The ministry of health of the Republic of Belarus

Educational institution «Gomel State Medical University»

Department of Otorhinolaryngology with a course of ophthalmology

Discussed at the meeting
department
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MANUALS

for 6th year students of faculty of foreign students on ophthalmology

TOPIC №2. PATHOLOGY OF THE EYELIDS, CONJUNCTIVA, LACRIMAL SYSTEM, CORNEA, SCLERA AND LENS. PATHOLOGY OF THE OF CHORIOIDEA AND RETINA. CHANGES IN THE ORGAN OF VISION IN GENERAL DISEASES.

The time 6 hours

Gomel, 2023

MOTIVATION FOR LEARNING TOPICS, TRAINING AND EDUCATIONAL PURPOSES, THE REQUIREMENTS TO THE ORIGINAL LEVEL OF KNOWLEDGE

Conjunctivitis and blefaritis refers to the inflammation or infection of the conjunctiva and eyelids. It is the most common presentation of eye redness in both primary care and the emergency department, therefore putting a large strain on the healthcare system. This activity will provide the learner with information regarding the etiology, pathophysiology, clinical presentation, evaluation, and management of blefaritis and conjunctivitis, and will review the role of the interprofessional team in improving the care of patients with this diseases and preventing transmission.

Corneal disease remains the leading cause of monocular blindness worldwide, especially affecting marginalized populations. Corneal opacities, which are largely caused by infectious keratitis, are the fourth leading cause of blindness globally and are responsible for 10% of avoidable visual impairment in the world's least developed countries. Approximately 2 million people develop a corneal ulcer every year in India alone. In the United States infectious keratitis is often associated with contact lens wear, but in developing countries it is more commonly caused by ocular trauma sustained during agricultural work.

Scleritis is a severe ocular inflammatory condition affecting the outer covering of the eye. Scleritis has high association with systemic disease. This activity reviews the evaluation and treatment of scleritis and highlights the role of the interprofessional team in caring for patients with this condition.

Cataracts are a major cause of visual impairment in older adults. When the transparency of the crystalline lens decreases enough to disturb vision, a clinically significant cataract exists. Such a decrease is usually the result of scattering of light rays or absorption in the axial part of the lens; similar changes in the peripheral parts of the lens may exist without loss of vision. Although these changes in the periphery are strictly cataractous in nature, surgical intervention is rarely warranted in the absence of visual symptoms.

Systemic diseases are diseases that involve many organs or the whole body. Many of these diseases also affect the eyes. In fact, an eye exam sometimes leads to the first diagnosis of a systemic disease.

The purpose of the class: To study inflammatory, allergic diseases of the eyelids, anomalies in the development and position of the eyelids, neoplasms of the eyelids. Pathology of the lacrimal producing and lacrimal discharge apparatus. Research methods of the lacrimal organs. Principles and methods of treatment of dacryocystitis. Methods of etiological diagnosis, characteristic clinical signs, causes of possible complications of gonoblennoe, diphtheria, adenovirus, pneumococcal, acute epidemic, spring conjunctivitis. Differential diagnosis of conjunctivitis. Chronic conjunctivitis. Pathogenesis, clinic, principles and duration of treatment, measures for the prevention of trachoma. Complications, outcomes.

To give students an understanding of the anatomical and topographic features of the structure of the cornea, sclera, and oculomotor apparatus. To study the pathology of diseases of the cornea, sclera, oculomotor apparatus of the eye, methods for their diagnosis and treatment.

To give students an idea of the types, frequency and methods of diagnosing the lens pathology. Classification of cataracts. Modern treatment principles: seamless cataract surgery. To acquaint students with abnormalities of the lens. Changes in Marfan's disease, Marchezani and other syndromes. Methods and terms of treatment, outcomes. To acquaint students with the frequency of vascular tract diseases among the common eye pathology. The structure of the diseases of the vascular tract (inflammatory, dystrophic processes, neoplasms, congenital anomalies). Severe outcomes as a cause of low vision and blindness.

Objectives of the lesson:

Student should know:

1. Clinic, diagnosis and treatment of inflammatory diseases of the eyelids.
2. Clinic, diagnosis and treatment of inflammatory conjunctival diseases.
3. Differential diagnosis of conjunctivitis.
4. Pathology of the lacrimal and tear-producing apparatus. Research methods of the lacrimal organs.
5. Pathogenesis, clinic, principles and duration of treatment, measures for the prevention of trachoma. Complications, outcomes.
1. General symptoms, classification of keratitis. Methods for the diagnosis of keratitis.
2. Complications, outcomes of injuries and diseases of the cornea. Keratoplasty.
3. Inflammation of the sclera: episcleritis, scleritis, abscesses of the sclera (clinic, diagnosis, treatment).
4. Definition and classification of strabismus.
5. Dates, stages and succession in the treatment of friendly strabismus.
6. Types and frequency of the lens pathology. Diagnostic methods for the pathology of the lens.
7. Indications for surgical treatment of the lens pathology. Methods of cataract extraction. Cryoextraction. Phacoemulsification. Indications, terms of surgical treatment, outcomes.
8. Congenital cataracts. Classification of cataracts in children. Indications for surgical treatment, depending on the size of the cataract, its location, visual acuity, age of the child.
9. Intraocular correction of aphakia.
10. Vascular tract inflammation. Etiology, pathogenetic mechanisms of development of uveitis.
11. Dystrophic diseases of the iris and ciliary body. Forms (chronic ciliary body dysfunction, Fuchs syndrome). Differential diagnosis with anterior uveitis. Clinic, course, treatment principles.

12. Ophthalmic symptoms in cardiovascular, neurological, infectious diseases, HIV infection, blood diseases and endocrine pathology.

***Student must be able to*:**

1. Instillation drops in the conjunctival cavity.
2. Determine the sensitivity of the cornea.
3. Write out prescriptions for anti-inflammatory drops.
4. Determine the nature of vision.
5. Determine the strabismus angle.
6. Instillation drops in the conjunctival cavity.
7. Determine the sensitivity of the cornea.
8. Write out prescriptions for anti-inflammatory drops.
9. Determine the nature of vision.
10. Perform lens biomicroscopy.
11. Inspection of eye environments in transmitted light.
12. Assign treatment to a patient with an initial cataract.
13. Assign treatment to a patient with acute iridocyclitis.

***Student must be able to*:**

1. Instillation drops in the conjunctival cavity;
2. Turn out the upper eyelid;
3. Write prescriptions for anti-inflammatory drops;
4. Massage the eyelids;
5. Conduct a tubular test;
6. Conduct a Schirmer test;
7. Instillation of drops and insertion of ointments and medicinal eye films into the conjunctival cavity, washing of the conjunctival cavity;
8. Determine the sensitivity of the cornea;
9. Prescribing prescriptions for medicines, drawing up medical documentation (outpatient records, medical records of inpatients, etc.);
10. Determine the nature of vision;

The student must perform the following practical skills:

1. Instillation of drops and insertion of ointments and medicinal eye films into the conjunctival cavity, washing of the conjunctival cavity.
2. Eyelid massage.
3. Prescribing prescriptions for medicines, drawing up medical documentation (outpatient records, medical records of inpatients, etc.).
4. Determining the presence of contents in a tear bag.
5. Check the patency of the lacrimal tubules and the lacrimal-nasal canal.
6. Anatomy, histology of the cornea, sclera.
7. Physiology of the oculomotor apparatus.
8. Study of optical media with transmitted light.
9. Biomicroscopy. Principle of operation. Opportunities.

10. Study of the optical media of the eye using lateral illumination. Advantage over conventional diffused lighting.

CHECKLIST OF QUESTIONS FROM RELATED SUBJECTS

1. Anatomy of the lacrimal and nasal canal.
2. The histological structure of the conjunctiva.
3. General diseases, systemic and syndromic lesions, accompanied by ocular symptoms.
4. Pharmacokinetics and pharmacodynamics of ophthalmic anti-inflammatory drug.
5. Keratitis. General symptoms, classification of keratitis. Features of the keratitis clinic in children. Exogenous (infectious bacterial, fungal and secondary catarrhal keratitis), endogenous (herpetic, tuberculous, syphilitic) keratitis. Keratitis caused by shingles virus. Neuroparalytic keratitis. Vitamin keratitis. Keratitis of unknown etiology. Diagnostic Methods The average duration of keratitis of various etiologies. Principles and duration of treatment. Complications, outcomes of injuries and diseases of the cornea. Keratoplasty.
6. Congenital malformations of the cornea. Micro and macrocornea, keratoconus, keratoglobus. Dystrophy and degeneration of the cornea. Tumors of the cornea.
7. Inflammation of the sclera: episcleritis, scleritis, abscesses of the sclera. Anomalies in the color and shape of the sclera. Blue sclera syndrome, melanosis, staphylomas.
8. Anatomy, histology of the lens.
9. Anatomy, physiology of the vascular tract.
10. Gene, genomic and chromosomal mutations leading to the development of congenital cataracts.

CHECKLIST OF CONTROL QUESTIONS FOR THE LESSON

1. Inflammatory diseases of the eyelids. Diseases of the edge of the eyelids. Different types of blepharitis. Diseases of the cartilage of the eyelids, sebaceous and meibomian glands. Conservative and surgical treatment. Allergic diseases of the eyelids. Neoplasms of the eyelids.
2. Anomalies of development and position of the eyelids: microblepharon, cryptophthalmos, ankyloblepharon, coloboma, blepharochalasis, blepharophimosis, eversion, inversion, epicanthus, lagophthalmos. Congenital and acquired ptosis. Complications of ptosis (amblyopia, strabismus). Indications, principles and methods of surgical treatment.
3. Pathology of the lacrimal and tear-producing apparatus. Research methods of the lacrimal organs. Pathology of the lacrimal glands. Sjögren's syndrome. Pathology of the tear ducts. Pathology of the lacrimal openings, lacrimal tubules, the lacrimal sac and the lacrimal and nasal canal. Dacryocystitis of the newborn. Chronic dacryocystitis. Principles and methods of treatment of dacryocystitis. Phlegmon and fistulas of the lacrimal sac. Congenital abnormalities of the lacrimal gland (absence, underdevelopment, omission).

4. Conjunctivitis. Classification. Methods of etiological diagnosis. Frequency, pathogens, pathways of infection, characteristic clinical signs, causes of possible complications of gonoblennoe, diphtheria, adenovirus, pneumococcal, acute epidemic, spring conjunctivitis. Differential diagnosis of conjunctivitis. Chronic conjunctivitis.

5. Chlamydial conjunctivitis (trachoma, adult paratrachoma, conjunctivitis with inclusions of newborns, epidemic chlamydial conjunctivitis, chlamydial conjunctivitis with Reiter's syndrome, zoonotic nature).

6. Trachoma. Pathogenesis, clinic, principles and duration of treatment, measures for the prevention of trachoma. Complications, outcomes.

7. Degenerative conjunctival changes.

8. Tumors of the conjunctiva.

9. Types and frequency of the lens pathology. Diagnostic Methods Classification of cataracts. Modern principles of treatment. Specific gravity in the structure of low vision and blindness.

10. Age-related (senile) cataracts. Cortical, brown, mixed. Clinic. Stages of development of cataracts. Conservative treatment in the initial stages. Indications for surgery. Methods of cataract extraction. Cryoextraction. Phacoemulsification. Indications, terms of surgical treatment, outcomes. Secondary (postoperative cataracts). Causes of occurrence, clinic, treatment.

11. Complicated cataracts. The occurrence of cataracts on the basis of common diseases (diabetes mellitus), common infections (diphtheria, smallpox, malaria), with ocular processes (glaucoma, myopia, uveitis, retinal pigment degeneration, retinal detachment), as a result of poisoning with mercury, nitrites, protein starvation, ionizing radiation, damage, etc. The clinical picture of these types of cataracts. The prognostic value of the occurrence of complicated cataracts in general diseases. Treatment of cataracts depending on the etiology of the process and the degree of clouding of the lens.

12. Congenital cataracts. Classification of cataracts in children. Indications for surgical treatment, depending on the size of the cataract, its location, visual acuity, age of the child. Treatment of obstructive amblyopia.

13. Intraocular correction of aphakia. Aphakia, its signs, the principles of aphakia correction for distance and near vision. Correction of unilateral aphakia. Pathology of the lens.

14. Anomalies of the development of the lens. Changes in the lens in Marfan's disease, Marchezani and other syndromes. Methods and terms of treatment, outcomes.

15. The frequency of diseases of the vascular tract among the common eye pathology. The structure of the vascular tract diseases (inflammatory, dystrophic processes, neoplasms, congenital malformations). Severe outcomes of diseases of the vascular tract as a cause of low vision and blindness.

16. Vascular tract inflammation. Etiology, pathogenetic mechanisms of development of uveitis: infectious-metastatic and toxic-allergic. Classification of uveitis with the course, localization, clinical and morphological picture, etiology. The main morphological, functional signs and developmental mechanisms of anterior uveitis (iridocyclitis); posterior uveitis (choroiditis); Panuveitis. Age features of the course and outcomes of uveitis. The diagnosis of diseases of the vascular tract, depending on the etiology of the clinical, laboratory, radiological and immunological picture (collagenous,

viral, tuberculous, syphilitic, toxoplasmosis, focal, etc.). Organization, principles, methods of general and local treatment of anterior and posterior uveitis, depending on the etiology and nature of the process. Complications of uveitis. Metastatic ophthalmia. Outcomes, prevention.

17. Anomalies of the choroid (aniridia, coloboma, polycoria, pupil ectopia, albinism, aplasia).

18. Dystrophic diseases of the iris and ciliary body. The frequency of the disease. The reasons for the occurrence. Forms (chronic ciliary body dysfunction, Fuchs syndrome). Differential diagnosis with anterior uveitis. Clinic, course, treatment principles.

19.

PRACTICAL PART OF THE LESSON

Work of students is carried out in the ophthalmology department in the presence of the teacher of the department in order to develop and consolidate practical skills. The acquired skills are consolidated in the training room when examining patients or at a seminar. In the classroom, students independently study modern clinical protocols for examination and treatment, methodological recommendations of the ministry of health of the Republic of Belarus.

The control of the final level of knowledge is carried out at a seminar or in the clinical analysis of a patient, an outpatient card or a medical card.

1. Analysis of the thematic patient.
2. Clinical analysis of outpatient cards, medical records.
3. Answers to questions of a computer test program in ophthalmology on the topic «Pathology of the eyelids, conjunctiva and lacrimal gland».

QUESTIONS FOR SELF STUDY AND ADDITIONAL RESEARCH TASKS

1. Diseases of the eyelids.
2. Pathology of the lacrimal and tear-producing apparatus.
3. Conjunctivitis. Classification. Methods of etiological diagnosis. Frequency, pathogens, pathways of infection, characteristic clinical signs, causes of possible complications of gonoblennorea, diphtheria, adenovirus, pneumococcal, acute epidemic, spring conjunctivitis. Differential diagnosis of conjunctivitis. Chronic conjunctivitis.
4. Trachoma. Pathogenesis, clinic, principles and duration of treatment, measures for the prevention of trachoma. Complications, outcomes.
5. Anatomy and histology of the cornea and sclera.
6. Surgical treatment of cornea.
7. Pathology of the lens. Classification. Anomalies of development. Treatment.
8. Age-related cataracts (senile). Stages of development. Modern methods of treatment. Correction of aphakia.
9. Complicated cataracts with common diseases and ocular pathology. Causes. Treatment.
10. Secondary postoperative cataracts, causes, treatment.

11. Congenital cataracts. Causes. Prevention Treatment. Prevention of deprivation and amblyopia.

12. Dystrophic diseases of the iris and ciliary body. Differential diagnosis. Clinic. Treatment.

13. Iridocyclitis. Causes. Clinic. Complications Treatment. Prevention

14. Uveitis. Classification. Symptoms Pathogenesis. The principles of treatment. Prevention

15. Choroiditis. Symptoms Etiology. Clinic. Treatment. Prevention

16. Anomalies in the development of the vascular tract. Clinic. Treatment options.

17.

Conjunctivitis

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

Etiological classification

1. Infective conjunctivitis: bacterial, chlamydial, viral, fungal, rickettsial, spirochaetal, protozoal, parasitic

etc.

2. Allergic conjunctivitis.

Irritative conjunctivitis.

4. Keratoconjunctivitis associated with diseases of

skin and mucous membrane.

5. Traumatic conjunctivitis.

6. Keratoconjunctivitis of unknown etiology.

Clinical classification

Depending upon clinical presentation, conjunctivitis can be classified as follows:

1. Acute catarrhal or mucopurulent conjunctivitis.
2. Acute purulent conjunctivitis
3. Serous conjunctivitis
4. Chronic simple conjunctivitis
5. Angular conjunctivitis
6. Membranous conjunctivitis
7. Pseudomembranous conjunctivitis
8. Papillary conjunctivitis
9. Follicular conjunctivitis
10. Ophthalmia neonatorum
11. Granulomatous conjunctivitis
12. Ulcerative conjunctivitis
13. Cicatrising conjunctivitis

To describe different types of conjunctivitis, a mixed approach has been adopted, i.e., some varieties of conjunctivitis are described by their etiological names and others by their clinical names. Only common

varieties of clinical interest are described here.

INFECTIVE CONJUNCTIVITIS

Infective conjunctivitis, i.e., inflammation of the conjunctiva caused by microorganisms 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.

Viral conjunctivitis

Acute conjunctivitis is a rather common disease, which may affect many people and impose economic and social burdens. Studies have shown that viruses cause up to 35–80% of all cases of acute conjunctivitis and between 65% and 90% of cases of viral conjunctivitis are caused by adenoviruses. In line with these studies, in our investigation Adenovirus was the most commonly isolated causative agent of acute conjunctivitis from both conjunctiva and pharynx samples (74.2% from conjunctiva samples, 32.3% from pharynx samples).

The second most frequently observed causative agents were Enterovirus 70 and Enterovirus 71, respectively. Previously, Li et al. isolated Coxsackievirus A24 as the second most common viral agent following adenoviruses for acute conjunctivitis, but no Enterovirus 70 was isolated. Additionally, adenoviruses were the most frequently identified agents in co-infections of acute conjunctivitis, which is concordant with our data.

Viral conjunctivitis, secondary to adenoviruses, is highly contagious, and the virus spreads through direct contact via contaminated fingers, medical instruments, swimming pool water, or personal items. Hand washing and isolation of the infected patients are essential to avoid transmission. None of the cases have a history of exposure to swimming pool water. In 35% of the cases, the contamination was intrafamilial transmission through hands and personal items. No clinicians involved in sample collection or in treating the patients were contaminated because proper precautions were taken.

Redness, itching, burning, watery discharge, foreign body sensation, follicular conjunctivitis, membrane formation, lymphadenopathy, and hemorrhages are common symptoms in viral conjunctivitis.

The diagnosis of the viral conjunctivitis is usually made on the basis of patient history and clinical findings. Viral cultures by conventional techniques are the gold standard, but may be insensitive for certain samples and take up to 21 days to develop the cytopathic effect. PCR is a useful technique that amplifies small amounts of viral DNA with great sensitivity and specificity. A laboratory confirmation of the virus-related etiology might aid the physician in making an accurate diagnosis and taking hygienic precautions, and therefore reduce the spread of the disease [3].

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 cases and as epidemics. 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 lacunate (Moraxella Axenfeld bacillus) is most common cause of angular conjunctivitis and angular blepharoconjunctivitis.

Pseudomonas pyocyanea is a virulent organism. It readily invades the cornea.

Neisseria gonorrhoeae typically produces acute purulent conjunctivitis in adults and ophthalmia neonatorum in new born. It is capable of invading intact corneal epithelium.

Neisseria meningitidis (meningococcus) may produce mucopurulent conjunctivitis.

Corynebacterium diphtheriae causes acute membranous conjunctivitis. Such infections are rare now-a-days.

C. *Mode of infection.* Conjunctiva may get infectedfrom three sources, viz, exogenous, local surrounding

structures and endogenous, by following modes :

1. *Exogenous infections* may spread: (i) directly through close contact, as air-borne infections or as water-borne infections; (ii) through vector transmission (e.g., flies); or (iii) through material transfer such as infected fingers of doctors, nurses, common towels, handkerchiefs, and infected tonometers.

2. *Local spread* may occur 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 repsonse.* 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 catarrhal or mucopurulent conjunctivitis.
Acute purulent conjunctivitis
Acute membranous conjunctivitis
Acute pseudomembranous conjunctivitis
Chronic bacterial conjunctivitis
Chronic angular conjunctivitis

ACUTE MUCOPURULENT CONJUNCTIVITIS

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

Common causative bacteria are: Staphylococcus aureus, Koch-Weeks bacillus, Pneumococcus and Streptococcus. Mucopurulent conjunctivitis generally accompanies exanthemata such as measles and scarlet fever.

Clinical picture

Symptoms

Discomfort and foreign body sensation due to engorgement of vessels.
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.

Sometimes patient may complain of *coloured halos* due to prismatic effect of mucus present on cornea.

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.

Chemosis i.e., swelling of conjunctiva.
Petechial haemorrhages are seen when the causative organism is pneumococcus.
Flakes of mucopus are seen in the fornices, canthi and lid margins.

Cilia are usually matted together with yellow crusts.

Clinical course. Mucopurulent conjunctivitis 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 toless intense form, the ‘chronic catarrhal conjunctivitis.

Complications. Occasionally the disease may be complicated by marginal corneal ulcer, superficial keratitis, blepharitis or dacryocystitis.

Treatment

1. *Topical antibiotics* to control the infection constitute the main treatment of acute mucopurulent 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 specturm antibiotics. Therefore, treatment may be started with chloramphenicol (1%), gentamycin (0.3%) or framycetin 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 newer antibiotic drops such as ciprofloxacin(0.3%), ofloxacin (0.3%) or gatifloxacin (0.3%) may be used.

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

3. *Dark goggles* may 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.

ACUTE PURULENT CONJUNCTIVITIS

Acute purulent conjunctivitis also known as *acute blenorrhea or hyperacute conjunctivitis* is characterised by a violent inflammatory response. It occurs in two forms: (1) Adult purulent conjunctivitis and (2) Ophthalmia neonatorum in newborn .

ACUTE PURULENT CONJUNCTIVITIS OF ADULTS

Etiology

The disease affects adults, predominantly males. Commonest causative organism is *Gonococcus;* but rarely it may be *Staphylococcus aureus* or *Pneumococcus.* Gonococcal infection directly spreads from genitals to eye. Presently incidence of gonococcal conjunctivitis has markedly decreased.

Clinical picture

It can be divided into three stages:

1. *Stage of infiltraton.* It lasts for 4-5 days and is characterised by:

Considerably painful and tender eyeball.

Bright red velvety chemosed conjunctiva.

Lids are tense and swollen.

Discharge is watery or sanguinous.

Pre-auricular lymph nodes are enlarged.

2. *Stage of blenorrhoea.* It starts at about fifth day, lasts for several days and is characterised by: Frankly purulent, copious, thick discharge trickling down the cheeks. Other symptoms are increased but tension in the lids is decreased.

3. *Stage of slow healing.* During this stage, pain is decreased and swelling of the lids subsides. Conjunctiva remains red, thickened and velvety. Discharge diminishes slowly and in the end resolution is complete.

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.

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* and *N. meningitidis*. Because of the resistant strains penicillin and tetracyline are no longer adequate as first-line treatment. Any of the following regimes can be adopted :

Norfloxacin 1.2 gm orally qid for 5 days

Cefoxitim 1.0 gm or cefotaxime 500 mg. IV qid or ceftriaxone 1.0 gm IM qid, all for 5 days; or _ Spectinomycin 2.0 gm IM for 3 days.

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.

Because of the resistant strains, intensive therapy with penicillin drops is not reliable.

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 per cent eye drops should be instilled once or twice a day if cornea is involved.

6. *Patient and the sexual partner* should be referred for evaluation of other sexually transmitted diseases.

ACUTE MEMBRANOUS CONJUNCTIVITIS

It is an acute inflammation of the conjunctiva, characterized by formation of a true membrane on the conjunctiva. Now-a-days it is of very-*very rare occurrence*, because of markedly decreased incidence of diphtheria. It is because of the fact that immunization against diptheria is very effective.

Etiology

The disease is typically caused by *Corynebacterium diphtheriae* and occasionally by virulent type of *Streptococcus haemolyticus*.

Pathology

Corynebacterium diphtheriae produces a violent inflammation of the conjunctiva, associated with deposition of fibrinous exudate on the surface as well as in the substance of the conjunctiva resulting in formation of a membrane. Usually membrane is formed in the palpebral conjunctiva. There is associated coagulative necrosis, resulting in sloughing of membrane. Ultimately healing takes place by granulation tissue.

Clinical features

The disease usually affects children between 2-8 years of age who are not immunised againstdiphtheria. The disease may have a mild or very severe course. The child is toxic and febrile. The clinical picture of the disease can be divided into *three stages:*

1. *Stage of infiltration* is characterised by: conjunctival discharge and severe pain in the eye.

Lids are swollen and hard.

Conjunctiva is red, swollen and covered with a thick grey-yellow membrane. The membrane is tough and firmly adherent to the conjunctiva, which on removing bleeds and leaves behind a raw area. Pre-auricular lymph nodes are enlarged.

2. *Stage of suppuration.* In this stage, pain decreases and the lids become soft. Themembrane is sloughed off leaving a raw surface. There is copious outpouring of purulent discharge.

3. *Stage of cicatrisation.* In this stage, the raw surface covered with granulation tissue is epithelised. Healing occurs by cicatrisation, which may cause trichiasis and conjunctival xerosis.

Complications

1. *Corneal ulceration* is a frequent complication in acute stage. The bacteria may even involve the intact corneal epithelium.

2. *Delayed complications* due to cicatrization include symblepharon, trichiasis, entropion and conjunctival xerosis.

Diagnosis

Diagnosis is made from typical clinical features and confirmed by bacteriological examination.

Treatment

A. *Topical therapy*

1. *Penicillin eye drops* (1:10000 units per ml) should be instilled every half hourly.

2. *Antidiphtheric serum* (ADS) should be instilled every one hour.

3. *Atropine sulfate* 1 percent ointment should be added if cornea is ulcerated.

4. *Broad spectrum antibiotic* ointment should be applied at bed time.

B. *Systemic therapy*

1. *Crystalline penicillin* 5 lac units should be injected intramuscularly twice a day for 10 days.

2. *Antidiphtheric serum* (ADS) (50 thousand units) should be given intramuscularly stat.

C. *Prevention of symblepharon*

Once the membrane is sloughed off, the healing of raw surfaces will result in symblepharon, which should be prevented by applying contact shell or sweeping the fornices with a glass rod smeared with ointment.

CHLAMYDIAL CONJUNCTIVITIS

Chlamydia lie midway between bacteria and viruses, sharing some of the properties of both. Like viruses, they are obligate intracellular and filterable, whereas like bacteria they contain both DNA and RNA, divide by binary fission and are sensitive to antibiotics. The chlamydia combinedly form the PLT group (Psittacosis, Lymphogranuloma venereum and Trachomatis group).

Life cycle of the chlamydia. The infective particle invades the cytoplasm of epithelial cells, where it swells up and forms the *'initial body'*. The initial bodies rapidly divide into *'elementary bodies'* embedded in glycogen matrix which are liberated when the cells burst. Then the 'elementary bodies' infect other cells where the whole cycle is repeated.

Jones' classification. Jones' has classified chlamydial infections of the eye into following three classes :

Class 1 : *Blinding trachoma.* Blinding trachoma refers to hyperendemic trachoma caused by serotypes A, B, Ba and C of Chlamydia trachomatis associated with secondary bacterial infection. It is transmitted from eye to eye by transfer of ocular discharge through various modes.

Class 2 : *Non-blinding trachoma.* It is also caused by Chlamydia trachomatis serotypes A, B, Ba, and C; but is usually not associated with secondary bacterial infections. It occurs in mesoendemic or hypoendemic areas with better socioeconomic conditions. It is a mild form of disease with limited transmission owing to improved hygiene.

Class 3: *Paratrachoma.* It refers to oculogenital chlamydial disease caused by serotypes D to K of *chlamydia trachomatis*. It spreads from genitals to eye and mostly seen in urban population. It manifests as either adult inclusion conjunctivitis or chlamydial ophthalmia neonatorum.

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. It is still one of the leading causes of preventable blindness in the world.

The word 'trachoma' comes from the Greek word for 'rough' which describes the surface appearance of the conjunctiva in chronic trachoma.

Etiology

A. *Causative organism.* Trachoma is caused by a Bedsonian organism, the Chlamydia trachomatis belonging to the Psittacosis-lymphogranulomatrachoma (PLT) group. The organism is epitheliotropic and produces intracytoplasmic inclusion bodies called H.P. bodies (*Halberstaedter* Prowazeke bodies). Presently, 11 serotypes of chlamydia, (A, B, Ba, C, D, E, F, G, H, J and K) have been identified using microimmunofluorescence techniques.

Serotypes A, B, Ba and C are associated with hyperendemic (blinding) trachoma, while serotypes D-K are associated with paratrachoma (oculogenital chlamydial disease).

B. *Predisposing factors.* These include age, sex, race, climate, socioeconomic status and environmental factors.

1. *Age.* The infection is usually contracted during infancy and early childhood. Otherwise, there is no age bar.

2. *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.

3. *Race.* No race is immune to trachoma, but the disease is very common in Jews and comparatively less common among Negroes.

4. *Climate.* Trachoma is more common in areas with dry and dusty weather.

5. *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.

6. *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 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 air-borne or water-borne 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 Sourth-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 percent of the world's blindness, being second only to cataract.

Clinical profile of trachoma

Incubation period of trachoma varies from 5-21 days. Onset of disease is usually insidious (subacute), however, rarely it may present in acute form. ***Clinical course*** of trachoma is determined by the presence or absence of secondary infection. In the absence of such an infection, a pure trachoma is so mild and symptomless that the disease is usually neglected. But, mostly the picture is complicated by secondary infection and may start with typical symptoms of acute conjunctivitis. In the early stages it is clinically indistinguishable from the bacterial conjunctivitis and the term '*trachomadubium*' (doubtful trachoma) is sometimes used for this stage.

Natural history. In an endemic area natural history of trachoma is characterized by the development of acute disease in the first decade of life which continues with slow

progression, until the disease becomes inactive in the second decade of life. The sequelae occur at least after 20 years of the disease. Thus, the peak incidence of blinding sequelae is seen in the fourth and fifth decade of life.

Symptoms

In the absence of secondary infection, symptoms are minimal and include mild foreign body sensation in the eyes, occasional lacrimation, slight stickiness of the lids and scanty mucoid discharge.

In the presence of secondary infection, typical symptoms of acute mucopurulent conjunctivitis develop.

Signs

A. ***Conjunctival signs***

1. *Congestion* of upper tarsal and forniceal conjunctiva.

2. *Conjunctival follicles.* Follicles look like boiled sagograins 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 (pathognomic of trachoma) follicular conjunctivitis.

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

Each papilla consists of central core of numerous dilated blood vessels surrounded by lymphocytes and covered by hypertrophic epithelium.

Grading of trachoma

McCallan's classification

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

_ *Stage* I (Incipient trachoma or stage of infiltration). It is characterized by hyperaemia of palpebral conjunctiva and immature follicles.

_ *Stage* II (Established trachoma or stage of florid infiltration). It is characterized by appearance of mature follicles, papillae and progressive corneal pannus.

_ *Stage* III (Cicatrising trachoma or stage of scarring). It includes obvious scarring of palpebral conjunctiva.

_ *Stage* IV (Healed trachoma or stage of sequelae). The disease is quite and cured but sequelae due to cicatrisation give rise to symptoms.

WHO classification

Trachoma has always been an important blinding disease under consideration of WHO and thus many attempts have been made to streamline its clinical profile. The latest classification suggested by WHO in 1987 (to replace all the previous ones) 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. 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.

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.

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.

5. *CO: Corneal opacity.* This stage is labelled when easily visible corneal opacity is present over the pupil. 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).

Sequelae of trachoma

1. *Sequelae in the lids* may be trichiasis, entropion, tylosis (thickening of lid margin), ptosis, madarosis and ankyloblepharon.

2. *Conjunctival sequelae* include concretions, pseudocyst, xerosis and symblepharon.

3. *Corneal sequelae* may be corneal opacity, ectasia, corneal xerosis and total corneal pannus (blinding sequelae).

4. *Other sequelae* may be chronic dacryocystitis, and chronic dacryoadenitis.

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. *The clinical diagnosis* of trachoma is made from its typical signs; at least two sets of signs should be present out of the following:

1. Conjunctival follicles and papillae
2. Pannus progressive or regressive
3. Epithelial keratitis near superior limbus
4. Signs of cicatrisation or its sequelae

Clinical grading of each case should be done as per WHO classfication into TF, TI, TS, TT or CO.

Follicular conjunctivitis by other agents, especially adenoviruses, can be misdiagnosed as chlamydial infections. The earliest and easiest method of laboratory diagnosis was by direct detection of inclusion bodies (Halberstaedter-Prowazek bodies) with Giemsa staining of conjunctival smears. The test has low sensitivity and specificity. Antigen detection assays such as DFA with monoclonal antibodies have sensitivity of 85%–90% and enzyme immunoassay (EIA) have sensitivity of 85%–90%. Both are used widely in routine. Tissue culture isolation of C. trachomatis in Hella cells or McCoy cells, etc., is the most specific method and considered the “gold standard”.C. trachomatis inclusions are detected either by Giemsa staining or immunofluroscence assay after 48–72 h of incubation. [6]

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, specially 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.

The ocular surface has been suggested as a site of infection with Coronavirus- 2 (SARS- CoV- 2) responsible for the coronavirus disease- 19 (COVID- 19). Patients infected with SARS-CoV-2 can present with symptoms of conjunctivitis, including eye redness, ocular irritation, foreign body sensation, tearing, and chemosis. These symptoms have more commonly affected patients with severe systemic symptoms of COVID-19, though they can rarely present as an initial manifestation of the disease.

Examination findings include those consistent with mild follicular conjunctivitis, including unilateral or bilateral bulbar conjunctiva injection, follicular reaction of the palpebral conjunctiva, watery discharge, and mild eyelid edema. Bilateral chemosis alone may represent third-spacing in a critically ill patient rather than a true ocular manifestation of the virus.

There have been no reports of COVID-19 patients experiencing blurred vision, subconjunctival hemorrhage, eyelid ecchymoses, conjunctival scarring, keratitis, or pseudomembrane formation.

Ocular manifestations of COVID-19 have so far been uncommon, with most patients experiencing mild conjunctivitis otherwise indistinguishable from other viral etiologies. Differential diagnosis includes a broad range of common ocular manifestations of eye redness and increased tearing:

- Other viral conjunctivitis (e.g., Adenovirus)
- Bacterial conjunctivitis
- Allergic conjunctivitis
- Herpes simplex virus keratitis
- Anterior uveitis
- Corneal abrasion
- Foreign body
- Dry eye syndrome
- Exposure keratopathy in an intubated patient
- Chemosis in a critically ill patient

Ocular manifestations of COVID-19 are currently thought to be self-limited. There are currently no reports of sight-threatening manifestations of COVID-19. There have been no complications of ocular manifestations of COVID-19 reported, though larger studies and long-term follow up of these patients have not yet been conducted. In the absence of significant eye pain, decreased vision, or light sensitivity, many patients can be managed remotely with a trial of frequent preservative-free artificial tears, cold compresses, and

lubricating ophthalmic ointment. A short course of topical antibiotics can be added to prevent or treat bacterial superinfection based on the patient's symptoms and risk factors (e.g., contact lens wear) [7].

Cornea diseases

Fundamental importance of the cornea for the eye. The cornea is the eye's optical window that makes it possible for humans to see. The ophthalmologist is only able to discern structures in the interior of the eye because the cornea is transparent. At 43diopters, the cornea is the most important refractive medium in the eye.

Shape and location: The cornea's curvature is greater than the sclera's curvature. It fits in to the sclera like a watch-glass with a shallow sulcus (the limbus of the cornea) making the junction of the two structures.

Embryology: The corneal tissue consists of five layers. The cornea and the sclera are formed during the second month of embryonic development. The epithelium develops from ectoderm, and the deeper corneal layers develop from mesenchyme.

Morphology and healing:

The surface of the cornea is formed by stratified non keratinized squamous epithelium that regenerates quickly when injured. Within 1hour, epithelial defects are closed by cell migration and rapid cell division. However, this assumes that the limbus stem cells in the limbus of the cornea are undamaged. Regular corneal regeneration will no longer be possible when the secells are compromised. An intact epithelium protects against infection; a defect in the epithelium makes it easy for pathogens to enter the eye.

A thin basement membrane anchors the basal cells of the stratified squamous epithelium to Bowman's layer. This layer is highly resistant but can not regenerate. As a result, injuries to Bowman's layer usually produce corneal scarring.

Beneath Bowman's layer, many lamellae of collagen fibrils form the corneal stroma. The stroma is a highly bradytrophic tissue. As avascular tissue, it only regenerates slowly. However, its avascularity makes it an immunologically privileged site for grafting. Routine corneal transplants can be performed with out prior tissue typing. An increased risk of rejectionneed only be feared where the recipient's cornea is highly vascularized, as maybe the case following chemical injury or inflammation. Such cases require either a tissue-typed donor graft or immunosuppressive therapy with cyclosporin.

Descemet's membrane and the corneal endothelium lie on the posterior surface of the corneal stroma adjacent to the anterior chamber. Descemet's membrane is a relatively strong membrane. It will continue to define the shape of the anterior chamber even where the corneal

Stroma has completely melted. Because it is a genuine basement membrane, lost tissue is regenerated by functional endothelial cells. The corneal endothelium is responsible for the transparency of the cornea. A high density of epithelial cells is necessary to achieve this. The corneal endothelium does not regenerate; defects in the endothelium are closed by cell enlargement and cell migration.

Diameter: The normal average diameter of the adult cornea is 11.5mm (10–13mm).

A congenitally small cornea (micro cornea, diameter less than10.0mm)or a congenitally large cornea (megalo cornea , diameter from13to15mm) is always an abnormal finding.

Nourishment: The five layers of the cornea have few cells and are unstructured and avascular. Like the lens, sclera, and vitreous body, the cornea is a bradytrophic tissue structure. Its metabolism is slow, which means that healing is slow. The cornea is nourished with nutritive metabolites (amino acids and glucose) from three sources:

Diffusion from the capillaries at its edge.

Diffusion from the aqueous humor.

Diffusion from the tear film.

Significance of the tear film for the cornea: The three layer pre corneal tear film ensures that the surface of the cornea remains smooth and helps to nourish the cornea. Without a tear film, the surface of the epithelium would be rough, and the patient would see a blurred image. The enzyme lysozyme contained in the tear film also protects the eye against infection.

Transparency: This is due to two factors.

- the uniform arrangement of the lamellae of collagen fibrils in the corneal stroma and the smooth endothelial and epithelial surface produced by the intraocular pressure.
- the water content of the corneal stroma remains constant at 70%. The combined action of the epithelium and endothelium maintains a constant water content; the epithelium seals the stroma off from the outside, while the endothelium acts as an ion pump to remove water from the stroma.

This requires a sufficiently high density of endothelial cells. Endothelial cell density is age-dependent; normally it is approximately 2500cells/mm2.At cell densities below 300 endothelial cells/mm2, the endothelium is no longer able to pump water out of the cornea, resulting in edema of the corneal stroma and epithelium. The epithelial as well as endothelial layer act as barriers and regulate the exchange between cornea, tear film and aqueous humor by selective diffusion.

Protection and nerve supply: The cornea is a vital structure of the eye and as a result extremely sensitive. It receives its ample sensory supply from the ophthalmic division of the trigeminal nerve. The slightest tactile sensation causes an eye closing reflex. Any injury to the cornea (erosion, foreign-body penetration, or ultraviolet keratoconjunctivitis) exposes sensory nerve endings and causes intense pain with reflexive tearing and involuntary eye closing.

The triad of involuntary eye closing (blepharospasm), reflexive tearing (epiphora), and pain always suggests a possible corneal injury.

Examination Methods

Nonophthalmologists can evaluate the transparency of the cornea (opacities of the stroma and epithelium suggest scarring or infiltration of the epithelium), its surface luster (lack of luster suggests an epithelial defect), and possible superficial corneal injuries used to measure the size of the cornea, and sensitivity can be tested with a cotton swab.

The ophthalmologist uses instruments to evaluate corneal morphology and function in greater detail.

Slit Lamp Examination

The slit lamp is the primary instrument used in evaluating the cornea. The ophthalmologist chooses between eight and up to 40 power magnification

For examining all levels of the cornea with a narrow beam of collimated light.

Direct illumination: The slit beam is moved over the entire cornea, thus thickness and depth of corneal findings can be estimated.

Indirect illumination: The light of the slit lamp is directed at the corneal limbus from the side. So we have a total reflection by the other wise completely transparent cornea. Subtle opacities or discrete corneal edema show up with this technique by not totally reflecting the slit lamp light.

Retrograde illumination: The cornea is illuminated by light reflected from the iris by a slit lamp beam directed straight into the eye. Subtle epithelial and endothelial findings or small blood vessels become visible.

Dye Examination of the Cornea: Defects in the surface of the cornea can be visualized with fluorescein or rose bengal solution (in either case, administer one drop of 1% solution). Since these dyes are not usually absorbed by the epithelium, they can be used to visualize loss of epithelium over a wide area (such as corneal erosion) and extremely fined effects (as in superficial punctate keratitis). Illumination with a cobalt blue filter enhances the fluorescent effect.

These dye methods can reveal corneal epithelial defects (cornea lerosion)even with out the use of a slit lamp, which is helpful in examining infants.

Keratoconus:

Definition: Conical, usually bilateral central deformation of the cornea with parenchymal opacification and thinning of the cornea.

Epidemiology: Keratoconus is the most frequently encountered deformation of the cornea. Occurrence is familial, although women are more likely to be affected than men.

Etiology: Keratoconus is probably a genetic disorder. It can occur in families with varying paths of hereditary transmission. Occasionally keratoconus is associated with trisomy 21 syndrome (Down syndrome) as well as with atopic dermatitis and other connective-tissue disorders such as Marfan syndrome.

Symptoms: The clinical course of the disorder is episodic; the increasing protrusion of the cornea usually produces bilateral irregular myopic astigmatism. Left untreated, in rare cases keratoconus can cause tears of Descemet’s membrane due to the continuous stretching. The entire cornea can then bulge out at this site. This is referred to as a cute keratoconus. Symptoms of a cute keratoconus include sudden loss of visual acuity accompanied by intense pain, photophobia, and increased tearing.

Diagnostic considerations: The diagnosis is usually made with a keratoscope or ophthalmometer (reflex images will be irregular). The examiner can also detect keratoconus without diagnostic aids by standing behind the patient and pulling the patient’s upper eye lids downward. The conical protrusion of the surface of the cornea will then be readily apparent due to the deformation of the margin of the eyelid (Munson’s sign).

Treatment: Degeneration of visual acuity can usually be corrected initially with eyeglasses; hard contact lenses will be required as the disorder progresses. However, after a certain point, the patient repeatedly will lose the contact lenses. Then the only possible treatment is penetrating keratoplasty (transplantation of a corneal graft from a donor into the patient’s cornea). Keratoconus management has significantly changed over the last two decades. The advent of new interventions such as cornea cross-linking, intrastromal

corneal ring segments, and combined treatments provide corneal clinicians a variety of treatment options for the visual rehabilitation of keratoconus patients. [5]

Prognosis: The prognosis for penetrating keratoplasty in treating keratoconus is good because the cornea is avascular in keratoconus.

Corneal disease remains the leading cause of monocular blindness worldwide, especially affecting marginalized populations. Corneal opacities, which are largely caused by infectious keratitis, are the fourth leading cause of blindness globally and are responsible for 10% of avoidable visual impairment in the world's least developed countries. Approximately 2 million people develop a corneal ulcer every year in India alone. In the United States infectious keratitis is often associated with contact lens wear, but in developing countries it is more commonly caused by ocular trauma sustained during agricultural work. In this review we explore the current literature and future directions of the diagnosis and treatment of infectious keratitis.

Proper diagnosis of keratitis is essential to determining treatment and achieving resolution of infection. The mainstay in diagnosis is still Gram stain and culture of corneal samples despite imperfect sensitivity. Gram and Giemsa stains are advantageous because they provide instant results, with Gram stain accurately detecting causative organism 60–75% of the time for bacterial cases and 35–90% in fungal cases. Giemsa has a sensitivity of 40–85% for diagnosing fungal cases. Blood and chocolate agar are most commonly used to culture bacteria, while Sabouraud's agar or potato dextrose are best for isolating fungus, and non-nutrient agar with Escherichia Coli overlay can be used to culture Acanthameoba. Thioglycollate broth is another option to identify aerobic or facultatively anaerobic bacteria, but contaminant is a problem and often it is difficult to determine if isolated organisms are the etiology of infection. Viral keratitis is diagnosed largely on clinical exam because of its characteristic dendritic appearance, but PCR is sometimes used to confirm diagnosis because of its high sensitivity.

There is still substantial room for exploration of novel methods of diagnosing infectious keratitis. In vivo confocal microscopy (IVCM) has grown in popularity in recent years due to its rapidity and high sensitivity in detecting larger organisms such as filamentous fungus, acanthamoeba, and Nocardia bacteria. Anterior segment optical coherence tomography (AS-OCT) has been used more recently to provide an objective measure of corneal infiltrate and/or scar size or to monitor corneal thinning during treatment.

Topical antibiotics remain the first-line treatment for bacterial keratitis. Clinicians weigh many factors when choosing an antibiotic regimen, including, broad-spectrum coverage, toxicity, availability and cost, and region-specific epidemiology of pathogens and resistance patterns. Indeed, a recent international survey of cornea specialists found that concerns over several of these factors were predictive of antibiotic choice.

The use of adjuvant corticosteroids has long been debated in the treatment of bacterial keratitis Proponents of the use of corticosteroids argue that they improve outcomes by reducing inflammation, thereby reducing scarring, neovascularization, and stromal melt. However, others argue that corticosteroids delay epithelial healing and may even worsen infection.

FUNGAL KERATITIS

Fungal ulcers often have worse outcomes than bacterial ulcers, and there is little evidence to guide treatment. Fungal keratitis represents a relatively small percentage of infectious keratitis cases in regions with temperate climates, however in tropical climates it can cause up to 50% of infectious ulcers. Contact lens wear has been identified as a risk factor for fungal keratitis in the United States and an outbreak of Fusarium keratitis among contact lens wearers was related to the ReNu Moistureloc contact lens solution. There have been no new FDA approved treatments since natamycin, a topical polyene, was introduced in the 1960's [6].

Topical Treatments

Effective treatment with topical natamycin 5% is limited by its poor penetration into the corneal stroma. Topical amphotericin B 0.3% to 0.5% is an alternative, but its use requires access to a compounding pharmacy and is limited by toxicity. Voriconazole, a newer generation triazole, has gained popularity in the treatment of fungal keratitis due to its excellent ocular penetration. In addition, in one in vitro study, voriconazole was the only drug tested in which 100% of fungal isolates commonly implicated in keratitis were susceptible. Although topical voriconazole failed to show improved outcomes compared with natamycin, there are several reasons that oral voriconazole may have efficacy in the treatment of fungal keratitis. First, intermittent dosing of topical medications may result in intervals of sub-therapeutic drug levels and oral medications may provide more steady-state drug levels at the site of infection. One study comparing aqueous samples after topical and oral voriconazole found that topical administration of voriconazole resulted in highly variable aqueous concentrations with troughs well below the MIC90 for most fungi while oral voriconazole provided therapeutic drug level that remained relatively constant Of note, in many case reports of successful treatment with topical voriconazole, oral and/or intravenous voriconazole was used in conjunction with the topical medication [7].

VIRAL KERATITIS

Herpes simplex virus (HSV) keratitis affects an estimated 500,000 people in the United States and an estimated 1.5 million globally. It is the most common cause of unilateral infectious corneal blindness in much of the developed world. Viral keratitis differs from bacterial and fungal keratitis in that it can become chronic and recurrent. Besides being a painful, sight-threatening infection, HSV keratitis has been shown to significantly impact quality of life even when patients are not experiencing an active infection. Less common forms of viral keratitis include varicella-zoster virus (VZV) keratitis, and cytomegalovirus (CMV) keratitis [8].

Topical Treatments

Topical treatments for viral keratitis include antiviral medications and adjuvant topical corticosteroids. The topical antiviral trifluridine is the most commonly prescribed topical antiviral medication for HSV keratitis in the United States. While it is effective in treating HSV keratitis, it has low bioavailability and causes ocular surface toxicity, so its use has become more limited as newer topical antivirals are developed. Topical acyclovir is the first line treatment for HSV keratitis in Europe as it has been shown to be just as effective as trifluridine with less ocular surface toxicity. Unfortunately, it is unavailable in the United States. Ganciclovir is a newer synthetic medication with more broad-spectrum antiviral coverage. In addition to treating HSV and VZV keratitis, topical ganciclovir is also effective in treating keratitis caused by CMV. Ganciclovir has been shown to be just

as effective as acyclovir, while causing less ocular toxicity. It may also be less likely to promote drug resistance.

Topical corticosteroids are also sometimes used as adjuvant therapy to topical antivirals. The Herpetic Eye Disease Study I (HEDS I) evaluated the effectiveness of corticosteroids in treating HSV stromal keratitis. In this randomized controlled trial 106 patients with active HSV stromal keratitis were randomized to receive either topical prednisolone phosphate or placebo, tapered over a 10 week period. All patients received topical trifluridine. HEDS I found that the median time to treatment failure was drastically shorter in the placebo group: 17 days in the placebo group and 98 days in the topical steroids group ($P<0.001$). Time to resolution of infection was significantly shorter in the group receiving topical corticosteroids, with a median of 26 days for those taking corticosteroids and 72 days for those taking placebo ($P<0.001$). Visual acuity at 6 months was similar across groups [8].

Oral Treatments.

Valacyclovir, a newer antiviral, is well tolerated and there is some evidence that it may have better ocular penetration. Additionally, the treatment dose for valacyclovir is 1g three times daily, as opposed to acyclovir which is 400mg five times daily (800mg five times daily for VZV), which aids in patient compliance. Oral valganciclovir is the preferred treatment for CMV stromal keratitis, but it has significant side effects, including aplastic anemia, which must be closely monitored.

SCLERITIS

Scleritis is a severe ocular inflammatory condition affecting the sclera, the outer covering of the eye. It can be categorized as anterior with diffuse, nodular, or necrotizing subtypes and posterior with diffuse or nodular subtypes. Scleritis can be visually significant, depending on the severity and presentation and any associated systemic conditions. The presentation can be unilateral or bilateral.

Etiology

Scleritis can be idiopathic or caused by infectious or noninfectious conditions. Additionally, associations with malignancy, autoimmune diseases, and surgically induced or medication side effects are causative factors. Viruses, bacteria, fungi, and parasites can cause infectious scleritis and are reported to occur in 4% to 10% of all cases. Choroidal melanomas and conjunctival tumors can create ocular inflammation that can mimic the signs and symptoms of scleritis. 50% of patients with scleritis will have an autoimmune condition, sometimes undiagnosed at the time of presentation. Rheumatoid arthritis and systemic vasculitic conditions are most commonly associated with scleritis.

Surgically induced scleritis has been associated with pterygium removal and scleral buckle procedures. Medications used to treat osteoporosis such as bisphosphonates have been found to cause scleritis; however, reports of this occurrence are rare.

Epidemiology

Limited, population-based studies have reported 10,500 cases of scleritis in the United States per year or an estimated four to six cases per 100,000 persons.

It affects patients in middle age, commonly between 47 to 60 years. Scleritis is more common in women, with a 60% to 74% predominance. Limited information is known about incidence of children besides case reports.

Pathophysiology

The exact pathophysiology of scleritis is still under investigation. The anatomical structure of the sclera includes an extracellular matrix of collagen, elastin, and proteoglycans that closely resemble the components of joints, causing it to be susceptible to inflammatory conditions such as rheumatoid arthritis. Additionally, the sclera is mostly avascular and requires transport of nutrients and removal of cellular wastes on the surrounding episcleral and choroidal vascular systems.

Histopathology

The disease's histopathological features are similar to those seen in vasculitic conditions. Scleral biopsies from patients with severe scleritis or necrotizing scleritis demonstrated vascular occlusions and infiltration, necrosis, and evidence of macrophages and T cells. The presentation can vary depending on the location and subtype of scleritis and can be unilateral or bilateral.

Anterior scleritis. Anterior from the rectus muscle's limbus-insertion

Occurs in 98% of cases Mild to moderate pain and tenderness; worse at night Pain with eye movement. A blue-violet hue of deep vessels; best seen in daylight or natural illumination. Photophobia, tearing, and possibly decreased vision. No vessel blanching with the installation of 2.5% topical phenylephrine.

Diffuse scleritis. Most common (45% to 61%). Extensive scleral edema; congestion of deep and superficial vessels. Can be localized or encompass the entire anterior sclera.

Nodular scleritis. Multiple, well-defined, and non-moveable nodules. Scleral edema and congestion of vessels. Usually more localized.

Necrotizing scleritis. With inflammation – intense congestion of vessels. Severe pain. Most severe form with the worst prognosis. Highest association with a systemic disease. Scleral thinning and exposure of choroid possible. Inflammation can spread to other ocular tissues such as the cornea, ciliary body, or trabecular meshwork.

Posterior scleritis. Behind the insertion of the rectus muscles. Occurs in 2% of cases. Can occur in conjunction with anterior scleritis. May present with decreased vision, with or without ocular pain. Retinal detachments, optic nerve swelling, and cotton wools spots can be seen on dilated examination.

The diagnosis is based on the clinical presentation and ocular exam with a detailed history and review of systems, targeted laboratory tests, and imaging studies.

Common Lab Tests

Antineutrophil cytoplasmic antibodies (ANCAs) – specific for granulomatosis with polyangiitis (Wegener granulomatosis)

Antinuclear Antibodies (ANA) – suggestive of systemic lupus erythematosus and other autoimmune conditions

Complete blood count(CBC) with differential – identify infectious or inflammatory processes

C-reactive protein – elevated in inflammatory conditions

ESR – suggestive of giant cell arteritis, inflammatory or infectious processes

HLA-B27 - more useful in cases of uveitis; rare association with scleritis

Lyme serology – rule out Lyme Disease, presents with recurrent diffuse anterior scleritis

Rheumatoid factor – suspect for rheumatoid arthritis

Angiotensin-converting enzyme (ACE) - suggestive of sarcoidosis

Serum lysozyme – suggestive of sarcoidosis

Rapid plasma reagin (RPR) – suggestive for syphilis

Fluorescent treponemal antibody absorption test (FTA-ABS) – suggestive for syphilis

Treatment / Management

The treatment and management of scleritis are designed to determine any causative factor, manage ocular inflammation, control ocular pain and symptoms, prevent sequela, and reduce recurrences.

Topical corticosteroid eyedrops

Can be used in mild cases; some report very limited success

Prednisolone acetate 1.0% or difluprednate 0.05% four times per day are options

Oral NSAIDs

Can start with one agent and switch to another agent if not effective

Indomethacin, commonly used, 50 mg, 3 times per day

Ibuprofen 600 mg, 3 times per day

Naproxen 500 mg, 2 times per day

Prognosis.

Visual prognosis is relatively good for patients with mild or moderate scleritis that respond well to the appropriate medical treatment and management of any underlying systemic condition. Necrotizing and posterior scleritis cases pose a higher risk of visual loss to the extent of the inflammation and involvement of other ocular structures.

Complications.

Sequela resulting from scleritis can vary depending on the severity of presentation and any associated autoimmune conditions and can include decreased vision, cataracts, increased intraocular pressure, scleral thinning or melting, and corneal thinning. [12]

Cataract

A cataract is present when the transparency of the lens is reduced to the point that the patient's vision is impaired. The term cataract comes from the Greek word katarraktes (down-rushing; waterfall), because earlier it was thought that the cataract was a congealed fluid from the brain that had flowed in front of the lens.

General symptoms. Development of the cataract and its symptoms is generally an occult process. Patients experience the various symptoms such as seeing only shades of gray, visual impairment, blurred vision, distorted vision, glare or star bursts, monocular diplopia, altered color perception, etc. to varying degrees, and these symptoms will vary with the specific type of cataract.

Diagnosis of a cataract is generally very unsettling for patients, who immediately associate it with surgery. One should therefore refer only to a cataract when it has been established that surgery is indicated. If the cataract has not progressed to an advanced stage or the patient can cope well with the visual impairment, one should refer instead to a "lens opacity."

Classification. Cataracts can be classified according to several different criteria.

-Time of occurrence (acquired or congenital cataracts).

- Maturity.

- Morphology.

No one classification system is completely satisfactory. We prefer the system shown in table

Acquired Cataract

Senile Cataract

Epidemiology. Senile cataract is by far the most frequent form of cataract, accounting for 90% of cataracts. About 5%of 70-year-olds and 10% of 80-year-olds suffer from a cataract requiring surgery. Ninety percent of cataracts are senile cataracts.

Etiology. The precise causes of senile cataract have not been identified. As occurrence is often familial, it is important to obtain a detailed family history.

Classification and forms of senile cataracts: The classification according to maturity follows the degree of visual impairment and thematurity, which earlier was important to determine the time of surgery. We follow a morphologic classification as morphologic aspects such as the hardness and thickness of the nucleus now influence the surgical procedure.

Nuclear cataract: In the fourth decade of life, the pressure of peripheral lens fiber production causes hardening of the entire lens, especially the nucleus.The nucleus takes on a yellowish-brown color (brunescent nuclear cataract).

This may range from reddish-brown to nearly black discoloration of the entire lens (black cataract). Because they increase the refractive power of the lens, nuclear cataracts lead to lenticular myopia and occasionally produce a second focal point in the lens with resulting monocular diplopia.

Nuclear cataracts develop very slowly. Due to the lenticular myopia, nearvision (even without eyeglasses) remains good for a long time.

Cortical cataract. Nuclear cataracts are often associated with changes in the lens cortex. It is interesting to note that patients with cortical cataracts tend to have acquired hyperopia in contrast to patients with nuclear cataracts, who tend to be myopic.

Whereas changes in nuclear cataracts are due to hardening, cortical changes are characterized by increased water content. Several morphologic changes will be apparent upon slit lamp examination with maximum mydriasis:

- Vacuoles. Fluid accumulation swill be present in the form of small narrow cortical vesicles. The vacuoles remain small and increase in number.
- Water fissures. Radial patterns of fluid-filled fissures will be seen between the fibers.
- Separation of the lamellae. Not as frequent as water fissures, these consist of a zone of fluid between the lamellae (often between the clear lamellae and the cortical fibers).
- Cuneiform cataract. This is a frequent finding in which the opacities radiate from the periphery of the lens like spokes of a wheel.

Cortical cataracts progress more rapidly than nuclear cataracts. Visual acuity may temporarily improve during the course of the disease. This is due to a stenopeic effect as light passes through a clear are a between two radial opacities.

Posterior subcapsular cataract. This is a special form of cortical cataract that begins in the visual axis. Beginning as a small cluster of granular opacities, this form of cataract expands peripherally in a disc-like pattern. As opacity increases, the rest of the cortex and the nucleus become involved (the usual spectrum of senile cataract).

Posterior subcapsular cataract leads to early, rapid, and severe loss of visual acuity. Near vision is usually significantly worse than distance vision(near-field miosis). Dilating eye drops can improve visual acuity in this form of cataract.

Mature cataract. The lens is diffusely white due to complete opacification of the cortex. A brownish lens nucleus is often faintly discernible. Where water content is increased, a lens with a mature cataract can swell and acquire a silky luster (in tumescent cataract in which the capsule is under pressure). The increasing thickness of the lens increases the resistance of the pupil and with it the risk of angle closure glaucoma.

Vision is reduced to perception of light and dark, and the interior of the eye is no longer visible. Cataract surgery is indicated to restore visual acuity.

Hyper mature cataract: If amature cataract progresses to the point of complete liquefaction of the cortex, the dense brown nucleus will subside within the capsule. Its superior margin will then be visible in the pupil as a dark brown silhouette against the surrounding grayish white cortex. The pressure in the lens capsule decreases. The contents of the limp and wrinkled capsular bag gravitate within the capsule. This condition, referred to as Morgagni's cataract, is the final stage in a cataract that has usually developed over the course of two decades. The approximate on set of the cataract can usually be inferred from such findings. Prompt cataract extraction not only restores visual acuity but also prevents development of phacolytic glaucoma. When the lens capsule becomes permeable for liquefied lens substances, it will lose volume due to leakage. The capsule will become wrinkled. The escaping lens proteins will cause intraocular irritation and attract macrophages that then cause congestion of the trabecular network.

Emergency extraction of the hypermature cataract is indicated in phacolytic glaucoma to save the eye.

Cataract in Systemic Disease

Epidemiology. Lens opacities can occasionally occur as a sign of systemic disease. Types of cataract in systemic disease:

Diabetic cataract. The typical diabetic cataract is rare in young diabetic patients. Transient metabolic decompensation promotes the occurrence of a typical radial snowflake pattern of cortical opacities (snowflake cataract). Transient hyperopia and myopia can occur. Diabetic cataract progresses rapidly. Senile cataracts are observed about five times as often in older diabetics as in patients the same age with normal metabolism. These cataracts usually also occur 2–3years earlier [5].

Galactosemic cataract. This deep posterior cortical opacity begins after birth. Galactosemia is a rare cause of early cataract in children lacking an enzyme required tometabolize galactose. The newborn receives ample amounts of galactose in the mother's milk. Due a lack of uridylyl transferase, or less frequently galactokinase, galactose can not be metabolized to glucose, and the body becomes inundated with galactose or with galactose and galactose-1- phosphate. If the disorder is diagnosed promptly and the child is maintained on a galactose-free diet, the opacities of the first few weeks of life will be reversible. Galactosemic cataract is the only form of cataract that responds to conservative therapy.

Dialysis cataract. Hemodialysis to eliminate metabolic acidosis in renal insufficiency can disturb the osmotic equilibrium of lens metabolism and cause swelling of the cortex of the lens.

Other rare metabolic diseases that can cause cataract include mannosidosis, Fabry disease, Lowe syndrome (oculocerebrorenal syndrome), and Wilsondisease (hepatolenticular degeneration).

Cataract with myotonic dystrophy. Opacities first occur between the ages of 30 and 50, initially in a thin layer of the anterior cortex and later also in the subcapsular posterior cortex in the form of rosettes. Detecting these opacities is important for differential diagnosis as cataracts do not occur in Thomsen disease (myotonia congenita) or Erb progressive muscular dystrophy.

Symptoms that confirm the diagnosis include cataract, active signs of myotonia (delayed opening of the fist), and passive signs of myotonia (decreased relaxation of muscles in the extremities following direct percussion of the muscle and absence of reflexes).

Tetany cataract. The opacity lies within a broad zone inferior to the anterior lens capsule and consists of a series of gray punctate lesions. Symptoms that confirm the diagnosis include low blood calcium levels, a positive hyperventilation test, and signs of tetany: positive Chvostek, Trousseau, and Erbsigns [13].

Dermatogenous cataract. This may occur with chronic neurodermatitis and less frequently with other skin disorders such as scleroderma, poikiloderma, and chromic eczema. Characteristic signs include an anterior crest-shaped thickening of the protruding center of the capsule.

Complicated Cataracts

This form of cataract can occur as a complication of any protracted intraocular inflammation, especially heterochromia, chronic iridocyclitis, retinalvasculitis, and retinitis pigmentosa. The result is a pumice-like posterior subcapsular cataract that progresses axially toward the nucleus. This form of cataract tproduces extreme light scattering.

Cataract after Intraocular Surgery

Cataracts usually develop earlier in the operated eye as compared to the opposite, unoperated eye after intraocular surgery. This applies especially to filtering operations. A secondary cataract will generally occur following vitrectomy and silicone oil tamponade [14].

Traumatic Cataract

The incidence of these lens opacities is higher in men than in women due to occupational and sports injuries. The following types of traumatic cataracts are differentiated.

Frequent traumatic cataract:

- Contusion cataract. Contusion of the eyeball will produce a rosette-shaped subcapsular opacity on the anterior surface of the lens. It will normally remain unchanged but will migrate into the deeper cortex over time due to the apposition of new fibers.

Rarer traumatic cataracts:

- Infrared radiation cataract (glassblower’s cataract): This type of cataract occurs after decades of prolonged exposure to the infrared radiation of fire without eye protection.

Characteristic findings include splitting of the anterior lens capsule, whose edges will be observed to curl up and float in the anterior chamber. Occupational safety regulations have drastically

reduced the incidence of this type of cataract.

- Electrical injury. This dense subcapsular cataract can be caused by lightning or high-voltage electrical shock.
- Cataract fromionizing radiation.

Etiology. Ionizing radiation (neutron, or gamma/x-ray radiation) has high energy that can cause ionization and formation of radicals in cellular tissue. The penetration depth in the eye varies with the type of radiation—i.e., the wavelength, resulting in characteristic types of tissue damage.

This tissue damage always manifests itself after a latency period, often only after a period of years. Common sites include the lens (radiation cataract) and retina (radiation retinopathy). This tissue damage is usually the result of tumor irradiation in the eye or nasopharynx. Radiation disorders have been observed in patients from Hiroshima and Nagasaki and, more recently, in Chernobyl [14,15].

Toxic Cataract

Steroid cataract. Prolonged topical or systemic therapy with corticosteroids can result in a posterior subcapsular opacity. The exact dose–response relationship is not known. Other toxic cataracts can result from chlorpromazine, miotic agents (especially cholinesterase inhibitors), and busulfan (Myleran) used in the treatment of chronic myelocytic leukemia.

Congenital Cataract

There are many congenital cataracts. They are either hereditary or acquired through the placenta [8].

Hereditary Congenital Cataracts

Familial forms of congenital cataracts may be autosomal-dominant, auto-somal-recessive, sporadic, or X-linked. They are easily diagnose don the basis of their characteristic symmetric morphology.

Forms of hereditary congenital cataract:

Lamellar or zonular cataract. Opacities are located in one layer of lens fibers, often as “riders” only in the equatorial region.

Nuclear cataract. This is a variant of the lamellar cataract in which initially only the outer layer of the embryonic nucleus is affected.

Coronary cataract. This is characterized by fine radial opacities in the equatorial region. Cerulean cataract. This is characterized by fine round or club-shaped blue peripheral lens opacities.

Most familial lens opacities do not impair vision and are not progressive. This also applies to rare lens opacities involving the capsule such as anterior and posterior polar cataracts, anterior pyramidal cataract, and Mittendorf’s dot (remnant of the embryonic hyaloid artery on the posterior capsule of the lens.

Cataract from transplacental infection in the first trimester of pregnancy

A statistical study by Pau (1986) cites the following incidences of congenital cataract with respect to systemic disease contracted by the mother during the first trimester of pregnancy:

- Rubella40–60%.
- Mumps10–22%.
- Hepatitis16%.
- Toxoplasmosis5%.

Most of these cases involved total cataracts due to virus infection contracted by the mother during early pregnancy. This infection occur red during the fifth to eighth week of pregnancy, the phase in which the lens develops. Because the protective lens capsule has not yet been formed at this time, viruses can invade and opacify the lens tissue. The most frequent cause of cataract is a rubella infection contracted by the mother, which also produces other developmental anomalies (Gregg syndrome involving lens opacity, an open ductus arteriosus, and sensorineural hearing loss). The cataract is bilateral and total and can be diagnosed by the presence of leukocoria (white pupil) and chorioretinal scarring secondary to choroiditis.

Treatment of Cataracts

Medical Treatment

In spite of theoretical approaches in animal research, the effectiveness of conservative cataract treatment in humans has not been demonstrated.

At present, no conservative methods are available to prevent, delay, or reverse the development of a cataract. Galactosemic cataracts are the only exception to this rule.

Surgical Treatment

Cataract surgery is the most frequently performed procedure in ophthalmology.

Earlier surgical techniques were dependent upon the maturity of the cataract. This is no longer the case in modern cataract surgery.

Optical indications. Restoration of visual acuity is by far the most frequent indication for cataract surgery.

- In the presence of bilateral cataracts, the eye with the worse visual acuity should undergo surgery when the patient feels visually handicapped.

However, this threshold will vary depending on the patient’s occupational requirements.

- In the presence of a unilateral cataract, the patient is often inclined to postpone surgery as long as vision in the healthy eye is sufficient.

Medical indications.

- In the presence of a mature cataract, it is important to advise the patient to undergo surgery as soon as possible to prevent a phacolytic glaucoma.
- In the case of retinal disease (e.g., diabetic retinopathy) a cataract extraction may be necessary to clear the optical axis for retinal diagnosis and laser treatment.

The prospect of a successful outcome is important for the patient. Most patients define a successful outcome in terms of a significant improvement in vision. Therefore, it is important that the patient undergoes a thorough preoperative eye examination to exclude any ocular disorders, aside from the cataract, that may worsen visual acuity and compromise the success of the cataract operation. Such disorders include uncontrolled glaucoma, uveitis, macular degeneration, retinal detachment, atrophy of the optic nerve, and amblyopia.

A detailed history of the patient’s other ocular disorders and vision prior to development of the cataract should be obtained before surgery.

Reliability of Cataract Surgery

Cataract surgery is now performed as a microsurgical technique under an operating microscope. Modern standardized techniques such as extracapsular cataract extraction (ECCE), phacoemulsification, and microincision cataract surgery (MICS), microsurgical instruments and specially trained surgeons performing many cataract operations (high-volume surgeons) have made it possible to successfully perform cataract surgery without serious complications in 99% of patients. The procedure lasts about 20minutes and, like the postoperative phase, is painless.

The risk of losing vision or the entire eye during a cataract extraction, with hemorrhage during surgery or endophthalmitis after surgery, is about 0.05%.

Usually cataract surgery is carried out on an outpatient basis. The patient can be hospitalized for 3 days, depending on the adequacy of postoperative care at home. Older patients who live alone may be unable to care for themselves adequately and maintain the regimen of prescribed medications for the operated eye in the immediate postoperative phase. The operation can be performed as an outpatient procedure if the ophthalmologist's practice is able to ensure adequate care.

Possible Types of Anesthesia

Cataract extraction can be performed under local anesthesia or general anesthesia. Today, most operations are performed under local anesthesia. Aside from the patient's wishes, the reare medical reasons for preferring one form of anesthesia over another.

General anesthesia. This is recommended for patients who are extremely apprehensive and nervous, deaf, or mentally retarded; it is also indicated for patients with Parkinson disease or rheumatism, who are unable to lie still without pain.

Local anesthesia (retrobulbar, peribulbar, or topical anesthesia).This is recommended for patients with increased anesthesia risks, and is the preferred approach in outpatient surgery.

Intraocular lens. In almost all cataract extractions, an intraocular lens (IOL) is implanted, preferably in the place of the natural lens (posterior chamber lens, PC-IOL). If for intraoperative reasons PC-IOL placement is not possible, an IOL is implanted in the anterior chamber (AC-IOL). An eye with anartificial lens is referred to as a pseudophakia. The different kinds of IOL sare discussed in the sectionon IOL technology.

Biometry. The refractive power of the lens required is determined preoperatively by biometry to reach the targeted refractive result. In a simplified fashion, IOL refractive power is determined by the refractive power of the cornea, IOL refraction constants, and the axial length, determined by ultrasonic measurement. More recent devices (e.g., the Zeiss IOL-Master) carry out noncontact biometri calculations with numerous additional parameters.

Postoperative refractive status. The usual recommendation is to target emmetropia or mild myopia (–0.25 to –0.5D). The patient will then only need glasses for reading. Postoperative hyperopia (need for glasses at far and near distances) is not satisfactory for the patient. If the patient's fellow eye does not need cataract extraction within a short period of time, the refractive

Difference between the two eyes should be not more than 2 to 2.5D, to avoid anisometric problems in binocular vision.

Intraocular Lens (IOL)

IOL. Every IOL consists of a central optical part (refractive element) and two haptics, to stabilize the IOL in the capsular bag, ciliary sulcus, or chamber angle. A distinction is made between:

- Monofocal IOLs, with only one specific focus (far or near).
- Multifocal IOLs, with a focus for far and near objects.
- Toric IOLs correct not only spherical ametropia but also up to3Dof astigmatism. The correct orientation of the IOL (orientation marks) is crucial.
- Accommodative IOLs are designed to move forward and backward in the eye to allow accommodation. However, with less than 0.75D of accommodative power, this approach does not currently meet expectations.

IOL design. The geometrical configuration of IOLs has been subject to constant development. The sharp posterior edge of the optical part serves as abarrier to lens epithelial cells migrating from the equator of the capsule toward the center, preventing secondary cataract. The sloping side part and the rounded anterior edge of the optic minimize glare and internal light reflections. Additional enhancements of the optical quality include multifocal steps and an aberration-optimized anterior IOL surface.

IOL material. Basically, IOLS can bedivided into nonflexible and flexible types, as well as one-piece IOLs (in which the haptics and optic are made of a single material with no connecting points) and three-piece IOLs (in which the optic and haptics are made of different materials such as polypropylene, and polyamide and connected to each other).

Nonflexible IOLs. These are mostly made of polymethylmethacrylate (PMMA).To implantanon flexible IOL, the incision needs to be larger than the diameter of the IOL (5.5–6.5mm). Modern nonflexible IOLs are one-piece IOLs.

Flexible IOLs are folded with a forceps or an injector system and are therefore implantable through 2.0–3.0 mm incisions with the same optic size as nonflexible IOLs. Flexible IOLs are made of silicone, acrylic, hydrogel, or collamer.

Alternative Methods of Correcting Refractive Errors

Cataract eyeglasses. The development of the intraocular lens has largely replaced the correction of postoperative aphakia with cataract lenses. Long the standard, this method is now only necessary in exceptional cases. Cataract eyeglasses cannot be used to correct unilateral aphakia, because the difference in the size of the retinal images is too great (aniseikonia). Therefore, cat-

aract eyeglasses are only suitable for correcting bilateral aphakia. Cataract ey eglasses have the disadvantage of limiting the field of vision (peripheral and ring scotoma).

Contact lenses (soft, rigid, and oxygen-permeable). These lenses provide a near-normal field of vision and are suitable for postoperative correction of unilateral cataracts, as the difference in image size is negligible. However, many older patients have difficulty in learning how to cope with contact lenses.

Surgical Techniques

The operation is performed on only one eye at a time. The procedure on the fellow eye is performed after about a week once the first eye has stabilized.

Historical milestones.

- Couching (reclination). Up until the 19th century, a pointed instrument was used to displace the lens into the vitreous body out of the visual axis.

- 1746. J.Daviel carried out the first extracapsular cataract extraction by removing the contents of the lens through an inferior approach.

- 1866. A. von Graefe carried out the first removal of a cataract through a superior limbal incision with capsulotomy.

Intracapsular cataract extraction. Until themid-1980s, this was the method of choice. Today, intracapsular cataract extraction is used only with subluxation or dislocationof the lens. The entire lens is frozen in its capsule witha cryophake and removed from the eye through a large superior corneal incision

Extracapsular cataract extraction.

The anterior capsule is opened (capsular rhexis). Then only the cortex and nucleus of the lens are removed (extracapsular extraction); the posterior capsule and zonule suspension remain intact. This provides a stable base for implantation of the posterior chamber intraocular lens.

Extracapsular cataract extraction with implantation of a posterior chamber intraocular lens is nowthemethodof choice.

Today, phacoemulsification (emulsifying and aspirating the nucleus of the lens with a high-frequencyultrasonic needle) is the preferred technique for removing the nucleus. Where the nucleus is very hard, the entire nucleus is expressed or aspirated. Then the softer portions of the cortex are removed by suction with an aspirator/irrigator attachment in an aspiration/irrigation

maneuver. The posterior capsule is then polished, and an intraocular lens (IOL) is implanted in the empty capsular bag. Phacoemulsification and IOL implantation require an incision only 3–6 mm in length. Where a tunnel technique is used to make this incision, no suture will be necessary as the wound will close itself.

Advantages over intracapsular cataract extraction. Extracapsular cataract extraction usually does not achieve the same broad exposure of the retina that intracapsular cataract extraction does, particularly when a secondary cataract is present. However, the extracapsular cataract extraction maintains the integrity of the anterior and posterior chambers of the eye, and the vitreous body cannot prolapse anteriorly as it does after intracapsular cataract extraction. At 0.1–0.2%, the incidence of retinal detachment after extracapsular cataract extractionis about ten times less than after intracapsular cataract extraction, which has an incidence of 2–3%.

Secondary Cataract

Epidemiology. Approximately 30% of cataract patients develop a secondary cataract after extracapsular cataract extraction.

Etiology. Extracapsular cataract extraction removes only the anterior central portion of the capsule and leaves epithelial cells of the lens intact along with remnants of the capsule. These epithelial cells are capable of reproducing and can produce a secondary cataract of fibrous or regenerative tissue in the posterior capsule that diminishes visual acuity.

Treatment. A neodymium:yttrium–aluminum–garnet (Nd:YAG) laser can incise the posterior capsule in the visual axis without requiring invasive eye surgery. This immediately improves vision.

Special Considerations in Cataract Surgery in Children

Observe changes in the child's behavior. Children with congenital, traumatic, or metabolic cataract will not necessarily communicate their visual impairment verbally. However, it can be diagnosed from these symptoms:

- Leukocoria.
- Oculodigital phenomenon. The child presses his or her finger against the eye or eyes, because this can produce light patterns the child finds interesting.
- Strabismus. The first signof visual impairment.
- The child cries when the normal eye is covered.
- The child has difficulty walking or grasping.
- Erratic eye movement is present.
- Nystagmus.

Operate as early as possible. Retinal fixation and cortical visual responses develop within the first 6months of life. This means that children who undergo surgery after the age of 1year have significantly poorer chances of developing normal vision.

Children with congenital cataract should undergo surgery as early as possible to avoid amblyopia. The prognosis for successful surgery is less favorable for unilateral cataracts than for bilateral cataracts. This is because the amblyopia of the cataract eye puts it at an irreversible disadvantage in comparison with the fellow eye as the child learns how to see [16].

Plan for the future when performing surgery. After opening the extremely elastic anterior lens capsule, one can aspirate the soft infantile cortex and nucleus. Secondary cataracts are frequent complications in infants. The procedure should therefore include a posterior capsulotomy with anterior vitrectomy to ensure an unobstructed visual axis. The operation preserves the equatorial portions of the capsule to permit subsequent implantation of a posterior chamber intraocular lens in later years.

Refraction changes constantly. The refractive power of the eye changes dramatically within a

short period of time as the eye grows. The refraction in the eye of a newborn is 30–35 diopters and drops to 15–25 diopters within the first year of life (myopic shift).

Refractive Compensation

Two main points need to be noted for optical correction of aphakia in a child age and whether the cataracts are unilateral or bilateral. Possible methods of correction include:

- Glasses can be fitted in older children with bilateral, but not unilateral aphakia. It should be noted that thick cataract glasses may be in appropriate due to their weight, for cosmetic reasons, due to prismatic distortionand ring scotoma.
- Contact lenses are a good option for unilateral and bilateral aphakia. The use of soft contact lenses in infants is difficult and requires intensive cooperation from parents. Usually they are well tolerated up to the age of 2years.
- IOL implantation is now a days a routine procedure in children over the age of 2 or 3 years. The problem in newborns is the myopic shift in the growing eye. An IOL power is there fore calculated by biometry that results in a certain amount of hyperopia, which is then corrected with glasses. During the child's subsequent growth, the hyperopia ideally moves back toward emmetropia.
- Orthoptic postoperative therapy is required. Unilateral cataracts in particular require orthoptic postoperative therapy in the operated eye to close the gap in relation to the

normal fellow eye. Regular evaluation of retinal fixation is indicated, as is amblyopia treatment.

Refraction should be evaluated by retinoscopy every 2 months during the first year of life and every 3–4 months during the second year, and contact lenses and eyeglasses should be changed accordingly.

LensDislocation

Definition:

- Subluxation (partial dislocation). The suspension of the lens (the zonule fibers) is slackened, and the lens is only partially within the hyaloid fossa.

- Luxation (complete dislocation). The lens is torn completely free and has migrated into the vitreous body or, less frequently, into the anterior chamber.

Etiology. Most frequently, it is due to trauma. Later in life, pseudoexfoliation may also lead to subluxation or luxation of the lens. Hereditary causes and metabolic disease produce lens displacement early, but on the whole are rare. Additional rare causes include hyperlysinemia (characterized by retarded mental development and seizures) and sulfite oxidase deficiency (which leads to mental retardation and excretion of cysteine in the urine). The most frequent atraumatic causes of lens dislocation are Marfan syndrome, homocystinuria, and Weill–Marchesani syndrome.

Symptoms. Slight displacement may be of no functional significance to the patient. More pronounced displacement produces severe optical distortion with loss of visual acuity.

Diagnostic considerations. The cardinal symptoms include tremulous motion of the iris and lens when the eye moves (iridodonesis and phacodonesis). These symptoms are detectable using a slit lamp examination.

Treatment. Optical considerations and the risk of secondary angle closure glaucoma due to protrusion of the iris and dislocation of the lens into the anterior chamber are indications for removal of the lens.

Literature

1. Khurana, A.K. Comprehensive ophthalmology/ A.K. Khurana, Aruj K Khurana, Brawna Khurana – 6thed. – New Delhi [etal.] : Jaypee Brothers Medical Publishers, 2015 – x, 623 p. : phot., col. ill., tab. + Review of ophthalmology : quick text review & MCQs.

2. Дравица, Л.В. Анатомия зрительного анализатора = Anatomy of the visual system : учеб.-метод. пособие по офтальмологии для студ. 4 курса лечеб. фак. и фак. по подг. спец. для зарубеж. стран мед. вузов / Л.В. Дравица, А. Альхадж Хусейн ; УО «ГомГМУ» , Каф. оториноларингологии с курсом офтальмологии. – Гомель : ГомГМУ, 2016. – 44 с. : табл., цв. ил.

3. Дравица, Л.В. Клинические методы исследования = Clinical methods for ocular examination : учеб.-метод. пособие по офтальмологии для студ. 4-6 курсов лечеб. фак. и фак. по подг. спец. для зарубеж. стран мед. вузов / Л.В. Дравица, А. Альхадж Хусейн, О.П. Садовская ; УО «ГомГМУ» , Каф. оториноларингологии с курсом офтальмологии. – Гомель : ГомГМУ, 2017. – 44 с. : табл., цв. ил.

4. Khurana, A. K. Review of ophatholmology : quick text review & MCQs [(multiple choise question)] : a free companion to «Comprehensive ophthalmology. - 6th ed. / A. K. Khurana, Aruj K Khurana, Brawna Khurana – 6th ed. – New Delhi [et al.] : Jaypee Brothers Medical Publishers, 2015. [vii], 190 p.

5. Molecular identification of viral agents associated with acute conjunctivitis: a prospective controlled study / Akçay E // The Brazilian journal of infectious diseases : an official publication of the Brazilian Society of Infectious Diseases . – 2017: – P.391-395. https://doi.org/10.1016/j.bjid.2017.03.016 Date of access: 01.04.2021

6. Chlamydial eye infections: Current perspectives / Satpathy G // Indian journal of ophthalmology. – 2017. № 65(2). – P. 97-102. https://doi.org/10.4103/ijo.ijo_870_16 Date of access: 01.04.2021

7. Ophthalmic Manifestations Of Coronavirus (COVID-19) / Hu K [et al.] // 2021: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2021 Jan –. http://www.ncbi.nlm.nih.gov/books/nbk556093/

8. Keratoconus Treatment Algorithm / Andreanos K D. [et al.] // Ophthalmology and therapy. – 2017. №6(2). P. 245–262. https://doi.org/10.1007/s40123-017-0099-1 Date of access: 01.04.2021

9. Update on the Management of Infectious Keratitis / Austin A [et al.] // Ophthalmology. – 2017. №124(11). P. 1678-1689. https://doi.org/10.1016/j.ophtha.2017.05.012 Date of access: 01.04.2021

10. Effect of Oral Voriconazole on Fungal Keratitis in the Mycotic Ulcer Treatment Trial II (MUTT II): A Randomized Clinical Trial / Prajna NV // JAMA ophthalmology, 134(12), 1365–1372 https://pubmed.ncbi.nlm.nih.gov/27787540/

11. Comparison of oral antiviral therapy with valacyclovir or acyclovir after penetrating keratoplasty for herpetic keratitis / Goldblum D [et al.] // Br J Ophthalmol. – 2008. № 92(9). P. 1201-1205 https://doi.org/10.1136/bjo.2008.138065 Date of access: 01.04.2021

12. Lagina, A., & Ramphul, K. Scleritis. In StatPearls. StatPearls Publishing, 2021. http://www.ncbi.nlm.nih.gov/books/nbk499944/ Date of access: 01.04.2021

Cataract in diabetes mellitus / Kiziltoprak H [et al.] // World journal of diabetes. – 2019. # 10(3). P – 140-153 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6422859/

6. Bilateral hypocalcemic cataract after total thyroidectomy in a young woman: case report / Daba KT [et al.] // BMC ophthalmology. – 2019. №19: 233. https://doi.org/10.1186/s12886-019-1224-9

7. Visual outcome and complications of cataract extraction after pars plana vitrectomy / Rey A [et al.] // Clinical ophthalmology . – 2018 №12. – P. 989–994 https://doi.org/10.2147/opth.s161223

8. Congenital cataract - clinical and morphological aspects / Tătaru CI [et al.] // Romanian journal of morphology and embryology = Revue roumaine de morphologie et embryologie. – 2020. №61(1). P. – 105-112 https://doi.org/10.47162/rjme.61.1.11

9. Intraocular lens optic capture in pediatric cataract surgery / Xie YB [et al.] // International journal of ophthalmology. – 2018. №11(8). – P.1403-1410 https://doi.org/10.18240/ijo.2018.08.24