МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ`Я УКРАЇНИ
Харківський національний медичний університет

STUDY GUIDE FOR SELF-WORK IN OPHTHALMOLOGY

Part 3

МЕТОДИЧНІ ВКАЗІВКИ ЩОДО САМОПІДГОТОВКИ З ОФТАЛЬМОЛОГІЇ

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Topic: "Pathology of cornea"

I. Topic Relevance

The external eye is the most crucial part of the body exposed to the outside world. The normal structure and function of the healthy eye rely on homeostasis of the entire body for protection against an adverse environment. Genetics and nutrition determine the embryogenesis and growth of the eye. Intact vascular and nervous systems ensure stable metabolism, and the immune system maintains surveillance.

The epidermis and epithelium of healthy eyelids, conjunctiva, and cornea adhere tightly to their basement membranes. Regulation of cellular growth and metabolism are critical to the maintenance of an intact ocular surface and a transparent cornea. The underlying extracellular matrix of the eye's mucous membrane is rich in blood vessels. The anterior segment of the eye provides a clear, protected entrance for light that is to be processed by the visual pathways through the central nervous system.

II. Study Objective

Student should know the following subjects:

- Epidemiology, pathogenesis of corneal diseases.
- Clinic of keratitis.
- Treatment of keratitis.
- Evaluation of objective status of cornea.
- To give urgent help in the cases of keratitis.
- Describe the anatomy and molecular biology of the cornea.

Student should be able to:

- explain the pathogenesis of common disorders affecting cornea;
- recognize the distinctive signs of specific diseases of the cornea;
- outline the steps in an ocular examination for corneal disease and choose the appropriate laboratory and other diagnostic tests;
- apply the results of recent clinical research to the management of selected disorders of the cornea;
- perform Slit-Lamp Examination;
- perform an inspection with side illumination;
- perform an inspection corneal sensitivity.

III. Educational Objective

1. To facilitate the understanding of the importance of the patient's complete objective examination in order to establish correct diagnosis and indicate the surgical treatment.
2. To facilitate the learning of the principles of the medical ethics and the deontology in patients' examination.
3. To exemplify the importance of the subject knowledge.

IV. Interdisciplinary Integration:

Subject and proper chair	To know:	To be able to:
Anatomy	Topography of the cornea	
Histology	Histological texture of the cornea	To be able to describe normal status of cornea
Phatological anatomy	Main signs of inflammation	To know the clinic of keratitis
Propedeutics	Methods of examination of the patient	To collect anamnesis, perform inspection and objective examination
Therapy	Diseases which may be the cause of keratitis	To determine the scheme of investigation, including additional laboratory methods
Pediatric	Congenital Anomalies and Abnormalities	To make diagnosis of congenital anomalies and abnormalities of the cornea
Ophthalmology	External Examination	To evaluate changes in the eye
	Glaucoma	To know the main signs of congenital glaucoma

V. Lesson topic contents.

The cornea is a transparent, avascular tissue that measures 11–12 mm horizontally and 10–11 mm vertically. Its refractive index is 1.376, although a refractive index of 1.3375 is used in calibrating the keratometer to account for the combined optical power of the anterior and posterior curvatures of the cornea. The cornea is aspheric, although its radius of curvature is often recorded as a spherocylindrical convex mirror representing the central anterior corneal surface, also called the corneal cap.

The average radius of curvature of the central cornea is 7.8 mm (6.7–9.4 mm). The cornea thus contributes 74 %, or 43.25 diopters (D), of the total 58.60 dioptric power of a normal human eye. The cornea is also the major source of astigmatism in the optical system.

For its nutrition, the cornea depends on glucose diffusing from the aqueous humor and oxygen diffusing through the tear film. In addition, the peripheral cornea is supplied with oxygen from the limbal circulation.

The cornea has one of the body's highest densities of nerve endings, and the sensitivity of the cornea is 100 times that of the conjunctiva. Sensory nerve fibers extend from the long ciliary nerves and form a subepithelial plexus. Neurotransmitters in the cornea include acetylcholine, catecholamines, substance P, and calcitonin gene-related peptide.

Clinical Evaluation of Ocular Inflammation

In clinical practice, it is helpful to use key distinctive features to categorize a patient's problem. The major disease mechanisms of the outer eye that the clinician should recognize by the history and examination are the following:

- Infection.
- Immune alteration.
- Neoplasia.
- Maldevelopment.
- Degeneration.
- Trauma.

Because redness is often a feature of infection, allergy, neoplasia, injury, and other conditions, the following information introduces the most common signs of ocular inflammation.

Corneal Signs of Inflammation

Inflammation can affect any layer of the cornea. The pattern of corneal inflammation, or *keratitis,* can be described according to the following:

Distribution: diffuse, focal, or multifocal.

Depth: epithelial, subepithelial, stromal, or endothelial.

Location: central or peripheral.

Shape: dendritic, disciform, etc.

The clinician should also note any structural or physiologic changes associated with keratitis, such as ulceration or endothelial dysfunction.

Punctate epithelial keratopathy is a nonspecific term that includes a spectrum of biomicroscopic changes from punctate epithelial granularity to erosive and inflammatory changes.

A key feature of *stromal inflammation* is the presence of new blood vessels. Active corneal blood vessels most commonly come from the limbal vascular arcades and migrate into the peripheral cornea. Cells can also enter the stroma from the tear film through an epithelial defect or, less often, from direct interlamellar infiltration of leukocytes at the limbus. Inflammatory cells enter from aqueous humor in the presence of endothelial injury. In a vascularized cornea, inflammatory cells can emanate directly from infiltrating blood and lymphatic vessels.

Endothelial dysfunction often accompanies corneal stromal inflammation and contributes to epithelial and stromal edema. Swollen endothelial cells called *inflammatory pseudoguttae* are visible by specular reflection as dark areas of the normal mosaic pattern. *Keratic precipitates (KP)* are clumps of inflammatory cells on the back of the cornea that come from the anterior uvea during the course of keratitis or uveitis.

Inflammation can lead to corneal opacification. Altered stromal keratocytes fail to produce some water-soluble factors and, consequently, make new collagen fibers that are disorganized, scatter light, and form a nontransparent scar. Scarring can also incorporate calcium complexes, lipids, and proteinaceous material. Dark pigmentation of a residual corneal opacity is often a result of incorporated melanin or iron salts. Corneal inflammation can also lead to neovascularization. Superficial stromal blood vessels originate as capillary buds of limbal vascular arcades in the palisades of Vogt. New lymphatic vessels may also form but cannot be seen clinically. Subepithelial fibrous ingrowth into the peripheral cornea is called a *pannus* or vascularized pannus. Neovascularization may invade the cornea at deeper levels depending on the nature and location of the inflammatory stimulus. Any vessel tends to remain at a single lamellar plane as it grows unless stromal disorganization has occurred.

Table 2-1

Ocular Infections of Global Importance

Disease Burden	No. of Visually Disabled	High Regional
Trachoma	5,800,000	Africa
Herpes simplex virus keratitis	1,000,000	Worldwide
Microbial keratitis	500,000	Worldwide
Toxoplasmosis	500,000	Latin America
Onchocerciasis	500,000	Africa, Latin America
Hansen disease	250,000	Southeast Asia
Neonatal conjunctivitis	200,000	Africa

Table 2-2

Common Causes of Corneal Inflammation

Finding	Examples
Punctate epithelial erosions	Dry eye syndrome Toxicity Atopic keratoconjunctivitis
Punctate epithelial keratitis	Adenovirus keratoconjunctivitis Herpes simplex virus epithelial keratitis Thygeson superficial punctate keratitis
Stromal keratitis, suppurative	Bacterial keratitis Fungal keratitis
Stromal keratitis, nonsuppurative	Herpes simplex virus stromal keratitis Varicella-zoster virus stromal keratitis Syphilitic interstitial keratitis
Peripheral keratitis	Blepharitis-associated marginal infiltrates Peripheral ulcerative keratitis caused by connective tissue diseases Mooren ulcer

Table 2-3

Causes of Corneal Edema

Type	Cause
Acute	Trauma (eg, epithelial defect, intraocular surgery) Inflammation (eg, infectious or immune-mediated keratitis, corneal graft rejection) Hypoxia (eg, contact lens overwear) Hydrops from ruptured Descemet's membrane (eg, keratoconus) Increased intraocular pressure
Chronic	Trauma or toxins (eg, intraocular surgery) Fuchs dystrophy Posterior polymorphous dystrophy Iridocorneal endothelial syndrome

Epithelial keratitis

CLINICAL PRESENTATION. Patients with epithelial keratitis complain of foreign body sensation, light sensitivity, redness, and blurred vision. HSV infection of human corneal epithelium manifests as areas of punctate epithelial

keratitis that may coalesce into one or more arborizing dendritic epithelial ulcers with terminal bulbs at the end of each branch. The cytopathic swollen corneal epithelium at the edge of a herpetic ulcer stains with rose bengal due to loss of cell membrane glycoproteins and subsequent lack of mucin binding by the cells. The bed of the ulcer stains with fluorescein due to loss of cellular integrity and absence of intercellular tight junctions.

Patients with HSV epithelial keratitis exhibit a ciliary flush and mild conjunctival injection. Mild stromal edema and subepithelial white blood cell infiltration may develop beneath epithelial keratitis. Following resolution of dendritic epithelial keratitis, nonsuppurative subepithelial infiltration and scarring may be seen just beneath the area of prior epithelial ulceration, resulting in a ghost image, or *ghost dendrite*, reflecting the position and shape of the prior epithelial involvement. Resolution typically occurs without additional therapy.

Focal or diffuse reduction in corneal sensation develops following HSV epithelial keratitis. The distribution of corneal hypoesthesia is related to the extent, duration, severity, and number of recurrences of herpetic keratitis. Sectoral corneal anesthesia may be difficult to detect clinically and is not a reliable sign of herpetic disease.

Other conditions that may produce dendritiform epithelial lesions include:

- Varicella-zoster virus.
- Adenovirus (uncommon).
- Epstein-Barr virus (rare).
- Epithelial regeneration line.
- Neurotrophic keratopathy (postherpetic, diabetes mellitus).

Bacterial Keratitis

Bacterial infection is a common sight-threatening condition. Some cases have explosive onset and rapidly progressive stromal inflammation. Untreated, it often leads to progressive tissue destruction with corneal perforation or extension of infection to adjacent tissue. Bacterial keratitis is frequently associated with risk factors that disturb the corneal epithelial integrity. Common predisposing factors include:

- Contact lens wear.
- Trauma.
- Contaminated ocular medications.
- Impaired defense mechanisms.
- Altered structure of the corneal surface.

The most frequent risk factor for bacterial keratitis is contact lens wear, which has been identified as such in 19–42 % of patients who develop culture-proven microbial keratitis. Epidemiologic studies have estimated the annual incidence of cosmetic contact lens-related ulcerative keratitis at 0.21 % for individuals using extended-wear soft lenses and 0.04 % for patients using daily-wear soft lenses. The risk of developing microbial keratitis increases significantly (approximately 15 times) in patients who wear their contact lenses overnight

and is positively correlated with the number of consecutive days lenses are worn without removal.

PATHOGENESIS. Bacteria have multiple mechanisms of adherence. For example, *S aureus* uses adhesins to bind to collagen and other components of the exposed Bowman's layer and stroma, while *P aeruginosa* can bind to molecular receptors exposed on injured epithelial cells.

Corneal inflammation begins with the local production of cytokines and chemokines that enable diapedesis and migration of neutrophils into the peripheral cornea from the limbal vessels. Some microorganisms produce proteases that disrupt the extracellular matrix. Enzymes released by neutrophils and activation of corneal matrix metalloproteinases exacerbate inflammatory necrosis. With antimicrobial control of bacterial replication, wound healing processes begin that may be accompanied by neovascularization and scarring. Progressive inflammation, on the other hand, may lead to corneal perforation.

CLINICAL PRESENTATION. Rapid onset of pain is accompanied by conjunctival injection, photophobia, and decreased vision in patients with bacterial corneal ulcers. The rate of progression of these symptoms depends on the virulence of the infecting organism. Bacterial corneal ulcers typically show a sharp epithelial demarcation with underlying dense, suppurative stromal inflammation that has indistinct edges and is surrounded by stromal edema. *P aeruginosa* typically produces stromal necrosis with a shaggy surface and adherent mucopurulent exudate. An endothelial inflammatory plaque, marked anterior chamber reaction, and hypopyon frequently occur.

LABORATORY EVALUATION. A wide variety of bacterial species can cause microbial keratitis. The prevalence of a particular causative organism depends on the geographic location and risk factors for the infection. Common and uncommon organisms causing bacterial keratitis are listed in Table.

Because keratitis can be caused by a wide variety of organisms, many of which have unique antimicrobial sensitivity profiles, it can be difficult to determine whether a corneal ulcer has an infectious etiology. Before initiating antimicrobial therapy for cases of suspected bacterial keratitis, the clinician should consider conducting microbiological diagnostic tests.

If the patient has already been treated with topical antibiotics, the medication should be stopped for 12–24 hours prior to culturing in order to enhance recovery of viable organisms. However, antimicrobial therapy should not be discontinued in severe or rapidly progressive corneal ulcers.

In addition to culturing the cornea, it may be helpful to culture contact lenses, contact lens cases, solutions, and any other potentially contaminating sources such as inflamed eyelids, because any of these might provide a clue to the causative organism in the event that corneal cultures are negative. This approach can also help identify the source of the infection.

MANAGEMENT. Currently, no single antibiotic agent is effective against all bacterial species causing microbial keratitis. Initial broad-spectrum therapy is recommended until the offending microorganism is identified in culture. If one type of bacteria is prominently identified on a stained diagnostic smear, therapy may initially be weighted toward that class of microorganism. Broad-spectrum therapy, however, should not be eliminated, as cultures may reveal a different class of microorganism. Once the offending microbe is identified, or the clinical response suggests the change, then appropriate monotherapy may be considered.

The route of antibiotic administration should be based on the severity of the keratitis. Subconjunctival and IV antibiotics in addition to frequent topical antibiotics are indicated in cases with suspected scleral and/or intraocular extension of infection.

Table 2-5

Causes of Bacterial Keratitis

Common Organisms	Uncommon Organisms
Staphylococcus aureus	*Neisseria* species
Staphylococcus epidermidis	*Moraxella* species
Streptococcus pneumoniae and other *Streptococcus* species	*Mycobacterium* species *Nocardia* species
Pseudomonas aeruginosa (most common organism in soft contact lens wearers)	Non-spore-forming anaerobes
Enterobacteriaceae (*Proteus, Enterobacter, Serratia)*	*Corynebacterium* species

Corticosteroids

The role of corticosteroid therapy for bacterial keratitis is controversial.

Surgery

Penetrating keratoplasty for treatment of bacterial keratitis is indicated if the disease progresses despite therapy, descemetocele formation or perforation occurs, or the keratitis is unresponsive to antimicrobial therapy. The involved area should be identified preoperatively, and an attempt should be made to circumscribe all areas of infection. Peripheral iridectomies are indicated, since patients may develop seclusion of the pupil from inflammatory pupillary membranes. Interrupted sutures are recommended. The patient should be treated with appropriate antibiotics, cycloplegics, and intense topical corticosteroids postoperatively.

Universal Precautions

Optimal infection control is based on the assumption that all specified human body fluids are potentially infectious. Many transmissible diseases of the external eye, such as adenoviral conjunctivitis, cause redness that immediately indicates infection. Other infectious agents, however, can be present on the ocular surface without causing inflammation. Human immunodeficiency virus (HIV), hepatitis B virus, hepatitis C virus, rabies virus, and the agent of Creutzfeldt-Jakob disease are not immediately obvious without systemic clues or laboratory testing. Every patient must be approached as potentially contagious. Guidelines for routine ophthalmic examinations include the following:

• Wash hands between patient examinations. Use disposable gloves if an open sore, blood, or blood-contaminated fluid is present. Using cotton-tipped applicators to manipulate the eyelids can also minimize direct contact.

• Avoid unnecessary contact. Eyedropper bottles used in the office should not directly touch the ocular surface of any patient. Individual sterile strips impregnated with dye are preferred where available.

• Disinfect all contact instruments after each use. Tonometer tips and pachymeter tips should be soaked in diluted bleach or hydrogen peroxide or cleansed with isopropyl alcohol after every use. Trial contact lenses must be disinfected between patients. BCSC Section 10, *Glaucoma,* discusses infection control in clinical tonometry in greater detail.

• Handle sharp devices carefully. Needles must always be discarded into puncture-resistant (sharps) containers.

VI. Structure and Organization of the Lesson

6.1. Duration of the lesson – 2,2 academic hours

6.2. Lesson Stages (table)

№	Basic lesson stages and their contents	Study objectives and their mastering level	Training and control methods	Materials for the methodic supply	Time in %
I 1 2 3	**Preliminary stage** Lesson organization. Study objectives setting. Control of basic knowledge, experiences, skills	 A – I A – II A – III	 Oral questions on topic, test computer or standard-paper program	Students' assessment register. Control questions on topic, A-Form tests	15
II 1	**Basic stage** Anatomy of the cornea	 A – II	 Oral questions on topic, test computer or standard-paper program	 Models, presentations, atlases, tests	75
2	Modern techniques of the cornea examination	A – III	Individual control	Slit-lamp, ophthalmoscope, lenses 13 Dptr and 20 Dptr, tests, patients	
3	Classification of main pathology of the cornea	A – III A – IV	Oral questions on topic, test computer or standard-paper program	Models, presentations, atlases, tests	

№	Basic lesson stages and their contents	Study objectives and their mastering level	Training and control methods	Materials for the methodic supply	Time in %
4	Modern methods of keratitis management	A – III A – IV	Oral questions on topic, test computer or standard-paper program	Models, presentations, atlases, tests, videos	
5	To make diagnosis of keratitis, prescribe treatment	A – III A – IV	Test computer or standard-paper program	Presentations, tests, patients	
III	**Final stage**				
1	Control and correction of professional skills and experience.	A – II A – III A – IV			
2	Making conclusion of the lesson (individual questioning, discussion of the examined patients' cases, revision of the diagnosis establishment methods).	A – IV		Out-patient card of the in-patient, students' assessment register, A-Form questions.	10
3	Home task			Theme outline of practical lessons, study guides for practical work	

VII. Materials for the methodic lesson supply.

1. Materials for the lesson preliminary stage.

A. Lesson topic control questions:

1. Main characteristics of cornea.
2. Blood supply of cornea
3. Sensitivity of cornea
4. Exogenous and endogenous keratitis
5. Clinic of keratitis
6. Prophylaxis of keratitis

B. Tests

1. The normal human cornea…

a. Has a refractive index less than air.

b. Is less than 1 mm thick.

c. Is innervated by terminal branches of the superorbital nerve from the first division of the trigeminal nerve (cranial nerve V).

d. Receives most of its oxygen supply for aerobic metabolism from the limbic circulation.

e. Efficiently transmits ultraviolet C (190–290 nm) light.

Answer – b. The normal cornea measures approximately 0.5 mm centraly and 0.7 mm peripherally. The refractive index of the cornea is 1.3 compared to the 1.000 of air, although most keratometers and topograph instruments assume a combined refractive index for the corneal surface S 1.3375. The cornea, one of the most sensitive tissues in the body, receive its sensory nerve supply from the long ciliary nerves derived from the ophthalmic division of the trigeminal nerve. Sleeping with prolonged eyelid closure results in lower oxygen levels and a shift from aerobic to anaerobic metabolism. The cornea absorbs ultraviolet radiation for wavelengths less than 290 nm (UVC). Ultraviolet photokeratopathy can result from exposure to a germicidal UVC lamp or to a welding arc.

2. Which of the following statements regarding the normal adult cornea is NOT true?

a. The extracellular matrix of the normal corneal stroma consists primarily of a glycosaminoglycan, keratan sulfate, attached to a core protein, lumican.

b. The sum of the imbibition pressure and swelling pressure of the cornea equals the intraocular pressure.

c. Keratocytes synthesize procollagen and collagen-degrading enzymes.

d. Collagen fibrils are assembled into fibers that form about 300 lamellae.

e. Keratocytes in one plane are prevented from contacting other cell layers by each lamella.

Answer – e. Proteoglycans of the cornea are composed of glycosaminoglycans attached to core proteins. Keratan sulfate is the major glycosaminoglycan in a normal adult cornea, although hyaluronan is present in the embryonic cornea and during wound healing. Keratan sulfate binds to lumican, keratocan, and osteoglycin. Interfibrillary expansion between negatively charged glycosaminoglycans produces a swelling pressure (SP) that is related to the imbibition pressure (IP) and intraocular pressure (IOP) by the following formula: IP = IOP – SP. Keratocytes synthesize the extracellular matrix of the corneal stroma, including matrix metalloproteases that degrade collagen, resulting in slow collagen turnover. About 300 lamellae are stacked parallel to the corneal surface, running from limbus to limbus. Scattered between these lamellae, keratocytes contact one another to form a three-dimensional network throughout the stroma.

3. Which of the following does NOT usually accompany or follow corneal inflammation?

a. Pannus.

b. Pseudoguttata.

c. Ghost vessels.

d. Lipid keratopathy.

e. Hassall-Henle bodies.

Answer – e. Corneal inflammation often produces transient endothelial

dysfunction, manifesting structurally as endothelial pseudoguttata and stromal edema. Neovascularization may lead to fibrovascular proliferation from the limbus, called a pannus. These vessels may deposit cholesterol, phospholipids, and neutral fats, resulting in lipid keratopathy. Resolution of disease often leaves faint ghost vessels. Hassall-Henle bodies are peripheral guttata that are more common with advancing age than with corneal inflammation.

4. Each of the following statements pertaining to corneal topography is true EXCEPT:

a.Computerized videokeratoscopy of a perfectly smooth, spherical globe should show a simulated keratometry measurement of zero.

b.The central portion of a normal cornea is steeper than the periphery.

c.The orthogonal simulated K values are determined from more points reflected from the central cornea than are the values that are obtained by keratometry.

d. With-the-rule astigmatism produces a vertical bow-tie topographic pattern.

e.A color map is a result of computerized analysis of placido disk photo-keratoscopy.

Answer – a. Topographic mapping of the cornea by a videokeratoscope is more efficient than keratometry because many more points are measured simultaneously. For example, regular astigmatism produces bow-tie patterns. Irregular astigmatism is more difficult to quantify. Topographic indices include the simulated keratometric (SIM K).

5. Recurrent corneal erosions…

a. *Are almost always associated with subepithelial haze.*

b. Are typically present within 1 month after a corneal abrasion.

c. May occur spontaneously.

d. Are uncommon over the age of 50 years.

e. Frequently require phototherapeutic keratectomy.

Answer – c. Corneal erosions may be a manifestation of corneal epithelial-based membrane dystrophy. It may be difficult to detect on slit-lamp biomicroscopy. Recurrent erosions may also follow trauma, often several months or even years after the initial injury. Recurrent corneal erosion can occur at any age, but its prevalence is higher after the age of 50. Acute erosion responds to patching. Preventive therapy includes lubrication and hypertonic agents. Debridement or stromal micropuncture may be effective if more conservative management fails.

6. Neurotrophic keratopathy…

a.Results from damage to cranial nerve VII.

b.Is rarely associated with bulbar conjunctival anesthesia.

c.May be associated with a persistent epithelial defect.

d.Is more likely with herpes simplex virus than with herpes zoster virus.

e.Produces decreased osmolarity of the tear film.

Answer – c. Neurotrophic keratopathy is frequently associated with a persistent corneal epithelial defect, usually located in the palpebral fissure just below midline. These neurotrophic epithelial defects are usually horizontally oval with rolled edges. When lubrication and more conservative measures fail, a tarsorrhaphy is often required. Damage to cranial nerve VII results in neuroparalytic keratopathy. Corneal anesthesia associated with cranial nerve V involvement is the principal cause of neurotrophic keratopathy, and damage to both nerves leads to serious ocular surface disease. Causes of neurotrophic keratopathy include ophthalmic zoster.

7. Which of the following statements concerning conjunctival xerosis is INCORRECT?

a. Bitot spots are composed of keratin and saprophytic bacteria.

b. Night blindness often precedes xerophthalmia.

c. Conjunctival keratinization can occur as a result of an intestinal malabsorption syndrome.

d. Children with vitamin A deficiency have fewer viral infections because of immune hyperreactivity.

e. Topical retinoic acid can affect conjunctival differentiation.

Answer – d. Approximately five million children, predominantly in developing nations, have xerophthalmia. Altered dietary intake, impaired intestinal absorption, or altered liver storage and metabolism are the most common reasons for conjunctival xerosis in the United States. Ingestion of vitamin A or carotenoids with provitamin activity, such as beta-carotene, results in the absorption of retinol that is combined in the liver with retinol-binding protein. Retinol is an essential component of rhodopsin in the rod photoreceptors and is required for maintenance of mucous membranes. Vitamin A deficiency results in suppression of cell-mediated immunity that can predispose to herpes simplex virus keratitis and measles. Conditions such as ocular cicatricial pemphigoid and Stevens-Johnson syndrome that result in conjunctival keratinization may respond to topical retinoic acid.

8. A 30-year-old woman with chronic irritation in both eyes is found to have mild papillary conjunctivitis, punctate staining of the superior cornea and conjunctiva of both eyes, and filaments attached to the upper limbus of one eye. Bulbar conjunctival biopsy of the involved area showed keratinization, acanthosis, and intracellular glycogen granules. **Which of the following diagnostic tests would be the most appropriate?**

a. Bacterial cultures of the eyelid margins.

b. Histamine level of the tear film.

c. Serum rheumatoid factor.

d. Thyroid function testing.

e. HLA typing.

Answer – d. Superior limbic keratoconjunctivitis (SLK) is characterized

by chronic keratoconjunctivitis with inflammation of the superior bulbar conjunctiva and adjacent cornea. Filaments of the limbus and superior cornea occur in approximately one third of patients. Thyroid disease may be an associated condition.

9. Which of the following statements regarding pigmentation of the cornea is the most accurate?

a. Iron lines associated with keratoconus run parallel with Vogt's striae.

b. A total hyphema associated with increased intraocular pressure produces yellow-green deposits within the cornea.

c. Corneal chrysiasis produces vortex opacity of the central cornea.

d. Chloroquine and related drugs occasionally produce conjunctival and corneal argyriasis with pigmentation in peripheral endothelial cells.

e. The yellow deposits of ochronosis often resemble pinguecula.

Answer – b. Gold (chrysiasis) and silver (argyriasis) deposits within the cornea stroma may occur from local or systemic use. A Fleischer ring associated with keratoconus is an iron line around the base of the cone. Other iron lines may be associated with a pterygium (Stocker line), filtering bleb (Ferry line), and other corneal surface irregularities. Corneal blood staining that complicates hyphema with elevated intraocular pressure begins as small yellow within the posterior stroma, caused by hemoglobin and its metabolites can eventually produce a rust-colored opacity.

10. The most important complications after keratoprosthesis surgery include…

a. Wound necrosis.

b. Uveitis.

c. Endophthalmitis.

d. Glaucoma.

e. All of the above.

Answer – e. Keratoprosthesis surgery is rarely performed because of vision-threatening complications. Advances in keratoprosthesis design seek to prevent tissue necrosis around the keratoprosthesis and to improve wound healing. Postoperative uveitis contributes to vitreous opacities, cystoid macular edema, and retinal detachment. Postsurgical endophthalmitis is generally a complication of impaired wound healing with perforation. Glaucoma has been the most common complication leading to visual loss.

11. Which of the following statements concerning a corneal abrasion is the most accurate?

a. A corneal abrasion can easily be differentiated from early bacterial keratitis during external examination in the emergency room by using fluorescein eyedrops.

b. A corneal abrasion should always be patched to limit pain and to prevent neovascularization.

c. Corneal trauma is best managed by a collagen shield to allow the administration of a topical corticosteroid.

d. Trauma often precipitates future corneal erosions. When associated with contact lens wear, the eye should not be patched because of the possibility of promoting infection.

Answer – e. A corneal abrasion is the traumatic removal of all or some of the epithelial layers of the cornea. Exclusion of microbial contamination can be difficult soon after the injury, especially when the abrasion is caused by a dirty foreign body or contact lens. A patch, collagen shield, or bandage contact lens can reduce discomfort but should be used cautiously when microbial keratitis is likely. Topical non-steroid anti-inflammatory agents can be useful to relieve discomfort.

Materials for lesson basic stage

1. Differential diagnosis of keratitis, erosion, leukemia

Clinical signs	keratitis	erosion	leukemia
Pain	+	+	-
Lacrimation	+	+	-
Photophobia	+	+	-
Decreasing of VA	+	+	+
Redness of the eye	+	+	-
Infiltration	+	-	-
Fluorescein test	+	+	-

2. Diagnosis of keratitis

1. History of disease.
2. To perform external examination.
3. To perform examination in side illumination.
4. To perform an inspection in transmitted light.
5. To examine corneal abrasion.
6. To examine the sensitivity of cornea.
7. Pachymetry.
8. Ophthalmometry.
9. Additional laboratory tests.

3. How to examine a patient

Algorithm	
1. To take medical history	1. Pay attention to systemic diseases
	Visual acuity testing is an essential part of the examination, and the refraction of a patient with an abnormal cornea requires special attention. If visual acuity is reduced because of corneal irregularity, it may be necessary to use a rigid gas-permeable (RGP) contact lens with overrefraction. One method is to obtain keratometry readings and select a large diameter hard contact lens with a base curve halfway between the two powers and with a power near the patient's spherical equivalent.

	Topical anesthesia helps reduce tearing as the spherical overrefraction is performed. The potential acuity meter provides another method for assessing visual acuity potential in a patient with focal corneal opacification or surface irregularity, but this instrument can give misleading results.
2. To perform external examination	External examination of the outer eye and adnexa begins with the examiner looking at the patient, preferably in daylight or bright room light, then proceeding to magnification with focal illumination. The simplest magnifying instruments are loupes and condensing lenses like those used for indirect ophthalmoscopy. Many handheld penlights and transilluminators are also available, and these tools are helpful at the bedside and for external surgical procedures. Pay attention to blepharospasm, lacrimation, injection of the eye etc.
3. To perform examination in side illumination	1. To sit in front of a patient 2. The lamp should be in the left side of him 3. To take lens 13,0–20,0 D into the left hand and focus the light on to the eye (5–7 sм).
4. To examine the sensitivity of cornea	1. To sit in front of a patient 2. To take hare of Frey and touch cornea in 5 dots (6, 9, 12 and 3 hours and central part) 3. To evaluate sensitivity like + or –
5. To examine corneal abrasion	To sit in front of a patient. To ask a patient to look up. To put fluorescein drops into his eye. To wash out the eye with furacilline 1 : 5 000. Check a patient with slit-lamp. Topical fluorescein is a nontoxic, water-soluble dye that is available in several forms: as a 0.25 % solution with an anesthetic (benoxinate or proparacaine), an antiseptic (povidone-iodine), and a preservative; as a 2 % non-preserved unit-dose eyedrop; and in impregnated paper strips. Fluorexon is a related macromolecular compound available as a 0.35 % non-preserved solution that will not stain most contact lenses. Fluorescence is easily detected with a cobalt blue filter. Fluorescein is most commonly used for planation tonometry and evaluation of the tear film. Tear breakup time is measured by instilling fluorescein, asking the patient to hold the eyelids open after one or two blinks, and counting the seconds until a dry spot appears. Fluorescein will stain punctate and macroulcerative epithelial defects (positive staining), and it can highlight non-staining lesions that project through the tear film (negative staining). Different disease states can produce various punctate staining patterns. Fluorescein that collects in an epithelial defect will diffuse into the corneal stroma and cause a green flare in the anterior chamber. In the dye disappearance test, the tear meniscus is observed for the disappearance of fluorescein. Prolonged presence of dye suggests either blockage of the drainage system or decrease in tear flow consistent with lacrimal deficiency. The Seidel test is used to detect seepage of aqueous humor through a corneal perforation. The examiner applies

	fluorescein using a moistened strip or concentrated drop to the site of suspected leakage and looks for a flow of clear fluid streaming through the orange dye under cobalt blue light.
6. To make diagnosis of keratitis and prescribe the urgent treatment	1. To perform external examination 2. To perform examination in side illumination 3. To perform an inspection in transmitted light 4. To examine corneal abrasion 5. To examine sensitivity of cornea 6. To prescribe treatment

1. Having worked in his garden, a 57-year-old man complains of moderate discomfort and redness in his right eye. You note a visual acuity of 20/25 in the right eye and 20/15 in the left eye. The right eye has mild hyperemia of the conjunctival vessels; the right cornea appears clear to penlight examination. You diagnose a probable allergy to pollen and advise the patient to use topical dexamethasone sodium phosphate 0.1 % for 3 days.

A. Give two reasons why your diagnosis of allergic conjunctivitis is unlikely to be correct.

Answer: Unless the patient has always had weaker vision in his right eye, this finding should alert you to the possibility of a more serious inflammation, such as iritis, keratitis, or glaucoma. Also, the patient did not complain of itching, which you might expect in an allergic reaction.

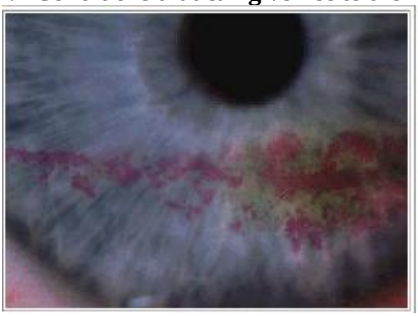
B. What other diagnostic techniques should you have performed to be certain that the cornea is normal?

Answer: If the patient has an early herpes simplex keratitis or if his cornea has been scratched by a twig, the epithelial disruption might not be easily seen during a penlight examination. However, it most likely would be rendered visible by fluorescein staining of the cornea.

C. Is there any hazard in your prescribed course of treatment? Explain.

Answer: Yes. The virulence of both herpes simplex and fungal infections, which can result from trauma involving organic material, is markedly potentiated by the application of topical corticosteroids to the eye.

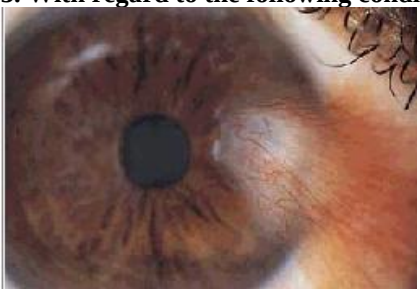
2. Conditions that can give rise to the following appearance include:



a. superior limbic keratoconjunctivitis.
b. Sjogren's syndrome.
c. Bell's palsy.
d. Thygeson's keratitis.
e. adenoviral keratitis.

a – F, b – T, c- T, d – F, e- F

3. With regard to the following condition:



a. the Bowman's layer is seldom involved.
b. there are three components (ie. a head, cap and body) to this tissue.
c. the whole abnormal structure is adherent to the corneal surface.
d. it is commoner in white than black population.
e. the following corneal topography may be taken from a patient with this condition.

a – F, b – T, c – T, d – F, e – T

4. The following are true about herpes simplex:

a. herpes keratitis in infants is caused mainly by type II herpes simplex.
b. ocular disease is the most common manifestation of primary type I herpes simplex.
c. herpes simplex can be contracted from pests.
d. ocular recurrence is usually caused by virus reactivation within the trigeminal ganglia.
e. ocular recurrence is more common than either genital or oral recurrence.

than ocular disease in primary type I herpes simplex.

5. With regard to herpes epithelial ulcer:

a. the sensation is reduced.
b. the disease usually presents initially as punctate keratitis.
c. geographical ulcer occurs as a result of inappropriate use of topical steroid.
d. the ulcer has typical tapering endings.
e. Rose Bengal stains infected epithelial cells.

a.T b.T c.T d.F e.T

6. The following are true about acyclovir

a. topical acyclovir is more effective than oral acyclovir in the treatment of herpes simplex epithelial keratitis.
b. valacyclovir is the prodrug of acyclovir.
c. the dosing frequency is less with valacyclovir than acyclovir.
d. oral acyclovir reduces the incidence and severity of postherpetic neuralgia.
e. oral acyclovir given daily can reduce the recurrent of herpes simplex epithelial. keratitis

a.F b.T c.T d.F e.T

7. In the treatment of herpes simplex ocular disease:

a. oral acyclovir has no effect on the development of stromal keratitis in the subsequent year when given during active herpes simplex keratitis.

b. oral acyclovir reduces the development of iritis in the subsequent year when given during active herpes simplex keratitis.

c. in patients with previous herpes simplex stromal keratitis, oral acyclovir reduces the likelihood of recurrent stromal disease.

d. stopping oral acyclovir in a patient with previous herpes simplex stromal keratitis causes rebound of herpes simplex disease.

e. oral acyclovir has no effect on herpes simplex iritis.

a.T b.F c.T d.F e.F

8. In the treatment of herpes simplex stromal disease, HEDS (Herpetic Eye Disease Studies) show that:

a. topical steroid reduces the duration of the disease.

b. patients on topical steroid have better visual outcome at six months review than the non-steroid treated group.

c. topical steroid increases the likelihood of epithelial disease.

d. systemic steroid is useful in the management of necrotizing stromal keratitis.

e. oral acyclovir has added benefit in patients treated with topical steroid and trifluridine.

a.T b.F c.F d.F e.F

Supporting materials required for learning

1. Participation in clinical duties on admission
2. Working in library

Recommended literature

1. Khurana A.K. Ophthalmology / A.K. Khurana. – New Delphi, 2006.

2. Lang Gerhard K. Ophthalmology: a short textbook / K. Lang Gerhard. – Stuttgart : New York, 2000.

3. Keiki R. Mehta Eye Care / R. Keiki. – Bombay, 1988.

4. Kanski Jack J. Clinical Ophthalmology: a systematic approach / J. Kanski Jack. – Wellington, 1994.

Навчальне видання

Методичні вказівки щодо самопідготовки з офтальмології

Частина 3

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STUDY GUIDE FOR SELF-WORK IN OPHTHALMOLOGY

Part 3