UNIT 19 PATHOLOGY OF CONJUNCTIVA, CORNEA, GLAUCOMA AND LENS

Structure

19.0 OBJECTIVES

After going through this unit, you will be able to understand:

o   pathology of the various diseases of cornea and conjunctiva;

●   pathological basis of glaucoma; and

●   normal lens and the pathological abnormalities including cataract.

19.1 INTRODUCTION

After going through section dealing with general pathology and microbiology you are familiar with various pathological processes including cellular injury, inflammation, death and neoplasia. This unit describes the pathology of the disorders affecting anterior segment of the eye. A quick review of the previous units describing ocular anatomy and general pathology will be extremely useful at this stage.

19.2 PATHOLOGY OF CONJUNCTIVA AND CORNEA

Cornea is the outer layer of the eyeball. Conjunctiva covers the outer layer sclera anteriorly and under surface of lids. When lids are separated, first structures of the eye visualised are conjunctiva and cornea.

19.2.1 Conjunctiva

Histologically conjunctiva consists of

a)   Nonkeratinized stratified columnar epitheliuni with goblet cells

b)   Substantia propria: loose connective tissue stroma

Palpebral conjunctiva is firmly adherent to the tarsus. Substantia propria of the bulbar conjunctiva is areolar and thus permits the chemosis.

The important disorders that can affect conjunctiva include:

- Acute and chronic conjunctivitis
- Trachoma
- Actinic Keratosis
- Dysplasia

  Intraepithelial Neoplasm

  Lymphoid Tumors

- Malignant Melanoma
- Nevus
- Pinguiucula/Pterygium
- Primary Acquired Melanosis
- Pyogenic Granuloma
- Squamous Papilloina
- Squamous Cell Carcinoma

a) Conjunctivitis

Conjunctivitis is inflammation of conjunctiva, manifested as redness, chemosis, discharge, watering and pain. It can be acute or chronic.

i) *Acute*

Congestion (Hyperemia), edema (chemosis) and discharge/exudation

Bacterial Conjunctivitis

Conjunctival smear shows polymorphonuclear cells and bacteria

Viral Conjunctivitis

Conjunctival smear shows lymphocytes

ii) *Chronic*

Follicular Conjunctivitis

Follicles are gray-white round to oval elevations having avascular center and vessels at the periphery, These are well-circumscribed focus of lymphoid hypertrophy as a result of reactive hyperplasia of conjunctiva's resident population of lymphocytes.

The overlying epithelium is usually thin.

Differential Diagnosis of Follicular Conjunctivitis

Infectious-acute
Adenovirus, Herpes simplex virus, Newcastle virus, enterovirus, inclusion conjunctivitis of adults

Infectious-chronic
Trachoma

Non-infectious
Pseudotrachoma, topical medications, cosmetics, physiological folliculosis of childhood

Papillary Hypertrophy

Papillae are pinkish elevations having central vascular tufts and pale avascular valleys with some epithelial proliferation and stromal hyperplasia.

b) Trachoma

Qne of the most significant causes of blindness in the world.

It spreads by direct,contact, secretions, poor hygiene. .

Characterized by bilateral kerataconjunctivitis, which may be asymmetrical.

Initial epithelial infection followed by subepithelial inflammation with follicles in substantia propria. '

Conjunctival smear shows polymorphonuclear cells and lymphocytes

Epithelial cells contain initial bodies, basophilic intracytoplasmic inclusions of Halberstaeder and Prowazek.

WHO Diagnostic Criteria (must have at least 2)

1) Follicles on the upper tarsus

2) Conjunctival scarring (Arlt's line)

3) Vascular pannus

4) Limbal follicles (>5, each >5mm) or remnants of limbal follicles (Herbert's pits).

c) Actinic Keratosis

Clinically, this lesion is characterized by a thickening of the conjunctiva with whitening or leukoplakia. Histologically, these lesions are very similar to actinic keratosis of the skin with the epithelium showing mild atypia with preservation of normal polarity and maturation. In addition, the underlying substantia propria may show degenerative changes similar to that seen in a pterygium.

d) **Dysplasia**

Conjunctival dysplastic processes probably arise secondary to chronic irritative stimuli such as solar irradiation. Clinically, these lesions are thickened and leukoplakic . Histopathologically, the atypical cells first appear along the basal layer and then progress to involve more and more of the epithelium until the entire epithelium is replaced by abnormal cells. The amount of cellular dysplasia can be sub-classified as mild, moderate, and severe. Mild atypia involves less than 25-33 per cent of the epithelium. Moderate atypia involves 33-75 % of the epithelium. Severe atypia involves greater than 75 per cent of the epithelium.
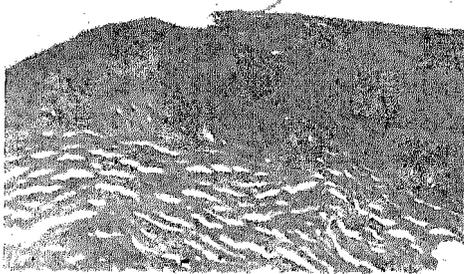
Fig.19.1(a): Conjunctival Dysplasia. Note the sharp demarcation of the cell type and tissue arrangement
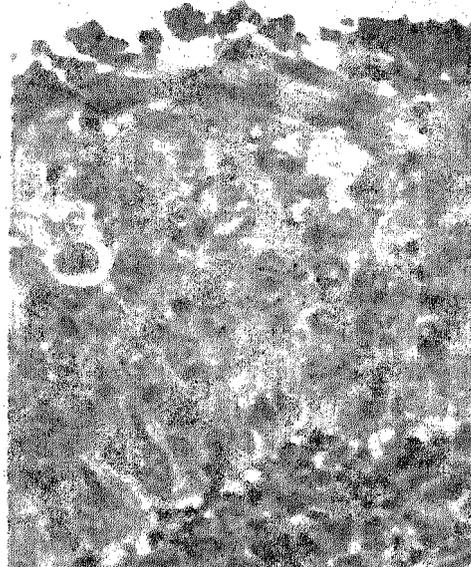
Fig. 19.1(b): Carcisoma in Situ

e) **Intraepithelial** Neoplasm

These lesions clinically appear as thickened, plaque-like lesions of the epithelium which show leukoplakia on the surface and increased vascularity. Pathologically, these lesions are characterized by dysplasia of the squamous cells in the epithelium which involves its full-thickness. This is in essence a 'carcinoma "in situ" and shows preservation of the epithelial basement membrane with no invasion into the underlying substantia propia.

f) Lymphoid **Tumors**

These lesions appear clinically as salmon-coloured nodules or patches. When the lesion is isolated and histologically shows no characteristics of malignancy, the diagnosis of benign lymphoid hyperplasia may be warranted. Malignant lymphoma , of conjunctiva may appear as the first evidence of systemic lymphoma or as part

of widespread involvement. The histopathologic diagnosis of lymphoid lesions can be quite difficult as many of these tumors fall into a "gray zone" between obviously benign lymphoid hyperplasia and frank lymphoma. Surface staining of fresh tissue is often necessary to differentiate these lesions. Malignant lymphomas are monoclonal and mostly composed of B-lymphocytes with sheets of cytologically similar cells with no germinal centers or vascularity, and no other cells such as plasma cells.

g) Malignant Melanoma

Conjunctival melanomas may be associated with primary acquired melanosis (75 per cent) or may arise from a pre-existing nevus or de-novo. Prognostically, the two most important pathological features concerning the likelihood of metastasis are the presence of a pagetoid growth pattern in a PAM component and the thickness of the invasive nodule. Dysplastic melanocytes may invade beyond the epithelium into the substantia propria or into the globe or lids.

h) Nevus

Conjunctival nevi begin to appear in childhood. It is unusual for it to first appear after the age of 35. Clinically, these lesions are elevated, variably pigmented, and may have subtle cystic spaces seen on slit lamp. Histologically, the nevus cells may lie purely at interface of the epithelium and substantia propria (junctional nevus), both within the epithelium and substantia propria (compound nevus), or totally in the subepithelial tissue (subepithelial nevus). Compound nevi of conjunctiva often show characteristic epithelial-lined inclusion cysts. These benign melanocytes form nests with minimal signs of cellular activity.

i) Pinguicula/Pterygium

These entities are benign, reactive, proliferative lesions of the conjunctiva characterized clinically by a yellow-white thickening with increased vascularity. Sunlight and other environmental exposure are thought to be predisposing elements. These lesions are both identical histopathologically with the pinguncula being limited to the area of the conjunctiva and the pterygium encroaching on the cornea in a wing like fashion. Histologically, these lesions are characterized by degeneration of collagen in the snbstantia propria of bulbar conjunctiva (elastoid and basophilic degeneration). The overlying epithelium is either thinned (atrophy) or thickened (proliferative-either dysplastic or hyperplastic) and shows no atypia. The pterygium encroaches on to the cornea and hence can interfere with vision and so should be excised. The destruction of the Bowman's layer by advancing fibrovascular tissue results in corneal scarring.

The recurrent pterygia lack the histo-pathologic features of elastotic degeneration and are more accurately an exuberant granulation tissue response.

j) Primary Acquired Melanosis

Primary acquired melanosis (PAM) is patchy, flat, acquired conjunctival pigmentation. Histologically, PAM may have one of two main patterns:

i) PAM without atypia is a histologic term denoting either increased pigmentation within the epithelium without hyperplasia of melanocytes or hyperplasia of the basal melanocytes lacking cytologic atypia.

ii) PAM with atypia denotes atypical melanocytes which may involve or replace the epithelium with spindle or epithelial cells.

PAM without atypia does not usually progress to melanoma, whereas almost half of the cases of PAM with atypia eventually progressed to melanoma.

k) Pyogenic Granuloma

Clinically this entity appears as a fleshy, red, often pedunculated lesion arising as a consequence of some other inflammatory process such as a chalazion, severe blepharitis, or foreign body reaction. The name is a double misnomer—the lesion is neither pyogenic nor a granuloma. Pathologically, it is an inflammatoiy lesion composed of granulation tissue (proliferative connective tissue—fibroblasts and Gbrocytes—and newly formed capillary channels) with interspersed acute and chronic inflammatory cells.

l) Squamous Papilloma

Squamous papillomas of the conjunctiva are benign lesions probably of viral etiology. Clinically, these lesions tire elevated with an irregular papillomatous surface. They may occur singly or multiply. They are histologically similar to squamous papilloma of skin, containing a branching fibrovascular core covered by acanthotic or thickened conjunctival epithelium with minimal atypia.

in) Squamous Cell Carcinoma

Clinically, these lesions appear quite similar lo conjunctival intraepithelial neoplasms, although they may show more extensive encroachment onto the cornea. Pathologically, these lesions are characterized by a marked dysplasia of the epithelium which has invaded through the epithelial basement membrane. These may be sub-classified as microinvasive which involves only the substantia propria or invasive which can involve the cornea, sclera, eyelid, or even orbit. The deep squamous cells may show signs of dyskeratosis and may also form nests with central keratin pearls.

Check Your Progress 1

1) Describe the histology of conjunctiva,

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2) List the types of conjunctivitis.

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3) List the WHO criteria for the diagnosis of Trachoma.

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19.2.2 Cornea

The cornea is a multilayered structure which is covered on the external surface by a stratified squamous, non-keratinized epithelium. The epithelium is firmly attached to the corneal surface by its basement membrane (Fig. 19.2). The majority of the cornea is made up of stroma. The anterior most stroma consists of a condensation of tissue called Bowman's layer. This layer does not regenerate following trauma or injury. The stroma itself is composed of regular lamellae of collagen with the predominant cell type being the keratocyte (a modified quiescent fibroblast). The posterior surface of the cornea is lined by an endothelium which consists of a monolayer of very metabolically active cells. The basement membrane of the corneal endothelium is called Descemet's membrane.

Fig. 19.2: Normal corneal histology

Important disorders that affect cornea are:

1) Corneal Abrasions

Abrasions can be traumatic or secondary to foreign body, or by improper use of contact lenses. The trauma results in partial or complete removal of a focal area of the epithelium on the cornea causing severe pain, watering and photophobia. Immediate treatment leads to a complete healing and regeneration of the epithelium.

2) Corneal Infections

a) *Bacterial Ulcer*

Bacterial ulcers often show initial destruction of epithelium and Bowman's layer followed by varying degrees of stromal desti-uction which is enhanced by collagenase produced by the injured epithelial cells, keratocytes, and inflammatory cells. The onset is acute and may be rapidly progressive. Pathologically, the infiltrate is composed of acute inflammatory cells such as polymorphoneutrophils (PMN's). The stroma shows collagen necrosis. The epithelium terminates abruptly at the margin of the ulcer. Common causative agents include Staph aureus, Pneumococcus, Strep, E. coli, Psoteus, Klebsiella, and Pseudomonas. If not treated properly, this entity may lead to corneal perforation or extensive scan-ing.

b) *Fungal Ulcers*

These lesions have a more insidious onset, seen at 8-15 days after trauma usually involving vegetative material, in immunocompromised subjects or after injudicious use of topical cortico-steroids. Agents include Candida, Aspergillus, Cephalospoiium, and Fusarium. Clinically, the lesion is usually associated with a hypopyon, has satellite lesions around the ulcer, and has an immune ring of Wessely (PMNs and plasma cells surrounding the central lesion with an intervening area of uninvolved tissue). Hislologically, its appearance is similar to bacterial ulcer except one often sees eosinophils and may see a granulomatous response. Special stains are required to see the fungal elements.

c) *Viral Ulcers/Keratits*

Herpes Simplex Keratitis

Herpes simplex keratitis is usually a self-limited epithelial disease characterized by a linear arborizing pattern of opacification and swelling of epithelial cells with central ulceration (dendrite formation). Histologically two structures in the involved epithelium and superficial stroma are diagnostic of HSV:

i)   Multinucleated giant cells and

ii) Intranuclear inclusions (inclusion of Lipschutz or Type A Cowdry inclusions) which are a densely-staining mass in the nucleus surrounded by a halo. Cellular infiltration may be PMNs and/or chronic inflammatory cells. A post-herpetic ulcer and a disciform (immune disease) keratitis involve deep stroma.

Herpes Zoster infection may involve the ophthalmic branch of the trigeminal nerve with lesions noted unilaterally in the classic distribution involving the face, scalp, and forehead. In addition, these lesions may involve the eyelids, as well as the side and tip of the nose. Ocular complications may occur in approximately 50 per cent of patients with herpes zoster ophthalmicus. Corneal involvement inay be seen in several different forms. Patients may have an interstitial or stromal keratitis from this infection which is characterized by vascularization deep in the corneal stroma just anterior to Descemet's membrane. In addition, herpes zoster inay lead to corneal ulceration and melting with eventual perforation. Histologicnlly, the cornea shows signs of a sterile melting with a moderate, predominately lymphocytic infiltrate.

d)   *Acanthamoeba Keratitis*

Acanthamoeba is a not so common but potentially very serious corneal infection that has been most frequently associated with contact lens wearers who do not take appropriate precautions in cleaning and sterilising their tenses. Acanthamoeba organisms are ubiquitous, free-living protozoan which are found naturally in soil and fresh water. Progression of this infection may lead clinically to a ring-shaped infiltrate of the cornea with infiltrates nlong the corneal nerve and hence a severe degree of associated pain. The clinical diagnosis is often difficult, and this condition may clinically be confused with fungal or herpetic keratitis. Histopathologically, the corneal stroma is invaded with multiple acanthamoeba cysts, as well as trophozoites, in areas of stromal necrosis and inflammation.

3)   Keratoconus

This is a non-inflammatory condition characterised clinically by a bilateral (and frequently asymetrical) central ectasia of the cornea with anterior protrusion in a cone-like fashion. This may give rise to myopia and irregular astigmatism. Histologically, the epithelium and the stroma of the cornea centrally are often thinned. There can also be multiple focal disruptions of epithelial basement membrane and Bowman's layer anteriorly. Central stromal thinning and anterior stromal scarring are usually present. In addition, Descemet's membrane may rupture in this condition resulting in acute corneal edema or hydrops.

4)   Band Keratopathy

This entity is characterized clinically by a band of brownish-staining material in the anterior cornea in the inter-palpebral zone. The condition is thought to be caused by chronic inflammation of the eye, as well as systemic disorders of calcium and phosphate. Pathologically, this entity is characterized by an intense calcification located underneath the epithelium nlong Bowman's layer and often involving the anterior stroma. Special stains arc necessary to confirm the presence of calcium. This is often associated with a pannus formation.

5)   Blood Staining

Corneal blood staining may occur in situations where there is long standing anterior chamber hemorrhage or hyphema present. This is more likely to occur if the intraocular pressure is also increased or if the corneal endothelium has been damaged. Breakdown products of red blood cells, such as hemoglobin, may diffuse into the stroma causing staining of the cornea. Histologically, the corneal blood staining is characterized by small light-red or brown staining globules or spheres which are located predominately between the corneal lamellae. Corneal blood staining may clear over a long period of time (months to years) beginning in the periphery.

6) **Dystrophies**

The corneal dystrophies are a group of hereditary disorders with bilaterally symmetrical clouding of cornea developing at variable age. They can be epithelial, stromal or endothelial. Few common dystrophies are described here.

a) *Lattice Dystrophy*

This entity is an autosomal dominant stromal dystrophy which is characterized clinically by multiple lines in the anterior stroma forming a lattice-like configuration. Histopathologically, the corneal stroma is invaded by an amyloid-like material. This material stains positively on Congo red staining and shows characteristic metachromasia and birefringence.

b) *Macular Dystrophy*

This is a recessive dystrophy of the coineal stroma which is characterized by cloudy opacities of the stroma which may coalesce and involve the entire stroma leading to corneal clouding. Histologically, the stroma is invaded by a mucopolysaccharides material which is seen best on Alcian blue stain.

c) *Meesman's Dystrophy*

This entity is an autosomal doininant coineal dystrophy which is characteiized by multiple tiny cysts or vacuoles in the epithelium. Histopathologically, the chasacteristic finding on this lesion consists of small cysts in the epithelium which is comprised of "peculiar substance".

d) *Reis-Buckler's Dystrophy*

This entity is an autosomal dominant anterior corneal dystrophy which is characteiized by multiple small discrete opacities seen centrally just under the epithelium which may have a honeycomb pattern. Histologically, this entity is characterized by scarring and thickening of collagen in the area of Bowman's membrane with loss of epithelial adhesions.

e) *Granular Dystrophy*

This is an autosomal dominant corneal stromal dystrophy which is characterized clinically by small opaque granules which are sharply defined with clear spaces between them. Histopathologically, the corneal stroma shows granular eosinophilic deposits which are thought to be hyalin in nature and are scattered throughout the stroma, although more commonly seen anteriorly. These deposits may be seen well on a trichrome stain, .

f) *Fuch's Endothelial Dystrophy*

This entity is thought to be a degenerative process as a result of endothelial dysfunction and occurs more frequently in women, Because endothelial cells are functioning poorly and are decreased in number, edema develops, first involving the stroma and then involving the epithelium. Epithelial bullae may rupture causing recurrent episodes of pain. Histopathologically, this entity is characterized by multiple wart-like exc escences on Descemet's membrane which are called guttata. In addition, there is a progressive loss of endothelial cells as well as a thickening of Descemet's membrane in this entity. It is a leading cause of Bullous Keratopathy.

7) **Pannus**

Clinically, this entity is characterized by the proliferation of fibrous or vascular tissue under the epithelium in the anterior cornea. This may be caused once again by chronic inflammation in the eye. Pathologically, Bowman's layer and the superficial stroma will be invaded by a mass of vascular, fibrovascular, or inflammatory tissue.

8) **Bullous Keratopathy**

The endothelial pump of the cornea may fail due to the abnormal functioning of
the endothelial cells or by a physical decrease in their number. Endothelial pump
system failure may lead to persistent corneal stromal edema, which if unrelieved
leads to formation of vesicles called "Bullae". Bullae may form between the
corneal epithelium and underlying Bowman's layer due to degeneration of the
epithelial basement membrane with loss of hemidesmosomes secondary to edema
of the basilar cell layer. In late stages, the bullous cavity is replaced by an
organized fibrous tissue ("plaque") interposed between the epithelium and
Bowman's layer. The endothelial cell layer is characteristically absent or markedly
attenuated. Bullous keratopathy continues to be seen after cataract surgery with or
without IOL implantation, but it's incidence has drastically decreased.

Check Your Progress 2

1) Discuss causes of corneal infection.

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2) List corneal dystropies.

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19.3 PATHOLOGY OF GLAUCOMA

The pathology of glaucoma will be discussed under following headings:

a) **Changes in the Angle/Trabecular Meshwork**

    i) *Open Angle Glaucoma*

        Open angle glaucoma is an entity in which the intraocular pressure is
        elevated, leading to optic nerve and retinal damage.with progressive
        loss of visual field. Although the angle is often normal in open angle
        glaucoma, some pathologic studies have found an increase in the
        compression or sclerosis of the trabecular bars with some interruption
        of the endothelial cell lining leading to high resistance to the aqueous
        outflow through the trabecular meshwork and sclemm's canal angle
        tissue (Fig. 19.3).
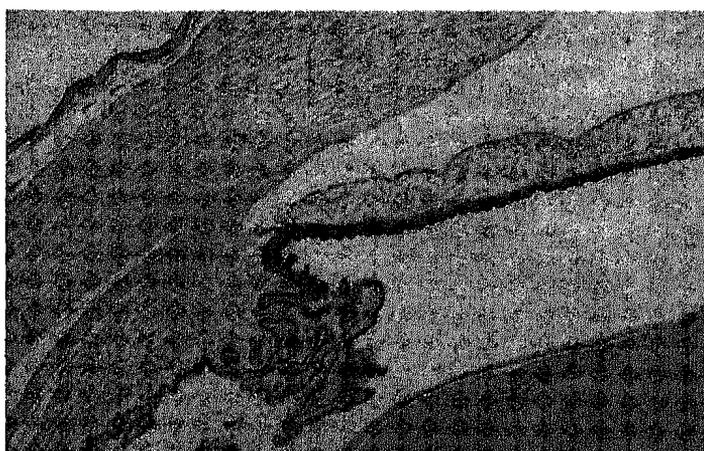
. **Fig. 19.3: Normal angle structures**

ii) *Angle Closure Glaucoma*

Glaucoma may also occur due to anything which causes the iris lo become opposed to the peripheral cornea, closing off the anterior chamber angle. It may be primary due to relative pupillary block or may be secondary to another eye disease (causing mechanical obstruction or inflammation with fibrotic changes). Another common cause of secondary angle closure glaucoma is neovascularization of the iris surface or rubeosis iridis. Histopathologically, the iris can be seen adherent to the posterior surface of the cornea, thus closing off the trabecular meshwork. In cases of chronic inflammation, the angle is closed off by a proliferation of fibrous tissue. In the case of neovascular glaucoma, there is a sheet of fine vascular tissue which grows along the surface of the iris with secondary fibrosis that lends to closure of the angle.

iii) *Secondary Glaucoma with Material in Trabecular Meshwork*

Depending on the source, cause and type of material obstructing the outflow, this can be further subdivided into the following:

Exfoliation Syndrome

This is found in older age groups and is characterised by deposition of PAS positive fibrils on the lens capsule, zonules, iris, ciliary body and trabecular meshwork. The deposition in the trabecular meshwork leads to secondary obstruction to the outflow and hence raised IOP.

Phacolytic Glaucoma

This occurs due to obstruction of the meshwork by the leaked lens proteins of a hypermatuse cataract. In addition to the protein, macrophages and vacuoles are also found at the angle.

Trauma Ghost Cell Glaucoma

It is due to deposition of the blood breakdown products (ghost erythrocytes) in the trabecular meshwork after intra-ocular hemorrhages. **Bemolytic Glaucoma** is due to hemoglobin laden macrophages obstructing the meshwork.

iv) *Secondary Open Angle Glaucoma*

Open angle glaucoma can occur due to any process which leads to invasion of the angle structures by abnormal cells. One entity that can do this is a malignant melanoma at the base of the iris or at the ciliary body. The melanoma cells may then invade the trabecular meshwork tissue leading to secondary glaucoma. Histopathologically, this is characterized by variably pigmented melanoma cells which are seen to directly invade the trabecular meshwork and other structures of the anterior chamber angle.

Note

Angle Recession *Glaucoma*

a) This specific entity, although different from all olhers, deserves a special mention here. A contusion injury of the globe may result in a secondary glaucoma in which the iris is recessed. This glaucoma may occur years following a blunt injury to the eye with contusion. Histopathologically, this entity is characterized by recession of the iris with a tear occurring at the face of the ciliary body. The anterior chamber angle is thus recessed posteriorly. The trabecular meshwork may appear normal initially and may later show mild signs of sclerosis, •

b) **Optic Nerve** *Changes*

Glaucomatous damage of the optic nerve is characterized by progressive increase in cupping with loss of neuroretinal rim. Clinically, this will appear as a deepening of the cup with a widening of the cup-to-disc ratio and progressive loss of

neuroretinal rim area. In its end stages, the cupping is quite extensive with almost complete loss of normal optic nerve head tissue. Seen on gross examination, the optic nerve is white and atrophic with a deep cupping and extreme nasal displacement of vessels. Histopathologically, the most prominent change seen in glaucoma is that of extensive cupping with posterior bowing and compressions of the tissues of the lamina cribrosa. There is often an undermining of the tissue at the rim of the scleral canal. There is extensive atrophy of axonal tissue and displacement of vessels from the retina. The optic nerve tissue itself may show marked atrophy of axons with increased gliosis in the optic nerve parenchyma. The retina shows atrophy or complete absence of the ganglion cell layer in end-stage glaucoma.

Fig. 19.4: Glaucomatous optic disc

Check Your Progress 3

.Describe optic nerve changes in glaucoma,

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19.4 PATHOLOGY OF LENS

The normal human crystalline lens occupies the space in the posterior chamber behind the iris and pupil (Fig. 19.5). The lens is attached to the ciliary body by multiple bundles of zonules. The lens itself is surrounded by a thick lens capsule, which is the basement membrane of the lens epithelial cells. Anteriorly, the lens capsule is thicker, and there is a monolayer of epithelial cells seen to underlie it. The lens epithelial cells migrate to the area of the equator and fan out forming lens cortical fibers. Normally there are no lens epithelial cells seen underlying the thinner posterior capsule. The lens nucleus and cortex are often difficult to distinguish on histopathologic examination.
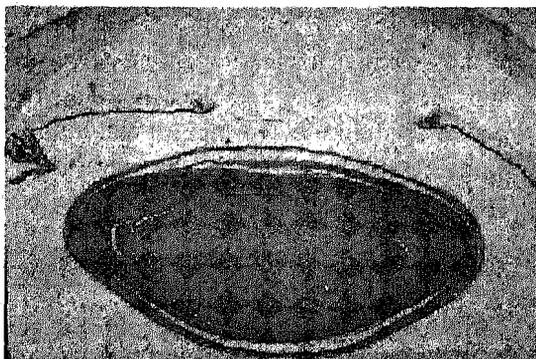
**Fig. 19.5: Normal Crystalline Lens.**

The common disorders of lens are:

- Cataracts

- Phacoanaphylactic Endophthalmitis

- Pseudoexfoliation

a) **Cataracts**

Progressive opacity of the normally clear ciystalline lens can lead to formation of a cataract (Fig. 19.6). These changes may involve both the nucleus, the cortex and the epithelium of the lens.

i) Nuclear changes are characterized by progressive crosslinking and insolubilty of 'crystalline proteins and also some accumulation of the urochrome pigment. This leads to a progressive hardening of the nucleus and discoloration which is initially yellow and. can eventually become a dark brown or brunescent colour (Fig. 19.7). The nuclear cataracts are difficult to assess histologically as they take on a subtle homogenous eosinophilic appearance.

ii) Cortical cataracts are generally associated with nuclear sclerosis and posterior sub-capsular cataracts. Cortical changes may begin as small peripheral water clefts with globular degenerative changes of the corlex. This may eventually coalesce into dense bands of opaque cortical material. Light microscopy shows the accumulation of eosinophilic globules (morgagnian globules) in slit like spaces between the lens fibres, which is a reliable sign of cortical degeneration.
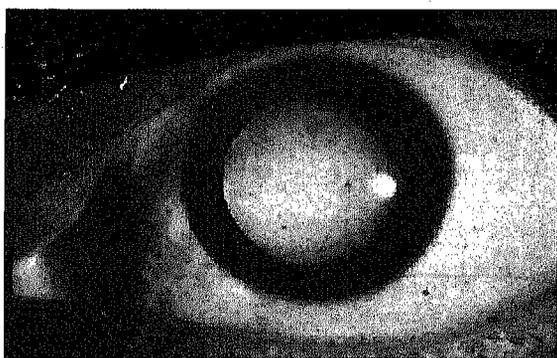
Fig. 19.6: Cortical cataract	Fig. 19.7: Nuclear cataract

iii) Posterior Subcapsular Calaractous changes begin with the epithelial disarray at the equator, with posterior migration of the lens epithelium. As these cells migrate posteriorly, they enlarge and swell (Bladder cells/Wedl cells). Also, anterior lens epithelial cells may undergo a fibrous metaplasia leading to a thick fibrous plaque between the anterior lens capsule and the anterior epithelial cells.

Congenital Cataract

Congenital cataracts are those which become apparent anywhere from birth to within the first six months of life. These cataracts may show many different patterns.The opacity may be confined to the area of the embryonic or fetal nucleus with clear cortex surrounding this. In addition, congenital cataracts may be manifest as a sutural type of cataracts which show a characteristic "Y" or dendritic pattern.

Morgagniun Cataract

A long-standing or very mature cataract may undergo liquefaction of the lens cortex. The dark brown, mature nucleus then sinks inferiorly in the fluid filled

capsular sac due to the forces of gravity. This may be noted clinically with the brown nucleus seen lying inferiorly with in the capsular sac (Fig. 19.8). Examination of a Morgagnian cataract grossly reveals a very hard, brunescent appearing nucleus with a markedly liquified cortex and loosely wrinkled capsular bag.
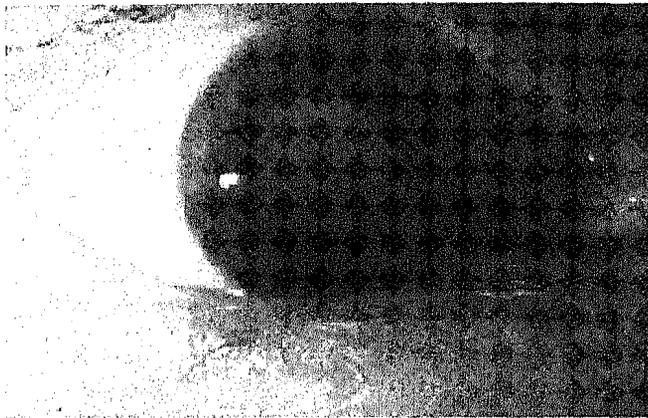
Fig. 19.8: Morgagnian cataract

Sommering Ring Cataract

Rupture of the lens capsule, either traumatically or iatrogenically, may lead to loss of the lens nucleus and much of the anterior and posterior cortex. Remnant peripheral or equatorial lens cortex, as well as proliferating lens epithelial cells in the periphery, form a Sommering's ring. This is characterized by a peripheral dough nut or ring-shaped configuration when viewed grossly. When viewed grossly in cross-section, the Sommering's ring appears to have a dumbbell configuration. Histopalhologically, a Sommering's ring catamct is characterized by proliferating lens epithelial cells in the periphery or equatorial region of the lens, as well as remnant, trapped degenerated lens cortex.

b)   **Phacoanaphylactic Endophthalmitis**

Phacoanaphylactic endophthalmitis is an inflammatory ocular condition secondary to rupture of the lens capsule, either traumatically or iatrogenically. This disease is thought to be due to an auto-immune process by which previously sequestered lens proteins are liberated or release through a ruptured lens capsule leading to an autosensitization. Histopathologically, this condition is characterized by a zonal. granulomatous inflammation which surrounds the area of ruptured lens capsule and cortical material. The innermost part of the reaction consists of polymorphonuclear cells and multi-nucleated giant cells. Finally, the outer-most layer of this zonal inflammation consists of lymphocytes and plasma cells which surround the epithelioid cells.

c)   **Pseudoexfoliation**

Pseudoexfoliation of the lens capsule or the exfoliation syndrome is a condition seen most commonly in Scandinavian or northern European people. It is characterized by a deposition of a white fluffy material on the anterior lens capsule with a relatively clear zone corresponding to the movement of the iris. In addition, this material can also be deposited on the zonules, iris pigment epithelium, ciliary epithelium, and trabecular meshwork. This condition may lead to glaucoma, as well as weakness of the zonules. This material is felt to be composed of abnormal basement material produced by all of the epithelial cells within the anterior segment of the eye. Histopathologically, this condition is characterized by tiny, pink eosinophilic-staining deposits on the anterior lens capsule which line up perpendicular to 'the edge of the lens capsule.

Check Your Progress 4

1) List the various morphological types of cataract.

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2) Discuss pathology of senile cataract.'

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3) Describe Morgagnian cataract.

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19.5 LET US SUM UP

In this unit, you have read about pathology of anterior segment of the eye in this unit. Corneal inlections, conjunctivitis, cataract and glaucoma are the frequently encountered in ophthalmic practice. Knowledge of their pathology will help you diagnose and understand their management more wisely. Role of an ophthalmic technician in screening lor glaucoma cannot be overemphasized. Early diagnosis is most effective measure to prevent irreversible blindness caused by this disorder. In next unit you will learn about pathology of lid, adnexa, orbit, optic nerve, uvea and retina.

19.6 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

1) Histologically conjunctiva consists of

    a) Nonkeratinized stratified columnar epithelium with goblet cells

    b) Substantia propria: loose connective tissue stroma. Palpebral conjunctiva is firmly adherent to the tarsus. Substantia propria of the bulbar conjunctiva is areolar and thus permits the chemosis.

2) Conjunctivitis

    a) Acute

        i)   Viral

        ii) Bacterial

    b) Chronic

        i)   Follicular

            1) Infectious— Acute and Chronic

            2) Non-infectious

        ii) Papillary hypertrophy

3) WHO Diagnostic Criteria for Trachoma (must have at least 2)

    a) Follicles on the upper tarsus

    b) Conjunctival scarring (Arlt's line)

    c) Vascular pannus

    d) Limbal follicles (>5, each >5 mm) or remnants of limbal follicles (Herbert's pits).

Check Your Progress 2

1) The important causes of corneal infections are:

    a) Bacterial Ulcer

    b) Fungal Ulcers

    c) Viral Ulcers

    d) Acanthamoeba keratitis

2) Corneal dystrophies can be listed as:

    a) Lattice Dystrophy

    b) Macular Dystrophy

    c) Meesman's Dystrophy

    d) Reis-Buckler's Dystrophy

    e) Granular Dystrophy

    f) Fuch's Endothelial Dystrophy

Check Your Progress 3

Glauconiatous damage of the optic nerve is characterized by progressive increase in cupping with loss of neuroretinal rim. Clinically, this will appear as a deepening of the cup with a widening of the cup-to-disc ratio and progressive loss of neuroretinal rim area. In its end stages, the cupping is quite extensive with almost complete loss of normal optic nerve head tissue. Seen on gross examination, the optic nerve is white and atrophic with a deep cupping and extreme nasal displacement of vessels. Histopathologically, the most prominent change seen in glaucoma is that of extensive cupping with posterior bowing and compressions of the tissues of the lamina cribrosa. There is often an undermining of the tissue at the rim of the scleral canal.

Check Your Progress 4

1) The common types of cataracts are

    a) Nuclear cataract

    b) Cortical cataract

    c) Posterior subcapsular cataract

    d) Congenital Cataract

    e) Morgagnian Cataract

    f) Sommering Ring Cataract

2) Progressive opacity of the normally clear crystalline lens can lead to formation of a cataract. These changes may involve both the nucleus, the cortex and the epithelium of the lens. Nuclear changes are characterized by progressive crosslinking and insolubilty of crystalline proteins and also some accumulation of the urochrome pigment. This leads to a progressive hardening of the nucleus and discoloration which is initially yellow and can eventually become a dark brown or brunescent colour. The nuclear cataracts are difficult to assess histologically as they take on a subtle homogenous eosinophilic appearance. Cortical cataracts are generally associated with nuclear sclerosis and posterior sub-capsular cataracts. Cortical changes may begin as small peripheral water clefts with globular degenerative changes of the cortex. This may eventually coalesce into dense bands of opaque cortical material. Light microscopy shows the accumulation of eosinophilic globules (morgagnian globules) in slit like spaces between the lens fibres, which is a reliable sign of cortical degeneration. Posterior Subcapsular Cataractous changes begin with the epithelial disarray at the equator, with posterior migration of the lens epithelium. As these cells migrate posteriorly, they enlarge and swell (Bladder cells/Wedl cells). Also, anterior lens epithelial cells may undergo a fibrous metaplasia leading to a thick fibrous plaque between the anterior lens capsule and the anterior epithelial cells.

3) A long-standing or very mature cataract may undergo liquefaction of the lens cortex. The dark brown, mature nucleus then sinks inferiorly in the fluid filled capsular sac due to the forces of gravity. This may be noted clinically with the brown nucleus seen lying inferiorly with in the capsular sac.