МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

Diseases of conjunctiva

Manual for individual work for English speaking foreign students

Захворювання кон'юнктиви

Методичні вказівки для індивідуальної підготовки студентів-іноземців з англійською мовою навчання

Затверджено
вченою радою ХНМУ.
Протокол № 11 від 26.12.2013.

Харків
ХНМУ
2014

Diseases of conjunctiva : manual for individual work for English speaking foreign students / compl. P.A. Bezditko, M.V. Panchenko, S.F. Zubarev et al. – Kharkiv : KNMU, 2014. – 16 p.

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Захворювання кон'юнктиви : метод. вказ. для індивід. підготовки студентів-іноземців з англ. мовою навчання / упор. П.А. Бездітко, М.В. Панченко, С.Ф. Зубарев та ін. – Харків : ХНМУ, 2014. – 16 с.

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THE REQUIRED MINIMUM OF BASIS SKILLS, WHICH ARE TO BE MASTERED BY FOREIGN STUDENTS STUDDING THE COURSE OF OPHTHALMOLOGY

Student should be acquainted with the following subjects
- The structure of the conjunctiva
- Prevalence, etiology and pathogenesis of conjunctivitis.
- Clinical manifestation and pathogenesis of acute bacterial conjunctivitis.
- Complications and treatment of the gonococcal keratoconjunctivitis.
- Clinical manifestation and pathogenesis of adult chlamydial conjunctivitis
  Clinical manifestation, classification and pathogenesis of the trachoma
- Complications and treatment of the trachoma
- Complications of the ophthalmia neonatorum.
- Adenoviral keratoconjunctivitis – symptoms, treatment
- Acute allergic rhinoconjunctivitis – clinical symptoms, treatment
- Vernal keratoconjunctivitis – clinical types
- Pinguecula, pterygium – complications and treatment

Student should be able to:
- examine of the conjunctiva by means of focal oblique illumination. To pay attention to: location, surface structure, color, conjunctiva.

CONTROL QUASTIONS
1. What are the main etiologic factors of gonococcal keratoconjunctivitis.?
2. What are the cardinal symtoms of the trachoma?
3. What are the cardinal symptomps of adenoviral keratoconjunctivitis?
4. What is the difference between pinguecula and pterygium?
5. What are the signs of acute conjunctivitis?
6. What are the main signs of the vernal conjunctivitis?
7. What are the main principles of local treatment of adenoviral conjunctivitis?
8. What are the main complications of trachoma?
9. What is giant papillary conjunctivitis?

Topic Relevance:

The conjunctiva is a transparent mucous membrane lining the inner surface of the eyelids and surface of the globe as far as the limbus. It has a dense lymphatic supply and an abundance of immunocompetent cells. Mucus from the goblet cells and secretions from the accessory lacrimal glands are essential components of the tear film. The conjunctiva is part of the defensive barrier against infection. The lymphatic drainage is to the preauricular and submandibular nodes, which corresponds to the drainage of the eyelids.

Conjunctivitis is one of the most common causes of an uncomfortable red eye. Conjunctivitis itself has many causes, including bacteria, viruses, Chlamydia, and allergies.

Clinical features of particular relevance to the differential diagnosis of conjunctival inflammation are: symptoms, discharge, conjunctival reaction, membranes, associated keratopathy and lymphadenopathy.

Clinical symptoms and signs

Non-specific symptoms include lacrimation, gritty irritation, stinging and burning. Itching is the hallmark of allergic conjunctivitis, although it may also occur to a lesser extent in blepharitis and dry eye. Pain, photophobia and foreign body sensation suggest associated corneal involvement.

Discharge:

1. Watery discharge is composed of a serous exudate and tears and occurs in acute viral or acute allergic conjunctivitis.
2. Mucoid discharge is typical of chronic allergic conjunctivitis and dry eye.
3. Mucopurulent discharge occurs in acute bacterial or chlamydial infections.
4. **Purulent** discharge is typical of gonococcal infection.

BACTERIAL CONJUNCTIVITIS

Acute bacterial conjunctivitis

Acute bacterial conjunctivitis is a common and usually self-limiting condition caused by direct eye contact with infected secretions. The most common isolates are *H. influenzae, S. pneumoniae, S. aureus,* and *Moraxella catarrhalis*.

1. Symptoms

- Acute onset of redness, grittiness, burning and discharge.
- Involvement is usually bilateral although one eye may become affected 1-2 days before the other.
- On waking, the eyelids are frequently stuck together and difficult to open.

2. Signs

- Diffuse conjunctival injection and an intense papillary reaction over the tarsal plates. The discharge is initially watery, mimicking viral conjunctivitis, but later it becomes mucopurulent. Superficial corneal punctate epithelial erosions are common.

3. Treatment

About 60% of cases resolve within 5 days without treatment. Antibiotics are frequently administered to speed recovery and prevent re-infection. In adults broad-spectrum antibiotic drops should be administered every 2 hours during waking hours for 5-7 days. Compliance in children is often better when using a gel or ointment. There is no evidence that any topical antibiotic is best at achieving clinical or microbiological cure.

1. Fusidic acid is a viscous gel which is useful for staphylococcal infections but not for most Gram-negative bacteria.
2. Drops include ofloxacin, chloramphenicol, ciprofloxacin, lomefloxacin, gatifloxacin, moxifloxacin, gentamicin, neomycin, framycetin and polymyxin B (in combination with bacitracin or trimethoprim).

3. Ointments provide higher concentrations for longer periods than drops but daytime use is limited because of blurred vision. Antibiotics available in ointment form include ofloxacin, chloramphenicol, gentamicin, tetracycline, framycetin and polymyxin B (in combination with bacitracin or trimethoprim).

Gonococcal keratoconjunctivitis

Gonorrhoea is a venereal genitourinary tract infection caused by *N. gonorrhoeae* which is capable of invading the intact corneal epithelium.

Diagnosis

1. Presentation is with acute, profuse, conjunctival discharge.
2. Signs - severe eyelid oedema and tenderness, intense conjunctival hyperaemia, chemosis, profuse purulent discharge. Pseudomembrane formation. Lympha-denopathy is prominent and, in severe cases, suppuration may occur. Peripheral corneal ulceration ensues if conjunctivitis is not treated appropriately. Central extension of ulceration, perforation and endophthalmitis.
3. Laboratory investigations

- Gram stain shows Gram-negative kidney-shaped diplococcic. Culture on enriched media such as chocolate agar or Thayer-Martin medium.

Treatment

The patient must be hospitalized if there is corneal ulceration.

1. Topical gentamicin or bacitracin is initially administered every hour.
2. Systemic Intramuscular ceftriaxone 250 mg daily for 3 days or 1g stat. Patients with keratitis require more aggressive treatment (up to 2g intravenously for 3 days) than those with only conjunctival involvement. Intramuscular spectinomycin 1g stat is an alternative on a named patient basis. Because of problems with multi-drug resistance it is important to determine local guidelines regarding antibiotic susceptibility and recommended treatment.

Meningococcal conjunctivitis

About 35% of the population are asymptomatic carriers of *N. meningitidis*. Meningococcal conjunctivitis is usually seen in children and is very rare in adults. It may be primary or secondary.

1. Primary conjunctivitis may appear as non-invasive disease and invasive disease characterized by systemic symptoms of fever, septicemia and meningitis, which is fatal in 10-15% of cases. The risk of developing invasive disease increases 10-20 times if prophylactic systemic antibiotics are not given acutely.
2. Secondary conjunctivitis can be spread to the eye during end-stage septicaemia but is extremely rare

- Diagnosis - acute conjunctivitis which may be associated with subconjunctival haemorrhages and preauricular lymphadenopathy.
- Keratitis develops in 30% of cases and may lead to ulceration and perforation.

Treatment - topical penicillin or cefotaxime drops, Systemic prophylaxis should be given to reduce the risk of meningitis.

- Patients with conjunctivitis should receive oral ciprofloxacin 750 mg stat; alternatives include intramuscular ceftriaxone 250 mg or cefotaxime 500 mg.
- Close contacts of patients with invasive disease should receive oral ciprofloxacin 500 mg stat but prophylactic treatment of contacts of patients with primary conjunctivitis is not required.

Adult chlamydial conjunctivitis

Chlamydia spp. are small, obligate intracellular bacteria but they cannot replicate extracellularly and hence depend on host cells. They exist in two forms: a robust infective extracellular elementary body and a fragile intracellular replicating reticular body. Adult chlamydial (inclusion) conjunctivitis is an oculogenital infection caused by serotypes D-K of *C. trachomatis.* Transmission is by autoinoculation from genital secretions although eye-to-eye spread may account for about 10% of cases. The incubation period is about 1 week.

- In males chlamydial infection is the most common cause of non-specific urethritis (NSU) and 'non-gonococcal urethritis' (NGU). It may also cause epididymitis and act as a trigger for Reiter disease. In females chlamydial infection may cause dysuria, pelvic inflammatory disease and perihepatitis (Fitz-Hugh-Curtis syndrome). Chronic salpingitis may result in infertility.

1. Presentation is with a subacute onset of unilateral or bilateral redness, watering, and discharge. Untreated, the conjunctivitis becomes chronic and may persist for several months.
2. Signs watering or mucopurulent discharge. Large follicles are often most prominent in the inferior fornix and may also involve the upper tarsal conjunctiva. Peripheral corneal infiltrates may appear 2-3 weeks after the onset of conjunctivitis. Tender preauricular lymphadenopathy. Neglected cases have less prominent follicles and develop mild conjunctival scarring and a superior pannus.
3. Special investigations - PCR to detect chlamydial DNA is the investigation of choice. Direct monoclonal fluorescent antibody microscopy of conjunctival smears is rapid and inexpensive Standard single-passage McCoy cell culture shows glycogen-positive inclusion bodies but requires at least 3 day.
4. Treatment - topical erythromycin or tetracycline ointment can be used initially for symptomatic relief. Systemic therapy should not be started prior to GU investigations. Systemic therapy is with one of the following:

- Doxycycline 100mg b.d. for 10 days.
- Azithromycin 1g as a single dose is particularly effective because it acts intracellularly.

Trachoma

Trachoma is chronic conjunctival inflammation caused by infection with serotypes A, B, Ba, and C of *C. trachomatis*. Initial infection is self-limiting and resolves without scarring but repeated infection, particularly if associated with bacterial conjunctivitis, can lead to blindness. Trachoma is associated with poverty, overcrowding, and poor hygiene. Sharing living space is also a risk factor, and there may be direct transmission from eye or nasal discharge. The fly is an important vector. Currently trachoma is the leading cause of preventable blindness in the world.

Active disease. Mixed follicular/papillary conjunctivitis associated with a mucopurulent discharge, in children under the age of 2 years the papillary component may predominate.

- Superior conjunctival follicles at the upper limbus may resolve to leave a row of shallow depressions. Superior epithelial keratitis and pannus formation.

Chronic disease. Linear or stellate conjunctival scars in mild cases, or broad confluent scars (Arlt lines) in severe disease. The entire conjunctiva is involved but the effects are most prominent on the upper tarsus.

Complications - trichiasis, distichiasis, corneal vascularization and cicatricial entropion. Severe corneal opacification. Dry eye caused by destruction of goblet cells and the ductules of the lacrimal gland.

Table 1. Modified WHO grading of trachoma

TF = trachoma follicles with five or more (>0.5mm) on the superior tarsus

TI = trachomatous inflammation diffusely involving the tarsal conjunctiva, which obscures 50% or more of the normal deep tarsal vessels

TS = trachomatous conjunctival scarring

TT = trachomatous trichiasis (at least one lash) touching the globe

CO = corneal opacity over the pupil sufficient to blur iris details

Management

1. Prevention involves regular face washing and control of flies by spraying.
2. Antibiotics. A single dose of azithromycin 20 mg/kg up to 1g reduces rates of active trachoma, but may need to be repeated after one year. Erythromycin 500 mg b.d. for 14 days is an alternative for women of childbearing age. Topical 1% tetracycline is less effective than oral treatment.
3. Surgery is aimed at relieving trichiasis and maintaining complete lid closure.

Ophthalmia neonatorum

Ophthalmia neonatorum (neonatal conjunctivitis) develops within 2 weeks of birth as the result of infection transmitted from mother to infant during delivery. It is serious because of the lack of immunity in the infant and immaturity of the ocular surface (no lymphoid tissue and relatively poor tear film).

1. *N. gonorrehoea* is now an uncommon although serious cause in developed countries. *C. trachomatis* accounts for the majority of cases in developed countries

and may also cause pneumonitis, otitis and rhinitis. Other pathogens include *S. aureus, S. pneumoniae, H. influenzae* and *Enterobacteriacae (Bacillus* spp., *E. coli, and Klebsiella s*pp.). Herpes simplex virus (typically HSV-2) is a rare cause and usually associated with generalized virus infection including encephalitis. Prophylaxis - povidone-iodine 2.5% is a cheap and effective agent against all of the common pathogens that cause ophthalmia neonatorum. It appears that a single application at birth is sufficient. Erythromycin 0.5% ointment or tetracycline 1% ointment is used by some.

1. Presentation is usually between 3 and 19 days after birth.
2. Signs**.** Usually bilateral eyelid oedema which may be severe in gonococcal infection. Discharge which is initially sero-sanguineous and later mucopurulent. A papillary conjunctival reaction which may occasionally be associated with pseudomembranes. Corneal complications are more severe with *N. gonorrhea* infection and include corneal ulcer and perforation. *C. trachomatis*, if untreated, can cause conjunctival scarring and peripheral corneal pannus.
3. Investigation. Gram stain of exudate for diplococci (gonorrhoea) and Giemsa stain for inclusion bodies. Cultures on chocolate agar or Thayer-Martin plates for *N. gonorrhoeae*. Immunofluorescence tests for chlamydia. PCR for chlamydia and neisserial DNA.

Urgent treatment is indicated in association with paediatric infectious diseases specialist. Chlamydial infection is treated with oral erythromycin ethyl succinate for 2 weeks. If pneumonitis is suspected treatment should be for 3 weeks. Erythromycin or tetracycline ointment is used in addition but not as sole therapy. Gonococcal infection requires ceftriaxone intravenously or intramuscularly, or cefotaxime. Other bacterial infections are treated with chloramphenicol or neomycin ointment q.i.d. Systemic antibiotics may be considered in severe cases. Herpes simplex infection requires systemic aciclovir for 14 days and topical aciclovir 5 times daily.

VIRAL CONJUNCTIVITIS

Adenoviral keratoconjunctivitis

Adenoviruses are icosahedral-shaped, unenveloped viruses with a linear, double-stranded DNA genome. There are 51 subtypes that affect humans and many cause clinical infection. Viral subtyping permits epidemiological tracing of outbreaks.

1. Adenoviral keratoconjunctivitis is the most common external ocular viral infection that may be sporadic or occur in epidemics in hospitals, schools and factories. The spread of infection is facilitated by the ability of the virus to survive on dry surfaces and the fact that viral shedding may occur for 4-10 days before clinical disease is apparent.
2. Transmission of this highly contagious virus is by respiratory or ocular secretions, and dissemination is by contaminated towels or equipment such as tonometer heads. Following the onset of conjunctivitis the virus is shed for about 12 days.

3. Precautions must be taken to avoid transmission following examination of patients with suspected adenovirus infection. Thorough washing of hands is important, as is meticulous disinfection of ophthalmic instruments. In addition, infected hospital personnel should not come in contact with patients and busy eye departments should have a separate 'red eye room' for management of patients with conjunctivitis.

The spectrum of adenoviral eye infection varies from mild and almost subclinical disease to full-blown infection with significant morbidity.

1. **Pharyngoconjunctival fever (PCF)** is caused mainly by serotypes 3, 7 and 11. It is spread by droplets within families with upper respiratory tract infection. Keratitis develops in about 30% of cases but is seldom severe.
2. **Epidemic keratoconjunctivitis (EKC)** is caused mainly but not exclusively by serotypes 8, 19 and 37. The virus is usually transmitted by hand to eye contact, instruments and solutions. Keratitis, which may be severe, develops in about 80% of cases.

Conjunctivitis

1. Presentation is usually with unilateral watering, redness, discomfort and photophobia; the contralateral eye is typically affected 1-2 days later, but less severely.
2. Signs - eyelid oedema and tender pre-auricular lymphadenopathy, follicular conjunctivitis. Severe infection may result in conjunctival haemorrhages, chemosis and pseudomembranes. Tender pre-auricular lymphadenopathy. The pseudomembranes resolve but may result in mild conjunctival scarring.
1. **Conjunctivitis** is treated symptomatically with artificial tears and cold compresses until spontaneous resolution occurs within 3 weeks. Topical steroids may be required for severe membranous conjunctivitis.

<u>Molluscum contagiosum conjunctivitis</u>

Molluscum contagiosum is a skin infection caused by a human specific double stranded DNA poxvirus which typically affects otherwise healthy children with a peak incidence between 2 and 4 years. Transmission is by contact with infected people and then by autoinoculation. Multiple, and occasionally confluent, lesions may develop in immunocompromised patients. A distribution in the chin-strap region is common in HIV positive patients.

1. Presentation is with chronic, unilateral, ocular irritation and a mild discharge.
2. Signs - A pale, waxy, umbilicated nodule on the lid margin associated with follicular conjunctivitis and mild mucoid discharge. Bulbar nodules may rarely occur in immunocompromised patients. Untreated long-standing cases may develop a fine epithelial keratitis or pannus.

Treatment. As there is no antiviral therapy for either agent management involves limitation of infection by education and infection control.

ALLERGIC CONJUNCTIVITIS

Acute allergic rhinoconjunctivitis

Atopy is a genetically determined predisposition to mount an allergic response to environmental allergens. Acute rhinoconjunctivitis is the most common form of ocular and nasal allergy affecting about 20% of the population. The following two clinical syndromes have been described, based on the pattern of exacerbations and the likely allergen.

1. Seasonal allergic conjunctivitis (hay fever), with onset during the spring and summer, is the commonest form. The most frequent allergens are tree and grass pollens, although the specific allergen varies with geographic location.
2. Perennial allergic conjunctivitis causes symptoms throughout the year with exacerbation in the autumn when exposure to house dust mites (*Dermatophagoides pteronyssinus*), animal dander and fungal allergens is greatest. It is less common and milder than seasonal allergic conjunctivitis.

1. Presentation is with transient, acute attacks of redness, watering and itching, associated with sneezing and nasal discharge.
2. Signs which resolve completely between attacks are: lid oedema, chemosis and a mild papillary reaction.

1. Mast cell stabilizers (sodium cromoglycate q.i.d., nedocromil sodium b.d. and lodoxamide b.d.) are effective for long-term use. There is no difference in benefit of a particular preparation except frequency of instillation.
2. Antihistamines (levocabastine, epinastine, emedastine b.d or q.i.d) when the patient is symptomatic. They are as effective as mast cell stabilizers and there is no difference in benefit between different preparations.
3. Combined antihistamines and mast cell stabilizers (olopatadine, ketotifen, azelastine b.d.).
4. Steroids are effective but rarely indicated.

Vernal keratoconjunctivitis

Vernal keratoconjunctivitis (VKC) is a bilateral, recurrent, disorder in which IgE and cell mediated immune mechanisms play important roles. It primarily affects boys and usually presents in the first decade of life (mean age 7 years); 95% of cases remit by the late teens and the remainder develops atopic keratoconjunctivitis. VKC is rare in temperate regions but in sub-Saharan regions of Africa it is a significant public health problem. In temperate regions about three-quarters of patients have associated atopy and two-thirds have a family history of atopy. Such patients often develop asthma and eczema in infancy. VKC may occur on a seasonal basis, with a peak incidence over late spring and summer although there may be mild perennial symptoms.

1. Symptoms consist of intense itching, which may be associated with lacrimation, photophobia, a foreign body sensation, burning and thick mucoid discharge. Constant blinking is also common and may be misdiagnosed as neurotic.

2. Palpebral disease primarily involves the upper tarsal conjunctiva and may be associated with significant corneal disease as a result of the close apposition between the inflamed upper tarsal plates and corneal epithelium. Diffuse papillary hypertrophy on the superior tarsus. Macropapillae (>1 mm) have a flat-topped polygonal appearance reminiscent of cobblestones.

- Mucus deposition between giant papillae. Decreased disease activity is characterized by less conjunctival injection and mucus production.

3. Limbal disease Limbal disease typically affects black and Asian patients. Gelatinous papillae on the limbal conjunctiva that may be associated with discrete white spots at their apices (Trantas dots). In tropical regions limbal disease may be very severe.

Mixed has features of both palpebral and limbal disease4. Keratopathy is more frequent in palpebral disease and may take the following forms: Punctate epithelial erosions involving the superior cornea are the earliest findings, epithelial macroerosions resulting from necrosis caused by toxins released from the inflamed conjunctiva. Shield ulcers and plaques may develop in palpebral or mixed disease when exposed Bowman layer becomes coated with mucus and calcium phosphate. This may result in poor wetting and delayed re-epithelialization.

- Pseudogerontoxon can develop in recurrent limbal disease. It resembles a local area of arcus senilis adjacent to a previously inflamed segment of the limbus.
- Peripheral superficial vascularization, especially superior, may develop following chronic inflammation and mucus deposition in the absence of ulceration.

a. *Mast cell stabilizers* are rarely effective as sole treatment, but they reduce the need for steroids. Lodoxamide and nedocromil sodium are more effective than sodium cromoglycate.

b. *Antihistamines* when used in isolation are as effective as mast cell stabilizers.

c. *Steroids* are indicated mainly for keratopathy although they may be required short-term for severe discomfort. Fluorometholone 0.1% is preferred as it has a low risk of causing ocular hypertension. Exacerbations should be treated intensively with prompt tapering. It is often possible to discontinue steroids between attacks.

d. *Acetylcysteine* is useful for mucous deposition and early plaque formation.

e. *Ciclosporin* q.i.d. may be considered in steroid-resistant cases. Improvement occurs after about 2 weeks of therapy but relapses occur if the drug is stopped suddenly.

2. Supratarsal steroid injection for non-compliant patients and those resistant to conventional therapy. The injection consists of 0.1 ml of either dexamethasone 4 mg/ml or triamcinolone 40 mg/ml after upper lid eversion. There is no clear benefit between the two steroids.

a. *Immunosuppressive agents* (steroids, ciclosporin and azathioprine) may be used in severe unremitting disease unresponsive to maximum tolerated topical therapy.

b. *Oral antihistamines* help sleep and reduce nocturnal eye rubbing.
c. *Superficial keratectomy* may be required to remove plaques. The epithelium is removed to the edge of the calcified region and a very superficial dissection performed. Medical treatment must be maintained until the cornea has re-epithelialized to prevent recurrences. Excimer laser phototherapeutic keratectomy is an alternative.
d. *Amniotic membrane overlay graft* with tarsorrhaphy or lamellar keratoplasty may be required for severe persistent epithelial defects with ulceration.

Atopic keratoconjunctivitis (AKC) is a rare bilateral and symmetrical disease that typically develops in young men following a long history of severe atopic dermatitis. About 5% of patients have childhood vernal disease. AKC tends to be chronic and unremitting with a low expectation of eventual resolution and is therefore associated with significant visual morbidity. Patients are sensitive to a wide range of environmental airborne allergens.

The diagnosis is clinical and there is no single laboratory test to distinguish AKC from VKC.

1. Symptoms are similar to VKC but often more severe and unremitting.
2. Eyelids - Red, thickened, macerated and fissured lids with chronic staphylococcal blepharitis and madarosis, tightening of the facial skin may cause lower lid ectropion and epiphora
3. Conjunctiva micropapillary conjunctivitis over the upper and lower tarsal plates and inferior fornix, giant papillae may develop with time, scarring and infiltration of the tarsal conjunctiva results in flattening of giant papillae and featureless appearance, cicatricial conjunctivitis may develop with inferior forniceal shortening, symblepharon formation and keratinization of the caruncle.
4. Keratopathy - punctate epithelial erosions over the inferior third of the cornea are common, persistent epithelial defects, plaque formation and peripheral superficial vascularization in response to chronic surface inflammation, predisposition to keratoconus, secondary bacterial and fungal infection, and aggressive herpes simplex infection

a. *Mast cell stabilizers* are effective and should be used throughout the year as prophylaxis against exacerbation and as steroid-sparing agents.
b. *Ketolorac* combined with a mast cell stabilizer.
c. *Antihistamines* are less effective than in VKC.
d. *Steroids* are effective short-term for severe exacerbations and keratopathy. A small number of patients require long-term low dose therapy for reasonable control.
e. *Acetylcysteine* for corneal mucus deposits.
f. *Ciclosporin* is an effective steroid-sparing agent in patients with severe disease. However, its efficacy as a first-line agent for long-term therapy warrants further studies.

g. *Antibiotics* and lid hygiene should be used for associated staphylococcal blepharitis.

2. Supratarsal steroid injections should be considered when topical treatment is ineffective.

a. Systemic - a*ntihistamines* for severe itching, a*ntibiotics* (doxycycline 50-100 mg daily for 6 weeks or azithromycin 500 mg once daily for 3 days) to reduce inflammation aggravated by blepharitis, c*iclosporin* in severe cases.

Giant papillary conjunctivitis

Giant papillary conjunctivitis (GPC) was originally described in association with soft contact lens wear, but has subsequently been recognized in association with a variety of mechanical stimuli of the tarsal conjunctiva including ocular prosthesis, exposed sutures and filtering blebs. The risk of developing GPC is increased by deposition of mucous and cellular debris on the contact lens surface (lens spoliation). GPC may develop in patients with mild allergic eye disease that is exacerbated by contact lens wear.

1. Symptoms consist of a foreign body sensation, redness, itching and loss of contact lens tolerance, often worse when the contact lens has been removed.

2. Signs - excessive mobility of the contact lens with upper lid lens capture, increased mucus production and coating of the contact lens, micropapillae on the superior tarsal conjunctiva.

- Macropapillae with focal scarring on the apices may develop in advanced cases.

1. Removal of the stimulus

- Stopping contact lens wear.
- Removal of exposed sutures, ocular prosthesis etc.

2. Cleaning contact lens or prosthesis

- Use of daily disposable lens.
- Rigid contact lens may be easier to clean effectively.
- Protein removing tablets.
- Polishing of prosthesis and cleaning with a detergent.

3. Mast cell stabilizers, which should be non-preserved in patients wearing soft contact lenses.

4. Topical steroids are rarely indicated but are safe to treat GPC associated with an ocular prosthesis.

DEGENERATIONS

Pinguecula

A pinguecula is an extremely common, innocuous, usually bilateral and asymptomatic condition.

1. **Signs.** Yellow-white deposits on the bulbar conjunctiva adjacent to the nasal or temporal limbus

2. **Treatment** is usually unnecessary because growth is very slow or absent.

Occasionally, however, a pinguecula may become acutely inflamed (pingueculitis) and require a short course of a weak steroid such as fluorometholone.

Pterygium

A pterygium is a triangular fibrovascular subepithelial ingrowth of degenerative bulbar conjunctival tissue over the limbus onto the cornea. Pterygia typically develop in patients who have been living in hot climates and may represent a response to chronic dryness and ultraviolet exposure.

Histology

A pterygium consists of collagenous degenerative changes in vascularized subepithelial stroma.

1. **Type 1** extends less than 2mm onto the cornea. A deposit of iron (Stocker line) may be seen in the corneal epithelium anterior to the advancing head of the pterygium. The lesion is often asymptomatic, although it may become intermittently inflamed. Patients who wear soft contact lenses may have symptoms earlier because large-diameter lenses rest on the elevated head of the pterygium and cause irritation.
2. **Type 2** involve up to 4mm of the cornea and may be primary or recurrent following surgery. They may interfere with the precorneal tear film, and induce astigmatism.
3. **Type 3** invade more than 4mm of the cornea and involve the visual axis. Extensive lesions, particularly if recurrent, may be associated with subconjunctival fibrosis extending to the fornices that may occasionally cause mild restriction in ocular motility.

Treatment

1. **Medical** treatment of symptomatic patients involves tear substitutes, and topical steroids for inflammation. The patient should also be advised to wear sunglasses to reduce ultraviolet exposure and decrease the growth stimulus.
2. **Surgery** is indicated for type 2 and 3 lesions. Simple excision is associated with a high rate of recurrence that may be more aggressive than the initial lesion. Numerous techniques aimed at preventing recurrence have been described. Currently the most widely used technique involves excision of the pterygium and covering of the defect with either a conjunctival autograft or amniotic membrane. Adjunctive treatment with mitomycin C and beta-irradiation may be used to minimize recurrence but may rarely be complicated by late scleral necrosis. Occasionally peripheral lamellar keratoplasty is required for deep extensive lesions.

BENIGN PIGMENTED LESIONS

Epithelial melanosis

Conjunctival (racial) epithelial melanosis is a benign condition due to increased melanin production that is often seen in dark-skinned individuals. Both eyes are affected but the intensity may be asymmetrical.

1. Presentation is during the first few years of life. The melanosis becomes static by early adulthood.
2. Signs - areas of flat, patchy, brownish pigmentation scattered throughout the conjunctiva but more intense at the limbus. The lesions may be more intense around the perforating branches of the anterior ciliary vessels or around an intrascleral nerve as they enter the sclera (Axenfeld loop).
Juxtalimbal pigmentation may extend onto the peripheral cornea.
With the slit-lamp the pigmentation is seen to be within the epithelium and therefore moves freely over the surface of the globe.
Secondary
Mascara deposits usually accumulate in the inferior fornix.
Adrenochrome deposits are tiny clumps of pigment on the tarsal or forniceal conjunctiva associated with the long-term use of adrenaline drops for glaucoma (Fig. 8.31b).

Congenital ocular melanocytosis
Classification
Congenital ocular melanocytosis is an uncommon condition characterized by an increase in number, size and pigmentation of melanocytes (Fig. 8.32a). It occurs in the following three clinical settings.
Ocular melanocytosis, the least common, involves only the eye.
Dermal melanocytosis involves only the skin and accounts for about one-third of cases.
Oculodermal melanocytosis (naevus of Ota) which involves both skin and eye is the most frequent type.
Diagnosis
1. Signs
Multifocal, slate-grey pigmentation in the episclera that cannot be moved over the globe (Fig. 8.32b).
Occasionally the peripheral cornea may be involved.
2. Naevus of Ota is bilateral in 5% of patients, occurring frequently in Orientals and darker races but rarely in white people.
Deep bluish hyperpigmentation of facial skin, most frequently in the distribution of the first and second divisions of the trigeminal nerve
It may be subtle in fair-skinned individuals and is best detected by observation in good lighting.
Involvement of the third division of the trigeminal nerve and of the nasal and buccal mucosa is uncommon.
Ipsilateral associations
Iris hyperchromia is common.
Iris mammillations which are tiny, regularly spaced, villiform lesions are uncommon. They may also be found in patients with neurofibromatosis-1,

Axenfeld-Rieger anomaly and Peters anomaly.
Fundus hyperpigmentation can occur.
Trabecular hyperpigmentation, which is associated with glaucoma in about 10% of cases.
Uveal melanoma may develop in a small minority of white people.

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Навчальне видання

Захворювання кон'юнктиви

Методичні вказівки для індивідуальної підготовки студентів іноземців з англійською мовою навчання

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Комп'ютерна верстка О.Ю. Лавриненко

План 2014, поз. 112.
Формат А5. Ризографія. Ум. друк. арк.1,0.
Тираж 150 прим. Зам. № 14-3204.

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Свідоцтво про внесення суб'єкта видавничої справи до Державного реєстру видавництв, виготівників і розповсюджувачів видавничої продукції серії ДК № 3242 від 18.07.2008 р.

Diseases of conjunctiva

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