pigmented lesions of conjunctiva
- Congenital epitarsus
- Conjunctival telangiectasia
- Cavernoma of conjunctiva
- Developmental anomalies of caruncle

CONJUNCTIVAL CHORISTOMA

Choristoma refers to benign tumour consisting of microscopically normal tissue derived from germ cell layers foreign to that body site. Conjunctival choristoma possess little growth potential and contain both dermal and epithelial elements that are not normally found in the conjunctiva.

Types of conjunctival choristomas

There are four types of conjunctival choristoma:

- Solid epibulbar dermoid
- Diffuse dermolipoma
- Complex choristomas, and
- Single-tissue choristoma

SOLID EPIBULBAR DERMOID

Demography

- *Frequency.* The estimated worldwide incidence of limbal dermoids ranges from 1 case per 10,000 population to 3 cases per 10,000 population.
- *Race.* No racial predisposition exists.
- *Sex.* Limbal dermoids occur with equal frequency in males and in females.
- *Age.* Limbal dermoids are present at birth but may not be recognized until the first or second decade of life. They may also appear to enlarge as the body matures.

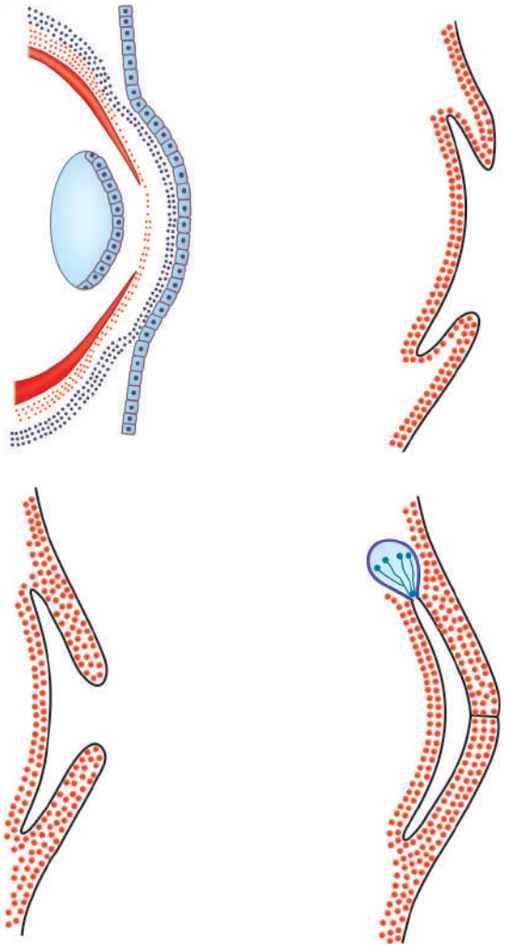

Fig. 1.1: Development of eyelids and conjunctiva.

- *Inheritance.* Limbal dermoids generally are not inherited, although some exceptions have been reported. Familial presentation of limbal dermoids in association with systemic disorders, such as Goldenhar syndrome, is well recognized and follows a multifactorial pattern of inheritance. Two rare forms of epibulbar dermoid (i.e. the annular limbal form, the corneal dystrophy form) presenting in multiple family members have been reported.

Pathophysiology

Several theories have been proposed to explain the development of limbal dermoids. Two important ones are:

1. *Theory of metaplastic transformation of the mesoblast.* According to this theory there occurs an early developmental error in between the rim of the optic nerve and the surface ectoderm.
2. *Theory of sequestration of the pluripotential cells* during embryonic development of the surrounding ocular structures, has also been suggested in pathophysiology.

Note. The exact pathogenesis probably varies from case to case.

Clinical features

Solid epibulbar dermoids are compact, pale yellow growths that typically occur unilaterally at the inferotemporal limbus (Fig. 1.2). Most limbal dermoids are superficial and only minimally involve the cornea and sclera. However, some tumours can penetrate deeply into the cornea, sclera, and conjunctiva.

Types. Based on the location, there are three broad categories of epibulbar dermoids:

- *First type of epibulbar dermoid is the limbal dermoid* which is the most common type, in which the tumour straddles the limbus. Limbal dermoids are usually superficial lesions but rarely may involve deeper ocular structures.
- *Second type of epibulbar dermoid* involves only the superficial cornea, sparing the limbus, the Descemet membrane, and the endothelium.
- *Third type of epibulbar dermoid* involves the entire anterior segment, replacing the cornea with a dermolipoma that may involve the iris, the ciliary body, and the lens.

Associations. Limbal dermoids may be associated with Goldenhar syndrome, linear naevus sebaceous syndrome, and encephalocraniocutaneous lipomatosis. Eyelid colobomas may also occur in association with limbal dermoids, which suggest the postulate that both anomalies may result from incomplete fusion of the lids with displacement of skin elements into the dermoid tumour.

Histological features

Histological examination reveals a thick, collagenous lesion that may contain hair, sweat glands, fat, sebaceous glands, or teeth (Fig. 1.3).

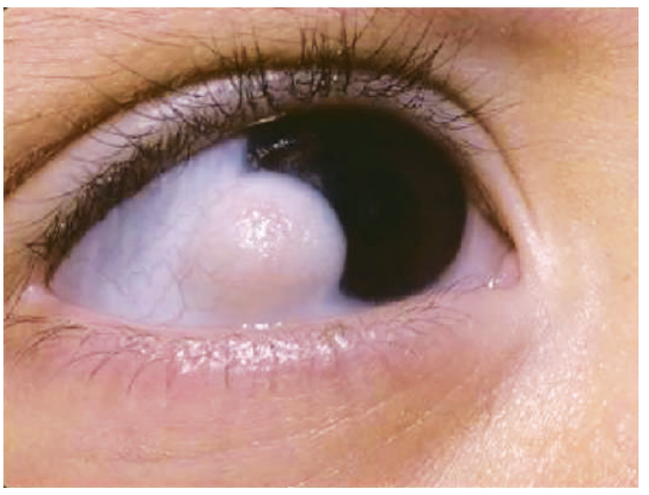

Fig. 1.2: Limbal dermoid.

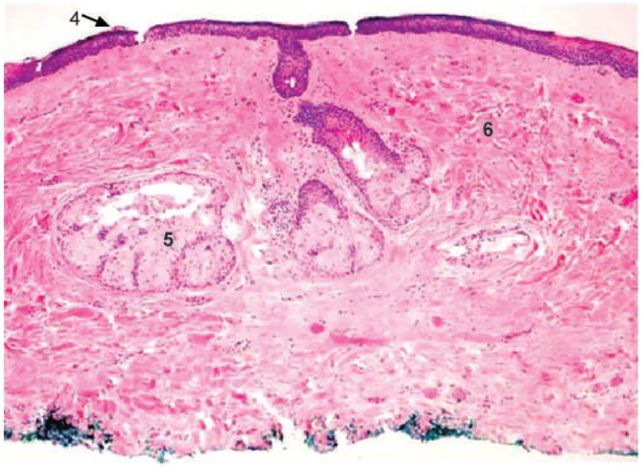

Fig. 1.3: Histological structure of limbal dermoid.

Treatment

Treatment of limbal dermoids may consist of:

Periodic removal of irritating cilia, and ***topical lubrication*** to prevent foreign body sensation.

Or

Excision of the dermoid if it is causing significant cosmetic disfigurement or interfering with vision. Surgical treatment should be instituted only when the risk of subsequent scar formation or surgical complications are outweighed by the likelihood of improving the patient's vision or cosmetic appearance.

Surgical excision usually involves:

- *A superficial sclerokeratectomy*, cutting flush with the surface of the globe (shaving the lesion off the cornea and sclera) is the procedure of choice for removal of the dermoid. Excised tissue always should be sent to the pathologist for examination.
- *Exposed sclera should be covered* by relaxing the adjacent conjunctiva and sewing it into the scleral defect.
- *Lamellar keratoplasty* can be performed to reinforce the site of excision, when a deep excision is necessary.
- *Amniotic membrane graft*. Large patches of bare sclera can be treated with application of single or multilayered amniotic membrane graft tissue. The amniotic membrane can be secured to underlying sclera using sutures and/or fibrin-glue adhesive.

DIFFUSE DERMOLIPOMA

Dermolipomas are less dense than solid epibulbar dermoids and contain more adipose tissue. These are true choristomas, because fatty tissue is usually not found anterior to the orbital septum. They are typically found on the superior temporal bulbar conjunctiva. These masses can extend from the limbus anteriorly to the posterior aspect of the globe and in the orbit between the superior and lateral rectus. It presents in adulthood and appears as soft, yellowish white, movable subconjunctival mass most frequently at outer canthus (Fig. 1.4). It consists of fatty tissue and the surrounding dermis-like connective tissue, hence the name lipodermoid.

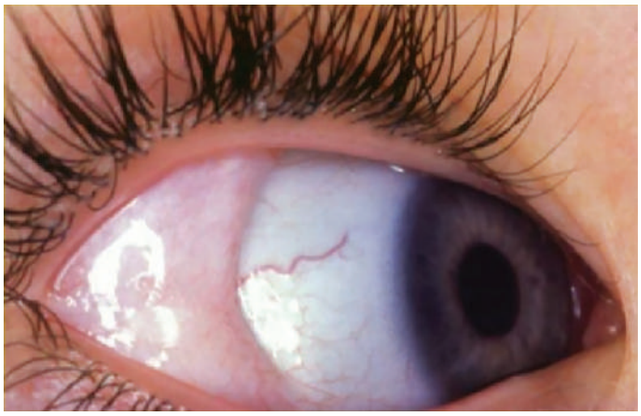

Fig. 1.4: Lipodermoid.

Treatment

Surgical excision is required only in the presence of significant cosmetic disfigurement. Usually, surgery is restricted to partial resection of the anterior portion of the tumour. Complete removal is usually not possible. Care must be taken during surgical removal not to damage the extraocular muscles, levator muscle, or lacrimal gland.

COMPLEX CHORISTOMAS

Complex choristomas consist of variable combinations of ectopic tissues such as cartilage, adipose tissue, smooth muscle, and acinar glands.

Clinical features

Clinically, these lesions resemble dermoids and lipodermoids. When acinar elements compose the majority of the tumour, complex choristomas may assume a fleshier, vascularized appearance with raised translucent nodules. These raised nodules have been referred to as ectopic lacrimal glands. Although mild growth may occur, especially during puberty, malignant transformation is rare.

Treatment

As these tumours also tend to invade deeply into the globe, excision is usually avoided. For cosmetic reasons, partial resection of the anterior portion of the tumour may be done.

SINGLE-TISSUE CHORISTOMA

Single-tissue choristomas include choristomas of lacrimal gland, respiratory tissue, and osseous choristomas.

Epibulbar osseous choristoma is the rarest type of choristoma of the eye. These are composed of firm deposits of bone and are most commonly found in the supero-

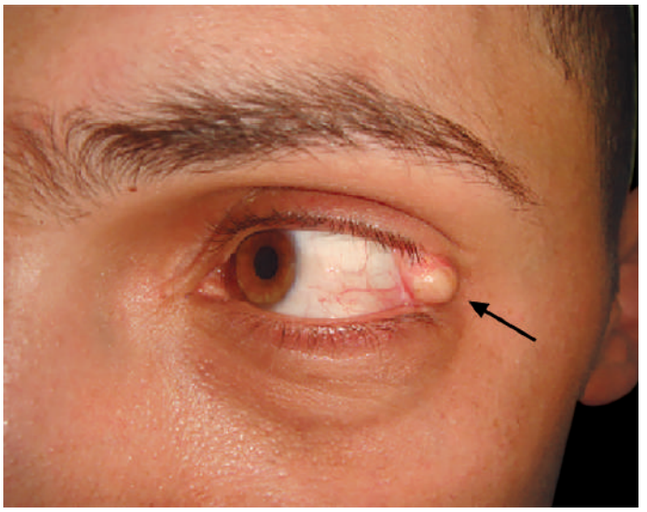

Fig. 1.5: Epibulbar osseous choristoma.

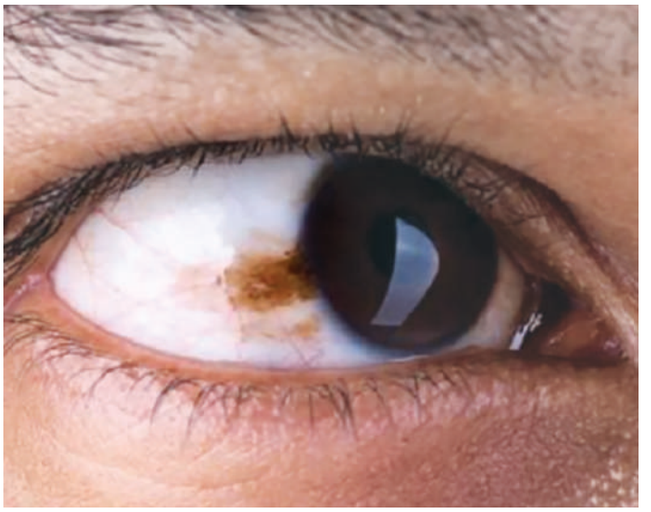

Fig. 1.6: Conjunctival naevus.

temporal conjunctiva (Fig. 1.5) occasionally, the mass is firmly attached to the sclera. As with all choristomas, osseous choristomas are believed to be congenital. Due to its location underneath the eyelid, the mass is usually detected once the child becomes old enough to palpate it. Osseous choristomas are solitary nodules that resemble dermoids. However, they can be differentiated from dermoids clinically because of their location about 5–10 mm posterior to the limbus and their more discrete borders.

Treatment. Usually, excision is performed for cosmetic reasons only.

CONGENITAL PIGMENTED LESIONS OF CONJUNCTIVA

Congenital melanocytic pigmented lesions of conjunctiva include:

- Conjunctival naevi
- Conjunctival epithelial melanosis
- Conjunctival subepithelial melanosis.

CONJUNCTIVAL NAEVI

Clinical features

Naevi or congenital moles are common pigmented lesions, usually presenting as grey gelatinous, brown or black, flat or slightly raised nodules on the bulbar conjunctiva, mostly near the limbus (Fig. 1.6). Melanocytic naevi are the most common tumours of the conjunctiva, accounting for 28% of all tumours. These lesions most commonly arise in the bulbar conjunctiva can also occur on caruncle, or plica semilunaris. They usually appear during early childhood and may increase in size at puberty or during pregnancy.

- Naevi present clinically as circumscribed, flat to slightly raised macules or papules.
- Naevi in children often lack pigmentation, but usually acquire pigmentation after puberty. However, up to 30% of naevi remain amelanotic.
- Naevi on the bulbar conjunctiva move freely over the sclera and appear well circumscribed without extension into the cornea. A common and characteristic feature of conjunctival naevi is the presence of intralesional cysts.
- Malignant melanoma will develop in less than 1% of conjunctival naevi. Clinical features particularly suggestive of evolving melanoma include extension into the cornea, attachment to the sclera, and development of multiple "feeder vessels" seen by slit-lamp examination. There are no specific clinical signs that can accurately predict malignant transformation in a conjunctival naevus.

Histopathology

Biopsy is indicated when a pigmented naevus shows clinical characteristics of possible malignancy such as rapid growth, change in shape and/or colour, recurrence after prior biopsy, and unusual location such as the palpebral conjunctiva or the fornix. Histologically, conjunctival melanocytic naevi are classified similarly as in the skin, including junctional, compound, and subepithelial naevi.

- *Junctional naevi.* About 5% of conjunctival naevi are junctional, characterized by nested but sometimes also lentiginous proliferations of type A or type B cells confined to the epithelium. They may show occasional mitotic activity. Most junctional naevi are found in patients in the younger age groups. Therefore, they are believed to be at an early stage in the evolution of compound naevi.

- *Compound naevi* are the most common type of conjunctival naevus, comprising about 70–78% of all naevi. A very characteristic and diagnostically useful feature of conjunctival naevi is induction of epithelial protrusions into the lamina propria and formation of intralesional epithelial cysts lined by conjunctival epithelium and goblet cells. These cysts are present in 50% of cases. It seems that cyst formation is a function of time, as they are less frequent in early lesions.
- *Subepithelial naevi* are the conjunctival counterpart of the dermal naevus and represent about 9% of all naevi. These are more prevalent in the older age groups and have predominantly type B or C nevomelanocytes in the substantia propria, without an intraepithelial component.

Treatment

Conjunctival naevi do not require treatment if clinically stable.

- *Excision or rebiopsy* is recommended in lesions that change in size or colour, recur, or show other clinical features of possible malignancy, or for cosmetic indications.
- Excision should be complete, whatever may be the indication.
- Reexcising of conjunctival naevi showing focal cytologic atypia is of no clinical benefit.

CONJUNCTIVAL EPITHELIAL MELANOSIS

Conjunctival epithelial melanosis (Fig. 1.7) develops in early childhood, and then remains stationary. It is found in 90% of the blacks. The pigmented spot freely moves with the movement of conjunctiva. It has got no malignant potential and hence no treatment is required.

CONJUNCTIVAL SUBEPITHELIAL MELANOSIS

Subepithelial melanosis may occur as:

- An isolated anomaly of conjunctiva (congenital melanosis oculi, Fig. 1.8) or

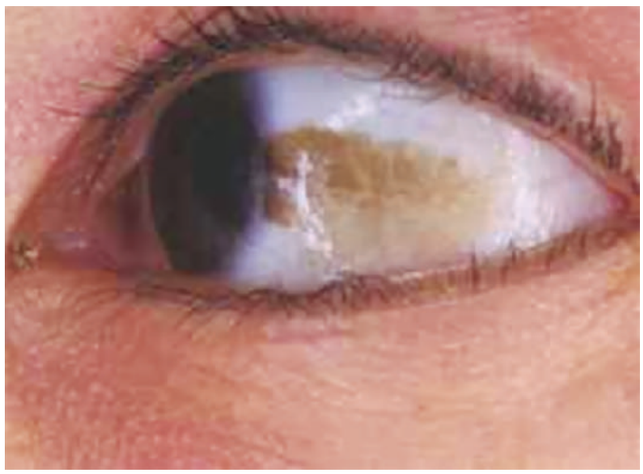

Fig. 1.7: Conjunctival epithelial melanosis.

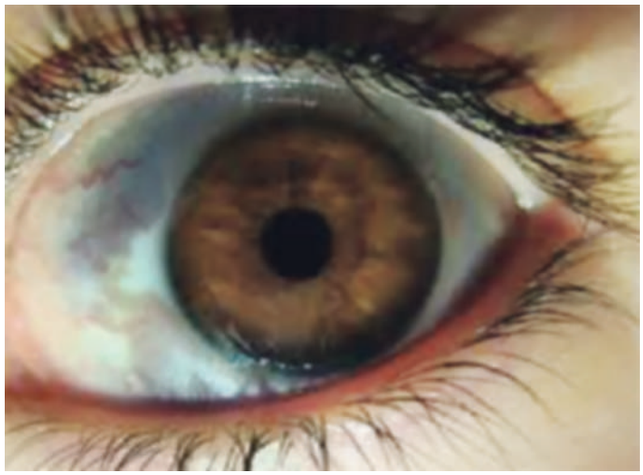

Fig. 1.8: Congenital melanosis oculi.

- In association with the ipsilateral hyperpigmentation of the face (oculodermal melanosis or naevus of Ota).

NAEVUS OF OTA

Melanosis bulbi associated with ipsilateral hyperpigmentation of the face, is also known as naevus of Ota or oculomucodermal melanocytosis. This condition was first described by Dr Ota, a Japanese physician, in 1939, and hence the term naevus of Ota. Naevus of Ota should not be confused with Mongolian spots. Unlike Mongolian spots, naevus of Ota does not disappear with time.

Demography

- *Age.* Naevus of Ota is a skin condition that is normally present at birth, but can occur during adolescence too.
- *Sex.* Both males and females are affected, but females are affected much more than males in a 5:1 ratio.
- *Race.* All racial and ethnic groups are at risk, though naevus of Ota is more frequent among the Japanese population and other Asian races compared to Europeans, Americans, or Africans.

Etiology

- Exact cause of naevus of Ota formation is not known.
- Some researchers believe that it may be formed due to abnormal accumulation of melanocytes (cells producing melanin) in the fetal development stage.
- Even though a congenital presentation is noted, but naevus of Ota is not a hereditary condition.

Clinical features

Naevus of Ota may not present any major signs and symptoms in most cases. The general features of the skin condition include:

- *Skin pigmentation*. It is a benign skin lesion that occurs as a hyperpigmented skin patch. The skin patch may be bluish to bluish-brown in colour.

- *Head and neck region* is mostly affected, especially the face; either one side, or both sides of the face may be involved.
- *Ocular melanosis* is seen in two-thirds of the cases, the sclera of the eye is affected. The condition may be unilateral or bilateral, meaning that either one eye or both eyes may be affected.

Complications

Complications from naevus of Ota may include:

- *Cosmetic concerns* and stress may occur in some cases.
- *Glaucoma* risk is higher if along with skin lesion is present in the eye(s).
- *Malignant melanoma* is known to develop rarely from the site of the lesion, and hence, close follow-up is important and necessary.

Treatment measures for naevus of Ota include:

- *No treatment* is generally required in mild cases. A regularly observation is all that may be required, i.e. a "wait and watch" approach may be followed.
- *Laser surgery* is found to be beneficial in case of cosmetic reasons.
- *Surgical excision.* Naevus of Ota can also be excised through electrocautery surgical procedure.

Prognosis of naevus of Ota is excellent even if no treatment is provided and only periodic observation maintained, since typically it is a benign skin condition.

EPITARSUS

Epitarsus is a peculiar condition which typically occurs as an apron-like fold of conjunctiva attached to the inner surface of the upper lid but occasionally as a bridge of tissue under which a probe may be passed.

Types: Etiologically epitarsus is of two types:

1. *Primary epitarsus*, occurring purely as a congenital anomaly; and
2. *Secondary epitarsus*, following neglected cases of conjunctivitis.

Congenital epitarsus

Epitarsus occurring as a congenital anomaly is rare. The deformity is almost invariably seen in the upper lid, though its bilateral occurrence in the lower lids has also been reported.

Four clinical varieties of epitarsus depending on the extent of the deformity reported are:

- Intrafornix
- Fornix-tarsal
- Fornix-limbal, and
- Interfornix (Fig. 1.9A)

Histopathological examination following resection of the fold shows moderately dense fibrovascular connective tissue covered by stratified squamous epithelium on both the sides (Fig. 1.10).

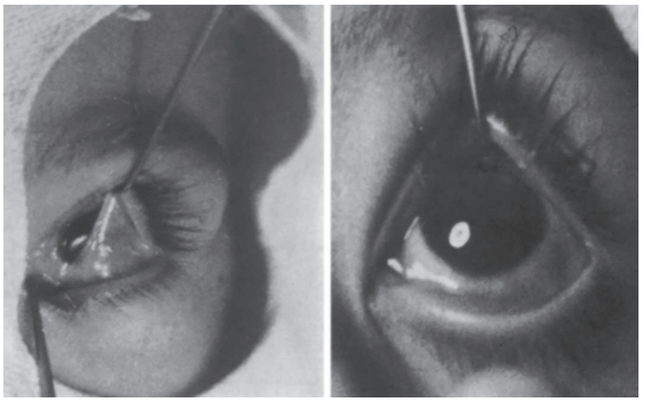

Fig. 1.9: A, Interfornix epitarsus—the membrane extending from upper fornix to lower fornix; B, After excision of the membrane, underlying eyeball is normal.

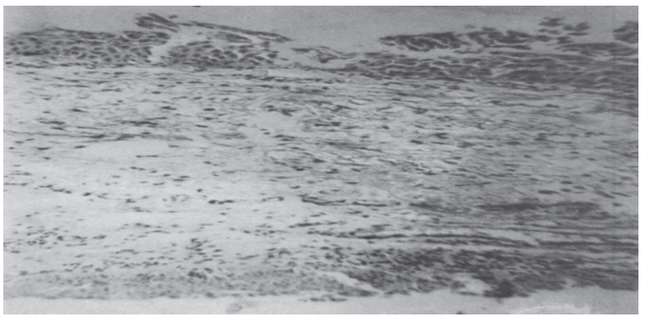

Fig. 1.10: The mucosa on either side comprises stratified squamous epithelium. Subepithelial soft tissue contains few mononuclear cells. (Haematoxylin and eosin ×120).

Treatment. Simple excision gives good cosmetic and functional results (Fig. 1.9B).

CONJUNCTIVAL TELANGIECTASIA

Conjunctival telangiectasia refers to abnormal, dilated conjunctival capillary formation, which usually develop between 3 and 5 years of age.

Clinical features

Conjunctival telangiectasia appears as dot-like, corkscrew, irregular vessels near the limbus (Fig. 1.11).

- Subconjunctival haemorrhage may occur from the telangiectatic vessels.
- Patients usually have no symptoms except the asymptomatic red spots on eye.

Associations include epistaxis and gastrointestinal bleeding.

Evaluation should include:

- *Complete ophthalmic history and eye examination* with attention to conjunctiva, cornea, lens, and ophthalmoscopy.
- *CT scan* may need to be considered for multisystem disorders.
- *Medical consultation* to rule out systemic disease.

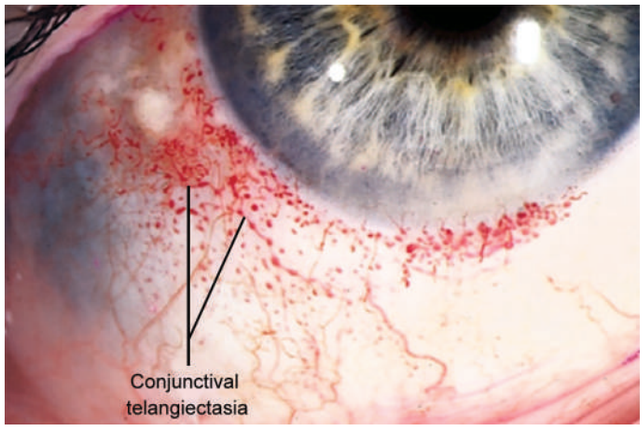

Fig. 1.11: Conjunctival telangiectasia appearing as dot-like, corkscrew, irregular vessels near the limbus.

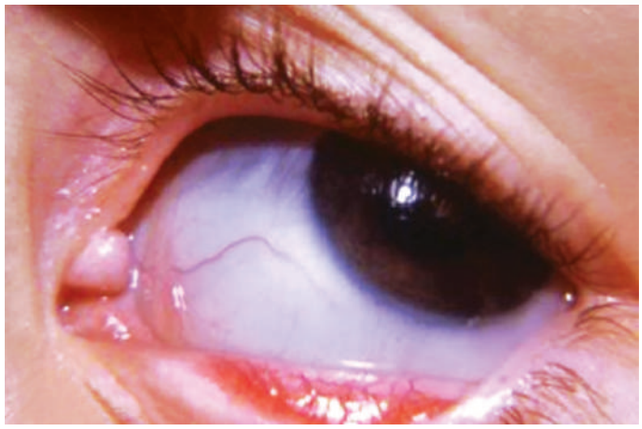

Fig. 1.12: Congenital bifurcated caruncle.

Differential diagnosis

Differential diagnosis consists of an idiopathic lesion, Osler-Weber-Rendu syndrome, ataxia-telangiectasia, Fabry's disease, and Sturge-Weber syndrome.

Management. No treatment is recommended.

Prognosis. Usually benign; may bleed; depends on etiology.

CONGENITAL ANOMALIES OF CARUNCLE

CONGENITAL BIFURCATED CARUNCLE

It is a rare anomaly which can be seen in the presence of normal plica semilunaris (Fig. 1.12).

OTHER CONGENITAL ANOMALIES OF CARUNCLE

Congenital anomalies of caruncle are in general rare. The rare case reports in the literature are on:

- *Dysplastic caruncle*, which may occur isolated or as part of Goldenhar syndrome.
- *Ectopic caruncle*
- *Congenital megacaruncle*
- *Supernumerary caruncles* are always unilateral and un-associated with other ocular abnormalities or Goldenhar syndrome.
- Caruncular dermoid has also been reported in the literature. Histopathology of caruncular dermoid shows a keratinizing epidermis-like surface and dense, thick collagen in place of substantia propria.

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Chapter

2

Conjunctivitis

Chapter Outline

DEFINITION AND CLASSIFICATION

DEFINITION

Inflammation of the conjunctiva (conjunctivitis) is classically defined as conjunctival hyperaemia associated with a discharge which may be watery, mucoid, mucopurulent or purulent.

CLASSIFICATION

Etiologically conjunctivitis can be classified as below:

A. Infective conjunctivitis

1. Bacterial conjunctivitis
- Acute bacterial conjunctivitis
- Hyperacute bacterial conjunctivitis
- Chronic bacterial conjunctivitis
- Angular bacterial conjunctivitis

Chlamydial conjunctivitis
- Trachoma
- Adult inclusion conjunctivitis
- Neonatal chlamydial conjunctivitis

2. Viral conjunctivitis
- Adenovirus conjunctivitis
  - Epidemic keratoconjunctivitis
  - Pharyngoconjunctival fever
- Enterovirus conjunctivitis
- Molluscum contagiosum conjunctivitis
- Herpes simplex conjunctivitis

3. Ophthalmia neonatorum (a separate entity)

4. Granulomatous conjunctivitis
- Parinaud oculoglandular syndrome.

B. Allergic conjunctivitis

1. Simple allergic conjunctivitis
- Hay fever conjunctivitis (rhinoconjunctivitis)
- Seasonal allergic conjunctivitis (SAC)
- Perennial allergic conjunctivitis (PAC)

2. Vernal keratoconjunctivitis (VKC)

3. Atopic keratoconjunctivitis (AKC)

4. Giant papillary conjunctivitis (GPC)

5. Phlyctenular conjunctivitis (PKC)

6. Contact dermoconjunctivitis (drop conjunctivitis).

C. Cicatricial conjunctivitis

- Ocular mucous membrane pemphigoid (OMMP),
- Stevens-Johnson syndrome (SJS),
- Toxic epidermal necrolysis (TeN), and
- Secondary cicatricial conjunctivitis.

D. Toxic conjunctivitis

- Toxic conjunctivitis secondary to molluscum contagiosum
- Chemical toxic conjunctivitis

INFECTIVE CONJUNCTIVITIS

Infective conjunctivitis, i.e. inflammation of the conjunctiva caused by micro-organisms is the commonest variety. This is in spite of the fact that the conjunctiva has been provided with *natural protective mechanisms* in the form of:

- Low temperature due to exposure to air,
- Physical protection by lids,
- Flushing action of tears,
- Antibacterial activity of lysozymes, and
- Humoral protection by the tear immunoglobulins.

BACTERIAL CONJUNCTIVITIS

There has occurred a relative decrease in the incidence of bacterial conjunctivitis in general and those caused by gonococcus and *Corynebacterium diphtheriae* in particular. However, in developing countries, it still continues to be the commonest type of conjunctivitis. It can occur as sporadic and epidemic cases. Outbreaks of bacterial conjunctivitis, epidemics are quite frequent during monsoon season.

Etiology

A. Predisposing factors for bacterial conjunctivitis, especially epidemic forms, are flies, poor hygienic conditions, hot dry climate, poor sanitation and dirty habits. These factors help the infection to establish, as the disease is highly contagious.

B. Causative organisms. It may be caused by a wide range of organisms in the following approximate order of frequency:

- *Staphylococcus aureus* is the most common cause of bacterial conjunctivitis and blepharoconjunctivitis.
- *Staphylococcus epidermidis* is an innocuous flora of lid and conjunctiva. It can also produce blepharoconjunctivitis.
- *Streptococcus pneumoniae* (Pneumococcus) produces acute conjunctivitis usually associated with petechial subconjunctival haemorrhages. The disease has a self-limiting course of 9–10 days.
- *Streptococcus pyogenes* (haemolyticus) is virulent and usually produces pseudomembranous conjunctivitis.
- *Haemophilus influenzae* (aegyptius, Koch-Weeks bacillus). It classically causes epidemics of mucopurulent conjunctivitis, known as 'red-eye' especially in semitropical countries.
- *Moraxella lacunata* (Morax-Axenfeld bacillus) is most common cause of angular conjunctivitis and angular blepharoconjunctivitis.
- *Pseudomonas pyocyanea* is a virulent organism, which readily invades the cornea.
- *Neisseria gonorrhoeae* typically produces acute purulent conjunctivitis in adults and ophthalmia neonatorum in newborn. It is capable of invading intact corneal epithelium.
- *Neisseria meningitidis* (Meningococcus) may produce mucopurulent conjunctivitis.
- *Corynebacterium diphtheriae* causes acute membranous conjunctivitis. Such infections are not known nowadays.

C. Mode of infection. Conjunctiva may get infected from three sources, viz. exogenous, local surrounding structures and endogenous, by following modes:

1. *Exogenous infections* are the commonest and may spread:
   - *Directly* through close contact, as airborne infections or as waterborne infections.
   - *Vector transmission* (e.g. flies)
   - *Material transfer* such as infected fingers of doctors, nurses, common towels, handkerchiefs, and infected tonometers.
2. *Local spread* may occur sometimes from neighbouring structures such as infected lacrimal sac, lids, and nasopharynx. In addition to these, a change in the character of relatively innocuous organisms present in the conjunctival sac itself may cause infections.
3. *Endogenous infections* may occur very rarely through blood, e.g. gonococcal and meningococcal infections.

PATHOLOGY

Pathological changes of bacterial conjunctivitis consist of:

1. ***Vascular response.*** It is characterised by congestion and increased permeability of the conjunctival vessels associated with proliferation of capillaries.
2. ***Cellular response.*** It is in the form of exudation of polymorphonuclear cells and other inflammatory cells into the substantia propria of conjunctiva as well as in the conjunctival sac.
3. ***Conjunctival tissue response.*** Conjunctiva becomes oedematous. The superficial epithelial cells degenerate, become loose and even desquamate. There occurs proliferation of basal layers of conjunctival epithelium and increase in the number of mucin-secreting goblet cells.
4. ***Conjunctival discharge.*** It consists of tears, mucus, inflammatory cells, desquamated epithelial cells, fibrin and bacteria. If the inflammation is very severe, diapedesis of red blood cells may occur and discharge may become blood stained.

Severity of pathological changes varies depending upon the severity of inflammation and the causative organism. The changes are thus more marked in purulent conjunctivitis than mucopurulent conjunctivitis.

CLINICAL TYPES OF BACTERIAL CONJUNCTIVITIS

Depending upon the causative bacteria and the severity of infection, bacterial conjunctivitis may present in following clinical forms:

- Acute bacterial conjunctivitis,
- Hyperacute bacterial conjunctivitis,
- Chronic bacterial conjunctivitis, and
- Angular bacterial conjunctivitis.

ACUTE BACTERIAL CONJUNCTIVITIS

Acute bacterial conjunctivitis is characterised by marked conjunctival hyperaemia and mucopurulent discharge from the eye. So, clinically, it is called acute mucopurulent conjunctivitis. It is the most common type of bacterial conjunctivitis.

Common causative bacteria are: *Staphylococcus aureus*, Koch-Weeks bacillus, *Streptococcus pneumoniae*, *Haemophilus influenzae* (Table 2.1). Mucopurulent conjunctivitis generally accompanies exanthemata such as measles and scarlet fever.

CLINICAL FEATURES

Symptoms

- *Discomfort, foreign body, grittiness, blurring and redness* of sudden onset (due to engorgement of vessels) are the usual presenting symptoms.
- *Mild to moderate pain* is often experienced by the patients.
- *Mild photophobia*, i.e. difficulty to tolerate light.
- *Mucopurulent discharge* from the eyes.
- *Sticking together of lid margins* with discharge during sleep.
- *Slight blurring* of vision due to mucous flakes in front of cornea.
- *Coloured halos*, may be complained by some patients due to prismatic effect of mucus present on cornea.

Signs (Fig. 2.1)

- *Flakes of mucopus* seen in the fornices, canthi and lid margins is a critical sign.
- *Conjunctival congestion*, which is more marked in palpebral conjunctiva, fornices and peripheral part of bulbar conjunctiva, giving the appearance of 'fiery red eye'. The congestion is typically less marked in circumcorneal zone.

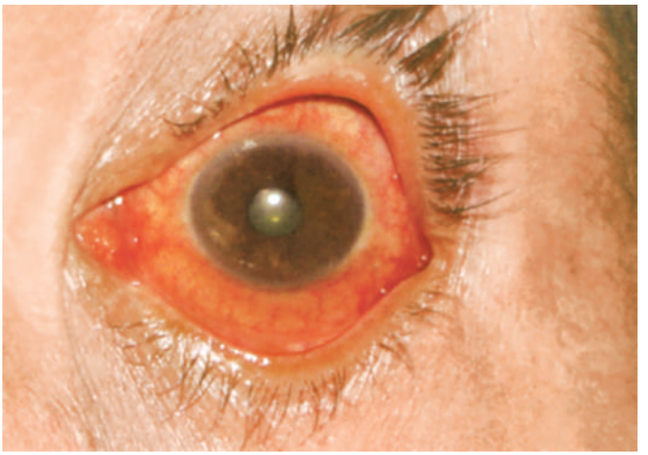

Fig. 2.1: Signs of acute mucopurulent conjunctivitis.

- *Chemosis*, i.e. swelling of conjunctiva.
- *Papillae* of fine type may be seen.
- *Petechial haemorrhages* are seen when the causative organism is *Streptococcus pneumoniae*.
- *Cilia are usually matted* together with yellow crusts.
- *Eyelids* may be slightly oedematous.

Clinical course

Acute mucopurulent conjunctivitis is usually bilateral, although one eye may become affected 1–2 days before the other. The disease usually reaches its height in three to four days. If untreated, in mild cases the infection may be overcome and the condition is cured in 10–15 days; or it may pass to less intense form, the 'chronic catarrhal conjunctivitis'.

Complications

Occasionally, the disease may be complicated by superficial punctate corneal epitheliopathy, marginal corneal ulceration, superficial keratitis, blepharitis, or dacryocystitis.

DIFFERENTIAL DIAGNOSIS

1. *From other causes of acute red eye* (Table 2.2).
2. *From other types of conjunctivitis.* It is made out from the typical clinical feature of disease and is confirmed by conjunctival cytology and bacteriological examination of secretions and scrapings (Table 2.3).

TABLE 2.1: Common pathogens causing acute, hyperacute and chronic bacterial conjunctivitis

Acute conjunctivitis	*Hyperacute conjunctivitis*	*Chronic conjunctivitis*
Staphylococcus aureus	*Neisseria gonorrhoeae*	*Staphylococcus aureus*
Streptococcus pneumoniae	*Neisseria meningitidis*	*Moraxella lacunata*
Koch-Weeks bacillus		*Proteus mirabilis*
Haemophilus influenzae		*Klebsiella pneumoniae*
		Escherichia coli

TABLE 2.2: Distinguishing features between acute conjunctitis, acute iridocyclitis and acute congestive glaucoma

Features	*Acute conjunctivitis*	*Acute iridocyclitis*	*Acute congestive glaucoma*
1. Onset	Gradual	Usually gradual	Sudden
2. Pain	Mild discomfort	Moderate in eye and along the first division of trigeminal nerve	Severe in eye and the entire trigeminal area
3. Discharge	Mucopurulent	Watery	Watery
4. Coloured halos	May be present	Absent	Present
5. Vision	Good	Slightly impaired	Markedly impaired
6. Congestion	Superficial con junctival	Deep ciliary	Deep ciliary
7. Tenderness	Absent	Marked	Marked
8. Pupil	Normal	Small and irregular	Large and vertically oval
9. Media	Clear	Hazy due to KPs, aqueous flare and pupillary exudates	Hazy due to oedematous cornea
10. Anterior chamber	Normal	May be deep	Very shallow
11. Iris	Normal	Muddy	Oedematous
12. Intraocular pressure	Normal	Usually normal	Raised
13. Constitutional symptoms	Absent	Little	Prostration and vomiting

TABLE 2.3: Differentiating features of common types of conjunctivitis

	Bacterial	*Viral*	*Allergic*	*Chlamydial (TRIC)*
(A) Clinical signs				
1. Congestion	Marked	Moderate	Mild to moderate	Moderate
2. Chemosis	++	±	++	±
3. Subconjunctival haemorrhages	±	±	–	–
4. Discharge	Purulent or mucopurulent	Watery	Ropy/watery	Mucopurulent
5. Papillae	±	–	++	±
6. Follicles	–	+	–	++
7. Pseudomembrane	±	±	–	–
8. Pannus	–	–	– (Except vernal)	+
9. Preauricular lymph nodes	+	++	–	±
(B) Cytological features				
1. Neutrophils	+	+ (Early)	–	+
2. Eosinophils	–	–	+	–
3. Lymphocytes	–	+	–	+
4. Plasma cells	–	–	–	+
5. Multinuclear cells	–	+	–	–
6. Inclusion bodies:				
Cytoplasmic	–	+ (Pox)	–	+
Nuclear	–	+ (Herpes)	–	–
7. Micro-organisms	+	–	–	–

TREATMENT

1. Topical antibiotics to control the infection constitute the main treatment of acute bacterial conjunctivitis. Ideally, the antibiotic should be selected after culture and sensitivity tests but in practice, it is difficult. However, in routine, most of the patients respond well to broad spectrum antibiotics. Therefore, treatment may be started with chloramphenicol (1%), or gentamicin (0.3%), or tobramycin 0.3% or framycetin 0.3% eye drops 3–4 hourly in day and ointment used at night will not only provide antibiotic cover but also help to reduce the early morning stickiness. If the patient does not respond to these antibiotics, then the quinolone antibiotic drops such as ciprofloxacin (0.3%), ofloxacin (0.3%), gatifloxacin (0.3%) or moxifloxacin (0.5%) may be used.

2. Irrigation of conjunctival sac with sterile lukewarm saline once or twice a day will help by removing the deleterious material. Frequent eyewash (as advocated earlier) is, however, contraindicated as it will wash away the lysozyme and other protective proteins present in the tears.

3. Dark goggles should be used to prevent photophobia.

4. No bandage should be applied in patients with mucopurulent conjunctivitis. Exposure to air keeps the temperature of conjunctival cul-de-sac low which inhibits the bacterial growth; while after bandaging, conjunctival sac is converted into an incubator, and thus infection flares to a severe degree within 24 hours. Further, bandaging of eye will also prevent the escape of discharge.

5. No steroids should be applied, otherwise infection will flare up and bacterial corneal ulcer may develop.

6. Anti-inflammatory and analgesic drugs (e.g. ibuprofen and paracetamol) may be given orally for 2–3 days to provide symptomatic relief from mild pain especially in sensitive patients.

Preventive measures to reduce risk of transmission to the close contacts

- Frequent handwashing, and
- Avoidance of sharing towel, handkerchief and pillow with others.

HYPERACUTE BACTERIAL CONJUNCTIVITIS

Hyperacute bacterial conjunctivitis also known as acute purulent conjunctivitis or *acute blennorrhoea* is characterised by a violent inflammatory response.

It occurs in two forms:

1. Adult purulent conjunctivitis
2. Ophthalmia neonatorum in newborn (*see* page 27).

HYPERACUTE CONJUNCTIVITIS OF ADULTS (GONOCOCCAL CONJUNCTIVITIS)

ETIOLOGY

The disease affects adults, predominantly males.

- *Gonococcal infection* directly spreads from genitals to eye. Presently, incidence of gonococcal conjunctivitis has markedly decreased.
- *Other pathogen* causing hyperacute conjunctivitis is *Neisseria meningitidis* (Table 2.1).

CLINICAL FEATURES

Gonococcal conjunctivitis

Onset is hyperacute (12 to 24 hours).

Symptoms include:

- *Pain* which is moderate to severe.
- *Purulent discharge*, which is usually copious.
- *Swelling of eyelids*, which is usually marked.
- *Mild photophobia*, i.e. difficulty to tolerate light.
- *Sticking together of lid margins* with discharge during sleep.
- *Slight blurring* of vision due to mucous flakes in front of cornea.

Signs are as follows (Fig. 2.2):

- *Eyelids* are tense and swollen.
- *Tenderness* is marked.
- *Discharge* is thick purulent, copious trickling down the cheeks.
- *Conjunctiva shows* marked chemosis, congestion and papillae, giving bright red velvety appearance. Frequently, a pseudomembrane may be seen on the conjunctival surface (Fig. 2.3).
- *Preauricular lymph nodes* are usually enlarged and tender.

Associations. Gonococcal conjunctivitis is usually associated with urethritis and arthritis.

COMPLICATIONS

1. *Corneal involvement* is quite frequent as the gonococcus can invade the normal cornea through an intact epithelium. It may occur in the form of diffuse haze and oedema, central necrosis, corneal ulceration or even perforation.

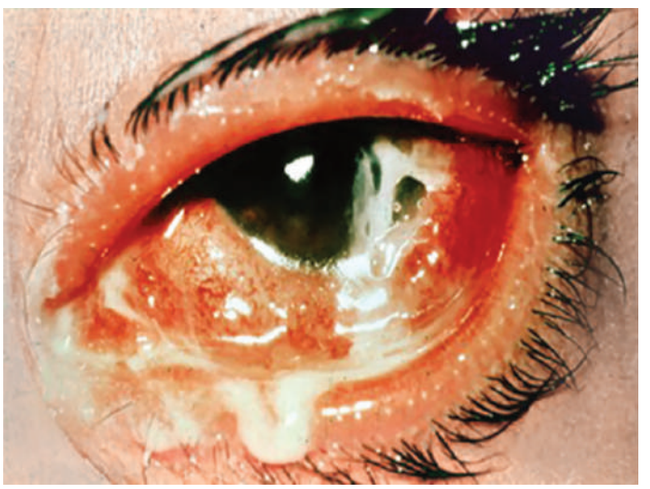

Fig. 2.2: Hyperacute conjunctivitis.

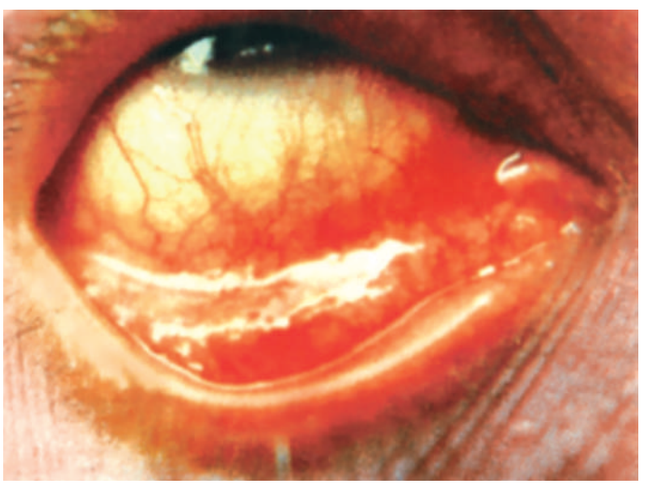

Fig. 2.3: Pseudomembranous conjunctivitis.

2. *Iridocyclitis* may also occur, but is not as common as corneal involvement.
3. *Systemic complications*, though rare, include gonorrhoea arthritis, endocarditis and septicaemia.

TREATMENT

1. Systemic therapy is far more critical than the topical therapy for the infections caused by *N. gonorrhoeae*. Any of the following regimes can be adopted:

- *Third generation cephalosporin* such as cefoxitin 1.0 g or cefotaxime 500 mg IV qid or ceftriaxone 1.0 g IM qid, all for 5 days; should be preferred treatment.
- *Quinolones* such as norfloxacin 1.2 g orally qid for 5 days, or
- *Spectinomycin* 2.0 g IM for 3 days, may be used alternatively.

All of the above regimes should then be followed by a one week course of either doxycycline 100 mg bid or erythromycin 250–500 mg orally qid.

2. Topical antibiotic therapy, presently recommended includes ofloxacin, ciprofloxacin or tobramycin eye drops or bacitracin or erythromycin eye ointment every 2 hours for the first 2–3 days and then 5 times daily for 7 days.

3. Irrigation of the eyes frequently with sterile saline is very therapeutic in washing away infected debris.

4. Other general measures are similar to acute mucopurulent conjunctivitis.

5. Topical atropine 1% eye drops should be instilled once or twice a day if cornea is involved.

Note. Sexual partner should also be treated with systemic antibiotics. Further, both the patient and the sexual partner should be referred for evaluation of other sexually transmitted diseases.

Meningococcal conjunctivitis

Hyperacute conjunctivitis caused by *Neisseria meningitidis* is comparatively milder than gonococcal conjunctivitis. It may be of two types: Primary and secondary.

Primary meningococcal conjunctivitis is extremely rare in adults and can be:

- *Invasive disease*, which is followed by systemic meningococcal disease.
- *Non-invasive disease*, i.e. isolated conjunctival infection.

Treatment of meningococcal conjunctivitis

- *Topical treatment* in similar to gonococcal conjunctivitis.
- *Systemic treatment* includes intravenous penicillin or intravenous cefotaxime or ceftriaxone (in penicillin-resistant cases).

Note. *Close contacts* of invasive meningococcal conjunctivitis should receive prophylaxis with a single dose of ciprofloxacin 500 mg or rifampin 600 mg twice daily for two days.

CHRONIC BACTERIAL CONJUNCTIVITIS

Chronic bacterial conjunctivitis also known as *'Chronic catarrhal conjunctivitis'* or *'simple chronic conjunctivitis'* is characterised by mild catarrhal inflammation of the conjunctiva. Chronic bacterial conjunctivitis lasts more than 3 weeks and is often associated with blepharitis.

ETIOLOGY

A. Predisposing factors

1. *Chronic exposure* to dust, smoke, and chemical irritants.
2. *Local cause of irritation* such as trichiasis, concretions, foreign body and seborrhoeic scales.
3. *Eye strain* due to refractive errors, phorias or convergence insufficiency.
4. *Abuse of alcohol,* insomnia and metabolic disorders.

B. Causative organisms (Table 2.1)

- *Staphylococcus aureus* is the commonest cause of chronic bacterial conjunctivitis. It colourizes the eyelid margin and then causes direct infection of conjunctiva or conjunctival inflammation through the exotoxins released.
- *Gram-negative rods* such as *Proteus mirabilis, Klebsiella pneumoniae, Escherichia coli, Moraxella lacunata, Serratia marcescens*, and *Branhamella catarrhalis* are other rare causes.

C. Source and mode of infection. Chronic conjunctivitis may occur:

1. *As continuation of acute mucopurulent conjunctivitis* when untreated or partially treated.
2. *As chronic infection* from associated chronic dacryocystitis, chronic rhinitis or chronic upper respiratory catarrh.
3. *As a mild exogenous infection* which results from direct contact, airborne or material transfer of infection.

CLINICAL FEATURES

Symptoms of simple chronic conjunctivitis include:

- *Burning and grittiness* in the eyes, especially in the evening.
- *Mild chronic redness* in the eyes. Feeling of heat and dryness on the lid margins.
- *Difficulty in keeping the eyes open.*
- *Mild mucoid discharge* especially in the canthi.
- *Watering*, off and on is often a complaint.
- *Feeling of sleepiness* and tiredness in the eyes.

Signs. Grossly the eyes look normal but careful examination may reveal following signs:

- *Congestion* of posterior conjunctival vessels which is mild and diffuse.
- *Mild papillary hypertrophy* of the palpebral conjunctiva.
- *Follicles,* may also occur
- *Conjunctival* thickening
- *Sticky look* of surface of the conjunctiva.

- *Lid margins* may show congestion, telangiectasis, loss of lashes and blepharitis.
- *Cornea* may develop marginal corneal ulcer.

TREATMENT

1. *Eliminate predisposing factors* when associated.
2. *Topical antibiotics* such as chloramphenicol, tobramycin or gentamicin should be instilled 3–4 times a day for about 2 weeks to eliminate the mild chronic infection.
3. *Astringent eye drops* such as zinc–boric acid drops provide symptomatic relief.
4. *Treatment of blepharitis*, which is usually associated needs to be done by good lid hygiene with warm compresses, and eyelid scrubs followed by rubbing of combination of antibiotic and corticosteroid eye ointment.
   - Systemic therapy with oral tetracycline 250 mg 4 times a day, or doxycycline 100 mg 1–2 times a day, may be needed for severe cases of blepharitis.

ANGULAR BACTERIAL CONJUNCTIVITIS

It is a type of chronic conjunctivitis characterised by mild grade inflammation confined to the conjunctiva and lid margins near the angles (hence the name) associated with maceration of the surrounding skin.

ETIOLOGY

1. *Predisposing factors* are same as for 'simple chronic conjunctivitis'.
2. *Causative organisms. Moraxella-Axenfeld* (MA) is the commonest causative organism. MA bacilli are placed end to end, so the disease is also called 'diplobacillary conjunctivitis'. Rarely, staphylococci may also cause angular conjunctivitis.
3. *Source of infection* is usually nasal cavity.
4. *Mode of infection.* Infection is transmitted from nasal cavity to the eyes by contaminated fingers or handkerchief.

PATHOLOGY

The causative organism, i.e. MA bacillus produces a proteolytic enzyme which acts by macerating the epithelium. This proteolytic enzyme collects at the angles by the action of tears and thus macerates the epithelium of the conjunctiva, lid margin and the skin, the surrounding angles of eye. The maceration is followed by vascular and cellular responses in the form of mild grade chronic inflammation. Skin may show eczematous changes.

CLINICAL FEATURES

Symptoms include:

- Irritation, burning sensation and feeling of discomfort in the eyes.
- History of collection of dirty-white foamy discharge at the angles.
- Redness in the angles of eyes.

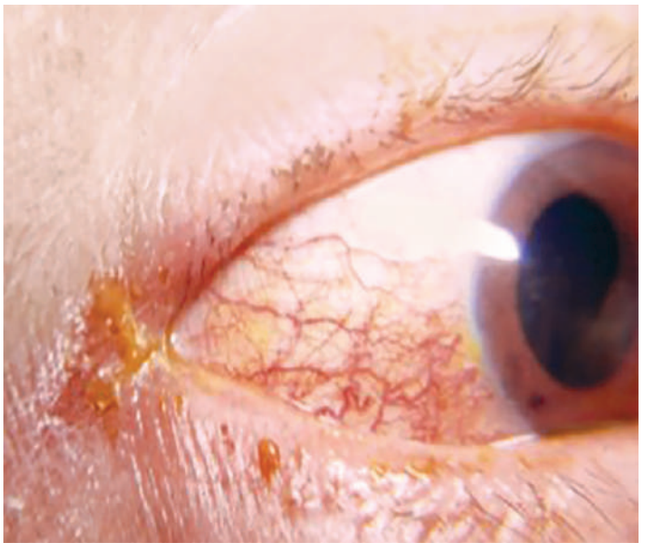

Fig. 2.4: Signs of angular conjunctivitis.

Signs include (Fig. 2.4):

- *Hyperaemia of bulbar conjunctiva* near the canthi.
- *Hyperaemia of lid margins* near the angles.
- *Excoriation of the skin* around the angles.
- *Foamy mucopurulent discharge* at the angles is usually present.

Complications include blepharitis and shallow marginal catarrhal corneal ulceration.

TREATMENT

A. Prophylaxis includes treatment of associated nasal infection and good personal hygiene.

B. Curative treatment consists of:

1. *Oxytetracycline* (1%) eye ointment, 2–3 times a day for 9–14 days will eradicate the infection.
2. *Zinc lotion* instilled in daytime and zinc oxide ointment at bed time inhibits the proteolytic ferment and thus helps in reducing the maceration.

CHLAMYDIAL CONJUNCTIVITIS

Chlamydia, earlier classified as a separate organism in between bacteria and viruses, has now been classified as bacterium belonging to the family Chlamydiaceae having two genera: Chlamydia and Chlamydophila.

Characteristics of Chlamydia

- Small, obligate intracellular, Gram-negative bacteria.
- Possess both RNA and DNA, ribosomes and cell wall similar to that of Gram-negative bacteria.
- Differ from most true bacteria is not having peptidoglycan.
- Lack the ability to produce their own ATP, therefore, use host's ATP (energy parasites).
- Multiply by binary fission.
- Inclusion bodies are basophilic in nature.

- Multiply in the cytoplasm of the host cell forming microcolonies or inclusion bodies which drape around the nucleus like a cloak or mantle (*chlamys* means mantle).
- Possess a genus-specific lipopolysaccharide-protein complex antigen.
- Exist in two morphologically distinct forms, namely elementary body (EB) and reticulate body (RB).

Life cycle of Chlamydia

Chlamydia exists in two morphological forms: The elementary body (EB) and reticulate body (RB). Life cycle of Chlamydia is shown in Figure 2.5:

- *Elementary bodies* (EBs) are extracellular infectious particles (Fig. 2.5A). These initiate infection by attaching to the susceptible host cells (Fig. 2.5B). After attachment, the EB enters the cytoplasm of the host cells within a vesicle (Fig. 2.5C), where it increases in size and differentiates into reticulate body (RB) (Fig. 2.5D).
- *Reticulate body* (RB) is thus intracellular, metabolically active form that divides by binary fission (Fig. 2.5E). Soon there occurs condensation of DNA within the RBs, disulphide bonds are formed in the outer membrane proteins and new EBs develop within the enlarging vesicle. The developing chlamydiaceal microcolony within the vesicle is termed inclusion body which is typically perinuclear and may contain 100–500 EBs (Fig. 2.5F).
- *Release of new* EBs into the extracellular space occurs following rupture of the inclusion body (Fig. 2.5G). The liberated EBs then infect the new cells where the whole cycle is repeated (Fig. 2.5G).

Ocular infections produced by Chlamydia

Ocular infections produced by Chlamydia in human beings are summarised in Table 2.4.

TRACHOMA

Trachoma (previously known as *Egyptian ophthalmia*) is a chronic keratoconjunctivitis, primarily affecting the superficial epithelium of conjunctiva and cornea simultaneously. It is characterised by a mixed follicular and papillary response of conjunctival tissue, pannus formation and in late stages cicatrization giving rough appearance. The word 'trachoma' comes from the Greek word for 'rough' which describes the surface appearance of the conjunctiva in chronic trachoma. It is still one of the leading causes of preventable blindness in the world.

ETIOLOGY

A. Causative organism. Trachoma is caused by the bacterium *Chlamydia trachomatis*, biovar TRIC. The organism is epitheliotropic and produces intracytoplasmic inclusion bodies called *HP bodies* (*Halberstaedter-Prowazek bodies*). Presently, 12 serovars of *Chlamydia trachomatis* biovar TRIC (A, B, Ba, C, D, E, F, G, H, I, J and K) have been identified using microimmunofluorescence techniques.

- *Serovars A, B, Ba and C* are associated with hyperendemic (blinding) trachoma.
- *Serovars D to K* are associated with inclusion conjunctivitis (oculogenital chlamydial disease).

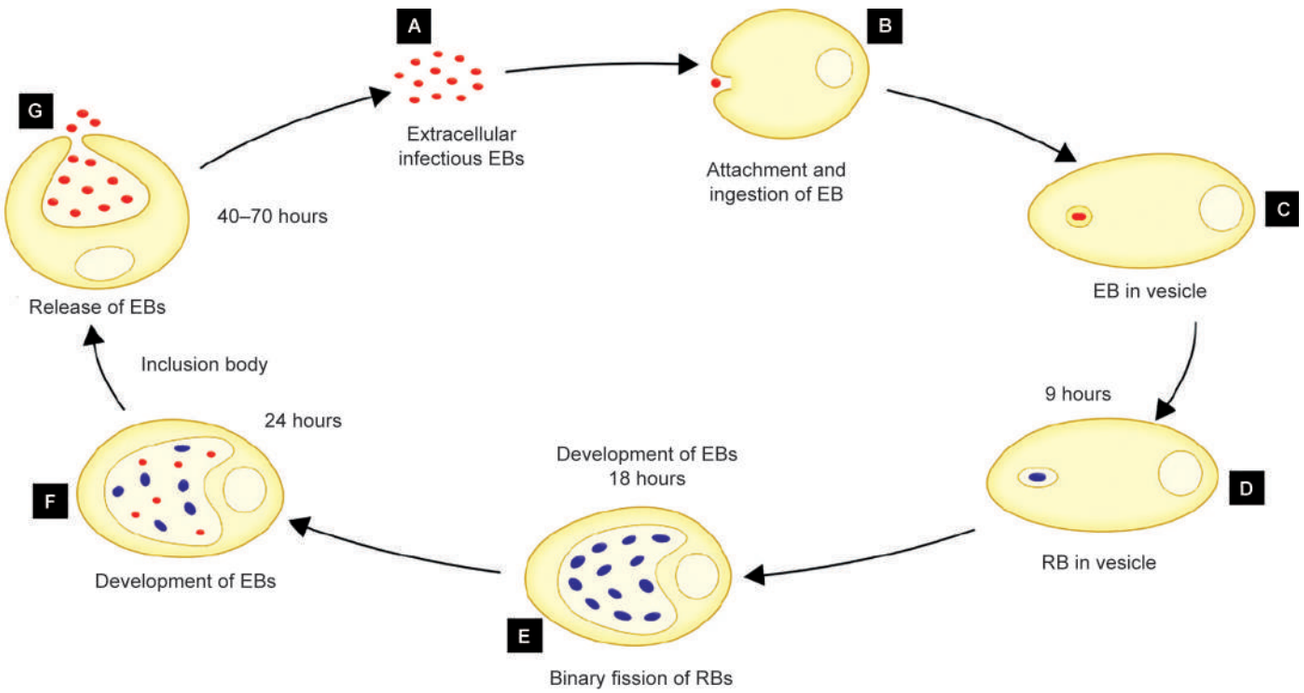

Fig. 2.5A to G: Life cycle of Chlamydia.

TABLE 2.4: Summary of ocular infections caused by Chlamydia

Family	*Chlamydiaceae*				
Genra	Chlamydia			Chlamydophila	
Biovar	*C. trachomatis* (TRIC agent) (humans)		*C. lymphogranulomatis* (humans)	*C. psittacosis* (animals)	*C. pneumoniae* (humans)
Serovar	A, B, Ba, C	D to K	L1, L2, L3	–	–
				–	–
Ocular disease	Hyperendemic trachoma	Paratrachoma (neonatal and adult inclusion conjunctivitis)	Lymphogranuloma venereum conjunctivitis		
Transmission	Eye to eye	Genitals to eye	Genitals to eye	–	–

B. Predisposing factors include:

- *Age.* The infection is usually contracted during infancy and early childhood. Otherwise, there is no age bar.
- *Sex.* As far as sex is concerned, there is general agreement that preponderance exists in the females both in number and in severity of disease.
- *Race.* No race is immune to trachoma, but the disease is very common in Jews and comparatively less common among Negroes.
- *Climate.* Trachoma is more common in areas with dry and dusty weather.
- *Socioeconomic status.* The disease is more common in poor classes owing to unhygienic living conditions, overcrowding, unsanitary conditions, abundant fly population, paucity of water, lack of materials like separate towels and handkerchiefs, and lack of education and understanding about spread of contagious diseases.
- *Environmental factors* like exposure to dust, smoke, irritants, sunlight, etc. increase the risk of contracting disease. Therefore, outdoor workers are more affected in comparison to office workers.

C. Source of infection. In trachoma endemic zones, the main source of infection is the conjunctival discharge of the affected person. Therefore, superimposed other bacterial infections help in transmission of the disease by increasing the conjunctival secretions.

D. Modes of infection. Infection may spread from eye to eye by any of the following modes:

1. *Direct spread* of infection may occur through contact by airborne or waterborne modes.
2. *Vector transmission* of trachoma is common through flies.
3. *Material transfer* plays an important role in the spread of trachoma. Material transfer can occur through contaminated fingers of doctors, nurses and contaminated tonometers. Other sources of material transfer of infection are use of common towel, handkerchief, bedding and surma-rods.

PREVALENCE

Trachoma is a worldwide disease, but it is highly prevalent in North Africa, Middle East and certain regions of South-East Asia. It is believed to affect some 500 million people in the world. There are about 150 million cases with active trachoma and about 30 million having trichiasis, needing lid surgery. Trachoma is responsible for 15–20% of the world's blindness, being second only to cataract.

CLINICAL AND PATHOLOGICAL FEATURES

Clinical features of trachoma can be described into two phases.

I. *Phase of active trachoma*

Phase of active trachoma usually occurs during childhood due to active chlamydial infection.

- *Incubation period* of active trachoma varies from 7 to 14 days.
- *Onset of disease* is usually insidious (subacute), however, rarely it may present in acute form.

Symptoms

Symptoms of active trachoma are determined by the absence or presence of secondary other bacterial infection (a very common situation).

▪ *In the absence of secondary infection*, a pure trachoma is characterized by following symptoms:

- Mild foreign body sensation
- Occasional lacrimation

- Slight stickiness of the lids
- Scanty mucoid discharge.

Note. The above symptoms are so mild that the disease is usually neglected so, the term trachoma dubium was suggested.

▪ *In the presence of secondary other bacterial infection*, typical symptoms of acute mucopurulent conjunctivitis develop (*see* page 12).

Signs

A. Conjunctival signs

1. *Congestion* of upper tarsal and forniceal conjunctiva.
2. *Conjunctival follicles.* Follicles (Figs 2.6A and B) look like boiled sago-grains and are commonly seen on upper tarsal conjunctiva and fornix; but may also be present in the lower fornix, plica semilunaris and caruncle. Sometimes, follicles may be seen on the bulbar conjunctiva (pathognomonic of trachoma).

▪ *Pathological structure of follicle.* Follicles are formed due to scattered aggregation of lymphocytes and other cells in the adenoid layer. Central part of each follicle is made up of mononuclear histiocytes, few lymphocytes and large multinucleated cells called Leber cells. The cortical part is made up of a zone of lymphocytes showing active proliferation. Blood vessels are present in the most peripheral part. In later stages, signs of necrosis are also seen. Presence of Leber cells and signs of necrosis differentiate trachoma follicles from follicles of other forms of follicular conjunctivitis.

3. *Papillary hyperplasia.* Papillae are reddish, flat topped raised areas which give red and velvety appearance to the tarsal conjunctiva (Fig. 2.7).

▪ *Pathologically* each papilla consists of central core of numerous dilated blood vessels surrounded by lymphocytes and covered by hypertrophic epithelium.

B. Corneal signs

1. *Superficial keratitis* may be present in the upper part.
2. *Herbert follicles* refer to typical follicles present in the limbal area. *Histologically* these are similar to conjunctival follicles.
3. *Progressive pannus*, i.e. infiltration of the cornea associated with vascularisation is seen in upper part (Fig. 2.8). The vessels are superficial and lie between epithelium and Bowman's membrane. Later on, Bowman's membrane is also destroyed. Pannus in active trachoma is progressive in which infiltration of cornea is ahead of vascularisation (Fig. 2.8A).
4. *Corneal ulcer* may sometime develop at the advancing edge of pannus. Such ulcers are usually shallow which may become chronic and indolent.

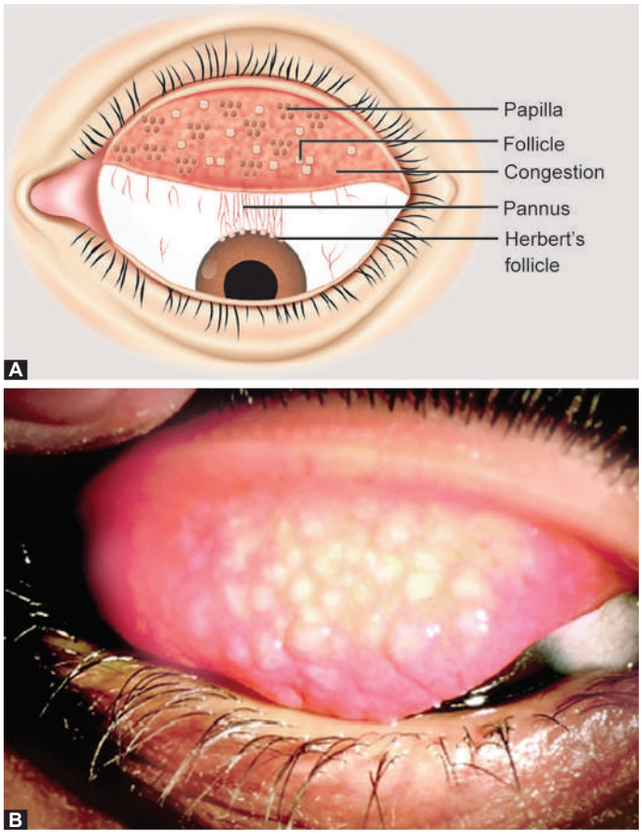

Fig. 2.6: Signs of active trachoma. A, Diagrammatic; B, Clinical photograph of trachomatous inflammation (TF) follicular.

II. *Phase of cicatricial trachoma*

Cicatricial phase of trachoma manifests in middle age. It results due to continued mild grade chronic inflammation. In fact recurrent infection elicits chronic immune response consisting of cell-mediated delayed hypersensitivity (type IV) reaction to the intermittent presence of chlamydial antigen, which is responsible for cicatricial phase of trachoma. The end stage of cicatricial trachoma is also referred to as sequelae of trachoma. This phase is characterized by following clinical features.

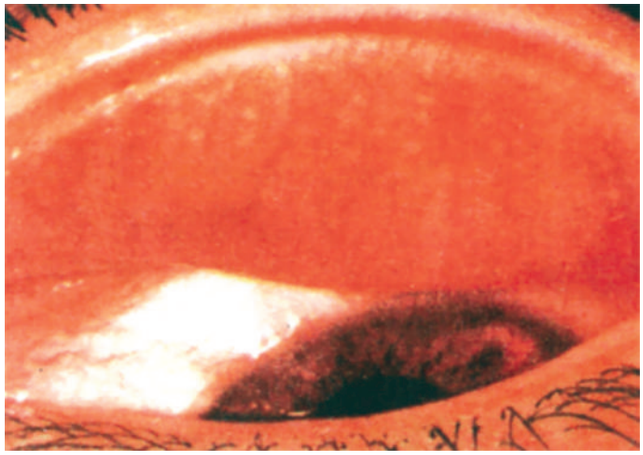

Fig. 2.7: Trachomatous inflammation (TI) intense.

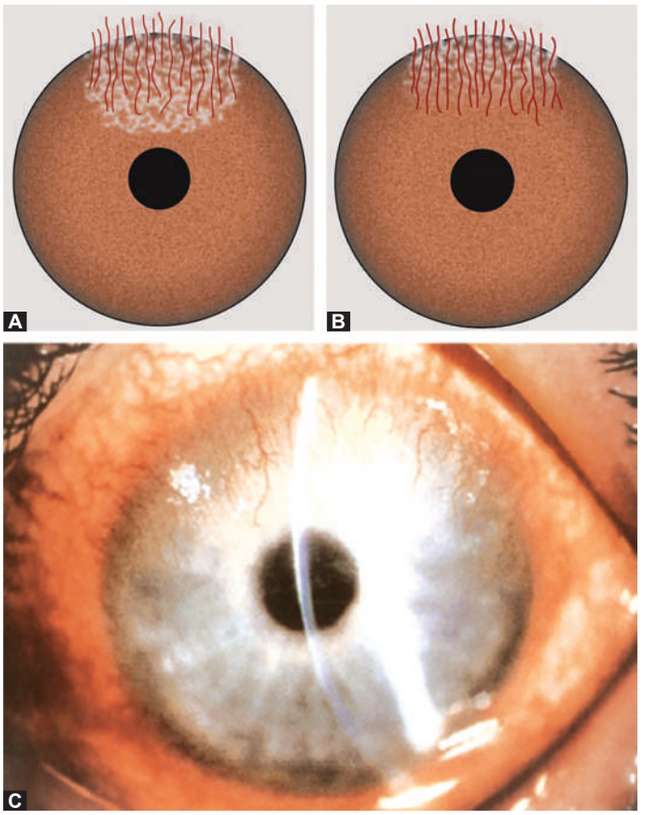

Fig. 2.8: Trachomatous pannus. A, Progressive; B, Regressive (diagrammatic); C, Clinical photograph.

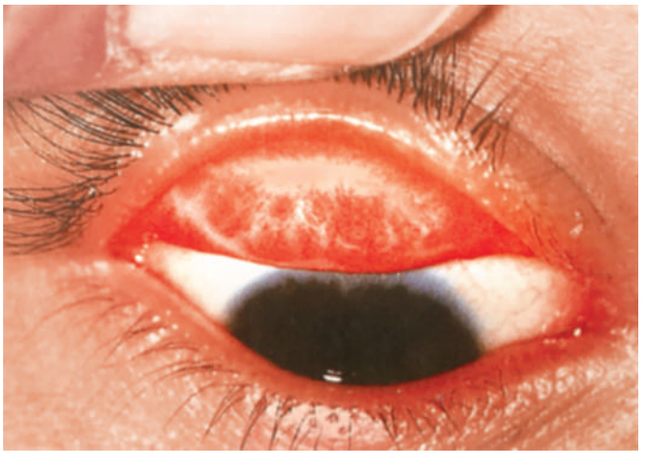

Fig. 2.9: Trachomatous scarring (TS).

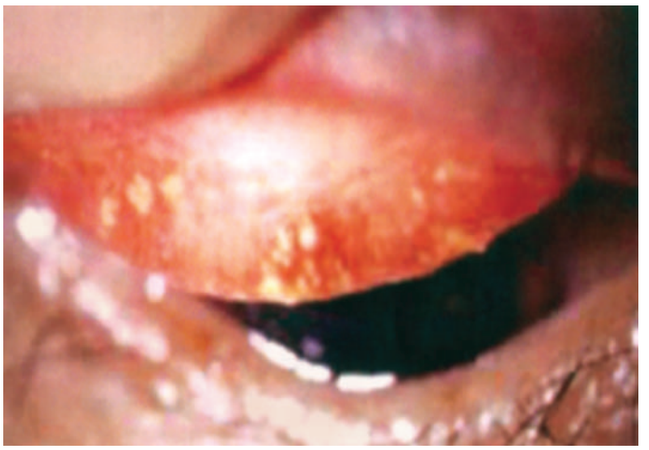

Fig. 2.10: Concretions in upper palpebral conjunctiva.

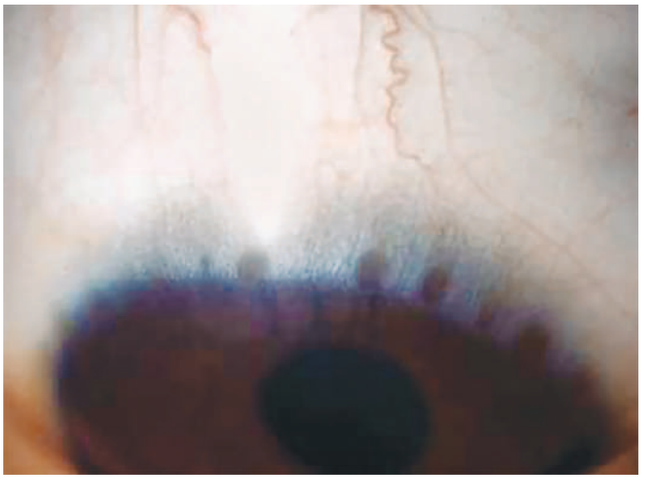

Fig. 2.11: Trachomatous Herbert's pits.

A. Conjunctival signs

i. *Conjunctival scarring* (Fig. 2.9), which may be irregular, star-shaped or linear. Linear scar present in the sulcus subtarsalis is called *Arlt's line*.

ii. *Concretions* are hard looking whitish deposits varying from pinpoint to 2 mm in size (Fig. 2.10). These are not calcareous deposits, but are formed due to accumulation of dead epithelial cells and inspissated mucus in the depressions called *glands of Henle*. Hence, the name is misnomer.

iii. *Other conjunctival sequelae* include *concretions, pseudocyst, xerosis* and *symblepharon*.

B. Corneal sign

i. *Regressive pannus* (pannus siccus) in which (Fig. 2.8B) vessels extend a short distance beyond the area of infiltration.

ii. *Herbert pits* are the oval or circular pitted scars, left after healing of Herbert follicles in the limbal area (Fig. 2.11).

iii. *Corneal opacity* (Fig. 2.12) may be present in the upper part. It may even extend down and involve the papillary area. It is the end result of trachomatous corneal lesions.

iv. *Other corneal sequelae* may be corneal ectasia, corneal xerosis and total corneal pannus (blinding sequelae).

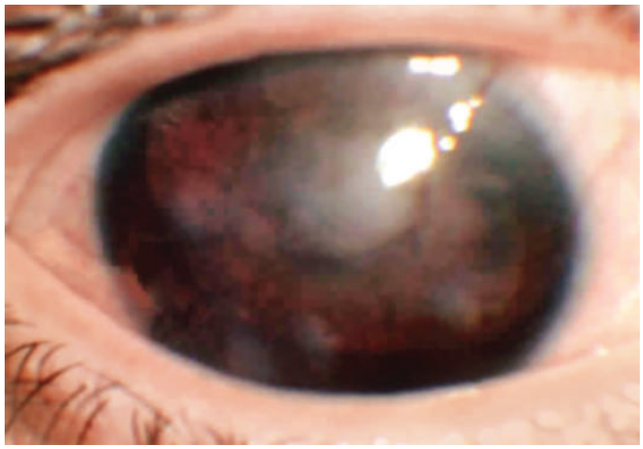

Fig. 2.12: Trachomatous corneal opacity (CO).

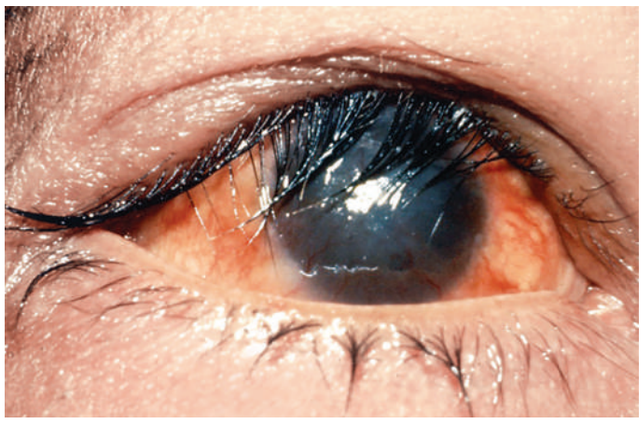

Fig. 2.13: Trachomatous trichiasis (TT).

C. Lid signs. Sequelae in the lids may be trichiasis (Fig. 2.13), entropion, tylosis (thickening of lid margin), ptosis, madarosis and ankyloblepharon.

D. Lacrimal apparatus sequelae may be chronic dacryocystitis, and chronic dacryoadenitis.

GRADING OF TRACHOMA

McCallan's classification

McCallan, in 1908, divided the clinical course of the trachoma into four stages:

1. *Stage I* (incipient trachoma or stage of infiltration). It is characterized by hyperaemia of palpebral conjunctiva and immature follicles.
2. *Stage II* (established trachoma or stage of florid infiltration). It is characterized by appearance of mature follicles, papillae and progressive corneal pannus.
3. *Stage III* (cicatrising trachoma or stage of scarring). It includes obvious scarring of palpebral conjunctiva.
4. *Stage IV* (healed trachoma or stage of sequelae). The disease is quiet and cured but sequelae due to cicatrisation, give rise to symptoms.

WHO classification

The latest simplified classification suggested by WHO in 1987 is as follows (FISTO):

1. *TF: Trachomatous inflammation-follicular.* It is the stage of active trachoma with predominantly follicular inflammation. To diagnose this stage at least five or more follicles (each 0.5 mm or more in diameter) must be present on the upper tarsal conjunctiva (*see* Fig. 2.6). Further, the deep tarsal vessels should be visible through the follicles and papillae.
2. *TI: Trachomatous inflammation intense.* This stage is diagnosed when pronounced inflammatory thickening of the upper tarsal conjunctiva obscures more than half of the normal deep tarsal vessels (*see* Fig. 2.7).
3. *TS: Trachomatous scarring.* This stage is diagnosed by the presence of scarring in the tarsal conjunctiva. These scars are easily visible as white, bands or sheets (fibrosis) in the tarsal conjunctiva (*see* Fig. 2.9).
4. *TT: Trachomatous trichiasis.* TT is labelled when at least one eyelash rubs the eyeball. Evidence of recent removal of inturned eyelashes should also be graded as trachomatous trichiasis (Fig. 2.13).
5. *CO: Corneal opacity.* This stage is labelled when easily visible corneal opacity is present over the pupil (Fig. 2.12). This sign refers to corneal scarring that is so dense that at least part of pupil margin is blurred when seen through the opacity. The definition is intended to detect corneal opacities that cause significant visual impairment (less than 6/18).

COMPLICATIONS

The only complication of trachoma is corneal ulcer which may occur due to rubbing by concretions, or trichiasis with superimposed bacterial infection.

DIAGNOSIS

A. Clinical diagnosis of trachoma is made from its typical signs. Clinical grading of each case should be done as per WHO classification into TF, TI, TS, TT or CO.

B. Laboratory diagnosis. Advanced laboratory tests are employed for research purposes only. Laboratory diagnosis of trachoma includes:

1. *Conjunctival cytology.* Giemsa-stained smears showing a predominantly polymorphonuclear reaction with presence of plasma cells and Leber cells is suggestive of trachoma.
2. *Detection of inclusion bodies* in conjunctival smear may be possible by Giemsa stain, iodine stain or immunofluorescent staining, especially in cases with active trachoma.
3. *Enzyme-linked immunosorbent assay* (ELISA) for chlamydial antigens.
4. *Polymerase chain reaction* (PCR) is also useful.
5. *Isolation of Chlamydia* is possible by yolk sac inoculation method and tissue culture technique. Standard single-passage McCoy cell culture requires at least 3 days.

6. *Serotyping of TRIC agents* is done by detecting specific antibodies using microimmunofluorescence (micro-IF) method. *Direct monoclonal fluorescent antibody microscopy* of conjunctival smear is rapid and inexpensive.

DIFFERENTIAL DIAGNOSIS

1. Active trachoma with follicular hypertrophy must be differentiated from acute adenoviral follicular conjunctivitis (epidemic keratoconjunctivitis) as follows:

- *Distribution of follicles* in trachoma is mainly on upper palpebral conjunctiva and upper fornix, while in EKC lower palpebral conjunctiva and lower fornix is predominantly involved.
- *Associated signs* such as papillae and pannus are characteristic of trachoma.
- *Laboratory diagnosis* of trachoma helps in differentiation of clinically indistinguishable cases.

2. Active trachoma with predominant papillary hypertrophy needs to be differentiated from palpebral form of spring catarrh as follows:

- *Papillae* are large in size and usually there is typical cobble-stone arrangement in spring catarrh.
- *pH of tears* is usually alkaline in spring catarrh, while in trachoma it is acidic.
- *Discharge* is ropy in spring catarrh.
- *Follicles and pannus* may also be present in trachoma.
- *Conjunctival cytology* and other laboratory tests for trachoma usually help in diagnosis in clinically indistinguishable cases.

MANAGEMENT

Management of trachoma includes curative as well as prophylactic measures.

A. Treatment of trachoma

I. Treatment of active trachoma

Stage TF and TI of WHO classification constitute active trachoma in which acute infection is present, and therefore, treatment is directed at eliminating the Chlamydia organism.

Antibiotics, thus constitute the mainstay of treatment of active trachoma. These can be given topically or systemically or in combination.

1. *Topical therapy regimes* are best for individual cases and consist of:

- *Tetracycline* (1%) or erythromycin (1%) eye ointment twice daily for 6 weeks, or
- *Sulfacetamide* (20%) eye drops three times a day along with 1% tetracycline eye ointment at bed time for 6 weeks.

2. *Systemic antibiotic therapy regimes* include:

- *Tetracycline* or *erythromycin* 250 mg orally, four times a day for 3–4 weeks, or
- *Doxycycline* 100 mg orally twice daily for 3–4 weeks, constitute the traditional standard systemic therapy.
- *Azithromycin* 20 mg/kg body weight up to maximum 1 g as single oral dose is as effective as 6 weeks of topical therapy and so is presently considered the first drug of choice. It is not used in pregnancy and children below 6 years of age.

3. *Combined topical and systemic therapy regime.* It is preferred when the ocular infection is severe (TI) or when there is associated genital infection. It includes:

- *Tetracycline* (1%) or *erythromycin* eye ointment 2 times a day for 6 weeks; and
- *Azithromycin* single oral dose (first choice) or tetracycline or erythromycin 250 mg orally 4 times a day for 2 weeks.

II. Treatment of cicatricial (inactive) trachoma

Stages TS, TI and CO of WHO classification constitute the inactive trachoma during which infection is no longer present, i.e. only trachoma sequelae are present, and therefore, treatment is directed towards these sequelae as below:

Stage TS measures include:

- *Concretions* should be removed with a hypodermic needle.
- *Conjunctival xerosis* should be treated by artificial tears (lubricating drops).

Stage TI includes trichiasis and cicatricial entropion.

- *Trichiasis*, a few misdirected cilia, should be treated with permanent lash removal measures such as electrolysis, cryolysis, and radiofrequency epilation.
- *Bilamellar tarsal resection* is the surgical procedure of choice for multiple misdirected lashes.
- *Cicatricial entropion* should be corrected surgically.

Stage CO (corneal opacification) constitutes stage of marked visual disability or blindness. After treating other trachoma sequelae, following measures must be taken:

- *Penetrating keratoplasty (PK)* is indicated for significant corneal scarring. However, the outcome is less than optimum, as these patients have extensive corneal vascularisation.
- *Keratoprosthesis (KP)* is indicated in bilateral blind cases with extensive corneal scarring and ocular surface problems.
- *Punctal occlusion and lateral tarsorrhaphy,* which takes care of the coexistent ocular surface problems, may be useful adjuncts for increasing the success of the above surgeries.

B. Prophylaxis for trachoma infection and blindness

Since immunity is very poor and short lived, so reinfections and recurrences are likely to occur. So, prophylactic measures are essential.

WHO defines blinding trachoma elimination as:

- TF prevalence, 5% in 1–9 years children, and
- TI prevalence, 1 per 1000 in total population.

SAFE strategy

The WHO's GET 2020 program (Global Elimination of Trachoma by 2020), has adopted the so-called SAFE strategy for prophylaxis against trachoma infection and prevention of blindness.

SAFE strategy includes:

S : Surgery (tertiary prevention)
A : Antibiotic use (secondary prevention)
F : Facial hygiene (primary prevention)
E : Environmental changes (primordial prevention).

1. Environmental changes (primordial prevention). Flies and other fomites are the common causes of spread of trachoma. So, environmental sanitation will constitute the primordial prevention for trachoma. Recommended environmental sanitation measures include:

- Provision of water latrines and good water supply to reduce flies and improve washing habits,
- Refuse dumps,
- Sprays to control flies,
- Animal pens away from human household, and
- Health education to improve personal and environmental hygiene.

2. Facial hygiene (primary prevention). Facial hygiene is critical measure for primary prevention of trachoma and should include:

- *Frequent face wash* with clean water to eliminate the potentially infectious ocular secretions.
- *Avoidance of common use* of towel, handkerchief, surmarods, etc. are important facial hygienic measures to prevent spread of trachoma infections.

3. Antibiotics for prevention against trachoma (secondary prevention). Use of antibiotics constitutes the secondary prevention against trachoma. Current WHO recommendations for community-based mass antibiotic therapy in endemic areas are as follows:

i. *In areas with 10% or more prevalence of active trachoma (TF in children 1–9 years) recommendation are as below:*

- *Oral azithromycin* (single dose of 20 mg/kg up to 1 g), should be administered to all community members.
- *Tetracycline eye ointment* twice daily for 6 weeks is recommended for all pregnant women, children, 6 months and those allergic to macrolides.

Note. The mass antibiotic therapy should be given once in a year for continuous three years, after which reassessment of prevalence should be made. The annual treatment should be continued till the TF prevalence in 1–9 years children of that area becomes less than 5%.

ii. *In areas with prevalence between more than 5% and less than 10%,* the targeted antibiotic therapy is recommended only among family members and close contacts of the patients.

iii. *In areas with prevalence less than 5%,* treatment of the patients only is recommended.

4. Surgery (tertiary prevention). Surgery for trichiasis and entropion constitutes tertiary prevention for trachomatous corneal blindness. WHO recommends bilamellar tarsal rotation surgery at community level for the affected persons.

ADULT INCLUSION CONJUNCTIVITIS

It is a type of acute follicular conjunctivitis associated with mucopurulent discharge. It usually affects the sexually active young adults.

ETIOLOGY

- *Serotypes D to K of Chlamydia trachomatis* are associated with adult inclusion conjunctivitis.
- *Primary source of infection* is urethritis in males and cervicitis in females.
- *Transmission of infection* may occur to eyes either through contaminated genital-hand-eye or genital-eye contact.
- *Spread of infection may also occur* through contaminated water of swimming pools (hence the name swimming pool conjunctivitis).

CLINICAL FEATURES

Incubation period of the disease is 5–14 days.

Symptoms are similar to acute mucopurulent conjunctivitis and include:

- *Ocular discomfort, foreign body sensation,*
- *Mild photophobia,* and
- *Mucopurulent discharge* from the eyes.

Signs of inclusion conjunctivitis are (Fig. 2.14):

- *Conjunctival hyperaemia*, more marked in the lower palpebral conjunctiva and fornix.
- *Acute follicular hypertrophy* predominantly of lower palpebral conjunctiva and fornix.
- *Superficial keratitis* in upper half of cornea. Sometimes, superior micropannus may also occur.
- *Pre-auricular lymphadenopathy* (non-tender) is a usual finding on the ipsilateral side.

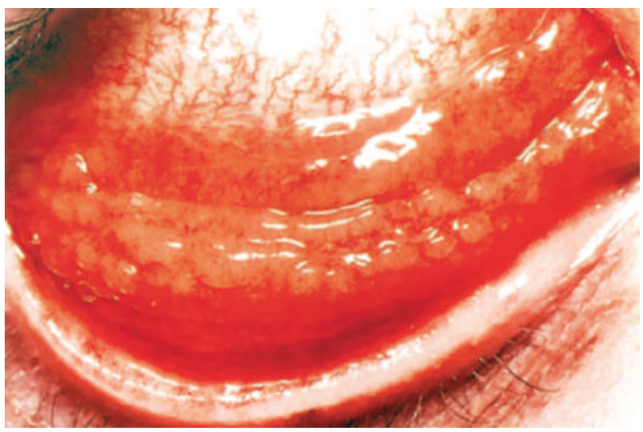

Fig. 2.14: Signs of acute follicular conjunctivitis.

Clinical course. The disease runs a benign course and often evolves into the chronic follicular conjunctivitis.

Investigations required, their role and status is same as described above for trachoma.

Differential diagnosis must be made from other causes of acute follicular conjunctivitis.

MANAGEMENT

Treatment

1. Topical therapy. It consists of tetracycline (1%) eye ointment 4 times a day for 6 weeks.

2. Systemic therapy is very important, since the condition is often associated with an asymptomatic venereal infection. Commonly employed antibiotics are:

- *Azithromycin* 1 g as a single dose repeated after one week is currently drug of choice, a 3rd dose is required in 30% of cases.
- *Tetracycline* 250 mg four times a day for 3–4 weeks, or
- *Erythromycin* 250 mg four times a day for 3–4 weeks (only when the tetracycline is contraindicated, e.g. in pregnant and lactating females), or
- *Doxycycline* 100 mg twice a day for 3 weeks.

3. Referral to genitourinary specialist is mandatory. Sexual partners should be treated simultaneously. Attention should also be given to other sexually transmitted diseases, contact tracing and pregnancy testing.

Prophylaxis

- Improvement in personal hygiene.
- Regular chlorination of swimming pool water will definitely decrease the spread of disease.
- Patient's sexual partner should be examined and treated. Abstinence of sexual contact until completion of treatment.

VIRAL CONJUNCTIVITIS

Most of the viral infections tend to affect the epithelium, both of the conjunctiva and cornea; so, the typical viral lesion is a 'keratoconjunctivitis'. In some viral infections, conjunctival involvement is more prominent (e.g. pharyngoconjunctival fever), while in others cornea is more involved (e.g. herpes simplex).

Viral infections of conjunctiva include:

- Adenovirus conjunctivitis
- Herpes simplex keratoconjunctivitis
- Herpes zoster conjunctivitis
- Molluscum contagiosum conjunctivitis
- Poxvirus conjunctivitis
- Myxovirus conjunctivitis
- Paramyxovirus conjunctivitis
- Arbor virus conjunctivitis.

Clinical presentations of acute viral conjunctivitis include:

- Acute follicular conjunctivitis
- Acute haemorrhagic conjunctivitis.

ADENOVIRAL CONJUNCTIVITIS

Adenoviruses are the commonest causes of viral conjunctivitis. These are non-enveloped, double-stranded DNA viruses, which replicate within the nucleus of host cells. General reservoir of adenoviruses is only human. Fifty-one distinct human adenoviral serotypes have been described and are classified into six subgenera (A–F). More than half of all adenoviral subtypes (32) belong to subgenus D. With a few exceptions, most adenoviral conjunctivitis is caused by this genus.

Clinical types of adenoviral conjunctivitis include:

- Epidemic keratoconjunctivitis (EKC)
- Nonspecific acute follicular conjunctivitis
- Pharyngoconjunctival fever (PCF)
- Chronic relapsing adenoviral conjunctivitis.

EPIDEMIC KERATOCONJUNCTIVITIS (EKC)

It is a type of acute follicular conjunctivitis mostly associated with superficial punctate keratitis and usually occurs in epidemics, hence the name epidemic keratoconjunctivitis (EKC).

Etiology

EKC is mostly caused by adenoviruses type 8, 19 and 37 with type 8 being the classic cause. The condition is markedly contagious and spreads through contact with contaminated fingers, solutions and tonometers.

Clinical features

Incubation period after infection is about 8 days and virus is shed from the inflamed eye for 2–3 weeks.

Symptoms

Symptoms are similar to severe form of acute catarrhal conjunctivitis and include:

- *Redness* of sudden onset associated with watering, usually profuse, with mild mucoid discharge.
- *Ocular discomfort* and foreign body sensation.
- *Photophobia*, usually mild, becomes marked when cornea is involved.

Signs

I. Eyelids are swollen causing narrowing of palpebral aperture.

II. Conjunctival signs are:

- *Hyperaemia* is usually marked and prominent.
- *Chemosis* of conjunctiva is often present.
- *Follicles* of small to moderate size typically involving the lower fornix and palpebral conjunctiva form the characteristic feature (Fig. 2.15).

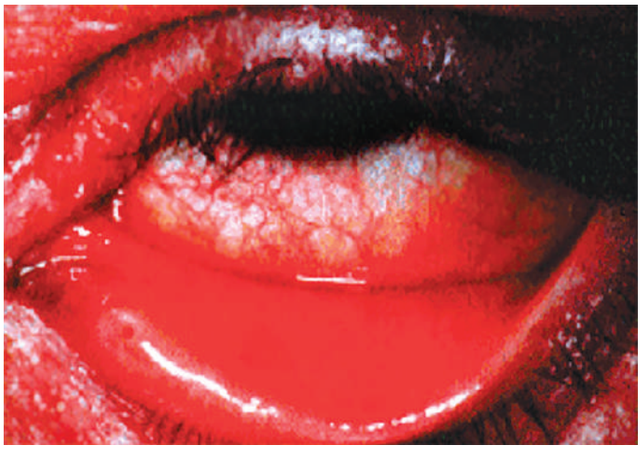

Fig. 2.15: Acute follicular adenoviral conjunctivitis.

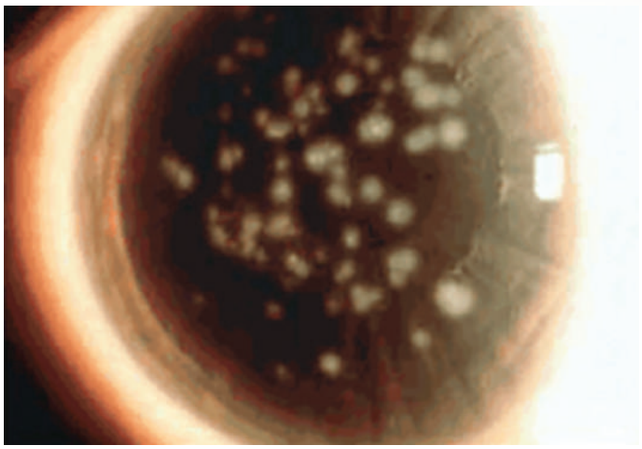

Fig. 2.17: Superficial punctate keratitis in EKC.

- *Papillary reaction* may also be seen in many cases.
- *Petechial subconjunctival haemorrhages* may be seen in severe adenoviral conjunctivitis (Fig. 2.16).
- *Pseudomembrane* lining the lower fornix and palpebral conjunctiva (Fig. 2.16) may be formed in about 3% patients with severe inflammation.

III. Corneal involvement occurs in about 80% of cases and is characterized by following lesions:

- *Epithelial microcystic* diffuse fine non-staining lesions are common during the early stage.
- *Superficial punctate keratitis* (SPK), a typical feature of EKC (Fig. 2.17), usually occurs after 10 days of onset of symptoms and lasts for 3 weeks even after subsidence of conjunctival inflammation.
- *Subepithelial infiltrates* may develop under the focal epithelial lesions in 20–50% of cases. These opacities may be initially disabling and may persist for months to years.

IV. Pre-auricular lymphadenopathy is associated in almost all cases of EKC.

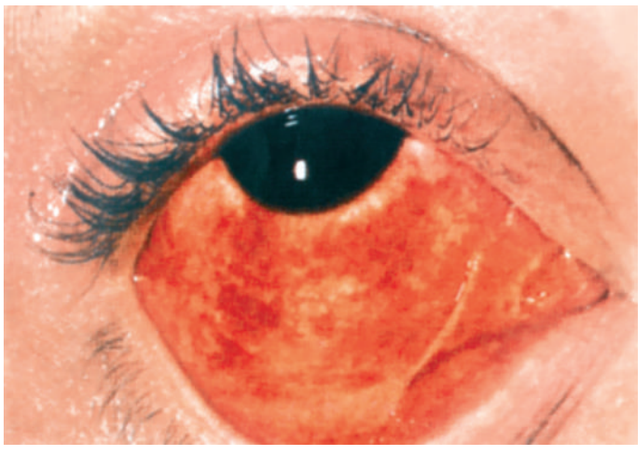

Fig. 2.16: Pseudomembrane and petechial subconjunctival haemorrhage in acute epidemic keratoconjunctivitis (EKC).

Differential diagnosis

EKC needs to be differentiated from *other causes of acute follicular conjunctivitis* which include:

- *Other types of adenoviral keratoconjunctivitis* such as:
- Nonspecific acute follicular conjunctivitis, and
- Pharyngoconjunctival fever.
- *Acute haemorrhagic conjunctivitis*
- *Herpes simplex virus conjunctivitis*
- *Systemic viral infections* such as herpes zoster conjunctivitis, measles, mumps and chikungunya virus conjunctivitis
- *Adult inclusion conjunctivitis.*

Differentiation is made from:

- *Typical clinical features* of each entity described.
- *Investigations* are required mainly for research purposes and in some nonresolving cases, and include:
  - *Conjunctival cytology* with Giemsa stain shows predominantly mononuclear cells in adenoviral conjunctivitis and multinucleated gaint cells in herpetic conjunctivitis.
  - *Polymerase chain reaction (PCR) test* is sensitive and specific for viral DNA.
  - *Point-of-care immunochromatography test* takes only 10 minutes to detect adenoviral antigens in tears and have excellent sensitivity and specificity.
  - *Viral cultures* are tedious and time consuming with variable sensitivity but 100% specificity.

Treatment

1. *Supportive treatment* for amelioration of symptoms is the only treatment required and includes:
   - Cold compresses, and sun glasses to decrease glare.
   - Decongestant and lubricant tear drops to decrease discomfort.
2. *Topical antibiotics* help to prevent superadded bacterial infections.

3. *Topical antiviral drugs* are not beneficial in adenoviral conjunctivitis. Recently promising results are reported with adenine arabinosides (Ara-A) and cidofovir.

4. *Topical steroids should not be used during active inflammation* as these may enhance viral replication and extend the period of infectivity. Weak steroids such as fluorometholone or loteprednol (0.5%) are indicated in patients with subepithelial infiltrates, and in those with membrane formation.

Prevention of spread of infection to the contacts

It is very important as the adenoviral conjunctivitis is highly contagious and patients may be infectious for up to 11 days after onset.

Transmission usually occurs

- From eye to fingers to eyes.
- Tonometers, contact lenses and eye drops are other routes of transmission.

Preventive measures include:

- Frequent handwashing and use of hand sanitizers,
- Relative isolation of infected individual,
- Avoiding eye rubbing and common use of towel or handkerchief sharing, and
- Disinfection of ophthalmic instruments and clinical surfaces after examination of a patient is essential.

NONSPECIFIC ACUTE FOLLICULAR CONJUNCTIVITIS

- *Most common* form of acute follicular conjunctivitis.
- *Caused by* adenovirus serotypes 1 to 11 and 19.
- *Clinical features* are of milder form of acute follicular conjunctivitis. Corneal involvement is not known.
- *Treatment and preventive measures* are similar to as described for EKC.

PHARYNGOCONJUNCTIVAL FEVER

Etiology. It is a highly infectious adenoviral infection commonly associated with subtypes 3, 4 and 7. *Transmitted* by three routes: Personal contact, fomites or through swimming pools or ponds.

Clinical features. Pharyngoconjunctival fever (PCF) primarily affects children and appears in epidemic form. It is characterised by an:

- Acute follicular conjunctivitis, associated with pharyngitis.
- Fever and pre-auricular lymphadenopathy.
- Corneal involvement in the form of superficial punctate keratitis is seen only in 30% of cases.

Treatment is usually supportive as described for EKC.

NEWCASTLE CONJUNCTIVITIS

Etiology. It is a rare type of acute follicular conjunctivitis caused by Newcastle virus. The infection is derived from contact with diseased owls; and thus the condition mainly affects poultry workers.

Clinically, the condition is similar to pharyngoconjunctival fever.

ACUTE HERPETIC CONJUNCTIVITIS

Acute herpetic follicular conjunctivitis is always an accompaniment of the 'primary herpetic infection', which mainly occurs in small children and in adolescents.

Etiology

The disease is commonly caused by herpes simplex virus type 1 and spreads by kissing or other close personal contacts. HSV type 2 associated with genital infections, may also involve the eyes in adults as well as children, though rarely.

Clinical features

Acute herpetic follicular conjunctivitis is usually a unilateral affection with an incubation period of 5–14 days. It may occur in two clinical forms—typical and atypical.

- In *typical form*, the follicular conjunctivitis is usually associated with other lesions of primary infection such as vesicular lesions of face and lids.
- In *atypical form*, the follicular conjunctivitis occurs without lesions of the face, eyelid and the condition then resembles epidemic keratoconjunctivitis. The condition may evolve through phases of no-specific hyperaemia, follicular hyperplasia and pseudomembrane formation.
- *Corneal involvement*, though rare, is not uncommon in primary herpes. It may be in the form of fine or coarse epithelial keratitis or typical dendritic keratitis.
- *Preauricular lymphadenopathy* occurs almost always.

Treatment

Primary herpetic infection is usually self-limiting.

- The topical antiviral drugs control the infection effectively and prevent recurrences.
- Supportive measures are similar to EKC.

ACUTE HAEMORRHAGIC CONJUNCTIVITIS

It is an acute inflammation of conjunctiva characterised by multiple conjunctival haemorrhages, conjunctival hyperaemia and mild follicular hyperplasia.

Etiology

The disease is caused by picornaviruses (enterovirus type 70) which are RNA viruses of small (pico) size. The disease is very contagious and is transmitted by direct hand-to-eye contact.

Clinical features

The disease has occurred in an epidemic form in the far East, Africa and England and hence the name 'epidemic