Term	Topic content
Cheilites (cheilitis)	The term «cheilitis» denotes not the cause of the disease, characteristics of its course and morphological changes, but only on the localization of the pathological process. The red border, the mucous membrane of the lips may be involved in this process in many dermatoses, characterized in most cases by diffuse inflammation. Lesion of the lips may be a manifestation of allergic, infectious diseases, cancer, and various other states. There is still no generally accepted classification of cheilites, and this makes it difficult to diagnose and leads to prescription of non-rational therapy.
Classification	Cheilites is divided into two groups: independent and symptomatic. The group of independent cheilites includes meteorological, exfoliative, glandular, contact and actinic cheilites. The group of symptomatic cheilites include atopic, of eczematous cheilitis (lip eczema), plasma cell, macrocheilitis (Rossolimo-Melkersson-Rosenthal's syndrome), cheilitis on the background of ichthyosis and caused by vitamin deficiency.
Meteorological cheilitis	Meteorological or simple cheilitis (cheilitis meteorological) is an inflammatory disease of the mouth caused by physical effects of various meteorological factors on the red border. Etiopathogenesis. The factors that cause development of meteorological cheilitis may be moisture, dryness, wind, heat, cold, dust. Often the disease develops in people who work outdoors, especially in adverse weather conditions. Clinical picture. In meteorological cheilitis, the red border of the lips is affected, usually the lower one, along its entire length. The lip becomes slightly erythematous, dry, often covered with small scales, patients suffer from dry or tight sensation, many lick their lips, which leads to increased dryness, peeling, and then the red border infiltration. When scales are removed, painful erosion is exposed and may bleed. The skin and mucous lips are unchanged. Diagnosis. The diagnosis is set based on past history and physical examination. Laboratory tests are not advisable.

	Differential diagnosis. It is necessary to differentiate meteorological cheilitis with allergic, actinic, atopic with cheilitis and dry form of exfoliative cheilitis. Treatment. To treat meteorological cheilitis, it is necessary to exclude or diminish the impact of meteorological factors. Recommendation: topical barrier creams, vitamin therapy (B,, B6, Bp, PP, C). Prognosis is favorable.
Exfoliative cheilitis	Exfoliative cheilitis (cheilitis exfoliativa), synonym: Mikulicz-KbmmeVs disease is a chronic disease that affects only the vermilion border. The skin and mucous membrane are never involved in the pathological process. Etiopathogenesis is not fully studied. The disease can be caused by neurogenic mechanisms and thyroid dysfunction. Clinical picture. There are two forms of the disease: dry and exudative, both are different phases of the same disease and can easily be transformed into each other. The exudative form of exfoliative cheilitis is characterized by appearance of grayish-yellow scales, crusts on the red border of the lips, which cover the red border with a layer from comer to comer of the mouth, beginning from the transition zone of the oral mucosa, Klein s line, till the middle of the vermilion border. Sometimes the crust is rather large and hangs from the lips like an apron. The disease is accompanied by severe burning and soreness, especially when the lips are closed, when eating and talking. Such patients almost always keep their mouth half open. The dry form of exfoliative cheilitis, just as exudative, characterized by localized lesions only on the red border of one or both lips. The lesion is in the form of a ribbon extending from the mouth corner to corner and from Klein's line to the middle of the red border of the lips. Commissure of the mouth remains free from lesions. Part of the red border belonging to the skin always remains unaffected. Gray or grayish brown flakes are tightly attached to the red border in the center and a little behind on the edges. Patient suffers from burning and dryness. After 5-7 days flakes easily exfoliate, exposing bare shiny red surface with no

	erosion. Diagnosis. Diagnosis of exfoliative cheilitis is divided into: - linical (based on past history and physical examination); - laboratory (thyroid function tests); - instrumental (histopathological study if necessary, if acanthosis, parakeratosis, hyperkeratosis is observed). Differential diagnosis. The exudative form must be differentiated from exudative form of actinic cheilitis, pemphigus vulgaris, erosive and ulcerative form of lupus, other types of Cheilites. Treatment. Treatment of patients with exfoliative cheilitis is a difficult task and implies correction of concomitant neurological and endocrine pathology. Local therapy should include sanation of the oral cavity, use of keratoplasty means (oil solution of vitamin A and E, kartolin). Local corticosteroids are used in the exudative form. Prognosis is favorable. Subject to correction of endocrine and psychopathological disorders, quite stable remission is achieved.
Glandular cheilitis	Glandular cheilitis (cheilitis glandularis) is a disease that develops as a result of hyperplasia, hyperthyroidism, and often heterotypic form of the salivary glands in the vermilion border and the transition zone. Etiopathogenesis. The cause of glandular cheilitis can be a congenital anomaly in which a large number of small salivary mucous glands are located in the transition zone (Klein's zone) and red border, or the glacis may have normal location, and their ducts are moved to the surface of the red border. Under the influence of stimulation the glands become hypertrophied and produce discharge intensively. Clinical picture. There are primary and secondary glandular cheilitis. Primary glandular cheilitis is manifested mainly after puberty. In the area of the mucous membrane transition in the vermillion border of the lips, and sometimes on the red border there are prominent dilated entries of salivary glands in the form of red dots, excreting droplets of saliva. 5-10 seconds after the lips are dried, salivation from entries of the salivary glands become clearly visible, and saliva

	covers the lip like dew drops. Development of secondary glandular cheilitis is obviously due to the fact that the inflammatory infiltrate, characteristic of the underlying disease, irritates the salivary glands, causing their hyperplasia and hyperactivity. In this case, against the background of the main manifestations of the disease, more frequently on lip mucosa in the transition zone area there are enlarged entries of salivary glands excreting droplets of saliva. Frequent wetting of the red border with saliva in glandular cheilitis contributes to dryness, maceration and leads to chronic fissures. Later on, the mucosa and the red border may gradually coarsen. Diagnosis. Diagnosis of the disease based on past history and physical examination is not difficult. Differential diagnosis. Differential diagnosis of glandular cheilitis is easy due to clear clinical signs and presence of enlarged ducts of glands. Treatment Anti-inflammatory corticosteroid ointments, punctate electrocoagulation ofhypertrophied glands or cryodestruction are used to treat glandular cheilitis. If a patient with glandular cheilitis has a great number of abnormal glands, their surgical excision is performed. In secondary glandular cheilitis it is necessary to treat the underlying disease. Prognosis is favorable. At superficial electrocoagulation or cryodestruction clogging of ducts of salivary glands and formation of cysts may be observed.
Contact allergic cheilitis	Contact allergic cheilitis (cheilitis alergica contactis) is a disease, developing due to sensitization of the vermilion border or, less frequently - mucosa, to chemicals and occurs when lip mucosa direct contacts the allergen. Etiopathogenesis. Contact allergic cheilitis is a clinical manifestation of delayed- type hypersensitivity. Most often it is a reaction to chemicals used in lipstick, toothpaste and other cosmetic products, in particular fluorescent substances and eosin, rhodamine, etc. This disease mostly affects women. Clinical picture. In clinical terms, contact allergic

	cheilitis is manifested through severe burning and itching. Usually the process is localized on the red border of the lips, sometimes it extends slightly to the skin of the lips. At the point of contact with the allergen there occurs a rather well-defined erythema and slight peeling. In long-term course of the disease the red border of the lips become dry and has small transverse grooves and cracks. Diagnosis. Contact allergic cheilitis is diagnosed based on clinical examination findings and, where appropriate, allergy tests. Differential diagnosis. This disease is differentiated with dry forms of exfoliative cheilitis, actinic and atopic cheilitis. Treatment. In treatment of allergic contact cheilitis first of all it is necessary to eliminate a causative factor of the disease. If the clinical picture of the disease is mildly expressed, it is possible to prescribe only local treatment- corticosteroid ointments to be applied 5-6 times a day. In more severe cases, desensitizing therapy should be used. Prognosis is favorable subject to maximum elimination of the allergen.
Actinic cheilitis	Actinic cheilitis (cheilitis actinica) is a chronic disease caused by hypersensitivity of the red border of the lips to sunlight. Etiopathogenesis. the main causes of this cheilitis are delayed reaction to ultraviolet rays, i.e. actinic cheilitis develops in people with sensitization of the red border of the lips to solar radiation. The dry form of actinic cheilitis is considered to be elective precancer. Classification. There are exudative and xeronous (dry) forms of actinic cheilitis. Clinical picture. The exudative form of actinic cheilitis is often found in individuals with hypersensitivity to sun exposure, so the clinical picture is consistent with evidence of acute allergic contact dermatitis. In spring the red border of the lower lip in the xeronous (dry) form of actinic cheilitis turns bright red, covered with small dry silvery-white scales. The lesion covers the entire surface of the red border. A number of patients develop keratinization areas on the red border; sometimes verrucous mass occur. Diagnosis. Actinic cheilitis is diagnosed on the basis of medical history and physical examination. If

	necessary, a smear mark to exclude cellular atypia, and dermal biopsy is recommended. Differential diagnosis. There is actinic cheilitis with dry forms of exfoliative cheilitis, atopic and meteorological cheilitis. A characteristic diagnostic feature is the process exacerbation under the influence of insolation. Treatment. First of all, it is necessary to recommend that the patient avoids sun exposure and changes an occupation, if it is associated with prolonged stay in the open air. Hyposensitization drugs, nicotinic acid are prescribed. Topical steroids are used locally inactinic cheilitis. Prognosis is favorable, however in long-term keratosis is necessary to exclude malignancy.
Symptomatic cheilites	Symptomatic cheilites is a group of cheilites, which are one of the main clinical manifestations of the underlying disease. There are atopic cheilitis, eczematous cheilitis, plasma cell cheilitis, Miescher's granulomatous macrocheilitis, Meiji's rophoderm, and Rossolimo-Melkersson-Rosenthal's syndrome. Atopic cheilitis (cheilitis atopica) is one of the symptoms of atopic dermatitis, which is often the only manifestation of this disease at its certain stages. Etiopathogenesis. Atopic cheilitis is a genetically caused disease accompanied by disorders of the central and autonomic nervous system. It is more common in girls and boys aged 4 to 17 years old. Clinical picture. In clinical terms, atopic cheilitis affects the red border of the lips and always the skin, and the process is more intensive in the corner of the mouth. The part of the red border, adjacent to the oral mucosa and the oral mucosa remain unaffected. The disease manifests itself through itching, erythema and lip lichenification. Diagnosis and differential diagnosis. In exfoliative cheilitis, unlike atopic, a part of the vermilion border is always affected in the form of a strip from the Klein's line to the middle of the red border; a part of the vermilion border adjacent to the skin is intact; the process never affects the skin of the lips and does not cover the corners of the mouth; there is no erythema and lip lichenification; the course of the disease characterized by monotony and lack of remission. Past

	history of patients with actinic cheilitis shows a clear dependence of exacerbations on insolation; there is no frank lesion of the mouth comers characteristic of atopic cheilitis. In allergic contact cheilitis lichenification is only observed during prolonged course of the disease; there are no sores at the corners of the mouth; the course depends on direct contact with the allergen. In some cases, differential diagnosis of atopic cheilitis with symmetrical streptococcal or Candida bridoes can be quite difficult. In bridoes, localization of the lesion is limited only to the mouth comers; as a rule, lichenification is not observed. Treatment. Treatment includes prescription of antihistamines and sedatives, vitamins.
Macrocheilitis	Macrocheilitis (macrocheiliti). This is the name for Rossolimo-Melkersson- Rosenthal's syndrome Meiji's trophoderm, Miescher's granulomatous macrocheilitis, characterized by persistent lip swelling. At this time it is known that granulomatous Miescher's cheilitis is not an independent nosological form of the disease and is a variant of Rossolimo-Melkersson-Rosenthal's syndrome with development of granulomatous infiltrate in the affected tissue. Meiji's trophoderm refers to angiotrophoneurosis. Rossolimo-Melkersson-Rosenthal's syndrome combines a triad of symptoms: macrocheilia (persistent increase in the lip), neuritis of the facial nerve, folded tongue, and a chronic disease with a tendency to recur. The disease occurs both in men and in women at any age, but most often between 16 and 35 years old. Etiopathogenesis. The causes of Rossolimo-Melkersson-Rosenthal's syndrome are not fully clear. Neurological manifestations of Rossolimo-Melkersson- Rosenthal's syndrome often may include neuritis or facial nerve paresis and paralysis of the facial muscles. Clinical picture. The onset of the disease is sudden. During few hours the lips get swollen. Swelling lasts 3-6 days, seldom less, sometimes longer, even up to a month. At the same time there is swelling of the mucous membranes of the oral cavity. Examination reveals deformation of the lips, the increase in their volume. The lip is dense at palpation. The mucous membrane of the mouth may be edematous, its surface becomes uneven, with a white cushion along the line

	of the teeth compression. There develops quite clear coarsening of the mucosa, various degrees of folding and lobulation of the tongue. The course of Rossolimo-Melkersson-Rosenthal's syndrome is chronic. At the onset of the disease relapses are usually replaced by more or less prolonged remissions, during which all symptoms of the disease resolve. Later, macrocheilia and paralysis of the facial nerve become stable. Diagnosis. Some patients with Rossolimo-Melkersson-Rosenthal's syndrome may lack both the folding of the tongue and paralysis of the facial nerve. The only symptom of the disease in this case is macrocheilitis. The impairment of the facial nerve manifests through the face distortion to the healthy side, smoothing of the nasolabial fold. There are signs of the cranial nerve impairment (trigeminal, auditory, etc.). Along with a thorough dental examination of the patient, identification of odontogenic, tonsillogenic and other sites of infection it is necessary to determine sensitivity to bacterial allergens with leukolysis reaction, as well as the neurological status. Differential diagnosis. It is performed with angioedema, lymphangioma, hemangioma, collateral edema of the lips in periostitis of the upper or lower jaw. Treatment. Treatment of Rossolimo-Melkersson-Rosenthal's syndrome is performed in two directions: surgical and conservative. In surgical treatment, which is performed for cosmetic purposes, a part of lip tissue is excised. However, surgical treatment does not prevent recurrences of the disease. Conservative treatment includes corticosteroids, broad-spectrum antibiotics and synthetic anti-malarial drugs.
Eczematous cheilitis	Eczematous cheilitis (cheilitis eczematosa). All types of eczematous cheilitis are grouped together according to similarity of clinical manifestations, but they occur for different reasons. There are eczematous cheilites: caused by seborrheic eczema; microbial eczematous cheilitis; contact eczematous cheilitis. Cheilitis in seborrheic eczema. Cheilitis is one of the symptoms of seborrheic eczema, but only the lips may be affected. In clinical terms, it manifests through

	hyperemia of the red border of the lips, occurrence of easily separated flakes, bubbles. Skin of the lips and the red border is dry, cracks and flakes appear. Microbial eczematous cheilitis cheilitis. Re-develops against seborrheic eczema due pyococcal infection (mainly streptococcal) at the site of formation. Clinical manifestations correspond to microbial eczema. Contact eczematous cheilitis cheilitis. Develops due to an allergic reaction to a variety of chemicals (in the lipstick, toothpaste, etc.). The clinical picture is consistent with acute or chronic eczema and is quite varied: swelling of the lips, bright hyperemia, blisters with subsequent formation of crusts. Elimination of allergen leads to rapid resolution of cheilitis. Treatment In eczematous cheilitis treatment is similar to that of eczema. Topical steroids and antibiotics are used.
Viral infections whose main symptom is blistering	The group of skin viral infections characterized by formation of blisters (vesicles), which appearance is caused by the degeneration of epidermal cells, includes herpes simplex and herpes zoster.
Herpes simplex	Herpes simplex (herpes simplex) is a viral disease of skin and mucous membranes caused by herpes simplex virus (HSV).
Etiology and pathogenesis.	Herpes simplex virus belongs to the family of Herpesviridae, subfamily Alphaherpesvirinae type Simplexvirus. There are two antigenic serotypes of HSV* first (HSV-1) and second (HS V-2). Transmission of HSV-1 usually occurs in childhood, through a direct contact with a HIV-sick or infected person. This causes frequent localization of orofacial herpes lesions caused by HSV-1, in particular on skin areas around mouth (herpes labialis), nose (herpes nasalis), seldom on cheeks, eyelids and ears. In addition to skin, mucous membranes of the mouth (herpes stomatitis) may also be affected. Transmission of HSV-2 occurs mainly through sexual contact. HSV-2 is dominant in causing genital herpes infection (herpes genitalis) with the localization of lesions on skin and mucous membranes of the external genital organs of men and women. However, there is no stable relationship between antigen serotypes of HSV

	and localization of herpetic lesions on skin and visible mucous membranes (genital, extragenital). This is confirmed by the fact that about 20% of cases of genital herpes are caused by HSV-1 In the pathogenesis of herpes simplex virus, the development of chronic persistent infection in sensory ganglia is crucial. Penetrating through the mucous membranes of the oropharynx, conjunctiva, urethra, cervix, rectum, or skin micro-cracks in the process of initial infection, HSV reaches nerve endings and moves to sensory ganglia through the retrograde axon, where there occurs an acute infection, when the virus replicates in the cells of sensitive ganglion. Further, virus enters into the state of persistence, which provokes the latent course of herpes. Under certain conditions (primarily, due to the lack of immune control), there occurs activation of the virus; from ganglion, the activated virus migrates along the axon of the peripheral nerve and replicates in the epithelial cells. Except general weakening of the immune control, reproduction of virus is caused by a violation of local immunity in the area of the epidermis.
Epidemiology	At the present stage, herpes simplex is one of the most common uncontrolled human infections (uncontrolled human infections involve the inefficiency of vaccination or treatment methods, which allow achieving the complete elimination of the pathogen from the host's body). More than 50% of the population of developed countries and 100% of the population of developing countries are seropositive to HSV presense. The disease features pandemic nature. Genital herpes ranks first in the list of common human infections transmitted primarily through sexual contact. The problem of herpetic infection is aggravated by the growth of cases of abortive and atypical clinical course of the disease. There is an essential difference between the number of persons seropositive to HSV and the number of people with clinical manifestations of herpes simplex.
Clinic	**Herpes can be primary and simplex recurrent.** The disease begins with itching or burning, accompanied by the formation of groups of small strained vesicles amid a slightly edematous limited congested spot. The

	content of vesicles is transparent, becomes thick in 2-3 days. Vesicles feature a tendency to merge. After 3-5 days, vesicles dry up and form yellowish-gray crusts. After 6-8 days, crusts fall off, while secondary pigmentation is left in their place, which then disappears with no trace. **Primary herpes simplex.** Primary infection with HSV-1 occurs mainly in young children. In most cases, the primary manifestations ofherpes simplex are minor (redness, itching) and remain undetected. However, children infected with HSV-1 may develop primary herpetic gingivostomatitis. The disease is developed suddenly, with an increase in body temperature to 39-40 ° C and intoxication. Mucous membranes of cheeks, gums, lips, tongue, and throat are tonsils are covered with painful grouped vesicles. After their destruction, there occur painful erosions prone to a merge. Clinical manifestations of the inflammation subside in two to three weeks. **Primary genital herpes.** The primary episode of genital herpes occurs after an incubation period of 1 -7 days. In men, herpes rash is usually localized on the head and shaft of the penis and foreskin, while in women it is localized on small and large labia, vagina, clitoris, cervix, perineum, thighs and buttocks. On the background of significant erythema and edema, there develop grouped vesicles, first with clear, and then thick contents. On the ruins of vesicles, erosions, sometimes ulcers and cracks are formed. Subjectively, the rash is accompanied by a sensation of pain and itching. There develops painful bubonadenitis. Unlike further relapses, primary clinical episode of genital herpes features more severe and prolonged course (4-5 weeks). **Recurrent herpes simplex and recurrent genital herpes.** In most cases, the initial clinical episode ofherpes simplex is followed by clinical recovery. However, virus (HSV-1, HSV 2) is stored in the body in a latent form throughout a person's life, not causing any clinical symptoms. Approximately 90% of people infected with HSV are virus carriers. Under the influence of a series of factors that reduce protective capacity of the body, which include hypothermia, overheating, infectious diseases, especially colds, etc., there occur recurrences ofherpes simplex. Unlike

	primary clinical episode of the disease, clinical episode of recurrent herpes simplex virus features milder course. In recurrent herpes simplex, typical location of the lesion includes lips, face, cornea and conjunctiva of the eye, buttocks. At these sites, there develop grouped vesicles with clear content, accompanied by itching and burning. In further, painful erosions are formed, which may merge. On the surface of the erosion, exudate dries in the form of a crust. After the removal of crust, secondary spots are left. Clinical recurrences ofherpes simplex may occur over many years and decades, with varying frequency - from one or two a year to two to four per month. Compared with the initial episode, recurrences of genital herpes are also characterized by a mild course. Rash on skin and mucous membranes is rather sparse. Typical for herpes, lesions are located on skin and mucous membranes of the vulva. Genital herpes can cause diverse complications, including reproductive disorders, miscarriage, intrauterine infection of fetus, and be transmitted to a baby during childbirth. In case of transplacental infection, a newborn may develop growth retardation, encephalitis, chorioretinitis. In addition, due to chronic recurrent genital herpes patients may experience significant psychosomatic disorders.
Diagnostic	Diagnosis of herpes simplex is simple and based on presence of typical clinical symptoms: itching, grouped vesicular eruption, formation of erosions and crusts. The recurrent nature of the lesion is testified by similar clinical manifestations in the past; identification of high titers of antibodies to HS V during relapse; identification of antigen in the focus of clinical manifestation. The coincidence of the type identified from the source of HSV and type of HSV, to which the antibodies in a patient's serum are found, is the condition for diagnosis of recurrent herpes. However, it should be noted that serology and identification of viral antigen are not routine methods in the diagnosis of herpes simplex. They are applyied only in specific cases, when there is considerable doubt as to the clinical diagnosis of herpes simplex. In addition, high titers of antibodies to HSV with no clinical manifestations (recurrent episodes) are not the reason for the

	diagnosis of herpes simplex.
Differential diagnosis	In case of localization in the mouth mucosa, herpes simplex is to be distinguished from acantholytic pemphigus and polymorphous exudative erythema. In pemphigus, erosions are localized on a visually normal mucous membrane, they are not subject to epithelialization, Nikolsky's symptom is positive, acantholytic cells are found in the impression smears from the surface of erosions. Unlike herpes, polymorphous exudative erythema is characterized by seasonality index (spring and autumn), significant size ofbubbles and erosions on a dramatically inflamed background, layering of bloody crusts on the red border. Clinical manifestations of genital herpes localized on the genitals should be differentiated from syphilitic chancre. Unlike herpes, syphilitic erosion is characterized by smooth edges, saucer-like shape, hard bottom, indolence and peculiar regional lymphadenitis. In doubtful cases, the issue is finally resolved by microscopy examination of the material from erosions to detect the agent of syphilis – Treponema Pallidum.
Treatment	All currently existing methods and tools for treatment of herpes do not allow achieving complete elimination of pathogens (HSV-1, HSV-2) from the human body. Approaches to treatment of herpes simplex are determined by a clinical picture of the disease, severity of clinical course, frequency of relapses, as well as availability of comorbidity. In the antiviral therapy of herpes infections, preparations of acyclic nucleotides that have an ability to disrupt interaction of virus and cells, in particular inhibit reproduction of the virus through its virostatic action, play the major role. For treatment of infections caused by herpes simplex viruses, the drugs from a group of acyclic purine nucleosides are used: acyclovir, valacyclovir (valine aether of acyclovir) and famciclovir (pro-forma of penciclovir). In the form of topical preparations (cream), acyclovir is prescribed to reduce the intensity and duration of the recurrent episode of herpes

	simplex. Systemic prescription of purine nucleoside analogs (internal or parenteral) is used for treatment of primary manifestations of herpes simplex virus, as well as treatment of relapse (for active clinical manifestations). With frequent recurrences of skin herpes and genital herpes, acyclovir and valacyclovir may be prescribed in long continual courses (so-called long-term suppressive therapy). For a healthy sexual partner of a patient with recurrent herpes, prophylactic or preventive treatment has no medical meaning, since existing antiviral drugs are unable to eliminate the virus from a human body. In some countries, particularly Ukraine, Russia and Belarus, recombinant interferons and interferon inducers are used as a part of comprehensive treatment of the disease.
Prevention	Preventive measures against primary HSV infection in children are reduced to avoidance of contacts with adults that have active clinical signs of infection. Compliance with the principles of safe sexual behavior (monogamous sexual relationships, the use ofbarrier protection equipment) is the only way to prevent infection with HSV Secondary prophylaxis involves the avoidance of hypothermia or excessive sun exposure, as well as the appointment of prolonged systemic antiviral therapy.
Herpes zoster (shingles)	Herpes zoster (shingles) is an acute infectious disease of skin and mucous membranes caused by a neurotropic virus (varicella zoster), which is also a pathogen of chickenpox. The disease is characterized by the occurrence of unilateral grouped vesicular lesions within one to two dermatomes and accompanied by neurological pain.
Etiology and pathogenesis	Shingles and chickenpox are both caused by is the varicella-zoster virus, which belongs to the family of Herpesviridae. There is a hypothesis saying that after the primary attack of varicella (manifested in the symptoms of chickenpox), the virus penetrates into the cells of dorsal root ganglion, where it is stored in an asymptomatic condition (persistent) until the moment of reactivation caused by certain factors (hypothermia, stress, cancer, immune incompetence and others). Under the influence of these factors that weaken the body immune reactivity, the virus is activated, multiplies, causing inflammation of the ganglia. In

	further, the virus enters the sensory nerves, causing neuritis and neuralgia, spreads around sensory nerve endings in skin and causes formation of a characteristic rash, which is located along one of the nerves. Recurrences ofherpes zoster are rare; occur mainly in the background of a significant immune suppression, particularly in HIV infection and malignancy. People of any age may suffer from shingles, while elderly people are at a higher risk.
Clinical picture	The disease may begin suddenly or be precededed by a general malaise, headache, fever, neuralgia or paresthesia in the areas that are to be subject to rash. The most frequent localization of lesions includes the area of intercostal nerves, hence the name «shingles», while the rash is always localized on one side of the body (unilateral localization), rarely covers a little area on the opposite side (due to anastomotic innervation). The second most common place of the lesion is the area of trigeminal nerve. The lesion of the first and third branches of the trigeminal nerve causes the emergence of rash in the mouth mucosa. The eruption emerges in a paroxysmal way on congested skin, there appear clusters of vesicles with clear serous contents, which quickly becomes cloudy and shrinks in serous crusts. The rash of vesicles on each individual spot occurs simultaneously, but stains emerge sequentially, with an interval of several days. Foci of the lesions may be located fairly closely, forming almost a continuous line along the nerves. After rejection of crusts, there remain brownish-red spots, which gradually disappear. In typical cases, the disease continues for two to three weeks. Burning and pain along the affected nerve are observed subjectively, especially when the rash is localized on face and mucous membrane of the mouth cavity. In herpes zoster, mucous membranes are rarely affected; typically, simultaneously with a skin lesion of a certain area. Against the background of edematous (from one side) oral mucosa, there emerge vesicles, which are rapidly destroyed, forming painful erosions, often covered with gray-white pellicle. Subjectively, burning is marked in the affected areas. Very rarely, shingles can affect vaginal mucosa and bladder. The most common complication of herpes zoster is the development of a persistent pain syndrome, prone to a

	prolonged and persistent course after dermatological recovery (postherpetic neuralgia). Postherpetic neuralgia occurs in older patients, which significantly affects the quality of life. Timely administration of systemic antiviral therapy during the first 72 hours from the moment of rash emergence (erythema and vesicles) reduces the risk of this complication in a few times. Other complications of shingles include facial paralysis, meningitis, meningoencephalitis, arachnoiditis, vestibular disorders, pneumonia, paralysis of the diaphragm, bladder paralysis, paresis of the lower extremities, myelitis with the disorder of pelvic organs, etc.
Diagnostics	Diagnosis ofherpes zoster is usually based on clinical manifestations. Presence of a neurological pain syndrome preceding and accompanying rash, unilateral localization of lesions located along the corresponding nerve and herpetiform grouped localization of vesicles are considered.
Differential diagnosis	In some cases, shingles should be differentiated from bullous form of erysipelas, atopic dermatitis, and sometimes impetigo. Pain syndrome that precedes formation of rash can resemble chest pain and pain in myocardial infarction, pain with bowel obstruction, etc.
Treatment	. In the antiviral therapy of both herpes zoster and herpes simplex, an important place is given to the drugs of the group of acyclic nucleosides that feature a virostatic action. Acyclovir, 800 mg orally, 4-5 times a day for 7-10 days is used. Given poor bioavailability of acyclovir, the valacyclovir, 1000 mg orally 3 times a day for 7-10 days, and famciclovir, 250 mg 3 times a day orally for 7 days are widely used in treatment ofherpes. In severe cases of herpes zoster, systemic corticosteroids in high doses (40 - 60 mg of prednisone per day, with a gradual reduction of the dose) are prescribed. Topical preparations of acyclovir and penciclovir (creams) are applied locally. The need for analgesics depends on pain intensity. Usually, paracetamol or indomethacin is enough. In some cases, the need to use opioids or epidural anesthesia may arise. Expressed pain that occurs prior to or simultaneously with herpetic eruption indicates a possibility of

	prolonged neuralgia in the future. In prolonged neuralgia that persists after the rash recourse, analgesics and non-steroidal anti-inflammatory drugs (infometatsin etc.) are prescribed. Patients with herpes zoster should avoid exposure to cold, exercise and stress situations.
Prevention	Primary prevention of chicken pox (and later, herpes zoster) involves a specific vaccine immunization in childhood. Patients with active manifestations of herpes zoster should avoid contact with people (especially children) that have not had chickenpox. If shingles is diagnosed in a hospitalized patient, he is to be immediately isolated to prevent nosocomial infection. Prognosis. Shingles belongs to a group of diseases that are treated independently and feature no tendency to relapse (except in patients with immune deficiency). Elderly patients that received improper treatment may experience persistent pain syndrome, which leads to a deterioration in the life quality. Recurrences of herpes zoster testify significant violations of immune status. Such patients must be carefully examined for the presence of malignant neoplasms and HIV infections.
Cutaneous tuberculosis(Lupus)	is essentially an invasion of the skin by Mycobacterium tuberculosis, the same bacteria that cause TB of the lungs (pulmonary TB). **Mycobacterium tuberculosis** is a straight or slightly bent (rod-shaped), non-motile, non sporulated, unencapsulated bacillus, measuring from 1 to 10μm long by 0.2 to 0.6μm wide; its most important feature is that it becomes stained in red by fuchsin and does not discolor under the actions of alcohol and acid (acid-fast bacillus). Its cellular wall has a high lipid content which grants resistance against the action of chemical agents, though, it is susceptible to the action of physical agents (heat and ultraviolet radiation).5 This bacillus is a strict aerobic pathogen that requires certain conditions to grow and multiply: oxygen, nutrients and an adequate pH in the medium. It has approximately 4,000 genes with most of them involved in the mechanisms of immune system evasion. For example, 200 of the genes are involved in lipid metabolism. So much emphasis occurs because lipids are the main energy source of Mtb and therefore

	are directly responsible for its ability to multiply in host tissue and form cellular walls. It can also be considered a facultative intracellular parasite, since it is able to survive and multiply both outside and inside phagocytic cells.
Cutaneous tuberculosis classification	A. Exogenous cutaneous tuberculosis Tuberculous chancre and Tuberculosis verrucosa cutis B. Endogenous cutaneous tuberculosis a) By contiguity or autoinoculation (Scrofuloderma, orificial tuberculosis and some cases of lupus vulgaris) b) By hematogenic dissemination (Lupus vulgaris, tuberculous gumma and acute miliary tuberculosis) C. Tuberculids - Papulonecrotic tuberculid - Lichen scrofulosorum D. Cutaneous tuberculosis secondary to BCG vaccination
TB verrucosa cutis	• Occurs after direct inoculation of TB into the skin in someone who has been previously infected with mycobacteria • Presents as a purplish or brownish-red warty growth • Lesions most often occur on the knees, elbows, hands, feet and buttocks • Lesions may persist for years but can clear up even without treatment
Lupus vulgaris	• Persistent and progressive form of cutaneous TB • Small sharply defined reddish-brown lesions with a gelatinous consistency (called apple-jelly nodules) • Lesions persist for years, leading to disfigurement and sometimes skin cancer

Scrofuloderma	• Skin lesions result from direct extension of underlying TB infection of lymph nodes, bone or joints • Often associated with TB of the lungs • Firm, painless lesions that eventually ulcerate with a granular base • May heal even without treatment but this takes years and leaves unsightly scars
Miliary TB	• Chronic TB infection that has spread from the primary infection (usually in the lungs) to other organs and tissues via the bloodstream • Skin lesions are small (millet-sized) red spots that develop into ulcers and abscesses • More likely in immunocompromised patients, eg HIV, AIDS, cancer • The patient is generally sick • Prognosis is poor (many patients die even if diagnosed and treated)
Tuberculid	• Generalised exanthem in patients with moderate or high degree of immunity to TB because of previous infection • Usually in good health with no identifiable focus of active TB in skin or elsewhere • Erythema induratum (Bazin disease) presents as recurring nodules or lumps on the back of the legs (mostly women) that may ulcerate and scar. It is a type of nodular vasculitis. • Papulonecrotic tuberculid results in crops of recurrent crusted skin papules on knees, elbows, buttocks or lower trunk that heal with scarring after about 6 weeks. • Lichen scrofulosorum is an extending eruption of small follicular papules in young persons with underlying TB.
Diagnostic	• Skin biopsy • Tuberculin skin test • Sputum culture • X-ray and other radiological tests

Ttreatment	Patients with pulmonary or extrapulmonary TB disease need to be treated with antitubercular drugs. This usually involves a combination of antibiotics (isoniazid, rifampicin, pyrazinamide and ethambutol) given over a period of several months and sometimes years. Patients with latent TB infection but no active disease may also be treated with antitubercular drugs to prevent development of active disease. See tuberculosis screening. Occasionally surgical excision of localised cutaneous TB is recommended.