Chapter-2

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DERMATOVENEROLOGY

MINISTRY OF HEALTH OF THE REPUBLIC UZBEKISTAN
SAMARKAND STATE MEDICAL UNIVERSITY

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Despite the revolutionary changes that have taken place over the past decades, dermatology still remains an urgent problem. In active consideration, possible causes of the development of dermatoses are considered and pathogenetically sound approaches to the description of such patients are explored. A study based on studies showed the presence of persistent positive dynamics, both clinical and functional, in almost 25.2% of patients with dermatoses. One of the reasons for the resistance of the disease to the method of treatment may be the presence of concomitant therapy, which aggravates the course of the disease, the serious effectiveness of therapy and the worsening of the prognosis of the disease.

***Training manual** is intended for use by students of universities, masters, as well as for general practitioners.*

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TABLE OF CONTENTS

LIST OF ABBREVIATIONS AND SYMBOLS

♠ - trade name of the medicinal product
℘ — medicinal product is not registered
⊗ — canceled medicinal product AG - antigen
AGLS - antihistamine drugs AD - atopic dermatitis
ACTH - adenocorticotropic hormone ANF - antinuclear factor
ASD - Dorogov's antiseptic stimulator AT - antibody
NPP - antiendotoxin component BCG - Bacillus Calmette-Guerin
HIV - human immunodeficiency virus WHO - World Health Organization
HSV - herpes simplex virus HPV - human papillomavirus GC - glucocorticoids
GKP - glucocorticoid drugs GLP - glycoprotein
DM - dermatomyositis
DNA - deoxyribonucleic acid GIT - gastrointestinal tract IB - immunoblotting
IR - immune complex IL - interleukin
PI - protease inhibitor PPI - drug intake index
ELISA - enzyme immunoassay IFN - interferon
ICG - immunochromatographic reaction ICL - method of immunochemiluminescence

FOREWORD

The development in recent years of fundamental research in the field of immunology, biophysics and pharmacology has made it possible to make a breakthrough in elucidating individual links of pathogenesis, to improve the diagnosis and treatment of a number of dermatoses and sexually transmitted infections (STIs). The mechanisms of development of psoriatic arthritis and severe forms of psoriasis are clarified, methods of diagnostics and cytokine therapy are being improved. The possibilities of photodynamic therapy with the use of various photosensitizers are being expanded, non-steroidal external preparations are being used in the staged treatment of allergic dermatoses, methods of specific immunogenetic diagnosis of infectious diseases of the skin and genitourinary organs are being introduced. The skin performs many functions, has a large area, closely interacts with the internal organs and systems of the whole organism due to neurohumoral connections, and therefore is a projecting screen for various clinical stigmas, which are sometimes symptoms of very serious diseases. This underlines the importance and significance of dermatovenereology as a medical discipline. The authors analyzed new data in the field of dermatology, hereditary skin diseases and STIs and shared their experience.

The authors hope that "Dermatovenereology", based on the latest achievements of medical science, will become a reference book for dermatovenereologists and will contribute to improving the professional level of doctors and quality patient care.

Introduction

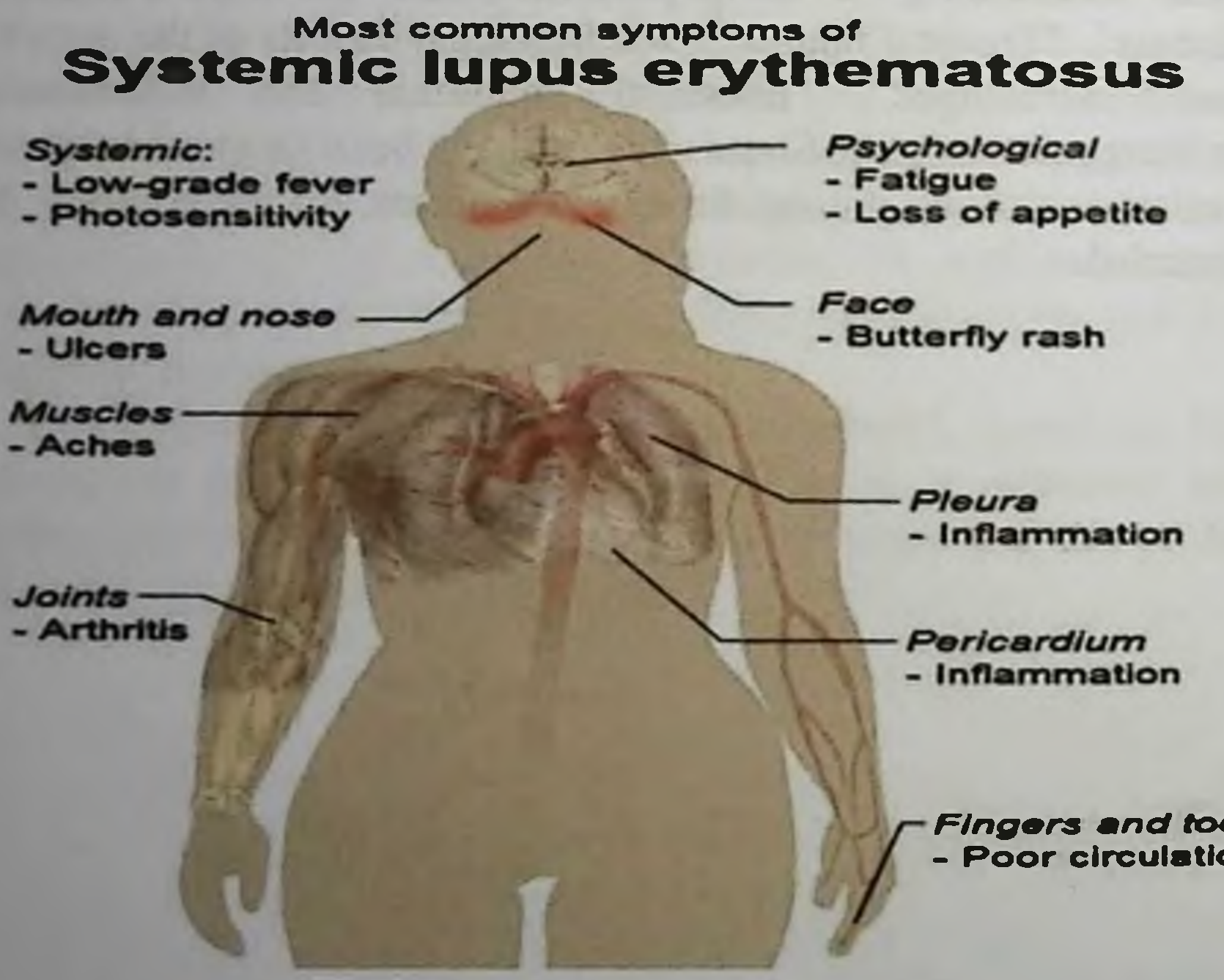
The skin performs many functions, has a large area, closely interacts with the internal organs and systems of the whole organism due to neurohumoral connections, and therefore is a projecting screen for various clinical stigmas, which are sometimes symptoms of very serious diseases. This underlines the importance and significance of dermatovenereology as a medical discipline. The authors analyzed new data in the field of dermatology, hereditary skin diseases and STIs and shared their experience. The brief edition of the national guide is a unique work and, in addition to the traditional sections on the specialty, includes a number of original ones: "Legal regulation of the organization of the provision of dermatovenereological care in modern conditions, ways to improve its quality and accessibility to the population", "Dermatological aspects of Lyme disease", "Tropical miases", "Medico-legal aspects of the activity of a dermatovenereologist", "Intestinal endotoxin and inflammation", "Peptide bioregulation", etc. Some chapters have been shortened due to the loss of relevance at the present time, while others, on the contrary, have been expanded.

CHAPTER 1. LEPROA, CUTANEOUS LEISHMANIASIS, CUTANEOUS TUBERCULOSIS.

LUPUS

Skin tuberculosis is a chronic infectious disease caused by Mycobacterium tuberculosis. Pathogen. Mycobacterium tuberculosis - rods 2.5 microns in length - are acid-fast. They do not form spores, they are characterized by polymorphism, their number increases every 20 hours. Among the causative agents of skin tuberculosis, 3 types of mycobacteria are known:

Mycobacterium tuberculosis hominis (human) - 94-95%;
Mycobacterium tuberculosis bovis (bovine) - 4-5%;
Mycobacterium tuberculosis avis (avian) - less than 1%.



Pic-1 Systemic lupus erythematosus

Pathogenesis. Mycobacterium tuberculosis enters the skin either primarily, i.e. exogenously, or secondarily, i.e. endogenously: hematogenously, lymphogenously or per continuitatem - from primary or

secondary foci, with pulmonary or extrapulmonary tuberculosis. The skin is far from being an ideal tissue of the human body for the development of Mycobacterium tuberculosis.

First, the surface temperature of the skin is several degrees lower than in the lungs; secondly, the oxygen content is significantly lower, and CO2 is higher than in the lung tissue; and, finally, the lung tissue is almost 95% water, and the water content in the skin

is no more than 76%. Therefore, tuberculosis of the skin and mucous membranes, unlike tuberculosis of the lungs, kidneys, other internal organs, bones and joints, is much less common.

Currently, 1 case of skin tuberculosis in Belarus accounts for almost 1,000 cases of pulmonary tuberculosis. Among the provoking factors are acute infections, injuries, endocrine disorders, especially diabetes mellitus, hypovitaminosis, malnutrition, and other conditions that lead to a decrease in nonspecific resistance of the body. A significant role in the pathogenesis of skin tuberculosis is played by the massiveness of infection, the virulence of mycobacteria, and the state of immunity.

Tuberculous inflammation is seen as a classic example of immune-based inflammation. The leading role in skin tuberculosis is assigned to T-cell immunity, and the significance of the humoral link of immunity, as well as the role of autoimmune reactions, remains debatable. At the first introduction of mycobacteria into the skin, a primary affect develops, and then the lymph nodes are involved in the process, since a primary tuberculosis complex is formed.

In this case, the entrance gates are injuries and skin defects, especially with its pustular diseases. As early as 3-4 weeks after infection, a large red-brown papule is formed at the site of mycobacteria introduction, and then a superficial painful ulcer up to 1-2 cm in diameter with a granular bottom with purulent discharge, soft and undermined edges. In some patients, the ulcer has a chancriform character with thickening of the edges.

During the second month, **lymphangitis** and **regional lymphadenitis** occur. Lymph nodes, initially mobile and isolated, soon become soldered between themselves and the skin, often open with the formation of ulcers, resembling scrofuloderma. The ulcer is scarred for several months, while

the repair process leads to petrification of the lymph nodes. Tuberculin tests in primary skin tuberculosis are often negative.

Classification of tuberculosis of the skin. For many years we have been using a classification according to which all forms of cutaneous tuberculosis are divided into localized and disseminated.

Localized forms include: lupus erythematosus, collicative tuberculosis of the skin, warty tuberculosis of the skin, ulcerative tuberculosis of the skin and mucous membranes.

Among the **disseminated forms** of skin tuberculosis are called papulonecrotic tuberculosis, indurative skin tuberculosis, lichenoid tuberculosis, or lichen scrofula, miliary disseminated lupus of the face.

Tuberculous lupus (lupus tuberculosis of the skin, or lupus vulgaris). If in the post-war years it affected mainly children, it currently occurs mainly in adults, more often in women suffering from tuberculosis of the lungs or lymph nodes. Favorite localization is the face. The primary element is a tubercle (lupoma) of a brownish-yellow or red-brown color.

Due to the appearance of a number of similar elements of the rash, after 1.5–4 months, plaques with scalloped outlines, a raised peripheral roller and a scar in the center are formed. New tubercles may appear on the emerging superficial whitish scar. The tubercles are characterized by a soft texture, they are characterized by 2 symptoms: a symptom of **"apple jelly"** during diascopy of the elements of the rash (but it can also be positive in the lupoid form of rosacea) and a "probe symptom" - a fossa on the surface of the tubercle after light pressure with a blunt probe (cause - death of collagen fibers in the focus of tuberculous inflammation).

With stronger pressure on the tubercle, the probe may fail, which is accompanied by soreness and the appearance of a drop of blood (this is not observed with rosacea). Tuberculous lupus can proceed according to one of the following clinical variants: - flat; -ulcerative, or mutilating; - tumorous; - warty, or papillomatous; - psoriasiform lupus erythematosus with an abundance of grayish-white scales on the surface.

There are cases of isolated lesions of the oral mucosa and nasal septum with perforation of the latter. The course of lupus vulgaris is perennial, worsening in winter. Possible complications, **erysipelas** and **lupus carcinoma** (squamous cell carcinoma with rapid metastasis).

Pathomorphology. The tubercles are based on an infectious granuloma with a predominance of epithelioid cells with caseous necrosis in the center, around which thereis an abundance of lymphocytes and giant Langhans cells. Tuberculosis bacilli are found. Differential diagnosis is carried out with serpiginous, or creeping, tubercular syphilis, grouped tubercular syphilis, tuber culoid leprosy, chromomycosis, cutaneous leishmaniasis, tuberculoid form. In favor of **tuberculous syphilis**, a pale red color with a bluish tint of tubercles, their densely elastic consistency, negative symptoms of a "probe" and "apple jelly", relatively rapid evolution (several months), the absence of relapses of tubercles on scars and positive treponemal serological tests will speak.

Pic-2 lupus

Tuberculoid leprosy foci are characterized by negative lupus symptoms, early loss of temperature and pain surface sensitivity, and sweating in the lesion. In the tuberculoid form of cutaneous leishmaniasis, the tubercles are painful on palpation, leaving retracted scars.

Foci of chromomycosis are more often localized on the trunk and extremities, lupus symptoms are negative, black oval or spherical bodies of the pathogen are found in biopsied pieces of skin from the focus. **Collicative tuberculosis** of the skin, or scrofuloderma. There are primary

scrofuloderma, which develops as a result of the hematogenous spread of mycobacteria from the primary focus with a sharp decrease in immunity, and the more frequently recorded secondary scrofuloderma, which occurs as a result of the lymphogenous spread of the pathogen from the lymph nodes affected by tuberculosis.

Primary scrofuloderma is characterized by the appearance in the subcutaneous tissue (mainly in the limbs) of small mobile and painless nodes, which subsequently increase in size and open with the formation of sluggishly granulating ulcers. Ulcers heal slowly with the formation of bridging scars with an uneven surface.

The favorite localization of secondary scrofuloderma is the neck, sternum, and collarbones. The primary element is a node in the subcutaneous tissue, the skin over which constantly turns red. After the disintegration of the nodes, deep ulcers with soft undermined edges are formed, and the bottom is covered with yellowish caseous masses, after the rejection of which retracted bridge-like scars form.

Histologically, in the deep layers of the dermis, a cellular infiltrate is determined in the form of a node, which consists of a mass of epithelioid cells forming a tubercle surrounded by lymphoid elements, in the center - cheesy necrosis. Mycobacteria are found along the periphery of the caseous area.

Differential diagnosis of scrofuloderma is carried out: - with **syphilitic gumma** (differences:different localization, the presence of a necrotic rod in the ulcer, stellate healing scar, positive treponemal seroreactions); - **actinomycosis** (differences: woody density infiltrate, liquid purulent and purulent-bloody discharge with the presence of yellow drusen); - **infiltrative-abscessed pyoderma** (differences: localization in the axillary and inguinal folds, on the buttocks; dense infiltrates, multiple communicating fistulas with bright hyperemia around).

Warty tuberculosis of the skin is a superinfection, in which mycobacteria enter the skin of already infected people, or autoinoculation, although the hematogenous route of entry is not excluded. The main contingent is surgeons, pathologists, veterinarians, butchers.

The main causative agent is M. tuberculosis bovis (bovine type). The primary element is the papule. Localization - open areas of the body, mainly the rear of the hands. Lesions of

various shapes and sizes, with clear boundaries, brownish-red color, with papillomatous, rough surface. It is possible to distinguish 3 zones of the focus: in the center - warty growths up to 1 cm high, then a zone of crusts, cracks with a bluish-reddish ridge along the periphery, the most extreme zone - a bluish corolla in the form of a border on the border with healthy skin. The focus is usually solitary.

Histology.There is **hyperkeratosis, papillomatosis**, a combination of inflammatory and granulomatous reactions in the dermis, mycobacteria are difficult to detect. Typical tubercles in the biopsy are not always detected, especially in the old elements of the rash. Differential diagnosis of warty tuberculosis is carried out: - with **ulcerative-vegetative pyoderma** (differences: acute inflammatory reaction around ulcers, lower limbs are more often affected,unpleasantly smelling serous-purulent discharge, a tendency to develop new ulcerative defects); - **chromomycosis** (difference: foci on the trunk and extremities,detection of the pathogen during mycological examination or biopsy); - **warty red lichen** (differences: predominant localization of foci on the legs, the absence of three zones in the lesion, no scarring, intense itching, different histological picture).

Ulcerative tuberculosis of the skin and mucous membranes. Occurs rarely. It is the result of massive autoinoculation in exudative tuberculosis of the lungs or gastrointestinal tract, less often of the genitourinary organs. The disease manifests itself by the appearance of miliary nodules of pink or yellowish color on the mucous membranes of the oral cavity or genital organs, around natural openings, which disintegrate relatively quickly with the formation of small painful ulcers with scalloped outlines, undermined edges and an uneven granular bottom (necrotic tubercles of a grayish-yellow color - grains Trill).

Ulcers can merge into long-existing extensive ulcerative surfaces. Regional lymph nodes are enlarged and painful. Histology. In the deep layers of the dermis, tuberculoid granulomas are observed, in the more superficial layers, an inflammatory reaction around the ulceration;

Mycobacterium tuberculosis is easily detected. Tuberculin reactions are often negative, but may be hyperergic.

Papulo-necrotic tuberculosis of the skin is actually allergic vasculitis due to sensitization by Mycobacterium tuberculosis. It appears symmetrically located on the skin of the face, extensor surfaces of the extremities, elbows, knees, buttocks with rounded papules 2–6 mm in size, bluish-red in color, in the center of which a pseudopustule with necrosis develops.

A small sore is covered with a tight-fitting crust, surrounded by a raised rim. Stamped scars remain in place of the sore. The disease lasts for months and years, worsening in winter. Tuberculin tests are positive. The superficial pustular form of papulonecrotic tuberculosis is called **Acnitis**, resembles **acne vulgaris**, and occurs in school-age children.

A deep pustular variety of papulonecrotic tuberculosis called Folliclis has been described in adolescents and young adults. Papulonecrotic tuberculosis is usually combined with tuberculosis of the lymph nodes, tuberculosis of the lungs, and bones.

Histology. In the dermis - non-specific inflammatory changes, then central necrosis is detected, around it - non-specific, granulomatous infiltrate. In the epidermis - **acanthosis, parakeratosis**. Mycobacteria are difficult to detect.

Differential diagnosis is carried out: - with **papulo-necrotic vasculitis** (differences: acute course, negative tuberculin reactions, the process is resolved in 2-3 weeks); - **lymphatoid papulosis** (differences: bluish-pink papules, plaques appear, the development cycle is 4-6 weeks, the disease lasts for years.

Histologically - a picture of true lymphoma). Bazin's erythema induratus (skin induration) is a vasculitis of the deep vessels of the dermis of tuberculous etiology. Women of middle age are more often ill. The predominant localization is the legs. The primary element is a node in the reticular layer of the dermis and subcutaneous adipose tissue.

The nodes are few, symmetrically located, 2–5 cm in diameter, densely elastic in consistency. The skin above them gradually turns red (red-cyanotic color), cold to the touch. In every 4-5th patient, the nodes ulcerate (the ulcerative form of Hutchinson). The edges of the ulcers are

steep or undermined, compacted. After healing, a smooth, retracted scar remains.

Histology. The inflammatory infiltrate is characterized by a granulomatous and lymphocytic-plasma cell reaction, swelling and proliferation of the endothelium of blood vessels, thrombosis and obliteration of their lumen are observed. In the future, a specific granulomatous reaction is formed.

Differential diagnosis is carried out: - with **erythema nodosum**, chronic form (differences: brighter color of the nodes, soreness, nodes do not ulcerate, localization - often the lateral surfaces of the legs); - **sarcoidosis coarse-nodular** (differences: one or more hemispherical nodes up to 3 cm in diameter, dense consistency, bluish-pink or red- brown in color, with a smooth surface, yellow-brown dust particles are visible during diascopy.

The nodes regress from the center, leaving temporary hyperpigmentation).

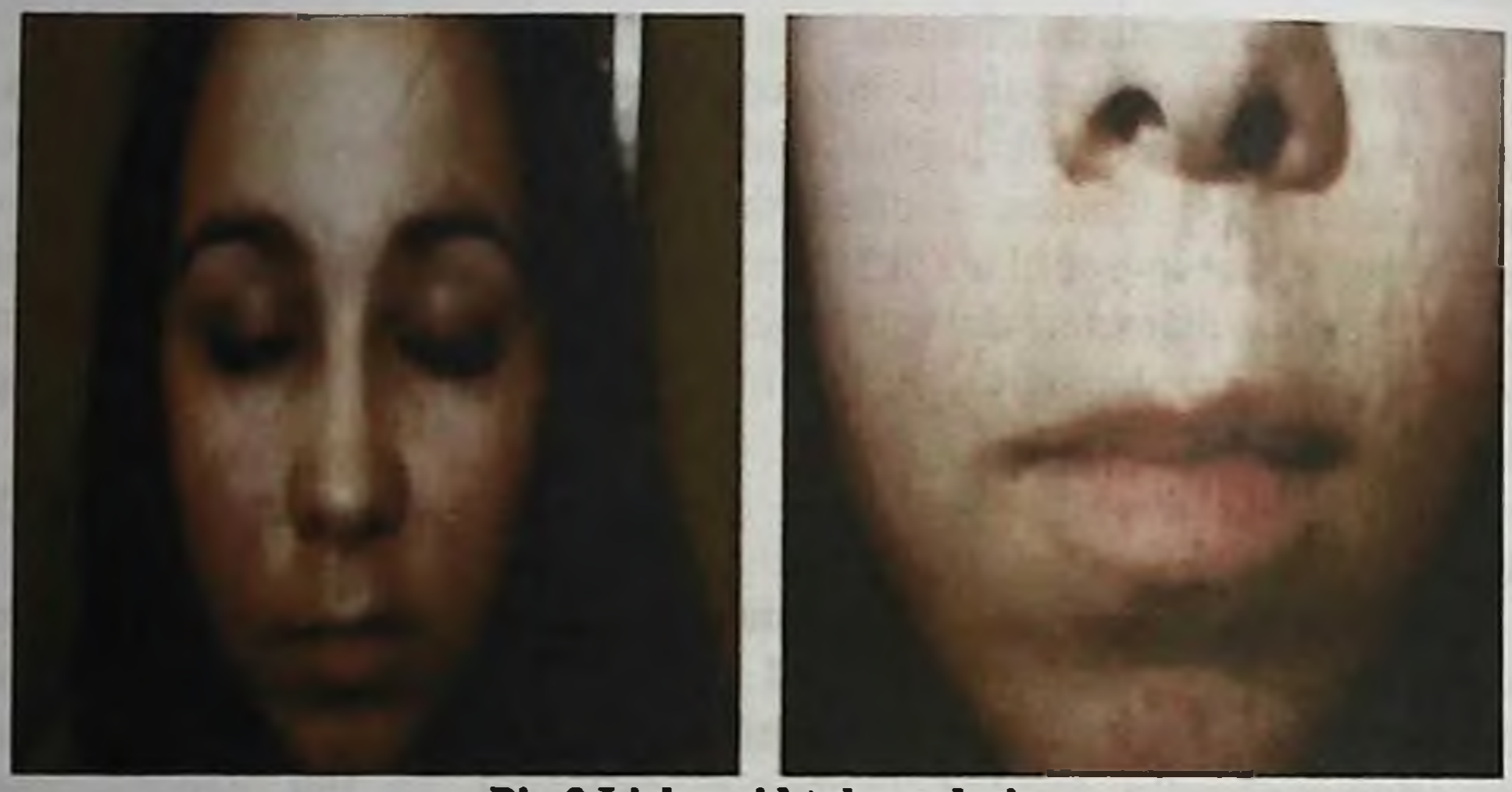
Lichenoid tuberculosis, or lichen scrofula. Currently, this form of tuberculosis is extremely rare and more common among debilitated children and adolescents with active tuberculosis of the internal organs.

Symmetrically located lichenoid, follicular or flat papules and tubercles of yellowish- reddish or brownish color appear on the skin of the chest, lateral surfaces of the body, which can be grouped, forming ring-shaped foci. The rashes leave superficial scars. The course of the disease is chronic, paroxysmal. Tuberculin tests are positive.

Histology. Epithelioid-cell granulomas are detected, located perifollicularly, in the
presence of a small number of lymphocytes, giant cells.

Differential diagnosis is carried out: - with **lichen planus** (polygonal shiny itchy papules with a central impression, with a predominant location on the inner surface of the upper limbs, on the legs and trunk, with a positive Koebner phenomenon, frequent lesions of the mucous membranes and nails; no data on the presence of internal tuberculosis organs); - **brilliant lichen** (differences: multiple isolated flat papules with a diameter of 1-2 mm with a shiny non-scaly surface, pale pink or normal in color; tuberculin reactions are negative).

Miliary disseminated lupus of the face. According to some scientists, this is a rare form of skin tuberculosis, it is a papular variant of localized papulonecrotic tuberculosis, according to others, it is a lupoid form of rosacea. The face is hurt. Miliary painless papules of yellowish-reddish color, hemispherical, with a pustule in the center, soft consistency are revealed.



Pic-3 Lichenoid tuberculosis

The phenomenon of "apple jelly" is determined. At the same time, the patient can see eruptive elements at different stages of development. The rash may leave small scars.

Histology. An infiltrate of a tubercular structure is found with the development of focal necrosis, surrounded by a large number of epithelioid cells, there are giant cells, a large number of tuberculosis bacilli.

Differential diagnosis is carried out: - with **rosacea** (differences: on the edematous and hyperemic skin of the face there are a large number of acne, papulo-pustular elements, telangiectasias, there are no indications of the presence of tuberculosis of the internal organs); - **small-nodular sarcoidosis** (differences: multiple, sharply demarcated, symmetrically located, dense tubercles and papules up to a pea size, with a smooth surface, the color of which varies from pale pink to purple and brown.

The phenomenon of motes is positive.Reactions to tuberculin are usually negative).

Diagnosis of tuberculosis of the skin. When establishing a diagnosis, one should take into account the history, clinical, microbiological and histological data, the detection of foci oftuberculosis in the internal organs, the results of tuberculin tests and trial treatment.

Treatment of patients with skin tuberculosis. Patients with skin tuberculosis are registered in regional anti-tuberculosis institutions, where they receive basic and anti-relapse therapy. Treatment begins immediately after the diagnosis is established in a specialized hospital.

Anti-tuberculosis drugs are divided into 3 groups: 1) highly active: isoniazid, ftivazid, tubozide, rifampicin; 2) medium activity: streptomycin, kanamycin, florimycin, ethambutol, ethionamide, pyrazinamide, lomefloxacin (maxakvin); 3) PASK, bepask. The treatment is combined, it starts with 3 drugs, for example: isoniazid + rifampicin +streptomycin.

After three months, streptomycin should be replaced with ethambutol or PAS, after another three months, PAS is changed to Bepask, which is better tolerated by patients. The duration of the first course of inpatient treatment is 9–12 months. Subsequently, annually for at least 2-3 years, two two-month courses of anti-relapse treatment arecarried out.

The complex treatment regimen also includes multivitamins, iron preparations, hepato- and angioprotectors. A prerequisite is the organization of high-calorie therapeutic nutrition. Table number 11 is assigned, rich in proteins, vitamins, calcium salts with limited salt.

Indications for deregistration of patients with skin tuberculosis are: - complete resolution of foci on the skin and mucous membranes with the formation of a mature scar; - positive results of clinical-laboratory-radiological examination in case of resolution of pulmonary or extrapulmonary tuberculosis process; - stating a clinical cure after a full-fledged basic and anti-relapse treatment; – results of a histological examination of the final skin biopsy in the area of the former lesion.

LEPROSY

Leprosy (syn.: leprosy (obsolete), Hansen's disease) is a chronic infectious disease caused by leprosy mycobacteria, characterized by

granulomatous lesions of the skin, mucous membranes, peripheral nervous and endocrine systems, internal organs, and also characterized by a long incubation period (from 1 up to 20 years or more), torpid protracted course with periodic exacerbations.

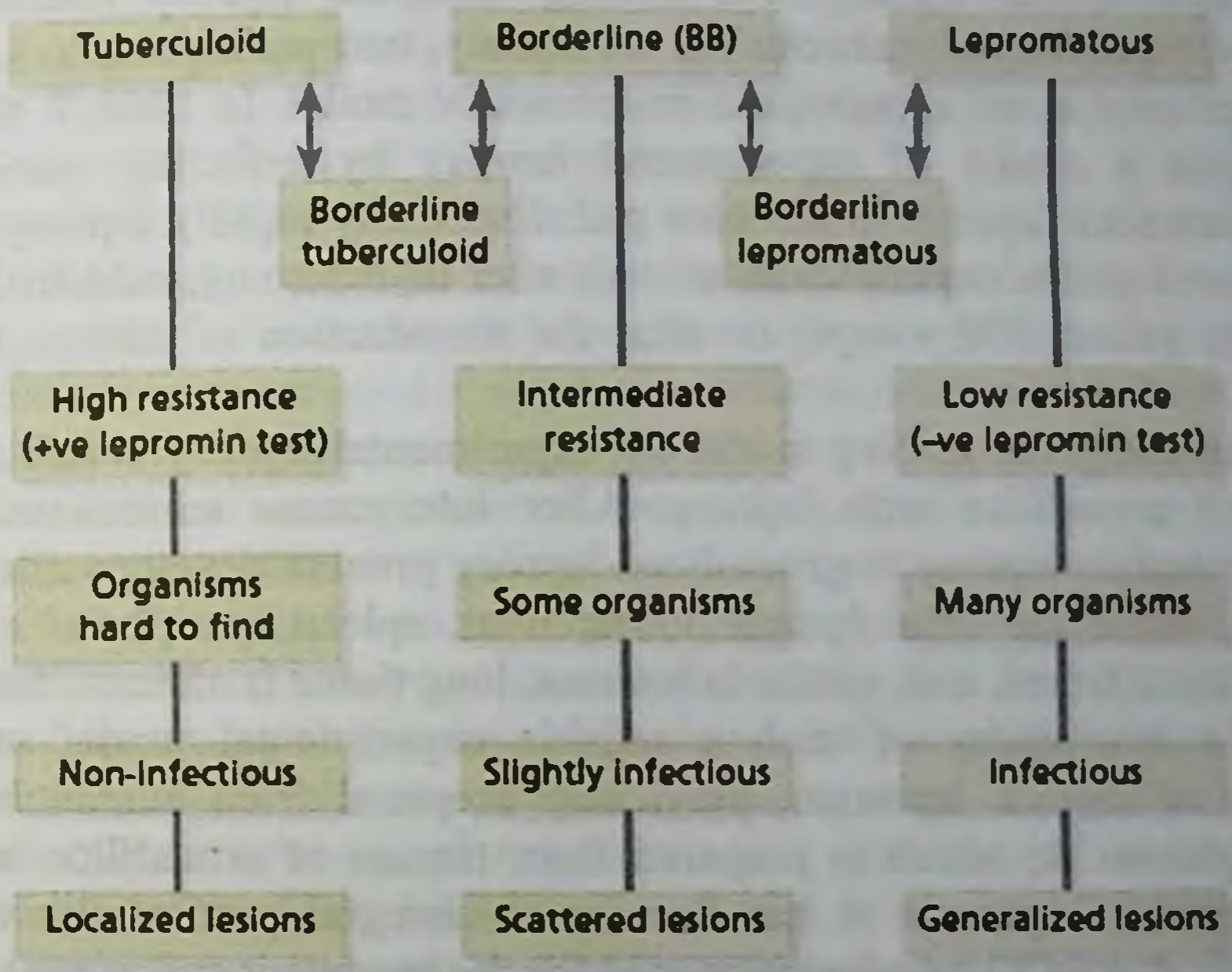
Pic-5 Leprosy

Prevalence. The disease has been known since ancient times. The peak incidence of leprosy in Europe fell on the XI-XIII centuries. Only the extensive state measures taken to combat leprosy, mainly the opening of a huge number of special isolation shelters-leper colonies, led to a significant reduction in the number of patients.

The exact number of patients with leprosy is not known, since not all countries inendemic regions report accurate data on the incidence. If in the 90s of the last century they talked about 10-12 million lepers, then in the last 5-7 years only 450-520 thousand patients are registered annually.

The disease is endemic in 53 countries of the world, mainly in countries with a hot and humid climate (Southeast Asia, Africa, South and Central America), where almost 2 billion people live. On the territory of the former USSR, leprosy endemic foci were considered: the Volga delta, the North Caucasus, Karakalpakstan, Kazakhstan. In the Republic of Belarus, since 1992, not a single patient with leprosy has been registered.

Etiology. The causative agent of the disease - mycobacterium leprosy (Mycobacterium leprae) was discovered by the Norwegian physician G. A. Gansen in 1873. This is the firstof the rod-shaped bacteria, the presence of which was associated with the development of the disease in humans. The disease of leprosy was considered shameful in society for a long time, which is why this nosology was more often called **Hansen's disease**.



Pic-6 Forms of Leprosy

Mycobacterium leprosy is an intracellular parasite that has many properties in common with tubercle bacillus. More often, these are straight or slightly curved rods measuring 1– 8 μm in length and 0.3–0.5 μm in width; sometimes club-shaped swellings and granular forms are found. Leprosy sticks have polymorphism, in addition to the typical ones, there are long, short, thin, large, curved, branched, segmented, degenerative (breaking into grains) forms.

The causative agent of leprosy does not grow on nutrient media used for growing Mycobacterium tuberculosis. This circumstance is explained by the obligate intracellular parasitism of the microorganism. In addition,

Hansen's bacillus is characterized by an unusually long reproduction cycle for bacteria - 12–14 days.

The causative agent of leprosy is characterized by a high degree of resistance. In human corpses, mycobacterium leprosy retains its full invasiveness for a long time. Outside the human body, the viability of the pathogen is rapidly lost.

Experimental leprosy. For a long time, rat leprosy (**Stefansky's leprosy**) caused by Mycobacterium rat leprosy, non-pathogenic to humans, has been used as an approximate experimental model. In 1960, I. Shepard developed a model of experimental leprosy by infecting mice with mycobacterium leprosy in the paw pad. Especially rapidly leprosy

developed in experimental animals after thymectomy, sublethal x-ray exposure (about 900 x-rays) or after the introduction of antilymphocyte serum.

Currently, the leading model for experimental leprosy is infection of 9-banded armadillos with leprosy. After intravenous administration of mycobacterium leprosy, a generalized leprosy process develops after 8–35 months, while the skin, lymph nodes, liver, spleen, peripheral nervous system are affected, and, unlike in humans, lung tissue is affected early.

The availability of such a reliable experimental model made it possible to replace lepromin, previously prepared from human leprosy, with lepromin A, which is prepared from tissues of armadillos infected with leprosy. Lepromin A has become an integral part of the vaccine (lepromin A + BCG vaccine) proposed for the treatment and prevention of leprosy.

Ways of transmission and pathogenesis of leprosy. The source of the disease is a sick person. The route of transmission of the infection is still unclear. Most likely, this is an airborne route, but other ways of getting the infection into the human body are possible - through the nasopharynx, damaged skin (bites, scratches, small wounds, tattoos), household items, and possibly by eating infected food and water. Infection occurs mainly during close and prolonged contact of healthy individuals with patients with leprosy.

Leprosy mycobacteria are found in large numbers in the nasal mucus of patients, in the discharge of the mucous membranes of the pharynx and

larynx, especially in lepromatous, **lepromatous-borderline leprosy** and in the presence of a reactive phase of leprosy.

Epidemiologists have calculated that such patients, during one act of sneezing, release over 2 million bacilli into the environment along with microdroplets of mucus. The main sources of infection are patients with small bacillary forms of leprosy, which account for 85-90% in endemic regions. In addition to nasal mucus, mycobacterium leprosy is found in semen, tears, urine, urethral discharge, and breast milk. A very important factor in the development of the infectious process is a decrease in the temperature of the external environment.

In addition to humans, the main reservoir and host of the causative agent of leprosy, armadillos are also naturally infected with leprosy (9-banded armadillos live in South America, which can get sick with leprosy), as well as chimpanzees and mangobey monkeys. Most people have a high natural resistance to leprosy infection, as evidenced by a positive lepromine test. Thousands of examples are known when, even after many years of close contact with patients with leprosy, people remained healthy.

The likelihood of contracting leprosy depends on age (in endemic areas, children aged 5 to 14 years and the elderly over 55–60 years of age are especially susceptible to leprosy), climate (wet tropical climate and an abundance of insect bites are especially predisposing to infection), social conditions (poor housing, poverty, starvation, poor general health), susceptibility of the body (children in a family of lepers get sick 5-6 times more often than their peers from families where there is no leprosy).

An important risk factor for the incidence of leprosy is living in a region endemic for leprosy. The largest percentage of patients with an advanced clinic of the disease falls on the age group of 30–50 years. Men and people of the Negroid race get sick more often. Factors that weaken the body's resistance (poor nutrition, alcoholism, drugs, infectious and colds, hard physical work) increased the risk of infection in a person who was in an endemic focus.

The activity of cellular responses of the immune response to mycobacterium leprosy (according to RBTL, leukocyte migration inhibition test, lepromine test) gradually decreases from TT to LL, reflecting the highest and lowest degrees of immunological resistance to

the disease, respectively. The intradermal lepromine test, proposed in 1919 by the Japanese K. Mitsuda, remains the test of choice, especially the late (on the 19th– 22nd day) reaction, which is what is called the **"Mitsuda reaction"**.

In patients with tuberculoid leprosy and in the majority (80–96%) of healthy people, the Mitsud test is positive, and in patients with lepromatous leprosy it is always negative. Since the Mitsud test can change during the transformation of one type of leprosy into another, as well as under the influence of treatment, it is not difficult to conclude that it has no diagnostic value.

The Mitsud test only reflects the state of the immunological reactivity of the macroorganism in relation to the causative agent of leprosy. The virulence of the causative agent of leprosy does not affect the nature of the disease. It has been proven that in the same family, in the presence of one source of infection and the same conditions, contact persons often develop different types of leprosy. The clinic of the disease depends entirely on the state of specific cellular immunity.

The causative agent of the disease multiplies in the peripheral nerves. In addition, mycobacteria leprosy are found in many organs, where they persist for a long time in endothelial cells and phagocytes. Despite the fact that the clinic of this disease is dominated by lesions associated with degenerative changes in the skin and peripheral nervous system, all organs and systems of the human body can be involved in the process, with the exception of the central nervous system and lungs. This fact allows us to classify this disease as a systemic disease.

Leprosy classification. The current working classifications for leprosy are the Madrid (1954) and the Ridley-Jopling (1973) classifications. The Madrid classification identifies2 polar types (tuberculoid and lepromatous) and 2 intermediate groups (undifferentiated and borderline (dimorphic). The Ridley-Jopling classification of leprosy is based on 4 criteria: clinical, bacteriological, immunological and histological.

In its original form, the authors of the classification considered leprosy as a continuous immunological process between **tuberculoid** (TT) and **lepromatous** (LL) types of leprosy, called polar, with the allocation of 3 intermediate - **borderline tuberculoid** (BT), **borderline**

(BB) and **borderline lepromatous** (BL) - and two additional groups - **undifferentiated** (I) and **subpolar leproomatous** type (LLs).

All these forms differ in the nature of clinical manifestations, laboratory parameters, prognosis and epidemiological significance.

Clinical presentation.Lepra infection is characterized by an unusually long incubation period.It is generally accepted that the average duration of the incubation period is 3– 5 years, but there are cases when this period can last 10–20 years or more.

Undifferentiated leprosy (I). In most patients, it is she who is the initial form of the disease. It is clinically manifested by the appearance of hypo- or depigmented spots on the scalp, lower back, buttocks, thighs, shoulders. The spots have clear boundaries, in their area temperature first disappears, then pain surface sensitivity, tactile sensitivity disappears later.

Sweating on the affected areas of the skin is sharply reduced or completely absent. In patients, polyneuritis develops early, more often than others, the ulnar and peroneal nerves are affected. They become painful, thickened, dense. The lesions are symmetrical. Vascular and trophic disorders, paralysis, contractures, amiatrophy (thenar, hypothenar), perforated foot ulcers can develop in the lesions.

In children, this form of leprosy may present as **erythema nodosum**. Hansen's bacilli are rarely found in scrapings of the nasal mucosa and in tissue juice from lesions and only isolated bacteria, and in peripheral nerves (with a biopsy) - more often.

The lymphocyte blast transformation test and the Mitsud test are usually positive. The histological picture is not typical. Undifferentiated leprosy evolves in three ways: it transforms into the TT form in 65–70%, into the lepromatous form (LL) in 5–10%, and remains stably undifferentiated in 25–35% of cases.

The **tuberculoid form of leprosy** is a mildly contagious type with a benign course, with damage to the skin and peripheral nerves. It develops against the background of high resistance and resistance of the body. The lepromine test is usually positive; Mycobacterium Hansen are found rarely and in small quantities.

Skin changes are initially superficial and are expressed by the appearance of hypopigmented or slightly erythematous spots, with a clear

contour; reddish-congestive polygonal papular rashes, often merging into plaques; border and sarcoid-like elements. Peeling is often observed on the surface of the elements. As a result of peripheral growth and resolution in the center of the pathological focus, a picture typical of TT leprosy is observed - large annular erythematous plaques, with a sharply defined, roller-like raised edge, consisting of small-papular elements of congestive or reddish-brown color (the so- called **curly tuberculoid**).

In the central part, a picture of gradually developing regression is observed - it begins to slightly flatten, sink, becomes hypo- or even achromic, covered with small bran-like scales; as a result, processes of skin atrophy develop. The number of foci that arise is different: the size of plaques and border elements can vary significantly - from 3-5 cm to very large elements that capture significant areas of the skin of the trunk and extremities. The asymmetry of the arrangement of elements is characteristic. The most important in the diagnostic aspect is the establishment in the area of foci of loss of temperature, pain and tactile types of sensitivity, the cessation of sweating.

Sarcoid-like (lupoid) rashes in TT leprosy are more often localized on the skin of the face and extremities, less often on the skin of the trunk. These are tuberculous elements ranging in size from 0.5 to 1.5 cm in diameter, with clear boundaries and a smooth surface, of a characteristic stagnant color, with a tendency to group. Along with skin lesions, nerve lesions can be observed in some cases.

First of all, the nerve trunks are affected, in the zone of which there are rashes. With the defeat of n. ulnaris, the patient has limited mobility of the V, IV, III fingers, up to the contracture of the V and IV fingers. With atrophy of thenar and hypothenar, the hand resembles a vulture's paw. With the simultaneous defeat of n. ulnaris and n. medianus, atrophy of the interosseous muscles occurs, and the hand takes on the appearance of a boat or a monkey's paw.

Histologically, an infective granuloma is represented by giant cells and a small numberof epithelial cells in the center.

Border leprosy. Skin changes in the borderline type of leprosy largely combine the features of TT and LL leprosy. At the beginning of the disease, these are numerous asymmetric erythematous spots, often of irregular shape, with blurred boundaries. However, there may be plaques with a distinct flat edge and a more convex central part. Pathognomonic for this form is the presence of "punched" or "stamped" spots and plaques ("cheese holes"), which are formed as a result of atrophy central to the plaque with elements of hypo- and depigmentation.

If the clinical picture is dominated by plaque rashes, then the skin in the areas of regression is flattened, the element first takes the form of an inverted saucer, and then rounded or strip-like border elements rising above the skin level with a clear steep inner edge. The color of the elements is stagnant, purple.

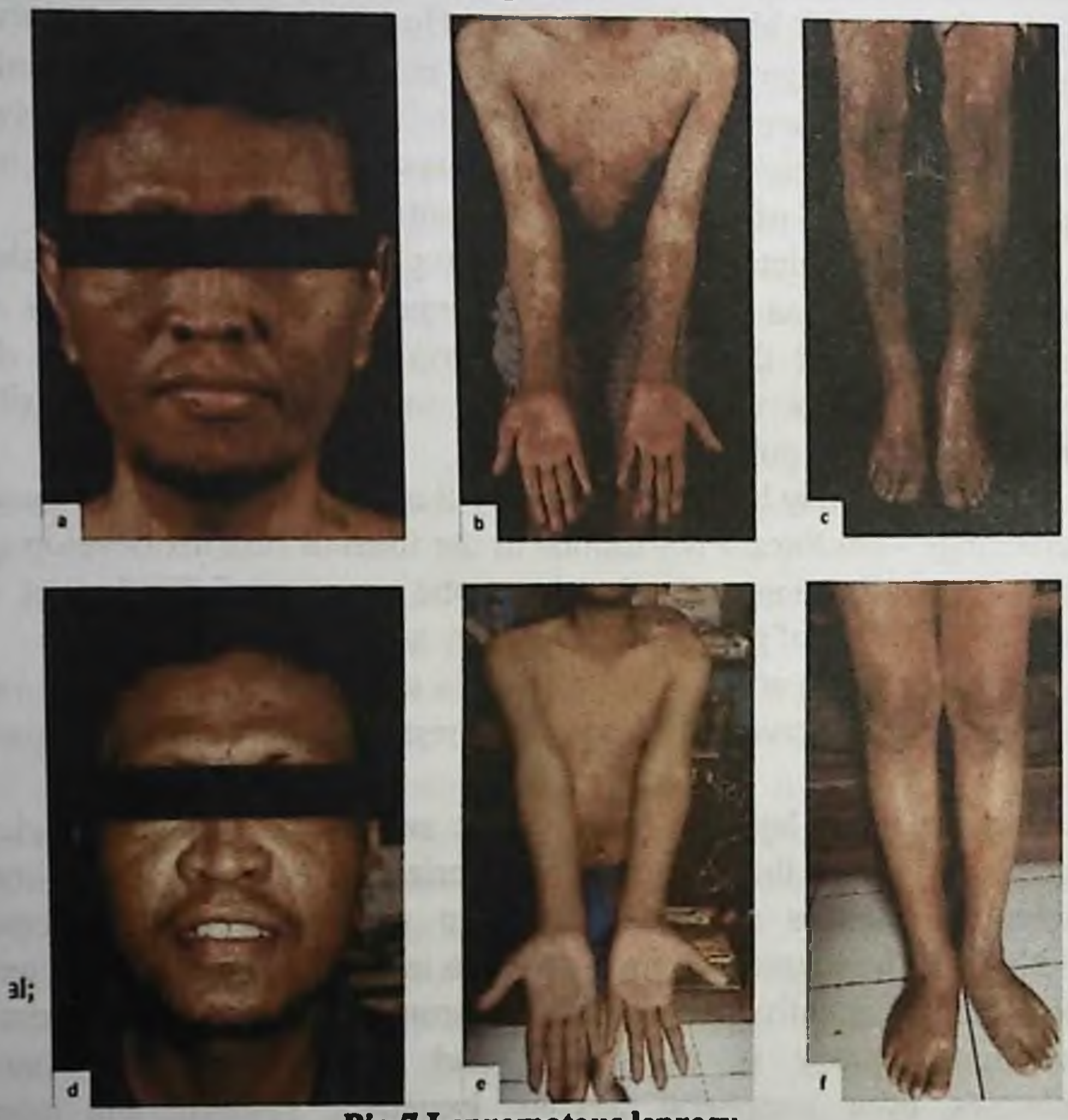
The process may be accompanied by the appearance of small bunches of screenings - satellites. PNS lesions in the form of neuritis develop quite early in borderline leprosy. Already at the stage of formation of skin elements, violations of peripheral sensitivity are detected in them.

Sweating in the area of skin rashes, as a rule, is not disturbed, vellus hair is partially preserved. The lepromine test is either negative or weakly positive.

Lepromatous leprosy is the most severe and epidemiologically dangerous form of the disease, characterized by the irreversibility of pathological changes and a tendency to progression. It develops in individuals with reduced body resistance to infection, which is detected by a persistently negative intradermal lepromine test. The process in lepromatous leprosy is widespread and affects the skin, mucous membranes, eyes, peripheral nervous, lymphatic systems, and internal organs.

A large number of granulomas are formed, literally "stuffed" with Hansen's mycobacteria. Initially, the process is symmetrical and is represented by numerous erythematous, erythematous-pigmented or erythematous-hypopigmentary spots of small size, with fuzzy contours, smooth, shiny surface. Most often, spots are localized on the skin of the face, extensor surfaces of the forearms and legs, as well as on the buttocks.

Note that sensitivity and sweating within the spots arenot disturbed at first. The formation of pathognomonic for lepromatous leprosy infiltrates and leprosy occurs gradually, over a long time.



Pic-7 Lepromatous leprosy

The initial red color of the spots becomes brown or yellowish (copper or rusty). The spotsbegin to take the form of limited, dermal or hypodermal plaques or significant areas of skin infiltration. Often, telangiectasias form on the surface of lepromas or lepromatous infiltrates. The processes of skin infiltration are accompanied by an increase in thefunction of the sebaceous

glands, as a result of which the surface of lepromas and infiltrates is smooth, shiny, "fatty", glossy.

The hair follicles and excretory ducts of the sweat glands are dilated, giving the skin an orange-peel appearance. Sweating in the initial stages decreases, and then completely stops. Gradually, the processes of atrophy of the hair follicles start, which first leads to the loss of vellus, and then other types of hair.

After 3–5 years or more, starting from the outside, there is hair loss of the eyebrows and eyelashes, beard and mustache, as well as the scalp. The consistency of the elements is densely elastic, deep-lying lepromas are palpable in the form of dense strands and knots and only slightly protrude above the level of the surrounding skin. The skin above them has a bluish-brown tint.

Usually dermal and hypodermal elements exist simultaneously, closely adjacent to each other, they form continuous bumpy conglomerates. When the pathological process is located on the skin of the face, natural wrinkles and folds deepen, the superciliary arches protrude sharply, the nose increases in size, the cheeks, lips and chin have a lobedstructure, all this greatly disfigures the patient's face, giving it the appearance of a "lion's muzzle" (**facies leonina**).

Moreover, infiltration of the skin of the face with leprosy LL, as a rule, does not pass to the scalp. Infiltrates also do not develop in the so-called immune zones - on the skin of theulnar and popliteal flexor surfaces, the pubis, the inner part of the eye sockets and the armpits. The favorite localization of lepromatous infiltrates is the forearm and lower leg; their depth is different.

Often inside such an infiltrate, you can feel the hypodermal leprosy. The skin over the infiltrates is colored bluish-brown. The course of the process does not have certain rules, lepromatous elements can occur singly or several elements ripen at once on a bright, erythematous background that mimics erysipelas, in the form of an acute outbreak. The latter is usually accompanied by a febrile state with severe toxic effects.

Leproms exist for an indefinitely long time - months and years. As a result, the element can be resolved with the formation of pigmentation,

cicatricial atrophy, or areas of retraction formed deep in the skin; disintegrate, with the formation of crater-like ulcers,

with undermined, overhanging edges, a grayish bottom and a viscous discharge containing Mycobacterium leprosy; gradually replaced by fibrous tissue - the nodesdecrease in size and become more dense.

While working in Guinea, I had to consult patients with mutilating leprosy, when the terminal phalanges of the fingers and sometimes all fingers were missing on the hands and feet, the limbs resembled a seal's paw. Very often, skin lesions are accompanied by lesions of the mucous membranes of the lips, tongue, soft and hard palate, larynx and nose.

In the initial period, damage to the nasal mucosa often manifests itself in the form of persistent rhinitis and frequent nosebleeds. Lepromas formed on the mucous membrane of the nasal septum gradually lead to its perforation and deformation of the nose.

With the localization of the lepromatous node on the mucous membrane of the larynx, the patient's voice becomes hoarse, up to its loss, an increase in the defect may also be accompanied by stenosis. Damage to the mucous membrane of the eyes leads to **episcleritis, keratitis** with a frequent outcome in leukoma, **iritis** and **iridocyclitis**. Often the first manifestation of the clinical picture may be a reaction from the lymphatic system. At the same time, peripheral lymph nodes increase, acquire an elastic consistency, are not soldered to surrounding tissues, and are painless on palpation. In the punctate of the lymph nodes, Hansen's mycobacteria are easily detected. In men, the clinic of orchiepididymitis often develops, as a rule, bilateral. The testicles and appendages are compacted, become tuberous.

The process ends with sclerosis and atrophy. Less commonly, the prostate gland and urethra are affected. In some patients, there is an increase in the liver and spleen. Superficial types of sensitivity remain undisturbed for a long time. A feature of leprosy neuritis is their ascending nature and "island" type of sensory disorders, which is caused by the destruction, first of all, of peripheral nerve endings in places of skin rashes, damage to the skin branches of nerves and individual trunks of the PNS.

The most vulnerable to mycobacteria leprosy are the areas of adduction of nerve trunks, and they are the first to be involved in the process. In some cases, stem neuritis is found, while the affected nerve trunks are palpated in the form of painful knotty strands. As a result, the patient begins to be disturbed by unbearable pains of a neuralgic nature, anesthesia, amyotrophy, etc.

The clinic of progressive peripheral sensitivity disorders is the main disability factor in patients with leprosy. When the immunological reactivity of the body changes, reactive states (exacerbations of the course of the leprosy process) are observed - a sudden change in the course of the disease, which develops in more than half of the cases.

There are reactive states of the first and second types. The reactive states of the first type include descending and ascending reactions of transformation. They occur in patients with

borderline lepromatous and **borderline tuberculoid leprosy** and are characterized by inflammation of existing foci of infection.

Downward transformation reaction - the transition of the disease towards the lepromatous form is observed in untreated patients. An ascending transformation reaction - a transition towards the tuberculoid form, occurs at the beginning of treatment. It is impossible to distinguish these reactions clinically.

With the same frequency, they can be accompanied by low-grade fever, new erythematous-papular rashes, increased symptoms of **polyneuritis** ("inflammation"). Type II reactive states include **erythema nodosum leprosy** and the **Lucio reaction.**

Erythema nodosa leprosy occurs in half of patients with lepromatous form, usually in the first 2 years after the start of treatment. Multiple inflamed subcutaneous nodes appear on the skin. Known as **Saint Lazarus leprosy**, Lucio's reaction occurs only in patients with diffuse lepromatous leprosy. Extensive, irregularly shaped superficial ulcers form onthe skin of the legs.

There is no single view on the formation of this reaction, some associate its development with emerging arteritis, eventually leading to vascular occlusion, others consider the Lucio reaction to be a variant of erythema nodosum lepromatous. **Lepromatous leprosy** is a multibacillary

leprosy, in smears of scrapings with leprosy and from the nasal mucosa, a large number of mycobacteria are detected, arranged in the form of clusters, balls, and in macrophages (Virchow cells) - like cigarettes in a pack.

Lymphocyte blast transformation test and Mitsud test were negative. Histomorphology of leprosy. The tuberculoid form of leprosy is characterized by the development of a granuloma reaching directly to the epidermis with clear foci of epithelioid cells surrounded by a border of lymphocytes. It is characterized by the development of signs of hypersensitivity - deep erosion of the epidermis, central caseosis, fibrinoid necrosis in the dermis, the presence of Pirogov-Langhans giant cells.

Most of the small nerves are destroyed or sharply infiltrated. It is not possible to detect mycobacteria by conventional methods. A similar picture is observed in the borderline tuberculoid form of leprosy. The difference is the presence in some places of the unaffected subepidermal zone, as well as thickened nerves and single mycobacteria. Signs of hypersensitivity are less pronounced.

The granuloma that develops in the borderline type of leprosy contains a fairly large number of pathogens. Structurally, it consists of diffusely located epithelioid cells, with a rare inclusion of lymphocytes and giant cells. The subepidermal zone is not infiltrated. Due to the proliferation of Schwann cells, the nerves are relatively easy to identify.

With borderline lepromatous leprosy, the granuloma consists mainly of macrophage cells, in which vacuolization is noted. Clusters of lymphocytes and a small number of epithelioid cells are detected. The subepidermal zone is not infiltrated. Nerve endings are

slightly infiltrated, perineurium dissection is revealed.

Mycobacterium leprosy is present in large numbers and diffusely distributed in the granuloma. The lepromatous form is accompanied by the development of granulomatous inflammation from macrophages at different stages of fatty degeneration, up to foam cells, and a small number of diffusely located lymphocytes. Nerves are often not changed, not infiltrated. The granuloma contains a huge number of pathogens, the clusters of whichresemble balls **("globl" type)**.

Diagnostics. In the early stages of the disease, the diagnosis of leprosy is difficult and is based on a correct assessment of the dermatological and neurological manifestations of the disease, functional and laboratory studies, as well as anamnesis data - living in an epidemic zone, contacts with patients with leprosy.

A thorough examination helps, revealing the slightest changes in the color of the skin, conducting studies of temperature, pain and tactile sensitivity, as well as functional tests for sweating (a test with intravenous injection of 5 ml of a 1% solution of nicotinic acid, a Minor test, a test with histamine, 1% pilocarpine, 2% diamine) and causing reflex erythema.

The presence of tuberculous-knotty and plaque elements of a peculiar brownish color with a greasy sheen, hair loss in the lesions with a violation of superficial types of sensitivity, thickening and pain on palpation of the peripheral nerve trunks facilitates clinical diagnosis. But in all cases, it is required to confirm the diagnosis by detecting Mycobacterium leprosy in tissue juice, in smears from the nasal mucosa or in histological preparations.

For bacterioscopic examination, scrapings are taken from the mucous membrane of the nasal cavity (on both sides of the septum), the contents of leprosy nodes, sputum, discharge of ulcers; during the period of fever examine the blood. Smears are stained according to Ziehl-Neelsen. In some cases, a biopsy of leprosy areas and a puncture of the lymph nodes are done.

The preparations determine the bacterial index (logarithmic Ridley scale) - the number of bacteria in the fields of view. With tuberculoid and borderline forms of leprosy, bacteria in smears may be absent, other reasons for a negative result may be previous antileprosy therapy, inexperience of a laboratory assistant.

Of the serological tests, the most commonly used are the complement fixation reaction (RCC) and the indirect hemagglutination test (RIHA). Recently, polymerase chain reaction (PCR) has been used to identify the DNA of Mycobacterium leprae, as well as to evaluate the effectiveness of the treatment. It is the most sensitive technique and can be used to diagnose any form of leprosy.

The treatment of leprosy should be directed towards the following principles. Firstly, sanitation of the body from mycobacteria leprosy; secondly, the prevention and treatment of reactive conditions; thirdly, the prevention and treatment of neurological complications of leprosy; fourthly, the rehabilitation of a patient with leprosy in the absence of sensitivity, as well as the social adaptation of the patient.

The effectiveness of treatment largely depends on the stage and characteristics of the course of the lepromatous process, the age of the patient, the compensatory capabilities of the body, the presence of concomitant pathology, and the competent selection of etiotropic therapy.

Treatment of patients with leprosy should be complex, consisting in the simultaneous appointment of 2-3 etiotropic drugs at once in combination with restorative, immunostimulating and vitamin preparations. The course of antileprosy treatment should be long and last at least 6 months for tuberculoid form, 9–12 months for undifferentiated and borderline leprosy, and at least 24 months for lepromatous and borderline lepromatous leprosy.

If the patient tolerates etiotropic therapy satisfactorily, a break between courses is not recommended. Currently, the most common and frequently used anti-leprosy drugs include sulfonic drugs (dapsone, DDS, avlosulfone, diucifon, solusulfone); antibiotics (rifampicin, minocycline and clarithromycin); long-acting sulfonamides (sulfadimethoxine, sulfamethodiazine, or chiron, sulfortomidine, or fanazil); clofazimine, or lampren, as well as some antibacterial drugs with anti-tuberculosis activity (ofloxacin, ethionamide and prothionamide).

Sulfonic drugs are bacteriostatic, the mechanism of their action is associated with inhibition of the synthesis of folic acid of the pathogen. They show their activity against a wide range of microorganisms, but mainly against Mycobacterium leprae, as well as Plasmodium, Pneumocystis carinii.

Of the antibacterial drugs, rifampicin is most often used, a semi-synthetic broad-spectrum antibiotic that has a bactericidal effect. Rifampicin inhibits bacterial RNA synthesis by inhibiting the pathogen's DNA-dependent RNA polymerase. Clofazimine (lampren, B 663) has been

used to treat leprosy since 1971. It is available in 50 and 100 mg capsules (orange powder).

WHO experts recommend the following method of its use: 300 mg on the first day ofeach month, on other days of the month, 50 mg daily. It is the drug of choice in the treatment of leprosy reactions, with this pathology it is prescribed 200–400 mg per day. Combines well with dapsone and rifampicin.

Its disadvantages are that it stains the skin bronze and the urine red. Thioamides (ethionamide, prothionamide) in terms of their bactericidal effect on mycobacterium leprosy occupy an intermediate place between dapsone and rifampicin, but they have a toxic effect on the liver. Assigned to 1 tablet (0.25 g) 2-3 times a day for 2-3 months as part of complex therapy.

Anti-leprosy therapy is prescribed more often by a chronically intermittent method, it is recommended to combine drugs taking into account their synergism. Specific therapy is combined with the appointment of hepatoprotectors, vitamins.

WHO experts on leprosy recommend the following combination of drugs for the treatment of patients with multibacillary leprosy: dapsone 100 mg daily, rifampicin 600 mg once a month, clofazimine 300 mg once a month, followed by 50 mg daily. Treatment with these drugs lasts 2 years without interruption. When resistance to rifampicin develops, it is replaced with either ofloxacin 400 mg daily, or minocycline 100 mg daily, or clarithromycin 500 mg daily for 6 months, and then for 18 months treatment with dapsone, clofazimine (50 mg daily) in combined with 100 mg minocycline or 400 mg ofloxacin.

For small bacillary leprosy, WHO experts recommend treatment for 6 months with dapsone 100 mg daily plus rifampicin 600 mg once a month. In the case of the development of reactive conditions, approaches to treatment will depend on their type. So, with a descending and ascending transformation reaction, prednisone is prescribed ata daily dosage of 40-60 mg orally, then gradually, over the next 2-3 months, the dose is reduced.

Of the antileprosy drugs at this stage of the disease, only clofazimine (lampren) can be used. Direct indications for the appointment of GCS in

this case is the development of neuritis, the threat of ulceration, a cosmetic defect (damage to the mimic muscles of the face).

In case of lepromatous reaction of the type of erythema nodosum in foreign countries, thalidomide is used at 100–300 mg orally at night (thalidomide is prohibited for use on the territory of the Republic of Belarus); rapid reduction in the dose of corticosteroids.

Patients with a developed Lucio reaction are recommended to carry out therapy in this way, however, in this case, the use of both prednisolone and thalidomide is ineffective. Rehabilitation surgery and orthopedics are widely used in case of damage to peripheral nerves. Careful foot skin care helps prevent the formation of neurotrophic ulcers. In the lepromatous form of leprosy, immunotherapy with the BCG vaccine can be used.

There is no specific prevention of leprosy, therefore, the earliest possible detection of patients with their subsequent isolation in infectious hospitals is of primary importance. At the same time, control monitoring of family members of the patient, who undergo in- depth examinations at least once a year for 3–10 years, is considered common practice.

In some cases, it is justified to prescribe preventive treatment to the relatives of the patient. For family members of a leper, as well as for attending medical personnel, no special rules of conduct are required, in addition to observing generally accepted norms of personal hygiene. Previously, children were separated from sick mothers and fed artificially.

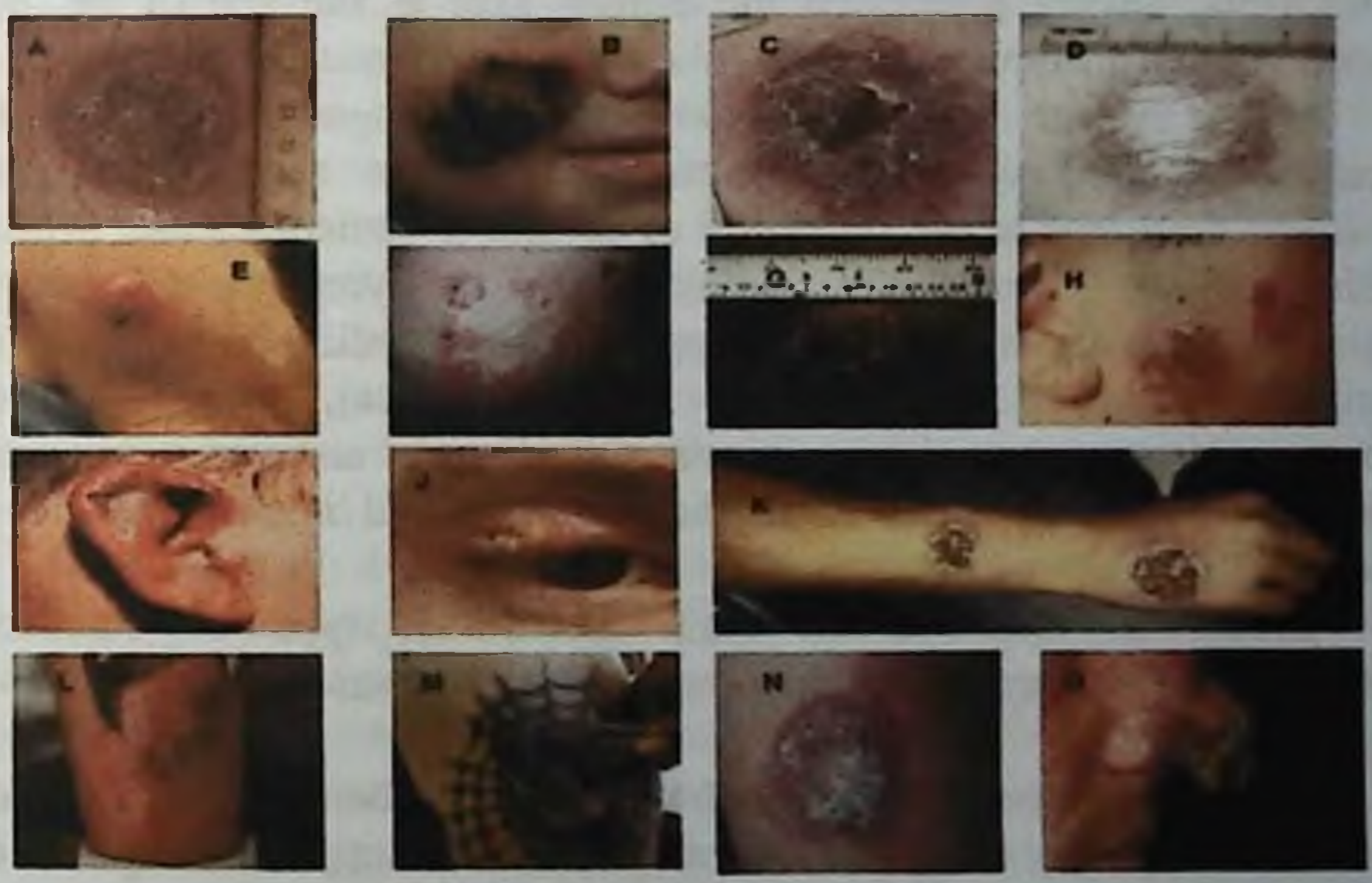
Currently, it is believed that if the mother is carefully treated and observes the rules of personal hygiene, the possibility of infection of the child is reduced to zero. Moreover, it has been shown that the child receives etiotropic drugs with milk, and therefore undergoes a course of preventive treatment.

Therefore, the child is transferred to an orphanage (or close relatives) only at the end of the mother's lactation period, as well as older children for the entire period of mother's treatment in a leper colony or in a special department. Children from a leper's family are not subject to military service.

Cutaneous leishmaniasis

Cutaneous leishmaniasis (syn.: **Borowski's disease, Pendin's ulcer**) is a transmissible parasitic skin disease caused by protozoan parasites Leishmania tropica, endemic to countries with a hot climate. This natural focal disease is distributed mainly in the countries of the Near and Middle East, from the CIS countries - in a number of republics of Central Asia (Turkmenistan, Uzbekistan). Only imported cases are registered in Belarus.

The causative agent of the disease. The causative agent of cutaneous leishmaniasis was discovered in 1898 by P.F. Borovsky, an intern at the Tashkent military hospital, in sections and contents of ulcerative defects while studying Penda ulcer in Turkestan. He discovered and described the oval bodies that filled the protoplasm of the cell, and attributed them to the type of protozoa - protozoa.



Pic-8 Cutaneous leishmaniasis

The causative agent of cutaneous leishmaniasis is called **Leishmania tropica**. Distinguish between L. tropica major (causes acutely necrotizing, rural type) and L. tropica minor (causes late ulcerative, urban type). Leishmania belong to the genus of the family Trypanosomidae

(Trypanosomidae), the order of protomonadids (Protomonadina), the class of flagellates, the type of protozoa.

In humans and other vertebrates, they have an intracellular, immobile leishmanial stage, and in the intestines of the carrier and on artificial nutrient media, they have a flagellated, mobile leptomonas stage. Later, the leishmanial stage was named amastigote, and the leptomonas stage, promastigote.

The reservoir of L. tr. major are small rodent gerbils, and the source of L. tr. minor - asick person, rarely dogs. Mosquitoes of the genus Phlebotomus act as carriers of Leishmania. They live in rodent burrows, damp corners, trash cans and crates. In the body of mosquitoes, Leishmania are in a mobile flagellar form (promastigote stage), in the human body - intracellularly in the form of immobile bodies (amastigote stage). Leishmania, having penetrated the human skin at the site of the bite, multiply in the protoplasm of cells (phagocytic mononuclear cells).

It is at the site of the introduction of the pathogen at the end of the incubation period that the leishmanioma-tubercle appears. Repeated mosquito bites lead to the formation of several leishmania. From primary leishmaniomas, the pathogen migrates along the lymphatic tract to the regional lymph nodes, causing the appearance of seeding tubercles. In the countries of the Old World (Europe, Asia, Africa), rural, urban and tuberculoid cutaneous leishmaniasis are found, and mucocutaneous leishmaniasis is also observed in the Americas and in the countries of Oceania.

Urban, or anthroponotic (late ulcerative), type of cutaneous **leishmaniasis** is characterized by a long incubation period - from 2 to 8-10 months.

Primary leishmanioma is a brownish-red tubercle with a smooth surface. Slowly increasing, after 5-6 months it turns into a node up to 2 cm in diameter, peeling appears on the surface and after 2-4 months or later the node ulcerates. A superficial ulcer of irregular shape is formed, covered with a serous-purulent dense film. The ulcer is surrounded by an elevated, moderately painful infiltrate ridge.

In some patients around the ulcer, "tubercles of seeding" appear. Having existed for about a year, the ulcer begins to clear itself of necrotic

masses, the infiltrate decreases, islands of epithelialization appear, and, finally, a scar forms. Throughout the disease, the general condition of the patient is not disturbed. This type of cutaneous leishmaniasis is now relatively rare.

Histologically, productive inflammation is determined in the dermis with the formation of an infectious granuloma, in which epithelioid cells, macrophages, Pirogov-Langhans giant cells are detected. In granuloma cells, leishmania are detected in the form of small bluish

oval bodies with red nuclei. The rural, or zoonotic (acutely necrotizing) type is characterized by a short incubation period (from 1–2 to 6 weeks), the rapid transformation of the tubercle into a furuncle-like node.

In the center of the node, necrosis develops with the formation of a crater-shaped ulcer up to 3-5 cm in diameter with purulent discharge, the edges of the ulcer are steep, and its bottom is uneven. The ulcer is characterized by a powerful inflammatory infiltrate of doughy consistency with edema. Around the primary ulcer often there are new ulcers, **circular lymphangitis, regional lymphadenitis**.

By the end of the 3rd month, the ulcer begins to clear itself of necrotic masses, papillomatous growths of granulations appear at the bottom of it. The ulcer scars from the center, the scarring process lasts 3-5 months. The scar is slightly depressed, hyperpigmented, with indistinct borders. The general condition of the patient suffers: soreness in the area of the ulcer, swelling of the feet and legs are concerned.

Histologically, a massive infiltrate is determined in the dermis - an infectious granuloma, consisting of epithelial cells, leukocytes and histiocytes, with necrosis in the center. Leishmania are found in large numbers in histiocytes. Those who have recovered from the rural type of cutaneous leishmaniasis remain immune to this pathogen.

Tuberculoid type of cutaneous leishmaniasis. It affects mainly young people, especially after healing of primary leishmanioma, more often in open areas of the body (face, hands), in place of former foci. The pathogenesis is based on the reactivation of Leishmania against the background of a decrease in general local immunity.

The disease acquires a long-term relapsing course and is manifested by recurring yellowish-brown tubercles grouped along the edge of the scars

with a smooth surface, 2–3 mm in diameter, slightly rising above the level of the skin. The tubercles exist for a long time - up to 2 years, after their resolution, an atrophic scar remains, on which new tubercles then appear.

If untreated, the disease lasts for many years, disfiguring the face. In skin biopsies, a small amount of Leishmania in macrophages is found. The disease leaves no immunity.

Diagnostics. The diagnosis of cutaneous leishmaniasis is made on the basis of anamnesis data (stay in an endemic area during the last 1-2 years, especially from June to November); clinical picture and the detection of Leishmania - "Borovsky's bodies" in preparations from the focus (scraping is done from under the edge of the ulcer or from a non-opening tubercle or a histological preparation from a biopsy).

Recently, in specialized microbiological laboratories, sowing on a special nutrient medium 3 N-agar has been practiced.

Differential diagnosis is carried out: - **with a boil** (soreness, the presence of a necrotic rod, evolution within 2 weeks); - **carbuncle** (pronounced dense and painful infiltrate, on which pustules and boils are located, a violation of the general condition: headache, fever,

malaise); - **chronic ulcerative pyoderma** (long-term torpid course with exacerbations of the process, slow scarring after rejection of necrotic tissue);

- **squamous cell carcinoma** (characterized by the development of one tumor, a rapid progressive course, infiltrating growth in depth, decay, with the formation of a deep painful ulcer with dense,torn edges, early metastasis); - **tuberculous syphilis** (characterized by the appearance of dense tubercles of a dark red color with a brownish tint, which do not have a tendency to peripheral growth and fusion; leaving mosaic scars after resolution, positive treponemal serological tests are determined in the blood);

- **tuberculoid form of leprosy** (rashes of tubercles of a reddish-cyanotic color on any part of the skin, prone to merging into continuous lesions rising above the skin, having sharp borders and a towering peripheral ridge; in the lesions there is no temperature and pain surface sensitivity, sweating, no vellus hair) ;

- **tuberculous lupus** (children are more likely to get sick, tuberculous tubercles are soft, they are characterized by symptoms of "apple jelly" and the phenomenon of probe failure, after healing, a superficial scar remains, on which new tubercles may appear again; tuberculin tests are positive, most often visceral tuberculosis is also detected at the same time).

Treatment. Pentavalent antimony preparations, in particular solyusurmin or glucantim, have proven themselves well. The daily dose is 0.35 ml of a 20% solusurmin solution per 1 kg of body weight intravenously daily, the course dose is 7.0 ml / kg of body weight. Recommend monomycin (not registered in the Republic of Belarus) 250,000 IU / m 3 times a day for no more than 10-12 days, doxycycline, metacycline, antimalarial drugs.

In recent years, ketoconazole, terbinafine (Lamisil) and itraconazole (Orungal) have been successfully used. Locally, cryodestruction of tubercles is more often used, chipping of the focus with solyusurmin or monomycin. Forecast. Complete healing of ulcers during treatment occurs in 5-8 weeks.

Prevention. Inoculation of burrowing rodents within a radius of 1.5–2 km, elimination of mosquito nesting sites, anti-mosquito treatment at home. Individual prophylaxis consists of vaccinations 3 months before leaving for endemic foci, the use of repellents and personal protective equipment against mosquitoes (canopies, nets).

Tests for knowledge control

1. Men mostly suffer from: a) lupus vulgaris; b) scrofuloderma; c) ulcerative tuberculosis of the skin; d) indurative erythema of Bazin; e) warty tuberculosis.

2. Women suffer more often: a) scrofuloderma; b) lupus vulgaris; c) lichenoidtuberculosis; d) warty tuberculosis; e) all of the listed forms.

3. Symptoms characteristic of lupus erythematosus: a) "apple jelly"; b) Asbo-Gansen; c)Pinkus; d) Koebner; e) hidden peeling.

4. What kind of tuberculous lupus practically does not occur? a) flat; b) ulcerative; c)mutilating; d) chancriform; e) verrucous.

5. Nodules in collicative tuberculosis: a) dense and painless; b) dense and painful; c) soft and painless; d) soft and painful; e) elastic and painful.

6. Favorite localization of warty tuberculosis a) legs; b) lateral surfaces of the body; c) neck; d) hands; e) genital and perianal areas. Sample answers: 1 d; 2 b; 3 a; 4 g; 5 B; 6 a. **Tests for knowledge control**

1. What clinical form is not included in the modern classification of leprosy? a) undifferentiated; b) tuberculoid; c) border; d) indurative; e) lepromatous.

2. The main route of infection with leprosy: a) through damaged skin; b) airborne; c) transplacental; d) transmissible (insect bites); d) kissing.

3. Which of the following indicators is not typical for tuberculoid leprosy? a) clear boundaries; b) rolled edges; c) fuzzy boundaries; d) less pronounced infiltration in the center; e) tendency to peripheral growth.

4. The most typical morphological element in lepromatous type of leprosy: a) erythematous spot; b) exudative papule; c) tubercle; d) bubble; e) node.

5. Which test has no diagnostic value for assessing sweating disorders in leprosy? a) test with nicotinic acid; b) test with histamine; c) potassium iodide test (Yadasson test); d) test with pilocarpine; e) iodine-starch test (Minor's test).

6. What drug is not used in the complex treatment of leprosy? a) dapsone; b) ampicillin;

c) rifampicin; d) lampren (clofazimine); e) ofloxacin.

7. Patients with lepromatous type of leprosy should receive continuous etiotropic therapy for at least: a) 6 months; b) 1 year; c) 9 months; d) 2 years; e) 6 years. Sample answers: 1 g; 2 b; 3 in; 4 d; 5 in; 6 b; 7 y.

Tests for knowledge control

1. The causative agent of cutaneous leishmaniasis are: a) viruses; b) bacteria; c) protozoa;

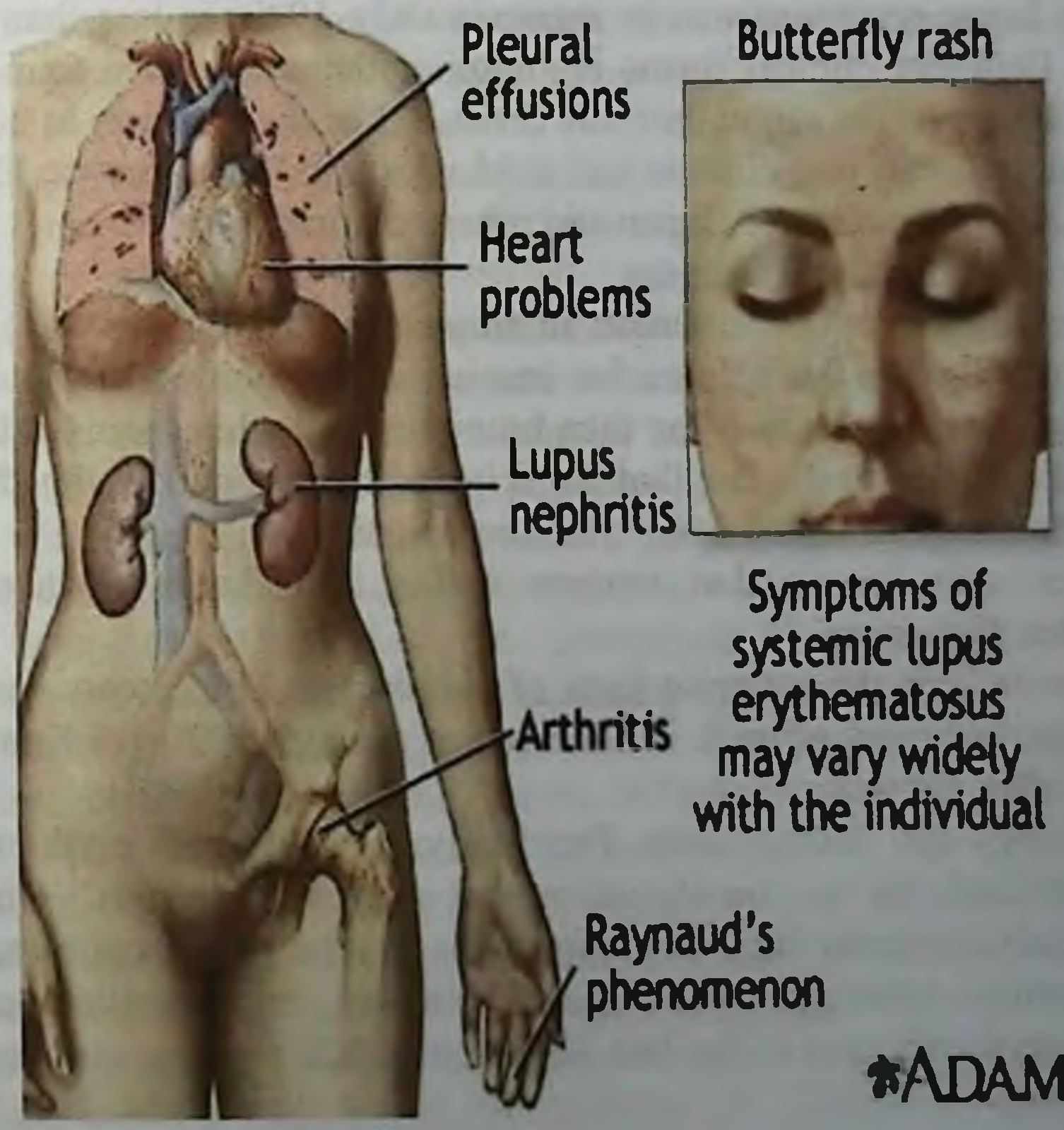
d) mushrooms; e) spirochetes.

2. The rural type of leishmaniasis is characterized by: a) an incubation period of 3-6 months or more; b) long course (7–10 months or more); c) only the lower extremities are affected; d) the ulcer begins to scar from the peripheral edge; e) strong immunity remains.

3. The urban type of leishmaniasis is characterized by: a) a short incubation period (up to 6 weeks); b) rapid evolution of precipitous elements; c) violation of the general condition of the patient; d) slow course (up to 1 year); e) high efficiency of penicillin therapy.

4. For the treatment of cutaneous leishmaniasis, three of the following drugs are used: a) monomycin, penicillin, chingamine; b) doxycycline, solusurmin, biseptol; c) ampicillin, glucantim, tavegil; d) penicillin, sulfadimethoxine, delagil; e) solyusurmin, monomycin, delagil.

Sample answers: 1 in; 2 d; 3 g; 4 days



CHAPTER 2 COLLAGENOSIS: LUPUS ERYTHEMATOSUS, DYSCHROMIA, SCLERODERMA, DERMATOMYOSITIS, VITILIGO, CHLOASMA, LENTIGO.

Lupus erythematosus

Lupus erythematosus (**scarring erythematosus**) Lupus erythematosus (lupuserythematodes; erythematodes) belongs to the group of connective tissue diseases - collagenoses. It is characterized by a long and persistent course, a variety of clinical manifestations. Refers to infrequently occurring diseases.

The number of patients with lupus erythematosus is a small proportion of the number of primary skin patients. The incidence of systemic lupus erythematosus is approximately 10 times less than that of discoid. Different clinical forms of lupus erythematosus are found in all climatic zones of the globe, but this disease is more common in countries with a humid maritime climate and cold winds: in Scandinavia, England, northern Germany, Greece, Japan and other countries.

Pic- 9 Lupus erythematosus

The disease is less common in tropical countries: in Brazil, Syria, Egypt, although in these countries insolation is significant. Blondes get lupus erythematosus more often than brunettes, and whites more often than blacks. This is due to the fact that the skin and body of dark-skinned people are more reliably protected from radiation irritation.

It is well known that women suffer from lupus erythematosus more often than men,

especially in the systemic form of the disease. The disease can begin at any age, but most often it is observed at the age of 20–40 Knowledge workers often get sick.

Etiology and pathogenesis. Previously, discoid lupus erythematosus was described as a manifestation of skin tuberculosis. However, subsequently, systemic lupus erythematosus was no longer correlated with the tuberculous etiology of this disease. However, the tuberculous etiology of lupus erythematosus in the late XIX - early XX century. enjoyed great

popularity, especially among dermatologists of the Viennese and French schools, but then this theory was rejected.

50 theories of syphilitic, streptococcal and viral etiology of lupus erythematosus also turned out to be untenable. The most modern theory of the autoallergic genesis of lupus erythematosus, linking the development of the disease with the occurrence of a special specific sensitization to deoxyribonucleic acid, which is formed during the breakdown of cellular proteins.

The antibodies to deoxyribonucleic acid that appear in the body of patients are actually autoantibodies directed against various components of the endothelium, which leads to damage to the skin, joints, serous membranes, renal glomeruli, blood cells and other formations of endothelial origin.

An example of such autoantibodies is the so-called lupus erythematosus factor, discovered by Khazerik, an antibody against leukocyte nuclei, the action of which owes its origin to LE cells. Like any antibody, lupus factor is a gamma globulin. It is assumed that the antigen-antibody reaction occurs in the connective tissue, which leads to its pathological changes.

Thus, altered immunological reactivity - autoimmune disorders - is of decisive importance in the development of the disease. The weak point of this theory is the uncertainty of various factors that cause the production of autoantibodies. In the light of the autoallergic theory, the role of various factors, which are often taken as causative factors, becomes more definite.

Chronic infections, endocrinopathies, pregnancy, cardiovascular lability and other diseases are conditions that contribute to the development of the disease, but are not necessary for its occurrence. Often, the appearance of clinical symptoms of the disease is provoked by solar insolation, cooling, cold wind, acute infections, mental and physical trauma, vaccination, childbirth, drug intolerance, etc.

All of these listed factors belong to the category of resolving factors that are important as a trigger in the development of the disease. Of particular importance in the development of skin manifestations of erythematosis is given to local trauma, primarily to sunlight, the influence of which explains the usual localization of rashes in open places,

exacerbation of diseases in the spring and summer, and its frequent onset after intense insolation.

An important role in the pathogenesis of **erythematosis,** in addition to radiation, can be

played by any other injury - mechanical, thermal, chemical. Cases of the appearance of lupus erythematosus foci at the site of surgical wounds, bruises, burns with hot oil, ashes, boiling water, on the scar after vaccination, at the site of X-ray therapy, erysipelas, vesicles and herpes zoster, insect bites, after surgical injuries, chemical burns, drug dermatitis, after 51 extrusion of acne, after the use of exfoliating ointments for the treatment of freckles and other traumas.

Cases of the development of the clinical picture of systemic lupus erythematosus after the administration of penicillin, streptomycin, tetracycline, griseofulvin, sulfonamides, sera and vaccines are described. Exacerbation of various clinical forms of lupus erythematosus can be triggered by pregnancy and childbirth.

The disease of lupus erythematosus is possible at the same time by several members ofthe same family. Sometimes there are two or more patients in the family, which is explained by a number of hereditary or acquired factors that predispose to the disease and are characteristic of this family.

Classification. It is considered the most acceptable division of lupus erythematosus into **discoid, intermediate** (disseminated lupus erythematosus and centrifugal erythema) and **systemsic** (acute, subacute and chronic) forms.

There are the following forms of lupus erythematosus:

1. **Chronic** (chronic scarring erythematosis).
2. **Acute, or systemic** (acute erythematosis), in which not only the skin is affected, butalso the internal organs.
3. **Subacute** (subacute erythematosis), characterized by less severity of clinical symptomsthan acute.

In addition, there are clinical varieties of the chronic form of erythematosis: **Bietta's discoid, disseminated, centrifugal erythema** and **the deep form of Kaposi-Irgang's lupus erythematosus.**

Discoid lupus erythematosus In the initial stage of the disease on the nose or on the cheeks, less often in other places, one or two small spots appear, the size of a lentil or more. Subjectively, they do not bother. Then the erythematous focus increases due to peripheral growth, infiltration and hyperkeratosis develop, and over time, skin atrophy.

In the developed focus of discoid lupus erythematosus, erythema, infiltration, hyperkeratosis and atrophy are observed. Erythema is constant, and at the beginning ofthe disease and during exacerbation - the leading sign of lupus erythematosus. The color of erythema is pink, sometimes with a bluish tint.

With exacerbation against the background of erythema, **petechial hemorrhages** may occur, and on the periphery of the foci there are separate dilated capillaries (telangiectasias). Over time, inflammatory phenomena, in particular infiltration, decrease in the center of the focus and atrophy develops, a pronounced infiltrated ridge covered

with small scales remains along the periphery of the focus.

The reaction from the epidermis is most characteristic of lupus erythematosus - this is increased keratinization - **hyperkeratosis.** On an erythematous background, whitish small dry scales appear, firmly associated with 52 hair follicle orifices. When the scales are removed, spines are found on their lower surface - this is nothing more than horny plugs extracted from the mouths of the follicles.

These spikes are called the **"symptom of a lady's heel."** In place of the extracted spines, the funnels of the mouths of the follicles are exposed. That is, in the focus of discoid lupus erythematosus there is follicular hyperkeratosis. When scraping the scales, soreness is noted. This is a symptom of Besnier-Meshchersky. Skin atrophy gradually develops.

The pattern of the skin disappears, it becomes thinner, easily folded, sometimes hyperpigmented along the periphery of the focus. The stronger the infiltration was, the greater the subsequent atrophy, up to the formation of depressed disfiguring scars.

Cicatricial atrophy develops especially rapidly in the foci located on the scalp. Hair in these places falls out and is no longer restored. On atrophied skin, relapses of lupus erythematosus may occur. Very rarely, cancer develops on old scars. The characteristic signs of discoid lupus

erythematosus - erythema, infiltration, hyperkeratosis and atrophy - are usually observed simultaneously in the same focus.

However, at the beginning of the disease, erythema prevails, and with the attenuation of the process, atrophy. In the lesions, three zones are distinguished. In the center - a zone of cicatricial atrophy, then a zone of hyperkeratosis, along the periphery - a zone of hyperemia. The most common localization of foci of discoid lupus erythematosus is the skin of the face.

Especially often the nose and cheeks are affected, that is, the places most exposed to the action of wind, cold and sunlight. Here, the contours of the hearth are often compared with the shape of a butterfly with outstretched wings. The lesion of the skin of the nose represents the body, and on the cheeks - the wings of a butterfly.

Much less often, foci of discoid lupus erythematosus are observed on the auricles, on the forehead, on the scalp, on the red border of the lips and on the oral mucosa. Relatively rarely, foci of discoid lupus erythematosus appear on the neck, chest, back, on the dorsum of the hands, palms, soles, in the genital area and armpits.

In most cases, the foci of this localization are combined with a lesion of the facial skin. An isolated lesion of the oral mucosa and genital organs is described. The affected red border of the lips looks as if smeared with collodion, which cracks and peels in places, dilated capillaries are visible on the periphery of the focus. Attempts to remove the scales are accompanied by soreness and bleeding.

On the red border of the lips, three forms of chronic focal or discoid lupus erythematosus are distinguished: typical; without clinically pronounced atrophy and hyperkeratosis;

erosive and ulcerative. With a typical form of lupus erythematosus, foci of oval outlines form on the red border of the lips.

The surface of the lesions is dry, purple-red in color, with persistently dilated vessels and a clearly defined infiltrate. The foci are covered with an even coating with densely set scales, the forcible removal of which leads to bleeding and significant pain. In the center of the lesion, atrophy of the red border of the lips is noted.

Histopathologically, with a typical form in the epithelium, **hyperkeratosis, acanthosis,** vacuolar degeneration of the basal layer, and in some places pronounced atrophy are determined. In the mucous membrane, a diffuse inflammatory infiltrate, a sharp expansion of blood and lymphatic vessels with perivascular infiltration from lymphocytes, histiocytes and plasma cells, destruction of collagen and elastic fibers are detected.

The form of lupus erythematosus without clinically pronounced atrophy and hyperkeratosis is characterized by the appearance of **diffuse congestive hyperemia** on the red border of the lips with the formation of scales on the surface, which peel off quite easily. There is no marked hyperkeratosis.

The pathohistological picture is characterized by alternating areas of acanthosis and thinned epithelium, a significant diffuse infiltrate, and a sharp vasodilation. The erosive- ulcerative form of lupus erythematosus on the red border of the lips is characterized by pronounced inflammation, swelling, the appearance of bright red foci, erosions and cracks covered with bloody-purulent crusts.

Patients usually complain of burning and itching of the skin. After healing, atrophic scars remain at the site of the lesions.

Differential diagnosis is carried out with **leukoplakia, lichen planus, actinic cheilitis, abrasive precancerous cheilitis.** There are cases when long-existing foci of lupus erythematosus, especially on the red border of the lips, which were more often irritated, were transformed into cancer. Foci of lupus erythematosus on the oral mucosa usually develop in the area of \u200b\u200bclosing teeth, on the hard or soft palate, on the tongue.

Foci are often manifested only by thickening and clouding of the epithelium, less often there are erosions or even superficial ulcerations. The edges of the foci are clearlydelimited, slightly raised, grayish in color.

Keratoconjunctivitis has been described. With deep lupus erythematosus Kaposi - Irganga infiltrates penetrate deep into the subcutaneous fatty tissue, sometimes they ulcerate. Rare pigmented, mutilating, pellagroid and hemorrhagic variants of lupus erythematosus are described.

Subjectively, most patients with **discoid lupus erythematosus** have itching and some are quite severe. Sometimes there is pain in the joints. Some patients have subfebrile temperature or various abnormalities in the blood, the functions of various organs and

systems.

Disseminated lupus erythematosus Skin rashes in the disseminated form of the disease are manifested by numerous, usually small foci such as discoid lupus erythematosus. They are located on the face, sometimes on the scalp, neck, upper chest, rarely in other places. The boundaries of the foci are clear. In addition to discoid-type foci (with hyperemia, infiltration, hyperkeratosis and atrophy), patients with disseminated lupus erythematosus quite often develop erythematous foci with a bluish tint, without pronounced infiltration and hyperkeratosis on the fingers and toes, in the area of the knee and less often elbow joints.

Cyanotic spots usually appear during an exacerbation of the process. Thus, rashes in disseminated lupus erythematosus are more or less similar in nature to rashes in the systemic form. With disseminated lupus erythematosus, common phenomena are observed more often than with discoid: low-grade fever, anemia, leukopenia, increased ESR, hypergamma globulinemia, traces of protein in the urine, Raynaud's phenomenon.

Half of the patients complain of pain in the joints, sometimes with deformity of the latter. In patients, a focal infection is detected (**tonsillitis, chronic tonsillitis, dental diseases, otitis media, sinusitis**, etc.). Under the influence of positive or negative factors, the disseminated form can be transformed into the discoid or systemic form of the disease, respectively.

Disseminated lupus erythematosus is considered a transitional form between discoid and systemic lupus erythematosus. Therefore, patients with disseminated lupus erythematosus require a deep examination and close systematic monitoring by doctors. Centrifugal erythema Centrifugal erythema - this form, first described by Biett, is called **Biett's form.** In the centers of centrifugal erythema there is no infiltration, no hyperkeratosis, noatrophy.

A typical picture of centrifugal erythema is characterized by the development of diffuse, somewhat edematous pink erythema, capturing the back of the nose and symmetrically on both cheeks in the form of a

"butterfly". Sometimes erythema is located only on the cheeks or on the back of the nose. In some patients, a slightly noticeable peeling is observed on the surface of the erythema, and after the disappearance of the erythema, a slight atrophy remains.

In patients with centrifugal erythema, subfebrile temperature, joint pain, erythematous rashes with a bluish tint on the phalanges of the fingers and knees, increased ESR, etc. are often observed. lupus. Practically, centrifugal erythema may be a precursor or manifestation of **systemic erythematosis.**

Patients with **centrifugal erythema** require a careful and in-depth examination, an appropriate regimen and strict follow-up.

Histologically, in chronic forms of lupus erythematosus in the epidermis, there is

follicular hyperkeratosis, vacuolar degeneration and atrophy of the cells of the basal layer. In the dermis - proliferation of cellular elements, edema and degeneration ofcollagen fibers. Systemic lupus erythematosus Develops spontaneously or during exacerbation of chronic erythematosis.

It can be acute, subacute and chronic. In the midst of acute lupus erythematosus, polymorphism of symptoms of damage to many organs and systems is characteristic. A variety of symptoms are conditionally divided into three groups: 1. General phenomena ofa sharply disturbed metabolism (fever, fatigue, general weakness to complete prostration in advanced cases, weight loss).

2. Diffuse damage to the connective tissue (fibrinoid degeneration of collagen) and as a result - damage to the serous membranes, **arthralgia, arthritis, pleurisy, pericarditis, endocarditis,** less often peritonitis); damage to the walls of blood vessels and, as a result, damage to internal organs, the central nervous system and skin (**nephritis, hepatitis,** gastrointestinal crises, enlargement of the spleen and lymph nodes, **phlebitis, Raynaud's phenomenon**, neuropsychiatric symptoms, various rashes, including hemorrhagic skinand mucous membranes, in rare cases - gangrene of the extremities).

3. Hematological changes: **anemia, leukopenia** (less often leukocytosis), **thrombocytopenia,** significantly increased ESR, **hypergamma globulinemia, hypocoagulation,** positive LE phenomenon,

sometimes nonspecific Wasserman reaction, Vidal, etc. It should be borne in mind that only in severe and advanced cases of systemic lupus erythematosus affect all or almost all of the above organs and systems (polysystemic lupus erythematosus).

At the onset of the disease, as well as in mild atypical cases, one or two systems can be affected, the clinical picture can be dominated by damage to only one of any organ, forexample, **nephritis, psychosis,** etc. (i.e., monosymptomatic lupus erythematosus). Somepeople with systemic lupus erythematosus may not have skin rashes. Joint damage - arthralgia or arthritis - is an almost constant companion of systemic lupus erythematosus. They are observed in almost all patients. In more than half of the patients, they are the first 56 sign of the disease. In some patients, joint pain appears months and even yearsbefore the development of a clear picture of systemic lupus erythematosus.

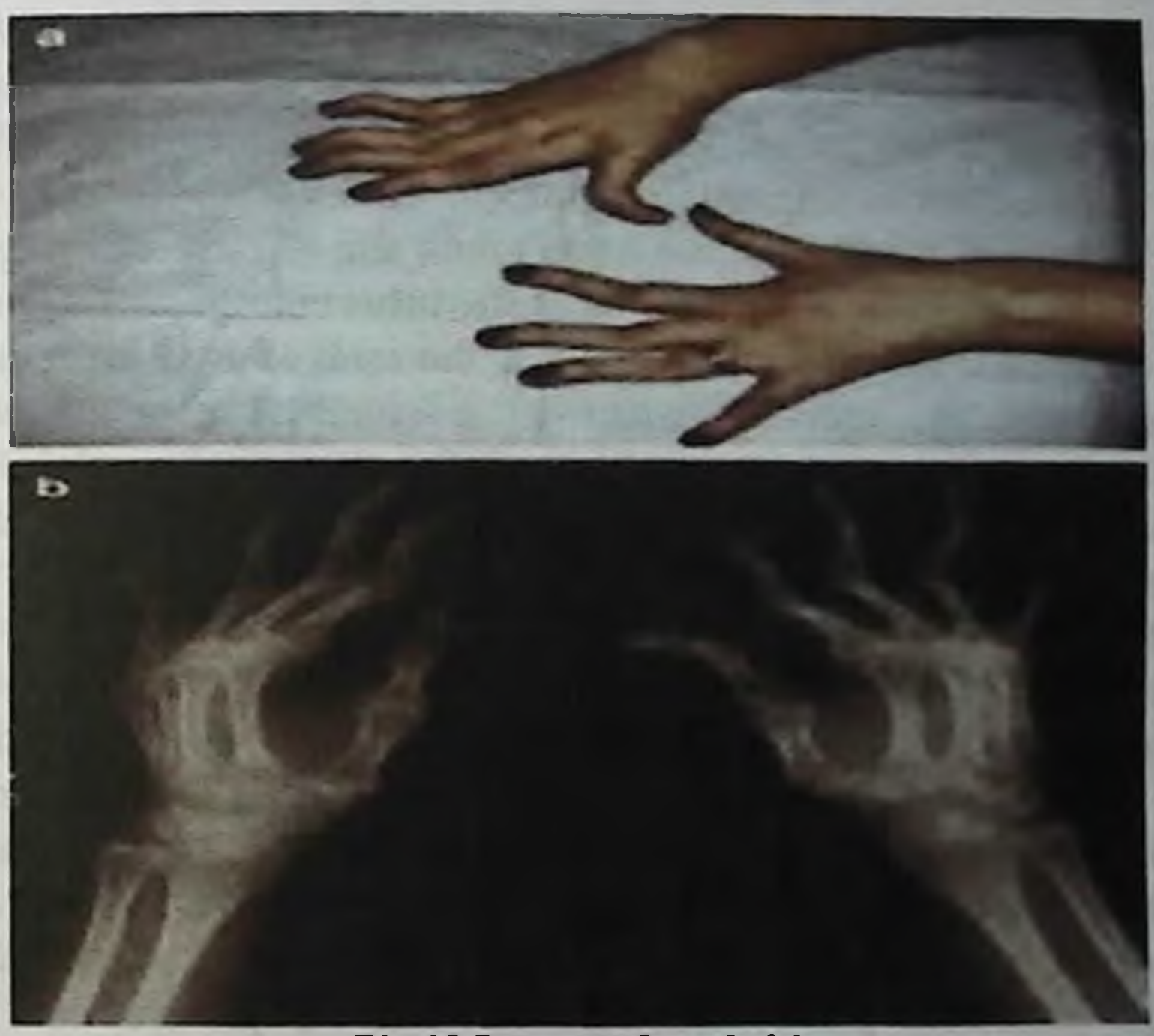
However, more often joint pains appear simultaneously with fever, skin rashes and other manifestations. First, the joints of the fingers are affected, and later other larger ones.

Lupus polyarthritis is no different from rheumatoid arthritis. Muscle damage. **Myalgia, myositis** and subsequently muscle atrophy are observed in 30-50% of patients with systemic lupus erythematosus. As a rule, myalgias develop simultaneously with arthralgias. Heart failure is observed in almost half of the patients. The endo-, myo- and pericardium may be affected, and the conduction of impulses may also be impaired.

There is an unpleasant sensation in the region of the heart, pain, tachycardia, systolic murmur at the apex, an increase in the boundaries of the heart, etc. The lungs and pleura are affected in about half of the patients. **Pleurisy** is sometimes even the first symptom of systemic lupus erythematosus, most often dry pleurisy. In cases of exudative pleurisy, there is usually little fluid.

Clinically, lupus lesions of the respiratory system are manifested by chest pain, cough, shortness of breath, sometimes hemoptysis, and other clinical and radiological symptoms. The lupus character of the lesion should be suspected in all cases of torpid, unexplained bronchopneumonia, especially if it recurs in the lower parts of the lungs and is resistant to antibiotic therapy.

Damage to the skin and mucous membranes in systemic lupus erythematosus is neither leading nor mandatory. With the discovery of the LE phenomenon, cases of systemic lupus erythematosus without skin rashes (lupus erythematosus without lupus erythematosus) are increasingly being identified.



Pic-10 Lupus polyarthritis

However, skin manifestations were found in almost all patients with systemic lupus erythematosus. Skin rashes in most patients with systemic lupus erythematosus have the following character: on the nose and on both cheeks there is a fairly symmetrical redness resembling the shape of a butterfly, essentially no different from centrifugal erythema. The more acute the course of lupus erythematosus, the more pronounced the swelling of

the skin in the area of erythema and the less clear the boundaries.

At first, the erythema has a pink character, then it becomes cyanotic, sometimes the surface is slightly flaky. With the disappearance of redness,

pigmentation and a slightly noticeable atrophy of the skin remain. Rashes can be on the face, neck, upper chest. On the open parts of the chest, erythema has the shape of a triangle, the boundaries of which resemble the Latin letter V. In very acute cases, the skin of the face, especially around the eyes, swells.

On the fingers, in the area of the nail ridges, cyanotic exudative spots or nodules sometimes appear. Occasionally, **erythema and skin atrophy** occur on the soles and palms. There are also hemorrhagic rashes, 57 rashes in the form of **urticaria, polymorphic or nodular erythema, herpes zoster;** rashes on the skin of the knee joints and legs, resembling chills, **chronic trichophytosis** and **papulonecrotic tuberculosis.**

Rashes on the mucous membranes in the form of erythematous spots, petechiae, vesicles, erosions occur in a small number of patients. Gastrointestinal symptoms are observed in almost all patients (poor appetite, coated tongue, nausea, vomiting, flatulence, abdominal pain, diarrhea, sometimes with an admixture of blood).

The cause of gastrointestinal disorders in systemic lupus erythematosus is damage to the walls of the vessels of the digestive tract, high temperature, general intoxication and damage to the central nervous system matter. In some cases, gastrointestinal crises are an early and dominant symptom of the disease, they can simulate **appendicitis, perforated gastric ulcer,** etc.

In rare cases, more severe lesions of the gastrointestinal tract are observed: **intestinal perforation, paralytic ileus, pancreatitis,** which endanger life. The liver is often affected. The state of protein fractions and sedimentary reactions of blood plasma is disturbed, changes in the pigment and antitoxic function of the liver are less common. Often the liver increases in size. The kidneys in systemic lupus erythematosus are affected in most patients.

Protein, erythrocytes, leukocytes, cylinders appear in the urine, as well as edema and other signs of kidney damage. **Glomerulonephritis, nephrosis, nephroso-nephritis** develop. In severe cases, **hypertension, edema, uremia,** and other symptoms of kidney failure develop.

Damage to the nervous system in systemic lupus erythematosus is manifested by a number of mental and neurological symptoms. In the

severe course of the disease, memory loss, depression, irritability, mental agitation, reaching psychoses with visual and auditory hallucinations, up to paranoid ideas, are noted.

Sometimes systemic lupus erythematosus begins with psychosis and patients, first of all, end up in psychiatric hospitals. Sometimes lupus erythematosus only provokes psychosis in patients who have a history of mental illness. There are various neurological

symptoms.

Often there is damage to the eyes, especially the retina. There are also **conjunctivitis, corneal ulcers, angiopathy of the fundus vessels.** The defeat of large vessels sometimes causes gangrene of the legs. Raynaud's phenomenon, nosebleeds (due to vascular damage) are often stated. Hematological changes are observed in all patients (anemia, leukopenia even at high temperature, which is important for differential diagnosis, sometimes leukocytosis, thrombocytopenia with hemorrhages, increased ESR, etc.).

With subacute lupus erythematosus, the severity of the process is less pronounced. The diagnosis of systemic lupus erythematosus is based on clinical and paraclinical parameters, with the detection of LE cells being of great importance. A typical LE cell is a mature polynuclear neutrophil (rarely an eosinophil or a monocyte), the nucleus of which is shifted in the form of a crescent to the cell periphery, and its center is occupied by a phagocytosed homogeneous mass of a round shape, consisting of phagocytosed and homogenized nuclei of disintegrated leukocytes. The composition of the phagocytosed mass includes deoxyribonucleic acid.

The LE cell is much larger than the normal leukocyte. Thus, the characteristic features of the LE cell are: 1) an increase in its size compared to normal leukocytes; 2) the homogeneity of the included mass within it; 3) differences in staining intensity between the lighter phagocytosed mass in the center of the cell and the more intensely stained and retracted to the periphery nucleus.

In the preparation, along with LE cells, **so-called rosettes** are sometimes seen. A rosette is a conglomerate of leukocytes located concentrically (due to bio- and chemotropism) around a lump of

homogeneous smoky mass. This mass is the homogenized nuclei of decayed leukocytes, contains deoxyribonucleic acid and, thus, is identical to the homogeneous mass of the LE cell. Some of the leukocytes can phagocytize the homogeneous mass and become LE cells.

LE cells and rosettes are the result of a pathological process - **nucleolysis.** Detection of LE cells is a pathognomonic sign of systemic lupus erythematosus. It serves as a formidable signal, indicating that protein compounds with antigenic properties have appeared in the patient's plasma (i.e., compounds to whose presence the body reacts as ifit were a foreign protein), and that a life-threatening nucleolysis process is taking place in the patient's body, which is an essential phase in the formation of LE cells.

Histologically: in the epidermis, vacuolar degeneration and atrophy of the cells of the basal layer, and in the dermis, leukocyte infiltration of the papillary and subpapillary layers. The main change is fibrinoid degeneration of the collagen substance, which is better expressed in the collagen of the internal organs, but is also found in the skin.

Treatment. Since various pathogenetic factors play a certain negative role in all forms of lupus erythematosus, it is necessary to examine the patient before starting treatment and,

if necessary, conduct appropriate pathogenetic therapy. Foci of focal infection, functional disorders of the gastrointestinal tract, endocrine, nervous and other systems are subject to treatment. Patients should stop smoking and drinking alcohol.

It is necessary to avoid taking medications that can cause allergic reactions and exacerbate the course of the underlying disease (antibiotics, sulfonamides, sera, vaccines, etc.). Protection from any trauma, radiation exposure, heat, frostbite, chemical and mechanical irritants, as well as mental trauma is important. Patients should be advised in sunny weather, especially in spring, to wear wide-brimmed hats, umbrellas, dark glasses, walk along the shady side of the street, and not use cold water and caustic soap to wash their faces.

Patients whose work is associated with prolonged exposure to the sun, cold, wind, should be transferred to work in closed warm rooms.

Patients should avoid drugs that increase skin sensitivity to ultraviolet rays (cologne, tar preparations, eosin, sulfonamides, etc.).

Protective ointments containing 5% quinine and salol (chininihydrochlorici, Phenyliisalicyliciaa 0.5; lanolini, aquaedestillataeaa 1.0; vaseliniad 10.0), 5–10% paraaminobenzoic acid (Ac. paraaminobenzoici, lanolini, aquaedestillataeaa 1.0; vaseliniad 10.0). Protective creams "Ray", "Shield", "Spring", "From sunburn", "Achromin", etc. For the treatment of chronic lupus erythematosus, antimalarial drugs (Aminoquinol, Amodiquin, Plaquenil, Chingamine, Rezokhin, Chloroquine, Delagil) are widely used.) at a dose of 0.25 g twice a day in cycles of 5–10 days with interruptions of 2 days and 5 days, depending on the clinical picture.

Preparations of the chloroquine series stimulate the activity of the adrenal cortex, affect the metabolism in the connective tissue, and have a pronounced photohyposensitizing effect. Corticosteroids are used to treat systemic lupus erythematosus. Prednisolone at a daily dose of 1 mg per 1 kg of body weight (for an adult, the daily dose is 50-80 mg).

Treatment with corticosteroids begins with loading doses, and after obtaining a therapeutic effect, the dose is gradually reduced to maintenance. Corticosteroids do not cure lupus erythematosus, but they quite reliably suppress life-threatening processes of autoaggression and thus help to save the life of patients, and often the ability to work for an indefinitely long time.

Thus, corticosteroid therapy allows you to gain time, during which persistent, prolonged, spontaneous remissions of the disease are possible. Corticosteroids are unconditionally indicated in patients with acute, severe systemic lupus erythematosus. Immediate treatment with sufficient doses of corticosteroids saves such patients from imminentdeath.

In severe cases, corticosteroid therapy is combined with cytostatics. Corticosteroids are also indicated for patients with a subacute course of the disease, if the kidneys, heart, central nervous system and other important organs are affected early.

Treatment of mild chronic or subacute systemic lupus is essentially the same as treatment of discoid and intermediate forms of lupus erythematosus. Treatment with vitamins (C, B1, B2, PP, B5, B6, B12,

B15) is of great importance. Vitamins increase the tolerability of corticosteroids and prevent some side effects of corticosteroid therapy.

Long-term use of corticosteroids causes the loss of calcium and potassium salts and sodium retention. Therefore, to prevent and treat **osteoporosis and myasthenia gravis,** as well as to improve protein synthesis, anabolic hormones (nerobol, methylandrostenediol, etc.) are prescribed. Potassium and calcium salts are also prescribed.

For local therapy, in some cases, corticosteroid ointments (mometasone furoate) are used. The prognosis of systemic lupus erythematosus is always serious, especially in patients with lupus nephritis.

The most common causes of death in patients with systemic lupus erythematosus are: 1) progressive nephropathy with symptoms of kidney failure; 2) vasculitis of the central nervous system, accompanied by hemorrhages, paralysis, convulsions, psychosis and other disorders; 3) secondary infection (before the advent of antibiotics - the main cause of death of patients). All patients with lupus erythematosus are on dispensary records.

SCLERODERMIA

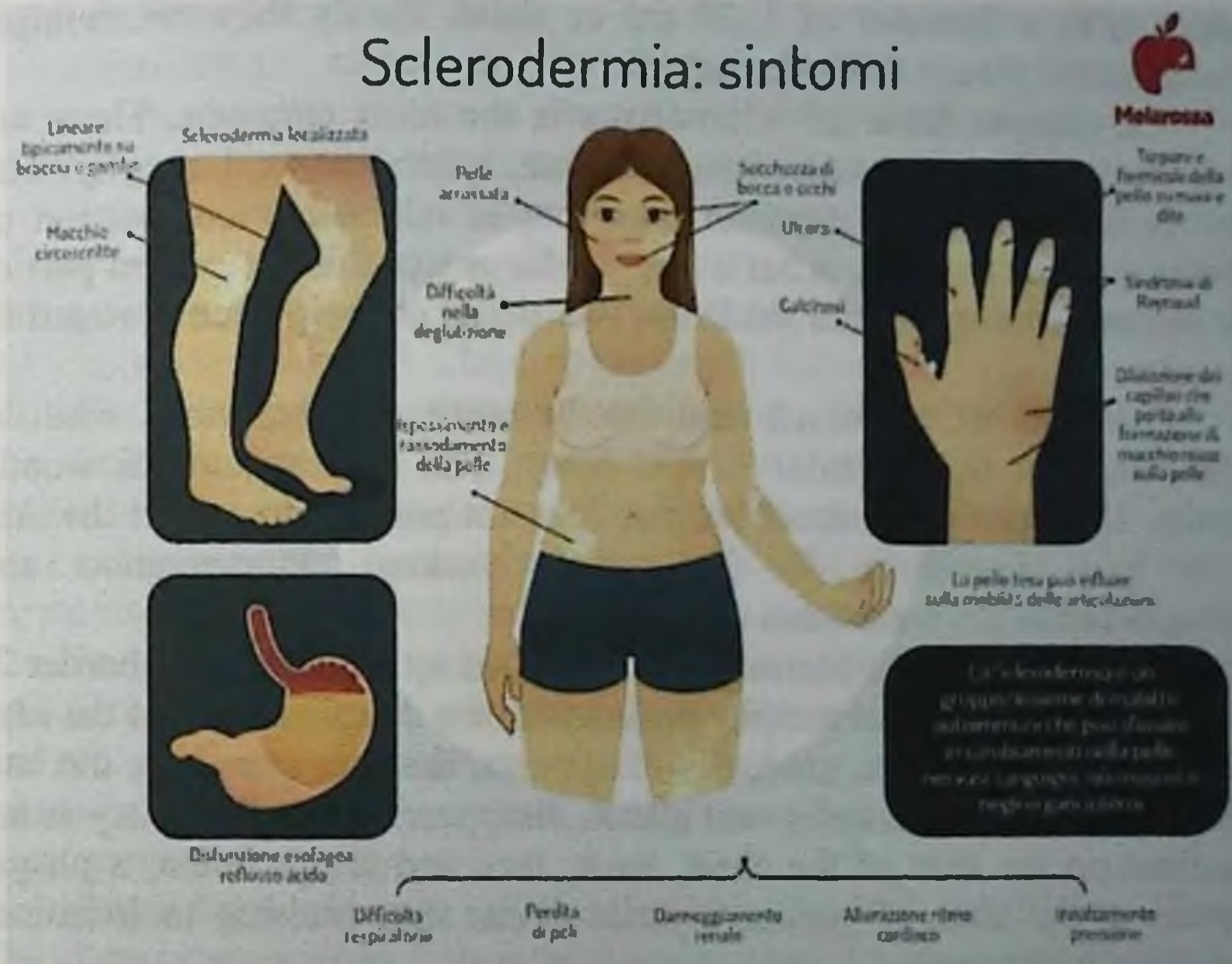
This is a group of dermatoses characterized by focal or diffuse thickening and thickening of the skin (and in some forms, subcutaneous tissue), resulting from inflammatory dystrophic processes in the connective tissue. These diseases belong to the group of collagenoses.

Epidemiology. It occurs most often between the ages of 30 and 50, and women are four times more likely to be affected than men. Etiology and pathogenesis. Scleroderma sometimes occurs after a severe injury to the central nervous system, after hypothermia, sometimes there is a connection with acute and chronic infectious diseases (**rheumatism, diphtheria, tuberculosis, typhoid fever, syphilis,** etc.), various kinds of toxicosis, severe mental illness.

Scleroderma has an infectious origin. The pathogenesis of scleroderma is closely related to the endocrine-vegetative system. The importance of hyper- or hypofunction of the parathyroid glands, which is associated with a violation of lime metabolism, is attached. Scleroderma

most often affects women. Patients present with **amenorrhea or dysmenorrhea.**

Basal metabolism in scleroderma can be disturbed, more often upwards.The pathogenesis is based on the disorganization of cellular and humoral immunity associated with the thymus-dependent system (T-



Pic-11 Scleroderma

lymphocytes are thymus-dependent, carrying out a cellular type of immunity, and B-lymphocytes are thymus-independent, carrying out humoral immunity).

However, the theory of auto-aggression has not yet been finally proven. With scleroderma, as with other collagenoses, collagen, elastic and reticular fibers and interstitial substance, cells, blood vessels and nerve endings suffer. Scleroderma occurs in both children and adults. Clinically, there are limited (sclerodermiacircumscripta) and diffuse (progressive,

generalized), or systemic (sclerodermiadiffusa, seu progressiva), scleroderma.

Limited scleroderma includes plaque, linear (strip-like), punctate (white spot disease), superficial (Pasini-Pierini idiopathic atrophoderma) forms. Plaque scleroderma is characterized by the presence of one or more lesions with a diameter of 1–20 cm or more. Rarely they are multiple (disseminated plaque scleroderma).

The **plaque form** of scleroderma is the most common. There are three stages of plaque scleroderma: edema, induration, and atrophy. The process begins with an erythematous spot or edematous compaction of certain areas of the skin. After a few weeks or months, the central part of the plaque turns white and thickens. The shape of the plaque is round or irregular.

The color of thickened skin can be white, pearly, waxy, whitish-yellowish, that is, the color of old ivory. The skin acquires a woody density. Her drawing is smoothed out. It is not possible to collect the skin in a fold. Sensitivity in the hearth weakens. Pigmentation and **telangiectasias** are sometimes observed.

Often, foci of scleroderma are surrounded by a lilac or lilac border 2-3 mm wide. The stage of atrophy begins with the disappearance of the lilac ring, then the induration gradually disappears, the skin atrophies, the hair falls out, the sebaceous and sweat glands disappear. More often plaques are localized on the skin of the chest, back, face and scalp. Often, a plaque appears at the sites of various injuries (from skin bruising to irritation, squeezing with a bra).

Linear, or strip-like, scleroderma. This form of localized scleroderma is relatively common, especially in childhood. The average age of patients with linear scleroderma is 7 years. Its most frequent localization is the forehead, and here the process spreads from above, from the scalp, subsequently often goes further to the back of the nose, very reminiscent of a scar formed as a result of a saber strike.

In some cases, linear scleroderma is combined with **facial hemiatrophy**. Less commonly, linear scleroderma occurs on the lower extremities, sometimes with partial ulceration. Sometimes linear scleroderma extends from the 62 buttocks throughout the thigh and lower

leg to the foot, inclusive. In some cases, there is a zosteriform arrangement of linear scleroderma, that is, along the course of the nerves. Sometimes linear scleroderma extends to the oral mucosa.

Extremely rarely, strip-like scleroderma occurs in an annular form (**scleroderma annularis**), the so-called **congenital amputation**. In early childhood, narrow fibrous

constrictions appear on the fingers or toes, less often on the extremities, causing a state of **chronic edema and elephantiasis** in the underlying areas, and then spontaneous amputation.

Annular scleroderma sometimes occurs on the penis. With linear scleroderma, three stages of the course of the disease are also distinguished. White spot disease, or punctate, scleroderma. Mostly adults, especially women, are ill. Children rarely get sick.

The clinical picture resembles white spots the size of a lentil or close to it. Color from chalky (old ivory) to whitish gray with a pearly sheen. Elements of dense consistency with a sunken or raised surface. The skin pattern on the spots is smoothed. In some cases, there is a lilac, reddish-brown ring around the periphery.

In general, the clinical picture is, as it were, a miniature of the manifestations of plaque scleroderma. In other cases, there is no colored rim around the spot (colorless variety). Sometimes, on the contrary, the spots are entirely erythematous in nature, disappearing only with the development of atrophy.

Superficial scleroderma, or idiopathic atrophoderma Pasini - Pierini. The disease most often occurs in females aged 10–20 years. Most often, foci from one to multiple appear on the back and other parts of the body, ranging in size from a small coin to a palm or more. Bluish-violet or brownish-blue with translucent vessels are formed, somewhat sinking plaques, the edges of which can be clear or gradually turning into unchanged surrounding skin. The surface of the lesions is smooth.

The seal at the base of the plaques is completely absent. The peripheral lilac ring is absent in most cases. Sometimes there is pigmentation. In some cases, plaques of atrophoderma subsequently develop typical scleroderma with tissue thickening. Sometimes there are foci of atrophoderma in combination with foci of plaque scleroderma.

This relatively frequent combination testifies to the closest connection of this peculiar clinical form with scleroderma. Apparently, Pasini-Pierini atrophoderma is a type of scleroatrophic process, closer to plaque scleroderma than to primary atrophies.

Diffuse (progressive, generalized), or systemic, scleroderma. This is a severe chronic systemic disease with a progressive course. Adults 20–30 years old, especially women, get sick more often. 63 The disease usually begins suddenly, in some cases after an infectious disease. Many patients have prodromal symptoms of the disease, expressed mainly in vasomotor disorders due to a special sensitivity to cold.

In addition, patients feel unwell, sometimes their temperature rises, joint pains appear, appetite decreases, insomnia develops, depression, etc. Patients lose weight, they develop muscle weakness, and blood pressure drops. The first skin changes occur on the hands and face, as well as on the neck, subsequently the process captures the entire or almost the entire surface of the body. If the disease is limited only to the limbs and face, they speak of **acrosclerosis.**

Clinically, there are three stages. In the first stage, the skin appears diffusely edematous, whitish-yellowish in color. The skin pattern is smoothed. In the second stage, the edema is replaced by a fibrous thickening of the skin, which folds with great difficulty. The process is especially pronounced on the back surface of the hands and feet. The face becomes mask-like, there are many telangiectasias, as well as more or less pronounced **dyschromia** - areas of pigmentation and hypopigmentation.

It is at this stage of the disease, which usually lasts for years, that patients are compared to mummies. This external similarity is especially enhanced in the third stage, when, as a result of sclerotic and atrophic processes, not only of the skin, but also of the deeper tissues of the hands, feet and other parts of the body, the skin fits snugly to the underlying bones.

The movements of patients are limited, any possibility of facial expressions is lost, food intake is difficult, since not only the skin, but also the mucous membranes of the cheeks, tongue, palate, pharynx and esophagus can be involved in the process. All these mucous membranes

are wrinkled, the mouth opening is narrowed to a small size (**microstomia**).

The head sometimes takes on a cranial appearance as all the proper soft tissues and skin atrophy. Often hair falls out, and a large number of telangiectasias appear. The fingers are usually compared with the "**fingers of the Madonna**": they are thin, covered with thinned, matte, ivory-colored skin, which lies completely motionless on the bones. In this case, the fingers are in a bent position, and they cannot be straightened in any way.

Often, small ulcers form on the fingers. **Sclerodactyly** is a form of scleroderma. They talk about it when scleroderma changes on the fingers are especially pronounced. Sometimes here the process ends even with partial mutilation. Fingers and nails are more like claws, they are twisted, lifeless.

Ulceration in diffuse scleroderma occurs almost exclusively on the hands and feet, in very rare cases - on the face (nose, lips). In rare cases, blisters appear on a scleroderma background. Muscles are affected in approximately 30–70% of patients. The skeletal muscles are predominantly affected.

Clinically, they manifest as **myositis, myasthenia gravis, and atrophy**. Degenerative and fibrotic changes can be primary, i.e., arising independently of scleroderma foci, and secondary, developing after deep scleroderma skin changes. **Progressive scleroderma** is a systemic disease of the entire vascular and connective tissue of the body. Indeed, with a thorough examination of patients with diffuse scleroderma, many of them observe certain extracutaneous changes.

According to the literature, visceral lesions can be detected in half of all patients with systemic scleroderma. Damage to the esophagus, gastrointestinal tract, lungs, kidneys, heart, blood vessels, nervous system, bones and joints is described. Histologically, scleroderma is manifested by edema, hypertrophy, homogenization, fibrinoid

degeneration and increased synthesis of collagen fibers, infiltration and atrophy. Diagnosis. In typical cases of scleroderma, diagnosis is not difficult. Sometimes it can be difficult to diagnose in the early stages of

the disease. Sometimes it is necessary to differentiate with pseudoscleroderma of the hands due to rheumatism.

But the latter is characterized by primary damage to the joints, atrophy of some muscle groups, thin and delicate skin, and the absence of scleroatrophic changes in the fingers. In the initial stage of diffuse scleroderma, there is sometimes a resemblance to **dermatomyositis**, but when observing the patient, typical signs of scleroderma appear, and doubts are dispelled.

The prognosis for limited scleroderma is favorable. With a systemic one, it is always serious. Fatal outcomes in systemic scleroderma are more common during the first 5 years of the disease. However, cases of a 20-year course of diffuse scleroderma are known, although the patients outwardly represented really, as it were, **"living relics"**.

Treatment. Patients with scleroderma, both limited and diffuse, should be examined in the most thorough manner in order to identify possible etiological and pathogenetic factors, of which neuro-endocrine disorders occupy the first place. There are no reliable methods of treatment, however, the available means and methods allow in most cases of limited scleroderma to cure it or significantly improve the course of the disease, and in the case of diffuse scleroderma, significantly alleviate the painful process.

For the treatment of all forms of scleroderma, hyaluronidase preparations (lidase, vitreous body) are widely used. The use of lidase is based on a presumed deficiency of hyaluronic acid. With the introduction of lidase, the permeability of blood vessels and connective tissue increases. The drug is used at 64 IU every other day subcutaneously. For a course of 12-15 injections.

You can conduct several courses at intervals of 4-6 weeks. The best effect of lidase has a limited form of scleroderma. 65 Often, with limited scleroderma, the vitreous body improves. Inject 2 ml every other day No. 20 per course. Courses can be repeated after 2- 3 months. There are observations that nicotinic acid enhances the action of lidase. It is advisable to use vasodilators (nikospan, no-shpa, andekalin, etc.).

In the phase of compaction and sclerosis, favorable results can be obtained with prolonged use of large doses of antibiotics (10–15 million units of penicillin per course). Vitamins A, C, PP, B15, E are shown. Against the background of regression, physiotherapeutic procedures are advisable: ultrasound, diadynamic therapy, electrophoresis of lidase, ichthyol, potassium iodide directly into the lesions, therapeutic mud, massage, therapeutic exercises. 3.3.

Scleroderma-like diseases Neonatal scleroma This severe disease begins in the first days of life. There are two forms of scleroma of newborns: **edematous** and **fatty.**

Edematous sclerema begins mostly on the shins, the process quickly spreads higher to

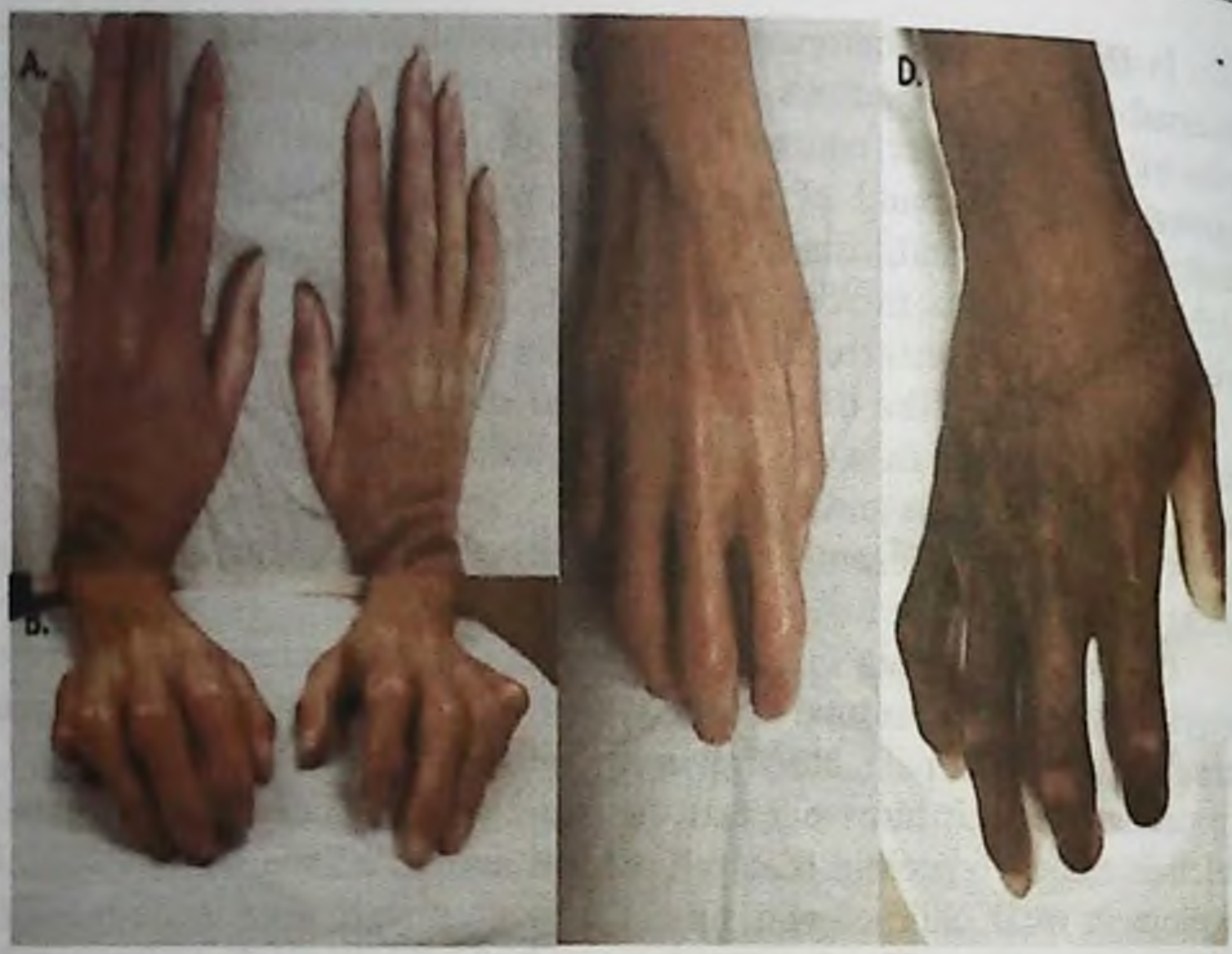
the back, capturing the genital area, and sometimes goes to the face, especially to the periorbital areas. The affected areas of the skin appear densely edematous, pale, blue-red or cyanotic.

At autopsy, it turns out that the entire body is saturated with fluid, which is richer in protein than the fluid in normal edema. Fatty sclera. It develops in weak children with pronounced atrophy, in the first days of life or somewhat later. The localization of the process is the same as in edematous sclerema, but the skin is extremely immobile, dense, tightly stretched, no fossa remains on it under pressure.

The skin color is waxy, sometimes with a cyanotic tint. Adipose tissue is pale, stearin- like consistency. Breathing and cardiac activity are disturbed, severe muscle weakness, **convulsions, drowsiness, pericarditis, peritonitis**, etc. develop. The duration of this form is 2–8 days, then death occurs.

The etiology and pathogenesis of both forms of sclerema remain unclear.

Treatment. Antibiotics, corticosteroids, vitamins, especially A, E, ascorbic acid, rutin. With the introduction of antibiotics and hormones into therapy, the prognosis of both forms of scleroma has become much more favorable. Scleredema of adults Bushke Adults are ill, more often women. There are isolated cases of the disease in children.

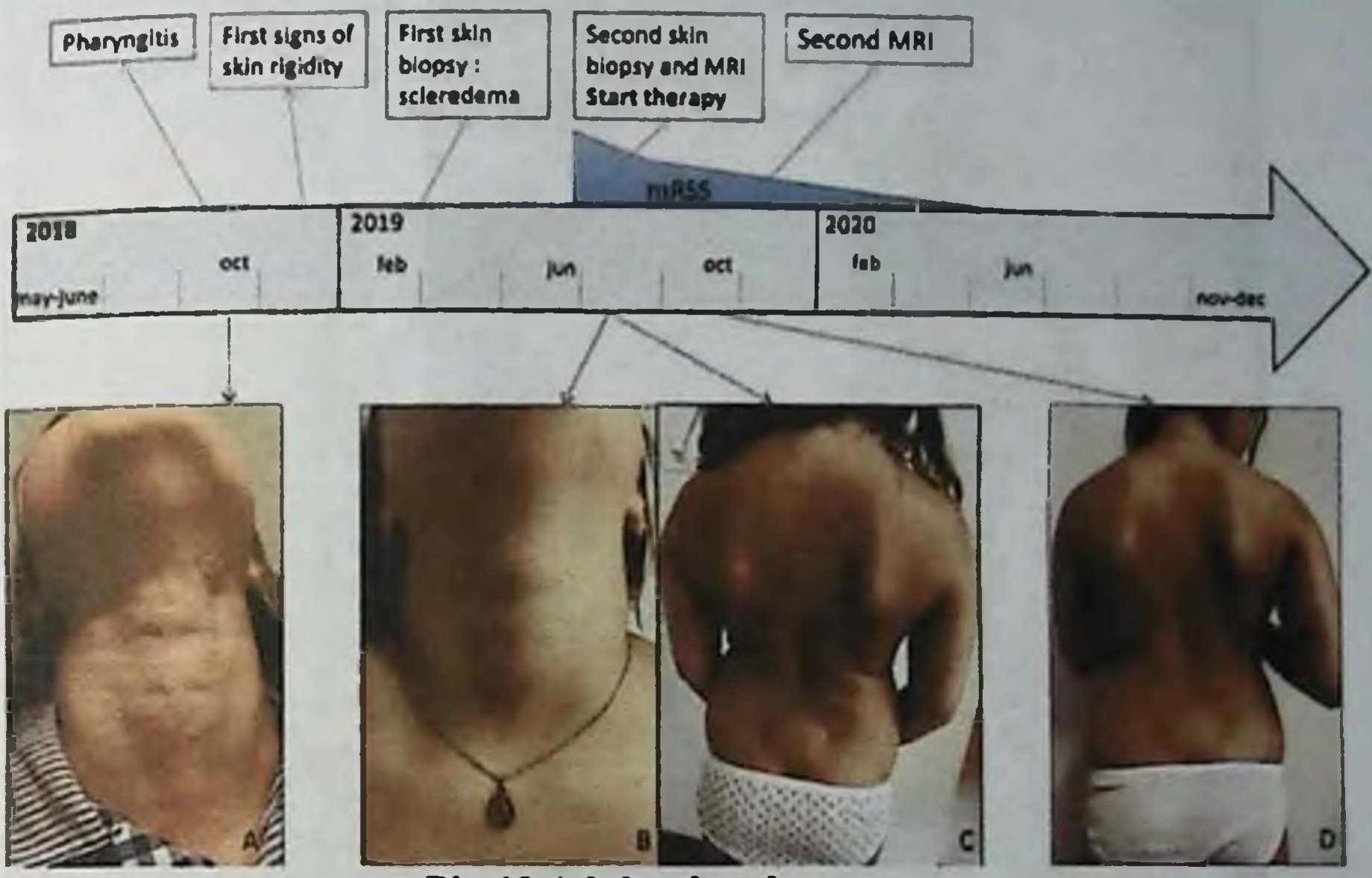


Pic-12 Edematous Sclerema

Adult scleredema almost always begins after some infectious disease, more often in the oral cavity, pharynx, upper respiratory tract, etc. Sometimes after an injury. Clinic. Suddenly there is a very dense swelling of the skin and subcutaneous tissue (paraffin density), first usually on the neck and face, then the process spreads to the shoulders, trunk and limbs.

The density and tension of tissues is noted. At the same time, the skin is smooth, devoid of its normal pattern, and no fossa remains under pressure. The skin is waxy in color, in some cases there is hyperemia. In some areas, **hyperkeratosis** is sometimes observed. The disease lasts 8-18 months, and sometimes lasts for years. The disease is always curable.

Treatment. Elimination of pathogenetic factors (foci of infection), the appointment of vitamins A, E, B15, thermal procedures, ultraviolet irradiation, ultrasound. In some cases, corticosteroids, lidase and antibiotics are used.



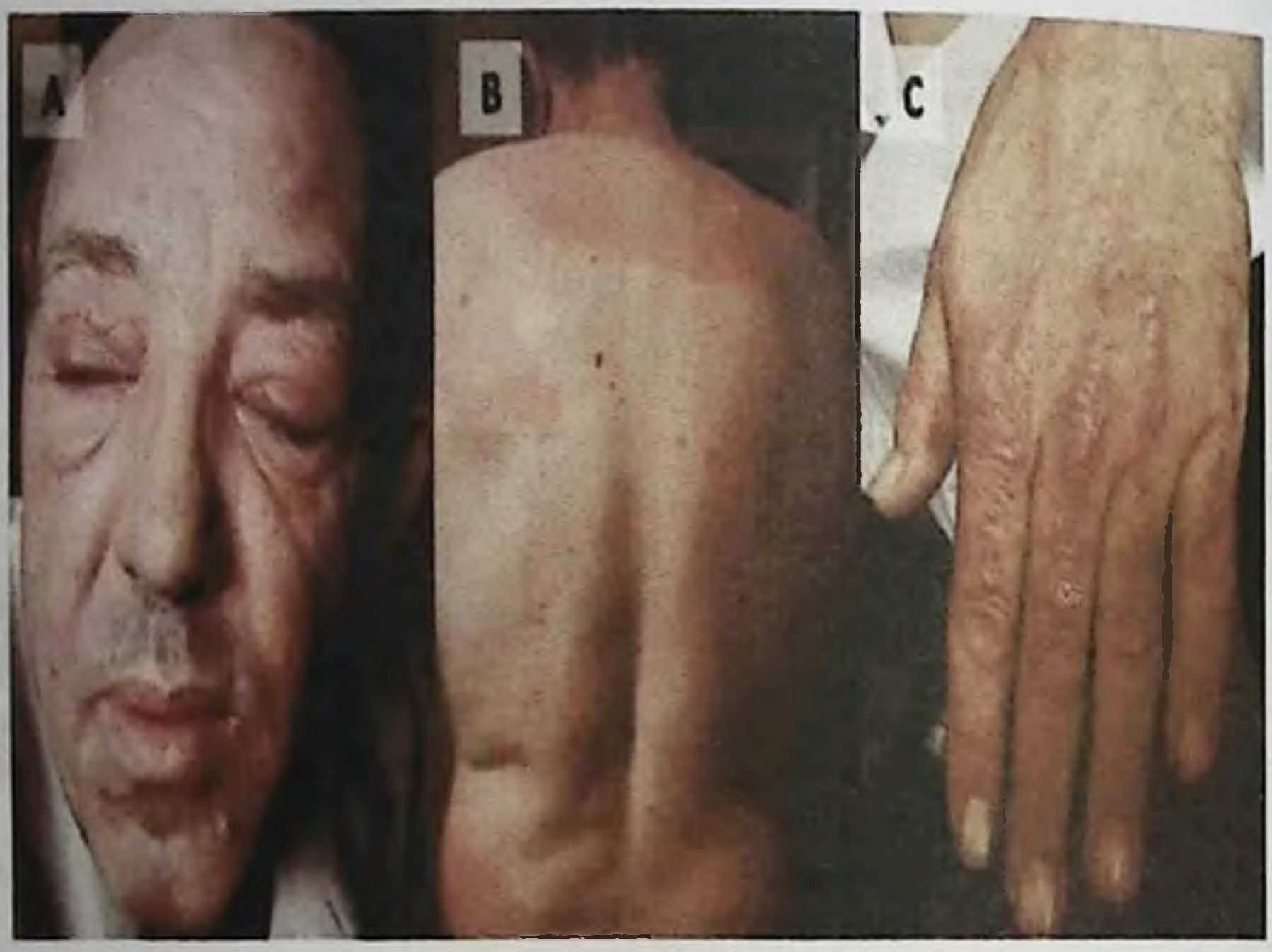
Pic-13 Adult scleredema

DERMATOMYOSITIS

Dermatomyositis is a severe systemic disease characterized by inflammatory purple changes in the eyelids and periorbital area, erythema of the skin of the face, neck and upper trunk, as well as flat purple papules in the joints of the hands, combined with polymyositis. It ranges from dermatomyositis with only dermatitis (dermatomyositis without myositis, amyopathic dermatomyositis) to **polymyositis** with only muscle inflammation (hypomyotic myositis).

Epidemiology. The disease is considered rare. The prevalence is about six cases per 1 million population (these data do not include patients who do not have muscle damage and do not require hospitalization). It affects older children and adolescents (juvenile dermatomyositis) and adults over 40 years of age. In people older than 55 years,

dermatomyositis occurs more often against the background of malignant tumors(paraneoplastic dermatomyositis).



Pic-14 Dermatomyositis

Etiology and pathogenesis. The etiology of dermatomyositis has not been established. A frequent combination of dermatomyositis with malignant tumors has been noted. However, the nature of the association of dermatomyositis with tumors has not been established. The autoimmune hypothesis for the development of dermatomyositis is currently considered the most recognized.

Systematicity, sensitization of lymphocytes to antigens of muscle tissue, cytotoxicity of lymphocytes to cultures of auto-, homo- and heterologous muscle, the presence of autoantibodies, including antinuclear, immune complexes, circulating and fixed on lymphoid cells of muscle infiltrate, testify in its favor.

Dermatomyositis is subdivided into **idiopathic, paraneoplastic and juvenile**, and along the course - into acute, subacute and chronic. An increased frequency of HLA-B8 / HLA- DR3 in children and HLA-B14 in

adults is genetically determined, which indicates a hereditary predisposition.

Clinic. The disease has a variety of clinical forms: from an isolated skin lesion (**amyopathic dermatomyositis**) to an isolated muscle lesion (**polymyositis**). Skin lesions are noted in adult patients and in children. Classification of dermatomyositis.

The following types of dermatomyositis are distinguished. I. **Adult dermatomyositis**: 1) classic, 2) primary, 3) paraneoplastic, 4) in combination with rheumatic diseases. II. **Amyopathic**. III. **Juvenile dermatomyositis**:

1) classic,
2) amyopathic,
3) hypomyopathic.

Provoking factors are often infections, insolation, cooling, drug therapy, vaccination. In

the prodromal period, fever, weakness, nausea, bone and muscle pain, and sweating are possible. Skin and muscle lesions may develop simultaneously or skin changes occur earlier; less often they are absent.

The most common localization of the process on open parts of the body. First there is a bright (flaming), edematous erythema, then acquiring a purple hue. A "syndrome of points" is formed. Urticarial, vesicular, hemorrhagic elements may appear on the affected areas. Sometimes there is soreness of the skin or itching.

In the future, erythema acquires a rich brown color, telangiectasias, atrophy of the epidermis, areas of hypo- and hyperpigmentation, capillaries on the fingers and palms, and painful paronychia develop. Mucous membranes can be affected in the form of **conjunctivitis, rhinitis, stomatitis, laryngitis, pharyngitis**. The striated muscles areaffected.

Severe fatigue, soreness and increasing weakness of the muscles of the shoulder and pelvic girdle are noted. Muscle soreness becomes so pronounced that patients cannot raise their arms, legs, or head. Muscle consistency gradually becomes more dense to woody due to myofibrosis. Contractures and muscle atrophy develop.

The skin above them has a lilac color, shiny, thickened, not folded. Often there is focal or diffuse muscle calcification. A visceral-muscular syndrome may occur when the muscles of the soft palate and pharynx are affected, manifested by dysphagia of the larynx, 68 intercostal muscles (shortness of breath, etc.), myocardium (myocarditis, etc.). Other visceral and neurological manifestations are possible.

Histopathology. The epidermis is thinned. The dermis is edematous, dystrophic changes in the hair follicles and sebaceous glands are observed. In the muscles, transverse striation is not detected, there is fiber dystrophy, in investments - infiltrates, mainly from lymphoid cells. Sometimes only dystrophic changes are visible in the muscle fibers.

The diagnosis at a typical picture does not cause difficulties. However, in all cases it must be confirmed by histological and other paraclinical studies.

Differential diagnosis is carried out with **systemic lupus erythematosus, scleroderma, Weber-Christian panniculitis, trichinosis.**

Treatment. In the paraneoplastic variant, the main focus should be on the elimination of the tumor. Corticosteroids are the main treatment for dermatomyositis.

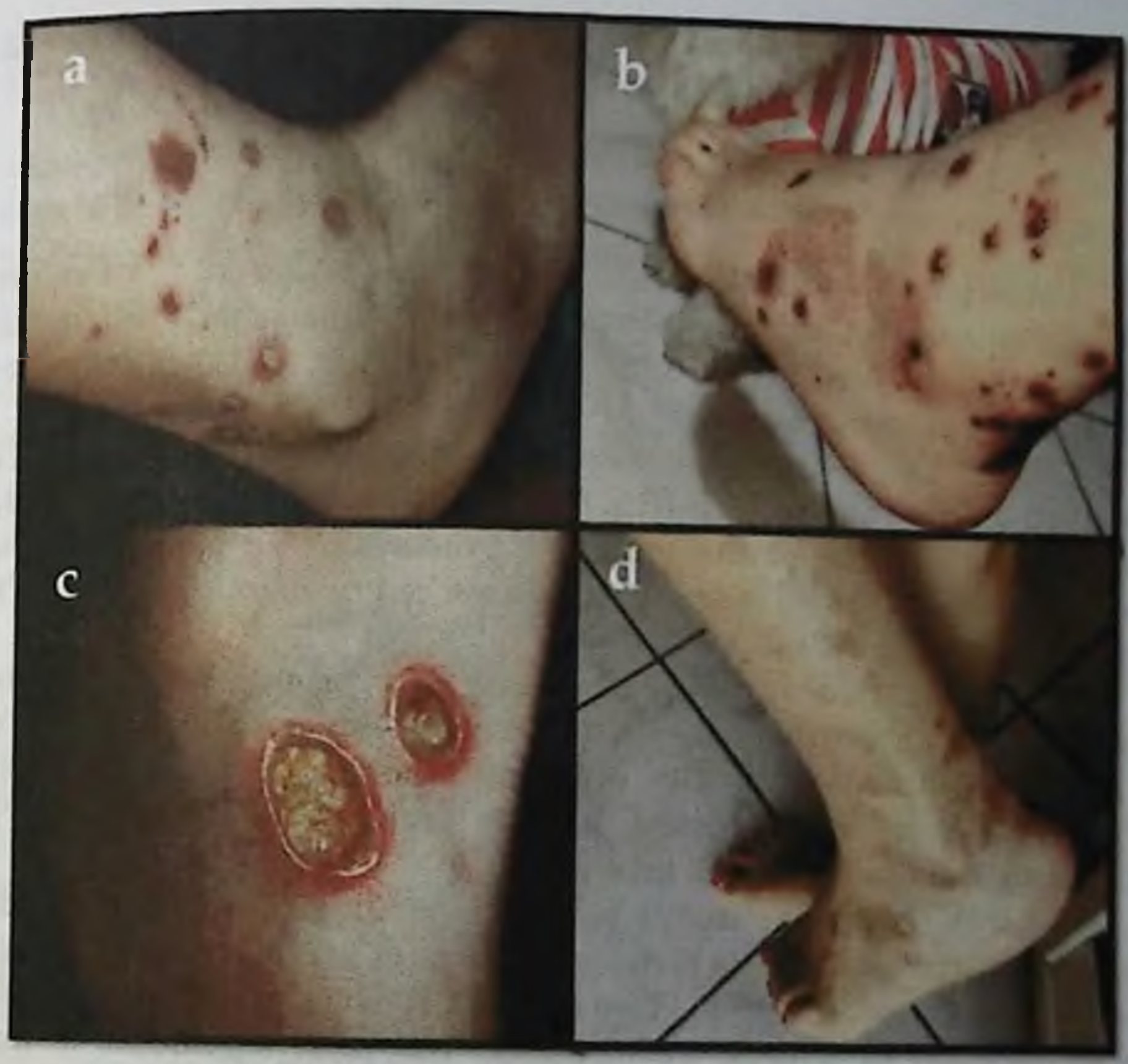
Treatment should begin with high doses of the hormone (at least 1 mg per 1 kg of body weight per day) and continue until the therapeutic effect is achieved, but not less than 1 month, followed by a slow decrease in the daily dose to maintenance.

Simultaneously with corticosteroid therapy, potassium, calcium preparations, periodically anabolic hormones (nerobol, retabolil), vitamins, oxygen therapy should be prescribed.

Those who have had dermatomyositis should be examined by a rheumatologist.

LEUKOCYTOCLASTIC VASCULITIS

Synonyms: allergic cutaneous vasculitis, leukocytoclastic angiitis, dermal necrotizing vasculitis, hypersensitivity angiitis, anaphylactoid purpura, immunocomplex vasculitis, superficial capillaritis, Gougerot-Ruiter arteriolitis.



Pic-15 Leukocytoclastic Vasculitis

Leukocytoclastic vasculitis is a symmetrical exanthema, constantly manifested by hemorrhage, characterized by subacute and chronic course, the pathogenetic basis of which is immunocomplex vasculitis of small and medium-sized vessels of the dermis. Leukocytoclastic vasculitis is most often an acute, predominantly neutrophilic, inflammatory reaction of small to medium-sized vessels that involves the entire vascular wall and leads to fibrinoid necrosis of the vessel walls.

The term "leukocytoclastic vasculitis" refers to the histological characterization of a group of diseases that cause acute neutrophilic inflammation and vascular lesions in the dermis of small to medium

caliber. The lesion may develop in the absence of an obvious defining internal pathology or serve as a skin manifestation of a systemic

disease (Schonlein-Henoch purpura, systemic lupus erythematosus,cryoglobulinemia).

Histological classification of leukocytoclastic vasculitis Leukocytoclastic vasculitis, polyetiological or idiopathic, occurs in Gougerot-Ruiter disease, Schonlein-Henoch disease, and hemorrhagic-necrotizing cutaneous vasculitis.

Leukocytoclastic vasculitis associated with autoimmune diseases is observed in **rheumatoid arthritis, polyarteritis nodosa, collagenosis, polyclonal cryoglobulinemia, pyoderma gangrenosum, paraproteinemia.** As an independent disease can be detected **in Sweet's syndrome** and **Behcet's disease.**

Leukocytoclastic vasculitis associated with neoplastic diseases is detected in lymphomas and plasmacytomas. This pathology with a granuloma pattern occurs with erythema nodosum, facial granuloma, erythema elevated and persistent, **allergic granulomatosis, Wegener's granulomatosis and giant cell arteritis.**

Etiology. Numerous factors have been considered as the cause of leukocytoclastic vasculitis, in particular antigens of microorganisms, drugs, foods, tumors or self-antigens. There may be a combination of causes, such as an infection or a drug. Pathogenesis. The most significant is the deposition of immune complexes in the vessel wall, which is confirmed by immunofluorescent and electron microscopic studies (IgG, components of the complement system (C)).

Deposition occurs primarily subendothelially in the wall of postcapillary venules. It is at the beginning of a chain of reactions that continues with complement activation, leukotaxis, release of cytokines and lysosomal enzymes, and subsequent destruction of the vessel wall. The consequence of this is the release of erythrocytes and necrosis.

This is a type III immune response. Damage to endothelial cells is undoubtedly primary in any type of vasculitis. This process is induced polyetiologically: infectious-allergic mechanism, drug-induced factors or

autoimmune processes. In this case, the leading role is played by foreign antigens, autoantigens, tumor antigens, medicines, food additives.

Damaged cells are localized mainly in the capillaries or postcapillary venules of the superior vascular plexus and, for their part, through the activation of cytokines and the expression of adhesion molecules, attract neutrophilic granulocytes that penetrate the vascular wall and are located perivascularly.

The release of granulocytes through endothelial cells is often followed by a lymphocytic reaction, which in many forms of vasculitis can end in the progressive stage with the formation of a granuloma. This kind of deposits of immunoglobulins and (or) complement in the walls of blood vessels contribute to the attraction of neutrophilic

leukocytes.

Provocative factors of leukocytoclastic vasculitis Slowing of blood flow probably plays a decisive hemodynamic role in the occurrence of a disease with superficial localization. Capillaries are found in the deeper dermis and between fat lobules, where they are the target organ of nodular vasculitis. Cold is another provoking factor: a decrease in skin temperature leads to vasoconstriction in the arterial capillary network, causing dilatation of the precapillary venules.

Due to the slightest irritation, primary manifestations of vasculitis may occur. The increased viscosity of the blood predisposes to superficial vasculitis, so that in the presence of pronounced fibrous deposits, a bleeding disorder should be suspected.

Recently, in this regard, anticardiolipin antibodies have been given importance as a pathogenetic factor in vasculitis. Bacterial infections are a common cause of clinical vasculitis as a reaction to bacteria or foreign proteins. The manifestations of the disease are preceded by streptococcal infections of the upper respiratory tract, which occurred 2–3 weeks before.

While such infections are most commonly associated with **acute leukocytoclastic vasculitis, mixed lymphocytic vasculitis** is more likely to have chronic infections (eg, dental granuloma) as precipitating factors. If the search for an inflammatory focus in the presence of vasculitis is unsuccessful, ex juvantibus systemic antibiotics can lead torecovery.

Provoking factors can be mycobacteria and mycoplasma infections, **syphilis, torpid gonorrhea, mycoses (intestinal candidiasis), hepatitis B infection, neonatal herpes (isolated cases).**

Medications and chemicals often provoke vasculitis, predominantly lymphocytic, and first of all we are talking about aspirin and systemic antibiotics, then there may be diuretics, sulfonamides. In rare cases, vasculitis is associated with malignomas and is probably due to tumor antigens. Most often they occur with lymphomas, but they also occur with breastcarcinoma, tumors of the gastrointestinal tract.

Identification and elimination of the above factors is an important prerequisite for the treatment of vasculitis.

Clinic. Leukocytoclastic vasculitis, as a rule, has a chronic relapsing course and is characterized by extremely diverse morphological symptoms, which gave rise to its confusing and abundant nomenclature. Rashes initially appear on the shins, but can also occur on other areas of the skin, less often on the mucous membranes.

Blisters, hemorrhagic spots of various sizes, inflammatory nodules and plaques, superficial nodes, papulonecrotic rashes, vesicles, blisters, pustules, erosions, superficial necrosis, ulcers, scars are observed. Rashes are sometimes accompanied by fever, general weakness, arthralgia, headache.

The resulting rash usually persists for a long period (from several weeks to several months), tends to relapse. The onset of the disease and its relapses are often provoked by acute infectious diseases (tonsillitis, influenza, acute respiratory viral infection), hypothermia, physical or nervous strain, less often by taking any medications or food intolerance.

Depending on the presence of certain morphological elements of the rash, various types of leukocytoclastic vasculitis are distinguished: hemorrhagic, urticarial, papulonodular, papulonecrotic, papular-ulcerative, necrotic-ulcerative and polymorphic.

However, often different elements are combined, creating a picture of polymorphic vasculitis. The hemorrhagic type is most characteristic of leukocytoclastic vasculitis. The most typical symptom in this variant is the so-called palpable purpura - edematous hemorrhagic spots of various sizes, usually localized on the legs and back of the feet, easily determined not

only visually, but also on palpation, which distinguishes them from the symptoms of other purpuras, in particular from the disease **Schamberg - Maiocchi**.

However, the first rashes in the hemorrhagic type are usually small edematous inflammatory spots that resemble blisters and soon transform into a hemorrhagic rash. With a further increase in inflammation against the background of confluent purpura and ecchymosis, hemorrhagic blisters can form, leaving deep erosions or ulcers after opening. Rashes are usually accompanied by moderate swelling of the lower extremities. Hemorrhagic spots may appear on the mucous membrane of the mouth and throat. The described hemorrhagic rashes that occur acutely after a cold (usually after a sore throat) and are accompanied by fever, severe arthralgia, abdominal pain and bloody stools constitute the clinical picture of **Shenlein-Genoch anaphylactoid purpura**, which isoften observed in children.

The urticarial type, as a rule, simulates a picture of chronic recurrent **urticaria**, manifesting itself with blisters of various sizes that occur on different parts of the skin. However, unlike urticaria, blisters with urticarial vasculitis are particularly persistent, remaining for 1-3 days (sometimes longer).

Instead of severe itching, patients usually experience a burning sensation or a feeling of irritation in the skin. Rashes are often accompanied by **arthralgia**, sometimes pain in the abdomen, i.e., signs of a systemic lesion. Examination may reveal **glomerulonephritis.** Patients also noted an increase in ESR, **hypocomplementemia**, an increase in lactate dehydrogenase activity, positive inflammatory tests, changes in the ratio of immunoglobulins.

Treatment with antihistamines usually does not work. Urticarial vasculitis is more common in middle-aged women.

The diagnosis is finally confirmed by histopathological examination of the skin, which reveals a picture of leukocytoclastic vasculitis. The papulonodular type is quite rare. It is

characterized by the appearance of smooth flattened inflammatory nodules of roundedoutlines the size of a lentil or a small coin, sometimes more, as well as small superficial, unsharply outlined edematous pale pink

nodes up to a hazelnut size, painful on palpation. Rashes are localized on the extremities, usually on the lower ones, rarely on the trunk and are not accompanied by pronounced subjective sensations. The **papulonecrotic type** is manifested by small flat or hemispherical inflammatory non-scaling nodules, in the central part of which a necrotic scab forms, usually in the form of a black crust. When the scab is torn off, small rounded superficial sores are exposed, and after resorption of the papules, small "stamped" scars remain.

The rashes are located on the extensor surfaces of the limbs and clinically completely simulate papulonecrotic tuberculosis, which should be carefully differentiated. The papular-ulcerative type begins with the appearance of small vesiculopustules resembling **acne or folliculitis**, quickly transforming into ulcerative lesions with a tendency to steady eccentric growth due to the disintegration of the edematous bluish-red peripheral ridge.

The lesion can be localized on any part of the skin, but more often on the shins, in the lower abdomen. After healing of ulcers, flat and hypertrophic scars remain, which retain an inflammatory color for a long time. The necrotic-ulcerative type is the most severe variant of leukocytoclastic vasculitis.

It begins acutely (sometimes with lightning speed) and takes a protracted course (if the process does not end in a quick death). Due to acute thrombosis of inflamed blood vessels, necrosis (infarction) of one or another area of the skin occurs, manifested by necrosis in the form of an extensive black scab, the formation of which may be preceded by an extensive hemorrhagic spot or bladder.

The process develops within a few hours, accompanied by severe local pain and fever. The lesion is more often located on the lower extremities and buttocks.

Purulent-necrotic scab persists for a long time. The ulcers formed after its rejection have a different size and shape, contain a purulent discharge, and slowly scar. The polymorphic type is characterized by a combination of various eruptive elements characteristic of other types of leukocytoclastic vasculitis. More often there is a combination of edematous inflammatory spots, hemorrhagic rashes of a purplish nature and superficial

edematous small nodes, which constitutes the clinical picture of the so-called three-symptom **Gougerot-Dupper syndrome** and **Rulter's polymorphic-nodular arteriolitis** identical to it.

Damage to other organs. Skin manifestations of leukocytoclastic vasculitis suggest that in many patients other organs are also affected by inflammatory vascular changes, such as joints (arthralgia, approximately 40%), kidneys (hematuria in 30%), gastrointestinal tract (30%, including with gastrointestinal bleeding), lungs (20%), central nervous system (10%).

The heart, liver, and muscles are less commonly affected. Practically significant is the factthat the scale of skin lesions does not have an inverse effect on the severity of damage to other organs. The course is subacute, to chronically recurrent, depending on the type and duration of the provoking antigenic stimulus. The prognosis depends on the severity ofthe systemic lesion; when only the skin is affected, it is favorable.

As a complication, a secondary bacterial infection may occur. Laboratory and special studies. General blood analysis. Platelets are normal. ESR increased. General urine analysis. Erythrocyte casts, albuminuria. Histoimmunopathology. A typical picture of leukocytoclastic vasculitis. In this case, the penetration of small dermal vessels by rapidly decaying neutrophils and the deposition of fibrin in the necrotic walls of the vessels and their surroundings occur.

The breakdown of neutrophils (i.e., leukocytoclasia) is recognized by the remnants of nuclei located in the tissues ("nuclear dust"). Erythrocytes penetrating the perivascular tissue are the histological substrate of purpura. This histological pattern varies depending on the clinical presentation and the stage of the disease.

Immunofluorescent microscopy shows the presence of intra- and perivascular deposits of immune complexes (C3, IgM, IgG) in fresh lesions. In unclear cases, a fresh focus is provoked by a histamine test. Diagnostics. When making a diagnosis, one should be guided by the following goals: objective confirmation of the diagnosis, taking into account damage to other organs, identifying provoking factors.

The clinical pathomorphological sign of vasculitis is a hemorrhagic rash that rises above the surface of the skin - palpable purpura, which is easy to recognize by pressing with a glass spatula. The **Konchalovsky-**

Rumpel-Leede test is positive (the appearance of petechiae on the skin of the shoulder and forearm after a short squeezing of the shoulder with a tourniquet or a rubber cuff of a sphygmomanometer is a sign of increased fragility and permeability of capillaries and precapillaries in vasculitis).

Platelets and clotting factors are normal. Biopsy with evidence of leukocytoclastic vasculitis confirms the diagnosis. Arthralgia, myalgia, headaches, gastrointestinal disturbances, hematuria, blood in the feces, positive C-reactive protein testify to a systemic lesion. Drugs, focal infection, autoimmune diseases, and tumors should be excluded as the cause of vasculitis.

The **American Rheumatological Association** has developed diagnostic criteria for leukocytoclastic vasculitis: age over 16 years, onset shortly after treatment with new drugs, palpable purpura, maculopapular rash, skin biopsy (presence of granulocytes in the perivascular space or in interstitial tissue).

The diagnosis is made when at least three of the five criteria listed are met.

Treatment. In an acute course and with a deterioration in the general condition, inpatient treatment is indicated. Bed rest is recommended as static factors contributing to vasculitis are eliminated. Corticosteroids are indicated for widespread skin lesions and acute vasculitis, as well as for systemic manifestations of the disease.

Recommended initial dose of prednisolone 60-80 mg / day with a gradual dose reduction (within 3-4 weeks) when a clinical effect is achieved. Sulphonic preparations (DDS, dapsone) are recommended for subacute and chronic cases or for exclusively cutaneous manifestations of vasculitis. The initial dose of the drug is 100-150 mg / day.

A weekly complete blood count is required (methemoglobinemia is possible, for the prevention of which vitamin C or ascorutin is prescribed one tablet three times a day). Non-steroidal anti-inflammatory drugs have analgesic, anti-inflammatory effects, normalize microcirculation, reduce the level of biologically active substances in the lesions. The following drugs are prescribed: indomethacin 0.025 g three times a day; Naprosin 0.25 g twice a day; butadione 0.15 g four times a day; pyrabutol or reopirin

0.025 g or 5 ml intramuscularly; mefenamic acid 0.5 g three times a day; course of treatment - 2-4 weeks.

Cytostatics are used in severe vasculitis or in cases of resistance to hormone therapy. Recommended cyclophosphamide (azathioprine) 0.05 g three times a day, as well as cyclosporine (sandimmun 2-10 mg per 1 kg of body weight per day), colchicine (1 mg per 1 kg of body weight per day), methotrexate. Antibiotics are prescribed for bacterial origin of vasculitis. One of the following drugs is recommended: oletethrin 0.25 g four times a day; metacycline 0.3 g twice a day; vibramycin 0.1 g once a day; tseporin intramuscularly

1.5 g two to three times a day.

The effect was noted as a result of the use of penicillin G at 3-4 million IU / day. In case of damage to the gastrointestinal tract and the presence of yersiniosis, treatment with tetracyclines is indicated for 10 days. Vitamin therapy. The use of vitamins with antioxidant action is recommended: tocopherol acetate (10-15 drops three times a day with meals for 3-4 weeks), ascorbic acid, rutin, quercetin, retinol palmitate (100 thousand IU / day for 2 weeks), as well as multivitamins (aevit, gendevit, undevit, dekamevit, kvandevit) one - two dragees two - four times a day after meals for 3-4 weeks.

Ascorbic acid has a wide spectrum of action: it stabilizes vascular permeability, improves metabolism, affects all types of metabolism, and stabilizes the sulfhydryl groups of many enzymes. Ascorbic acid is administered 5 ml of a 5% solution intravenously for 3–4 weeks, and then 0.3 g orally three times a day for 2–3 months.

At the same time, a synergist of ascorbic acid is prescribed - rutin, 0.05 g three to four times a day for 1-2 months. Antihistamines are recommended only in the active phase of vasculitis, since a high level of histamine is noted only in the first days of the disease. Therefore, drugs are used not only with antihistamine, but also with antiserotonin, antiacetylcholine, antibradykinin effects.

Apply suprastin 0.025 g three times a day; diazolin 0.1 g three times a day; tavegil 0.001

g twice a day; cyproheptadine (peritol) 0.004 g three to four times a day; reserpine 0.00025 g three times a day for 3-5 days. Angioprotectors

and antiplatelet agents are used to normalize the structure and function of the vascular wall, as well as the aggregant properties of blood cells.

The following drugs are recommended: parmidine 0.25 g three to four times a day; trental

0.2 g three times a day after meals with a gradual decrease in dose to once a day. Under the control of the coagulogram, escusan, esflazid, glivenol are used. local therapy. Zinc- based corticosteroid creams and ointments, troxevasin-gel, butadione ointment, aminocaproic ointment.

In the necrotic-ulcerative type, lotions or ointments with proteolytic enzymes (chymopsin, iruksol) are initially recommended, followed by disinfectant, anesthetic ointments and sponges, laser irradiation of the small power.

Test tasks Choose one or more correct answers.

1. The main clinical manifestations of discoid lupus erythematosus are 1) limited erythema 2) cicatricial atrophy 3) follicular hyperkeratosis 4) peeling loose scales

2. favorite places of localization of discoid lupus erythematosus 1) nose 2) stomach 3) cheeks 4) shins

3. basic advice for a patient with lupus erythematosus 1) sanitation of a focal infection 2) sports: cycling, skiing, mountain tourism 3) the use of sunscreen 4) resort therapy in the Crimea and the Caucasus

4. The difference between discoid lupus erythematosus and alopecia areata is that on 1) scalp cicatricial atrophy 2) face erythema, follicular hyperkeratosis 3) scalp skin is smooth 4) scalp peeling

5. limited scleroderma is characterized by 1) erythema, hyperkeratosis, cicatricial atrophy

2) erythema, lichenification, pigmentation 3) "medallion" spots, scales, pigmentation 4) lilac edematous spot, dense plaque, atrophy, pigmentation

6. 1) lidase 2) penicillin 3) nicotinic acid 4) acyclovir are used to treat scleroderma

7. The group of diffuse connective tissue diseases includes 1) lupus erythematosus 2) scleroderma 3) erythema nodosum 4) dermatomyositis

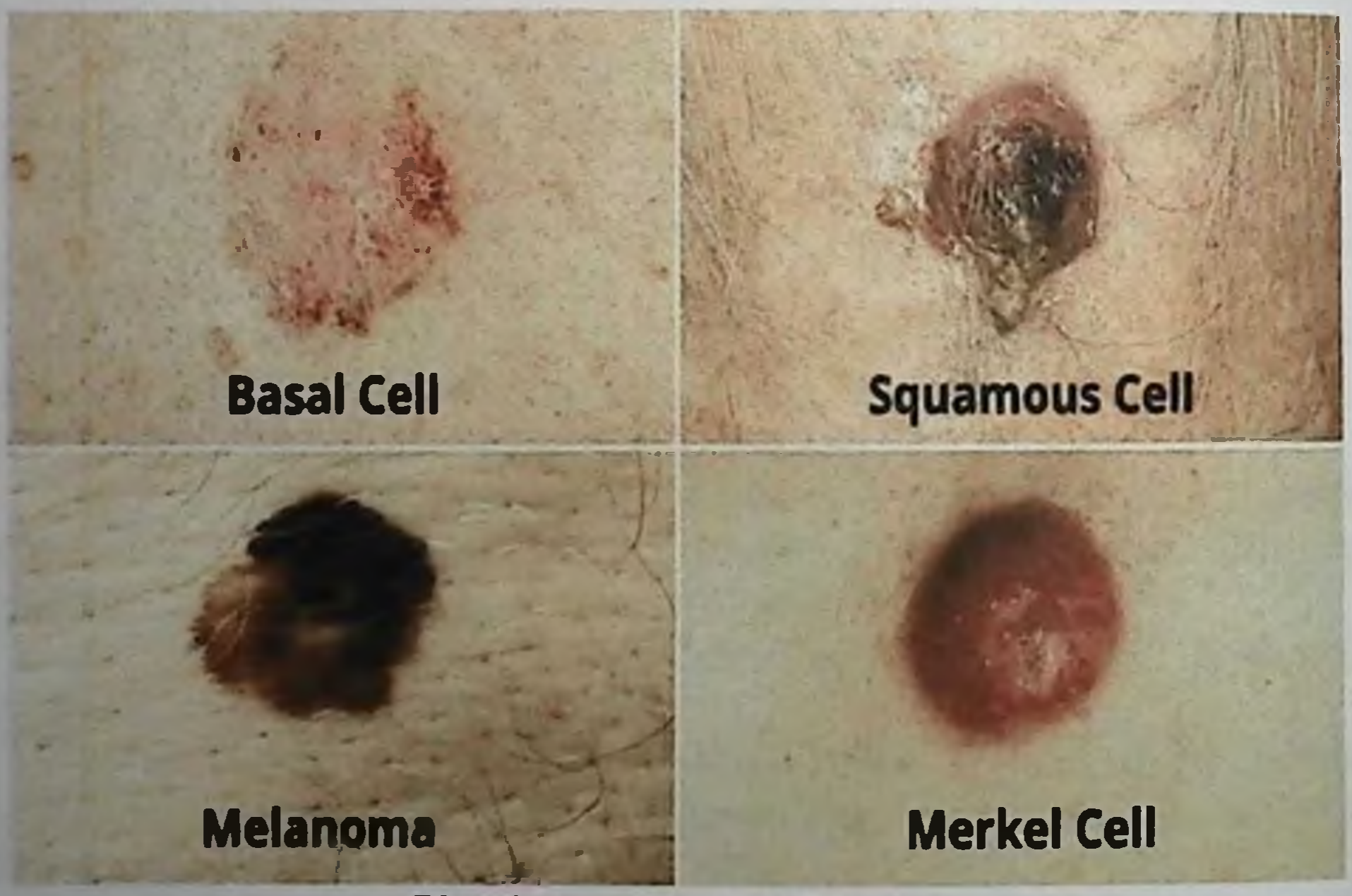
8. stages of discoid lupus erythematosus 1) erythematous 2) atrophic 3) hyperkeratotic-infiltrative

9. focal scleroderma is characterized by 1) induration 2) inflammatory edema 3)hypertrophy 4) atrophy 5) hyperpigmentation.

CHAPTER 3 BASICS OF ONCODERMATOLOGY. PRECANCER DISEASES. BENIGN AND MALIGNANT TUMORS OF THE SKIN. HIGHLIGHTS.

PRECANCER SKIN DISEASES.

This group of diseases includes such pathological conditions in which most often, but not necessarily, malignant tumors develop. Among the diseases of this group, xeroderma pigmentosum is an absolute precancer. It is a familial disease transmitted by an autosomal gene. From childhood, patients develop increased skin sensitivity to ultraviolet rays, photophobia develops, and inflammatory spots form on open areas of the skin, in place of which pigmentation, **telangiectasias, atrophic scars, and foci of hyperkeratosis** appear.



Pic-16 Precancer Skin Diseases

After a few years, the skin becomes dry, atrophic. **Keratoconjunctivitis, keratitis** develops, visual acuity decreases. On the affected skin at the age of 6-10 years, malignant tumors appear, more often **basaliomas and squamous cell carcinoma**

Treatment is carried out with vitamin A preparations, tumors are removed with liquid nitrogen, electrocoagulation, and surgically. It is necessary to be protected from insolation. **Senile, or solar keratosis,** occurs in older people on the skin of the back of the hands, face, less often on the trunk. A red-yellow spot appears with a small infiltrate at the base, on the surface of which dry, tightly-fitting scales are formed, upon removal of which bleeding is noted.

Thickening, bleeding and ulceration in the lesions 108 indicate malignancy. Diathermocoagulation, cryodestruction or laser therapy is used for treatment.

Senile keratoma (senile wart, seborrheic keratoma, basal cell papilloma) is localized on the face, hands, torso in the form of warty growths of a round or oval shape, yellowish or dark brown in color and has a soft oily texture.

Pic-17 Senile keratoma

Treatment is by surgical removal within healthy skin. **Bowen's disease** is manifested by the formation of several pale pink or dark brown plaques on the skin. Their surface is covered with scales and crusts,

sometimes with papillomatous growths. Individual plaques may coalesce and ulcerate.

Treatment of such patients should be carried out in oncological dispensaries. Paget's disease. Sick women over 40 years of age. The nipple of the mammary gland, the perineum, genitals, pubis, axillary cavity are affected.

There is an infiltrate of dark red color with peeling, weeping. After a few months, ulcers form. With localization in the perineal region, the disease acquires a warty shape, with damage to the nipple of the mammary gland, the nipple is retracted.

Lymph nodes are enlarged, firm, painless. Surgical treatment with removal of the mammary gland and regional lymph nodes, followed by radiation and chemotherapy.

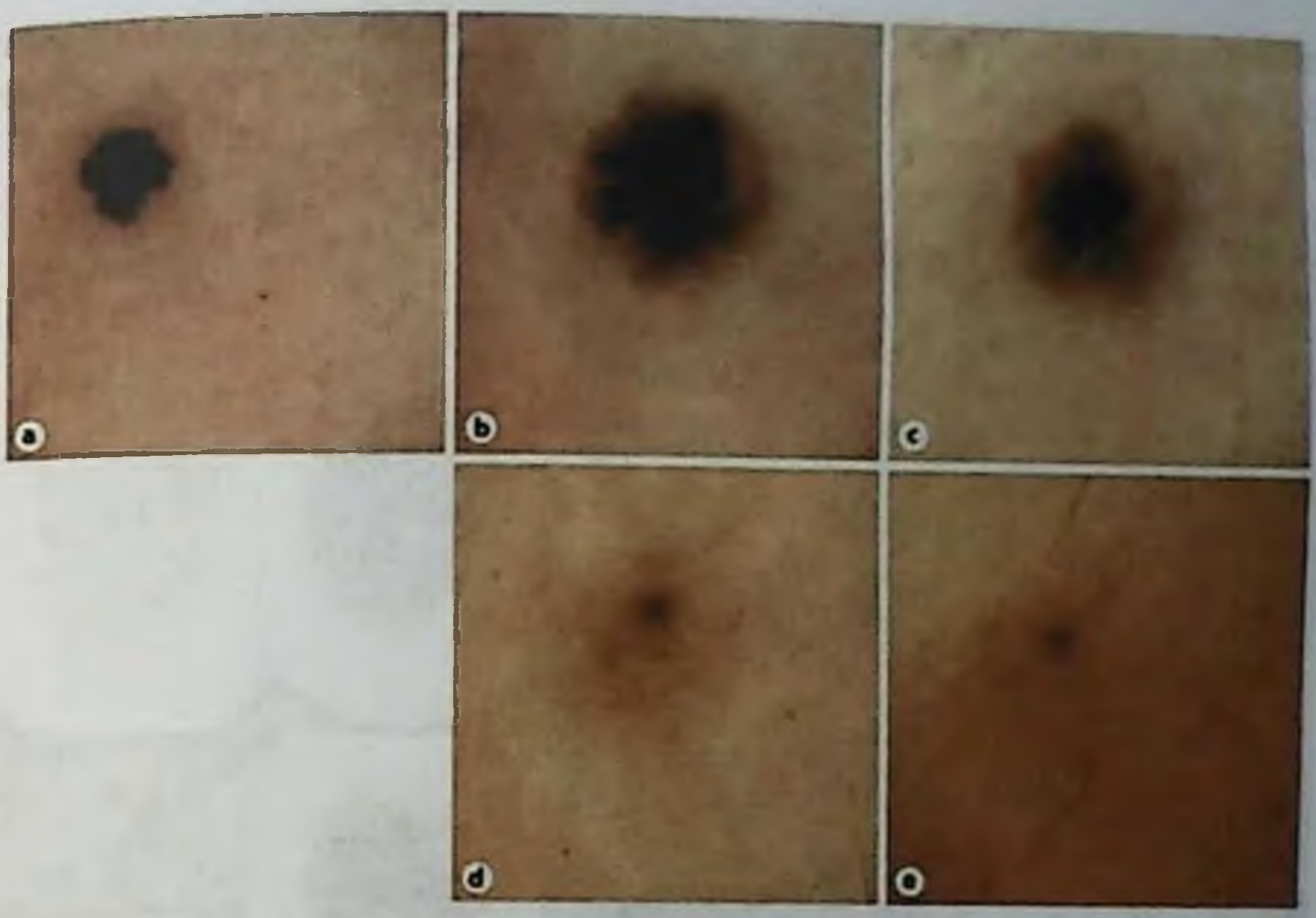
PIGMENT NEVI

Pigmented nevi (**birthmarks**) are formed as a result of accumulation in the epidermis or dermis of nevoid cells containing melanin. One of the varieties of acquired pigmented nevi is lentigo.

Lentigo is a clearly demarcated, smooth pigment spot of a rounded shape, dark brown in color, up to 1 cm in diameter.

Juvenile lentigo appears in childhood and is localized in all areas of the skin.

Senile lentigo occurs in old age on open areas of the skin. The disease is characterized by a benign course. **Melanoma-dangerous nevi** include a blue or blue nevus, which is manifested by a nodule of a rounded hemispherical shape, protruding above the surface of the skin, densely elastic, dark blue or bluish, less often dark brown. Its surface is smooth, devoid of hair. Localized on the skin of the face, buttocks, lower extremities. More often women and persons of middle age are ill. Surgical excision of the focus with the surrounding skin and subcutaneous tissue is shown.



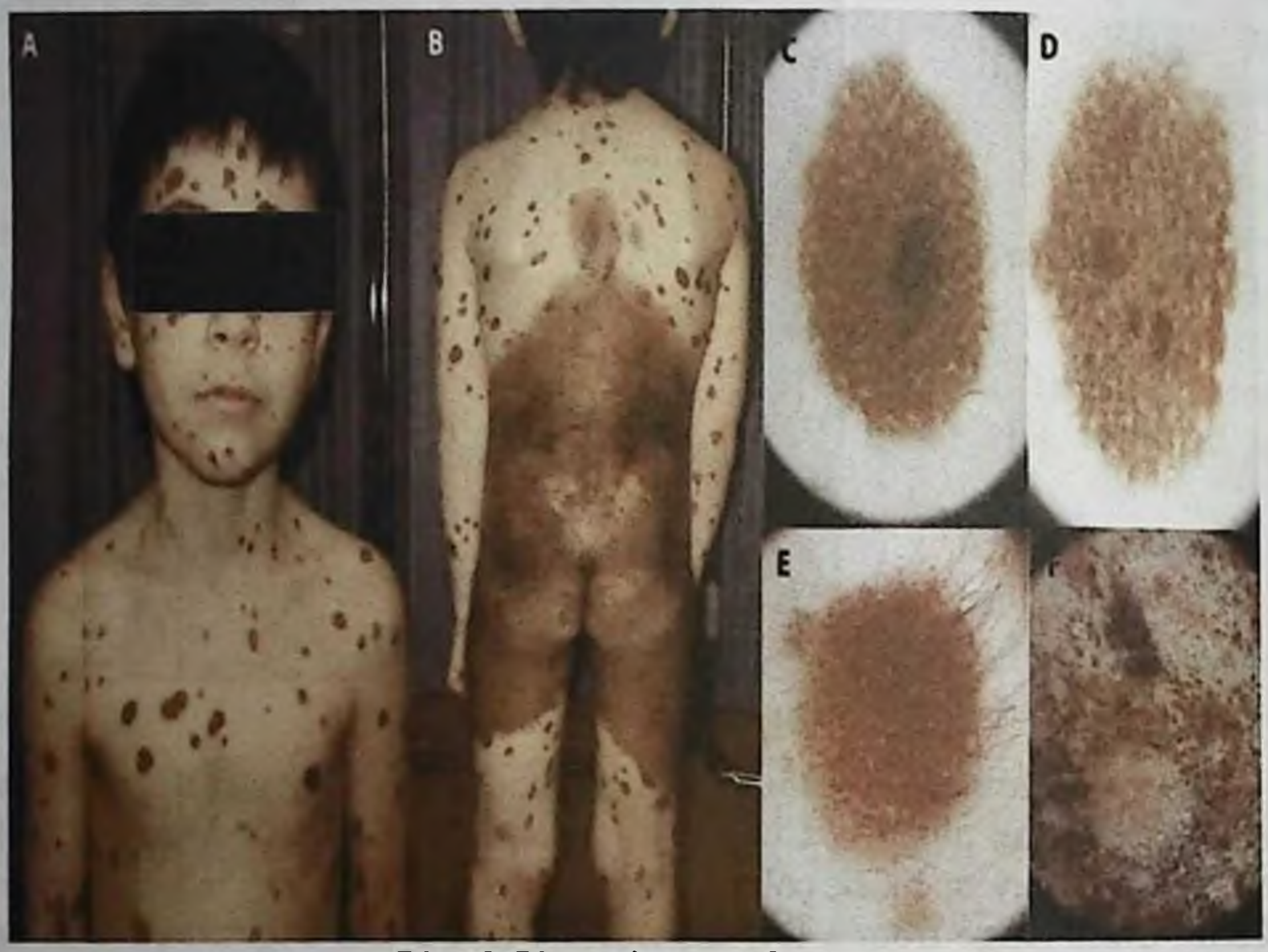
Pic-18 Pigmented nevi (birthmarks)

Nevus pigmentosa is the most dangerous **precancerous dermatosis**, especially during puberty. Clinically manifested as a flat nodule of dark brown, dark gray or black color. Its surface is smooth, dry, hair is always absent. Sizes range from a few millimeters to 4-5 cm. Pigmented nevi on the skin of 109 palms, soles and genitals in most cases are always borderline.

Nevus of Ota (eye - cutaneous melanosis, black-bluish eye - maxillary nevus, phakomatosis Oto - Sato). There are mild subtypes of orbital and zygomatic), moderately pronounced, intense and bilateral. It is clinically marked by dark brown pigmentation in various parts of the eye (conjunctiva, sclera), on the cheek, zygomatic region, and upper jaw,sometimes the red border of the lips and the mucous membranes of the soft palate, pharynx, larynx and nose are affected. Women get sick more often.

Giant pigmented nevus is a congenital disease. Its surface is bumpy, warty, with deep skin cracks, with areas of pronounced hypertrichosis. The color varies from dirty gray to black with a bluish tint. Sometimes the

lesion is combined with other congenital malformations (hydrocephalus, epilepsy). Cases of a giant nevus with the development of primary melanoma of the pia mater are described. Oncologist treatment.



Pic-19 Giant pigmented nevus

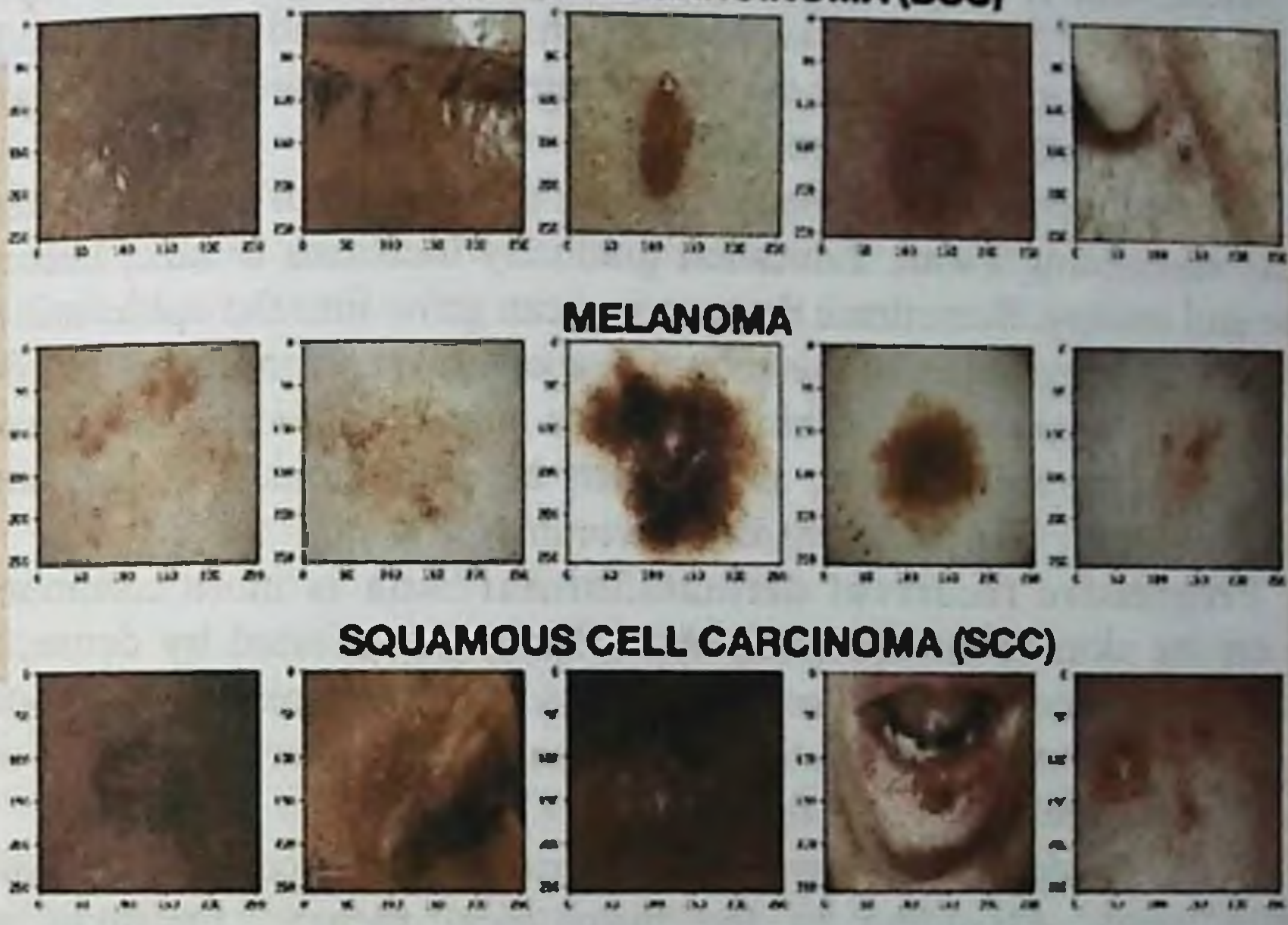
SKIN MALIGNANT TUMORS

There are many theories of cancer (embryonic, virogenetic, hereditary, somatic mutations,etc.).

Malignant tumors are characterized by infiltrating growth with destruction of the surrounding tissue and metastasis. There are two types of skin cancer: **basal cell** and **squamous cell**.

Pic-20 Skin Malignant Tumors **Basalioma, or basal cell carcinoma,** occurs predominantly in the elderly. Localized in the upper half of the face (upper lip, auricles, nose, cheeks, nasolabial folds). It is characterized by slow growth and the absence of metastases. There are several clinical variants of basalioma - superficial and invasive.

SKIN CANCER CATEGORY



Clinical manifestations begin with the appearance of a single flat or hemispherical dense papule up to 0.5 cm in diameter, slightly pink or normal skin color. The papule very slowly increases in size, an erosion or an ulcer is formed in the center, covered with a bloody crust. With an invasive form of basalioma, the ulcer penetrates inside and destroys the underlying tissues to the bone.

Squamous cell (spinocellular) cancer. Basalioma is less common, men are more often ill. The disease is characterized by rapid growth and metastases, malignancy. The skin process is localized at the site of transition of the skin into the mucous membranes of the lips, genital organs, and on the mucous membranes of the oral cavity.

A dense knot is formed in the thickness of the skin, rapidly increasing in size, growing into the deep layers of the skin and subcutaneous tissue, and undergoes disintegration with the formation of ulcers. The edges and

bottom of the ulcer are dense. The general condition of the body is disturbed, cachexia develops.

Treatment is surgical, radiation therapy. Skin sarcoma develops at the age of over 30years and is localized on the trunk and extremities. The disease appears more often at the site of injury, after radiation therapy, on **fibromas, angiofibromas, lipomas.**

Clinical manifestations begin with the appearance of a speck, a dense nodule resembling a wart. Education gradually increases in size, becomes dense and bumpy. Sometimes the sarcoma can grow into the epidermis and resemble the appearance of mushroom-shaped nodes or penetrate into deep tissues, the hypodermis. Skin sarcomas ulcerate, infiltrate, become inflamed. Metastasize rarely, more often hematogenously to the lungs, liver, skin, bones. The prognosis is unfavorable.

Progressive recurrent dermatofibrosarcoma is more common in men on the skin of the abdomen, chest, limbs. Manifested by dense, flat dermal or hypodermal nodules, painless on palpation, resembling foci of plaque scleroderma. In the future, very dense hemispherical tumors on a stalk or a wide base appear in their place.

Lymph nodes do not increase and dermatofibrosarcoma metastasizes in rare cases. According to the histological picture, **fusiform and polymorphic cell fibrosarcomas** are distinguished.

Treatment consists in the use of radiation and chemotherapy, surgery. Melanoma is a malignant tumor that develops from pigment-forming cells. It is localized more often on areas of the skin exposed to insolation, on the face, torso, and lower extremities. It often develops against the background of a pigmented nevus.

At first, a spot appears, resembling a large freckle, which gradually increases, thickens, redness and a stagnant corolla appear around. Then nodules form on the surface of the spot. Pigmented nodules and radial growths ("satellites") appear around the primary focus, indicating lymphogenous spread of the tumor and metastases. Regional lymph nodes are enlarged.

There are three main types of melanoma: **superficially spreading, malignant lentigo,** and **nodular.** Metastases of melanoma spread

lymphogenously or hematogenously. Treatment is surgical with chemotherapy.

Kaposi's sarcoma is a malignant disease of the skin and internal organs. There are 3 types of sarcoma: 1) idiopathic 2) due to immunosuppression, ionizing radiation, cytostatic drugs and corticosteroids in the treatment of cancer, 3) Kaposi's sarcoma caused by HIV infection.

Adult men are ill more often, children are seldom. The process is localized mainly on the skin of the feet, legs, back of the hands, less often on the auricles, cheeks, nose, back, abdomen, penis. The oral mucosa is affected in up to 12% of cases and is localized on the soft and hard palate, cheeks, pharynx, larynx, and tongue.

The disease begins either with the appearance of reddish - cyanotic spots with clear boundaries and a smooth surface, or with papules of a pinkish - cyanotic color, soft consistency. Subsequently, tumor-like elements are formed that rise above the skin, the color of which acquires a bluish tint.

At the site of the tumors, deep and painful ulcers are formed, the bottom of which is covered with bloody-necrotic discharge and warty growths. Kaposi's sarcoma is characterized by multifocality, prevalence and symmetry of rashes.

Skin manifestations are accompanied by excruciating pain, swelling. Kaposi's sarcoma can be a manifestation of AIDS, while it is characterized by a young age, the sudden appearance of common forms of papular and tumor-like formations, the absence of spotty rashes, atypical localization (head, neck, trunk, oral cavity), damage to internal organs and lymph nodes.

The diagnosis of Kaposi's sarcoma must be confirmed by histological examination. All patients with Kaposi's sarcoma should be tested for HIV infection.

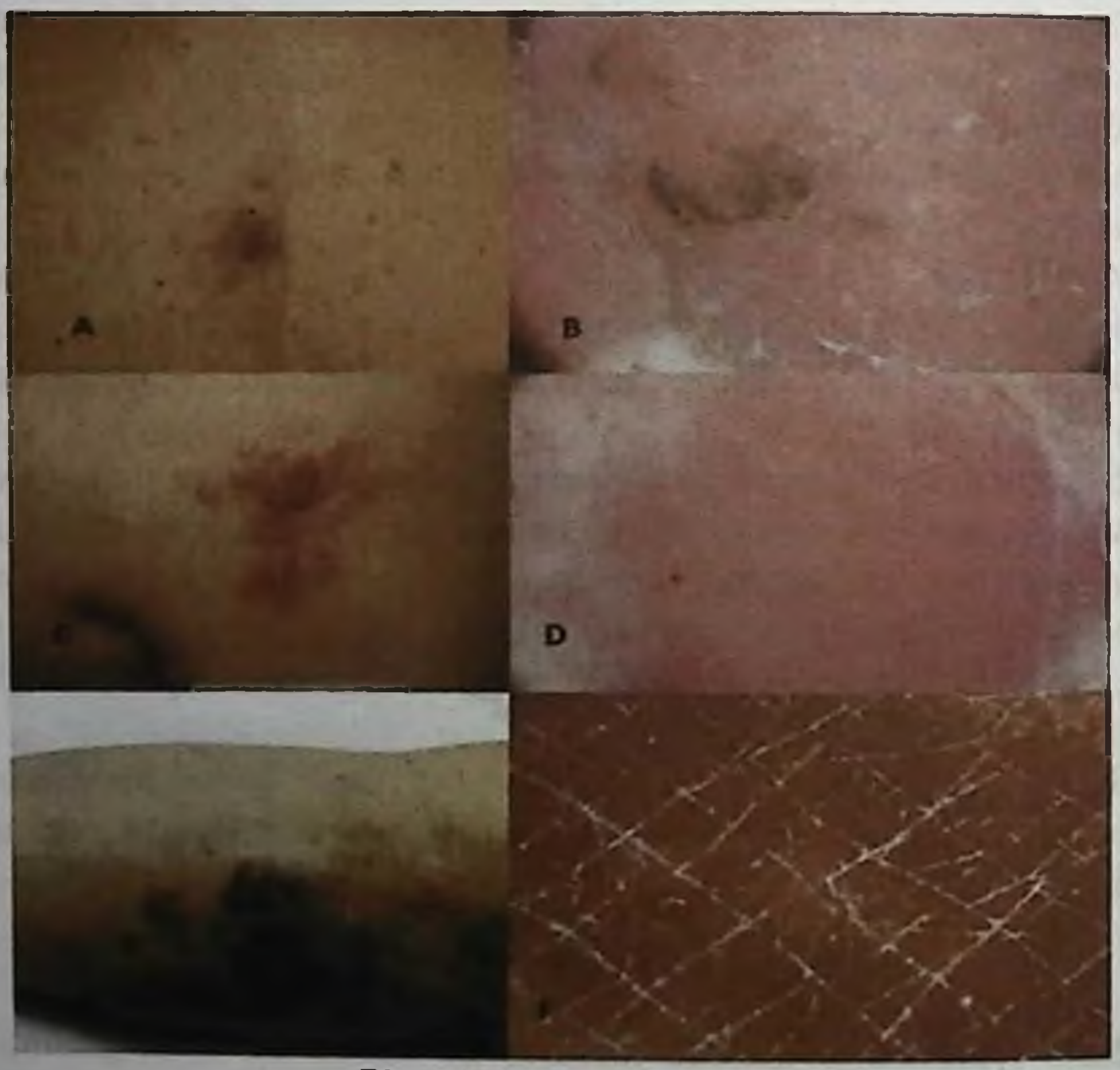
SKIN LYMPHOMAS

Skin lymphomas include diseases in which the main pathological process in the skin occurs in the form of malignant proliferation of lymphocytes and their derivatives - plasma cells. The development of

lymphomas is determined by general patterns characteristic of the tumor progression of all malignant neoplasms.

The etiology and pathogenesis of skin lymphomas remain not fully understood. One ofthe factors in the occurrence of malignant processes is a violation of the state of the immune system and the immunological function of the skin, which controls the regulation of growth and differentiation of various cells.

In addition, there is a tumor theory based on the instability of the genetic apparatus of cells or its mutations; the theory of the subsequent malignancy of the reactivity of the process due to constant antigenic stimulation of the skin by various factors of the external and internal environment.



Pic-21 Skin Lymphomas

Until now, the classification proposed by I.M. Raznatovsky, which more fully reflects the clinical and histological features, the rate of tumor progression and the degree of malignancy of the tumor process. According to this classification, skin lymphomas are divided into **slowly progressive monomorphic** and **polymorphic lymphomas**, and **rapidly progressive lymphomas**

Skin lymphomas with slow tumor progression. A. Monomorphic cell lymphomas: three-stage form; poikilodermic form with a variant of a universal lesion; nodular form; knotted form; spotted - infiltrative form; erythrodermic form; skin plasmacytoma. B. Polymorphocellular lymphomas: three-stage form; nodular form.

Skin lymphomas with rapid tumor progression: nodular-plaque (Gottron's reticulosarcomatosis); infiltrative - nodular form a) skin lymphosarcoma; b) erythrodermic form.

Clinical manifestations of skin lymphoma with slow tumor progression are nonspecific and are similar to **eczema, neurodermatitis, psoriasis, parapsoriasis** and other skin diseases, are characterized by a chronic and relatively benign course.

Morphological changes in the skin are manifested by the initial signs of impaired differentiation of lymphocytes. Tissue response to a developing tumor can be monomorphic (lymphocytes, histiocytes) and polymorphic (lymphocytes, histiocytes,plasma cells, eosinophils).

In this regard, all skin lymphomas with slow progression are divided into **monomorphic** and **polymorphic.** The pathological process may involve lymph nodes, internal organs, bone marrow, but the disease proceeds very slowly over many years and decades.

Monoform lymphomas of the skin include a three-stage monomorphic cell lymphoma, which manifests itself on the skin in three stages: patchy, patchy-plaque and tumor. In the spotted stage, spots of rounded outlines with clear boundaries appear on the skin of the trunk and proximal parts of the limbs.

The color of the spots is pale pink or brownish with a rough surface, which gives the impression of a grater due to the presence of follicular papules. There are no hairs in the lesions. In the future, due to the increase

in infiltration in the foci, plaques form and a spotty-plaque stage is formed, which is characterized by a long course and spontaneous remissions.

Gradually, the patchy-plaque stage can turn into a tumor stage; during morphological examination, lymphoblasts and prolymphocytes predominate in tumors.

The poikilodermic form proceeds for a long time and is benign. It is characterized by the appearance of dry, pale pink, slightly flaky lesions with indistinct edges in places subject to mechanical irritation. Over time, skin atrophy develops in the foci with telangiectasias, hair loss. Patients are concerned about the burning sensation, dryness and tightening of the affected skin.

The erythrodermic form begins on the limbs, then spreads throughout the skin in the form of hyperemia, edema, infiltration with peeling and severe itching. Large-lamellar peeling appears on the scalp, skin atrophy with hair loss. Lymph nodes are enlarged, dense. In the blood, leukocytosis, monocytosis, and accelerated ESR are noted. This form is prone to rapid generalization of the process.

The spotty-infiltrative form is more often localized on the skin of the chest and in the interscapular region in the form of bluish spots with clear boundaries, resembling the manifestations of spotted scleroderma. In the future, infiltration and cyanosis increase in the foci.

The nodular form is characterized by the appearance of dense cyanotic papules on the body, prone to grouping.

The knotty form is manifested by dense - cyanotic elastic nodes.

Plasmacytoma is characterized by single or multiple flat or nodular dark red infiltrates. Characterized by metastases to the lymph nodes and internal organs.

Polymorphic slowly progressive skin lymphomas are divided into three-stage and nodular forms. The three-stage form, previously described as mycosis fungoides, is T-cell lymphoma, since its association with T-lymphocytes has been proven. In its course, erythematous, plaque and tumor-like stages are distinguished.

The erythematous stage may present with erythema, blistering, psoriasis-like eruptions, and severe itching. After repeated remissions and relapses, the process enters the plaque stage. It is characterized by the

formation of infiltrative plaques of brick-red or bluish color, prone to peripheral growth and accompanied by severe itching. Skin atrophy remains in the lesions, hair falls out. Affected nails crumble, deform.

The duration of the first two stages of the disease is from 3 to 5 years.

The third stage is characterized by the formation of purple-red tumors with a bluish tinge. Tumors quickly increase in size and disintegrate, forming deep ulcers. Lymph nodes and internal organs are involved in the pathological process, the general condition of the body suffers.

Leukocytosis, monocytosis, eosinophilia develops, ESR increases. The nodular form begins with a rash of flat or hemispherical nodules of a cyanotic color, more often on the lower extremities, without subjective sensations. The nodules resolve spontaneously, leaving behind mild atrophy.

The remission period can be for a year or more. In the future, new nodules necrotic, leaving behind scars. The process can last for several decades, ending in death. Skin lymphomas with rapid tumor progression are characterized by a very high degree of malignancy and the formation of foci of decay on the rashes that have appeared.

Generalization of the process and death can occur within a few months of illness. The disseminated nodular-plaque form is localized on the trunk with the formation of cyanotic spots, against which plaques and nodes appear, and then rapidly decaying tumors. Very soon there comes a generalization of the process and a fatal outcome.

Infiltrative - nodular form begins with the appearance on the skin of the body of tumor- like formations of a bluish-red color with telangiectasias, prone to decay.

Lymphosarcoma of the skin is the most malignant form of lymphoma, characterized by the appearance of a single painless nodule of bluish-purple color with hemorrhagic rashes on its surface. It quickly ulcerates, metastasizes to the lymph nodes and internal organs. A few months later, death occurs.

The erythrodermic form refers to T - cell lymphomas, begins with the appearance of

severely itchy erythematous spots on the extremities, which quickly spread throughout the skin, turning into erythroderma. The subcutaneous lymph nodes are enlarged.

In the blood, mononuclear cells of white blood cells are found with a large basophilic nucleus, occupying about 4/5 of the volume of the cell - **Cesari cells**. Erythroderma with marked enlargement of the lymph nodes and the presence of these atypical cells in the peripheral blood is called **Cesari's syndrome**.

Diagnosis of skin lymphomas is based on clinical manifestations, on the data of mandatory morphological and cytological studies.

The treatment of lymphomas is one of the most difficult problems and depends on the form and stage of the disease, clinical and morphological features of the skin process.

In order to slow down the progression of the disease and curb the growth of pathological cells, hyposensitizing therapy (calcium preparations, sodium thiosulfate), antihistamines (tavegil, suprastin, phencarol, ketotifen, peritol, etc.), general strengthening (vitamins B, C, aloe extract, FIBS and etc.), adaptogens (Eleutherococcus extract, tincture of ginseng, Schisandra chinensis, aralia, etc.), immunomodulators (thymogen, sodium nucleinate, pentoxyl, methyluracil, thymalin, taktivin, immunoglobulin, etc.), enterosorbents (activated charcoal, polyphepan, smecta, belasorb, etc.), detoxifying agents (hemodez, polyglucin, trisol, etc.).

With pronounced clinical and morphological manifestations and an increase in signs of impaired cell differentiation, glucocorticoids (dexamethasone, triamcinolone, prednisolone, polcortalone), cytostatics (prospidin, methotrexate, 6-mercaptopurine, cyclophosphamide, vinblastine, etc.) are connected to the treatment.

External drug treatment consists in the use of antipruritic creams, corticosteroid and cytostatic ointments. Appropriate treatment must be prescribed to prevent complications from ongoing therapy. Patients with all forms of skin lymphoma are subject to dispensary observation.

A disability examination is carried out by the MREC at the patient's place of residence, which determines the disability group.

CHEILITS

Classification Cheilitis is divided into true (contact, actinic, exfoliative, glandular) and symptomatic (atonic, drug-induced, infectious, Melkersson-Rosenthal syndrome, cheilitis in various dermatoses). **CONTACT CHEILITS** is an inflammatory disease of the red border of the lips resulting from direct contact of the lips with various exogenous factors.

Simple contact cheilitis is an inflammatory skin disease of the red border of the lips that occurs in response to a single contact with an obligate (suprathreshold) irritant.

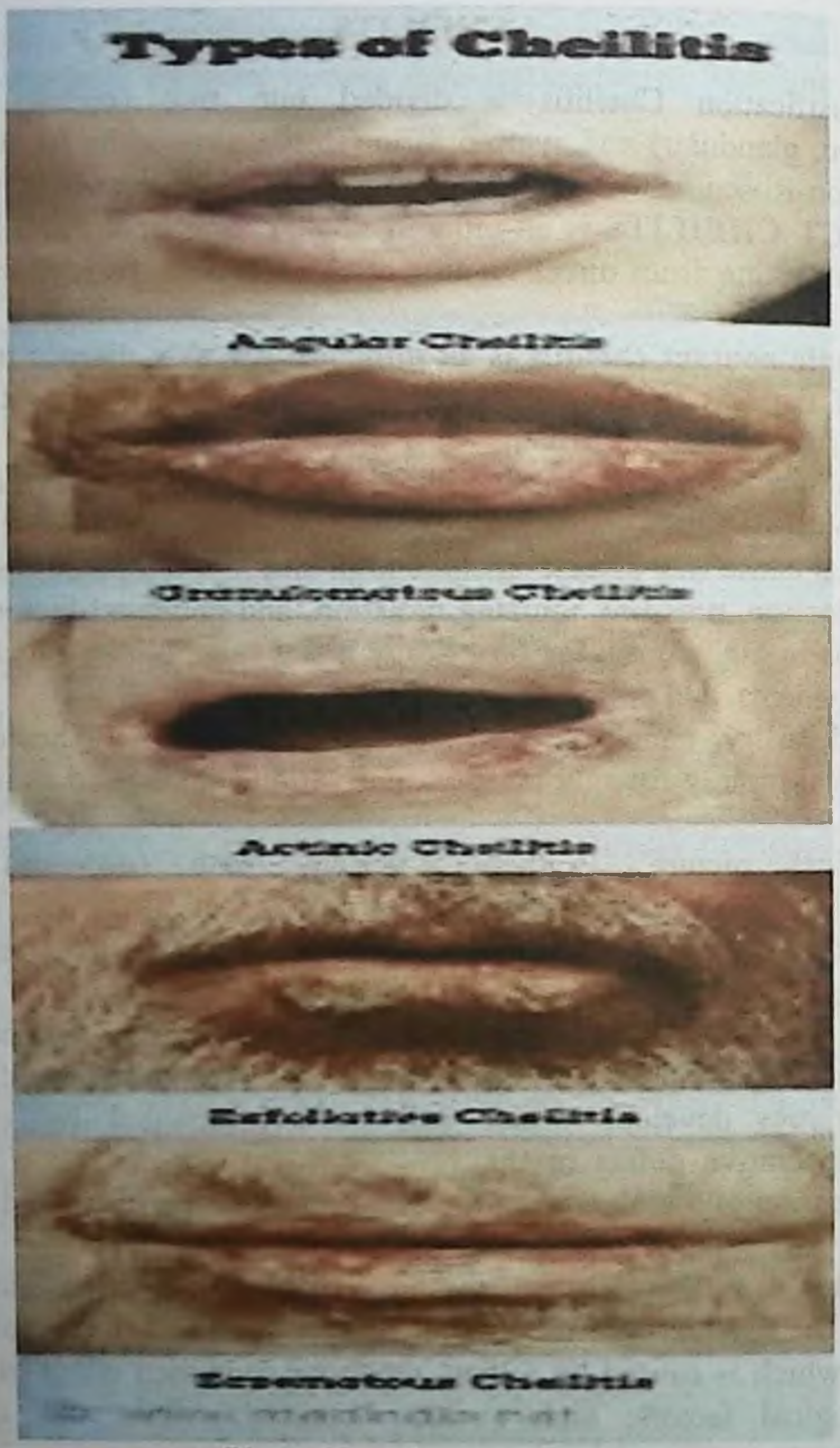
Etiology and pathogenesis. Simple contact cheilitis is caused by various obligate stimuli of a physical and chemical nature. The physical factors that cause this disease include high and low temperatures, electric current, friction, pressure, ionizing radiation, and chemical factors - acids and alkalis in high concentrations, heavy metal salts, blister agents, rocket fuel components. and other substances.

In dentistry, simple cheilitis occurs on contact with heated dental instruments, rapidly rotating nozzles, and various acids used in root canal treatment.

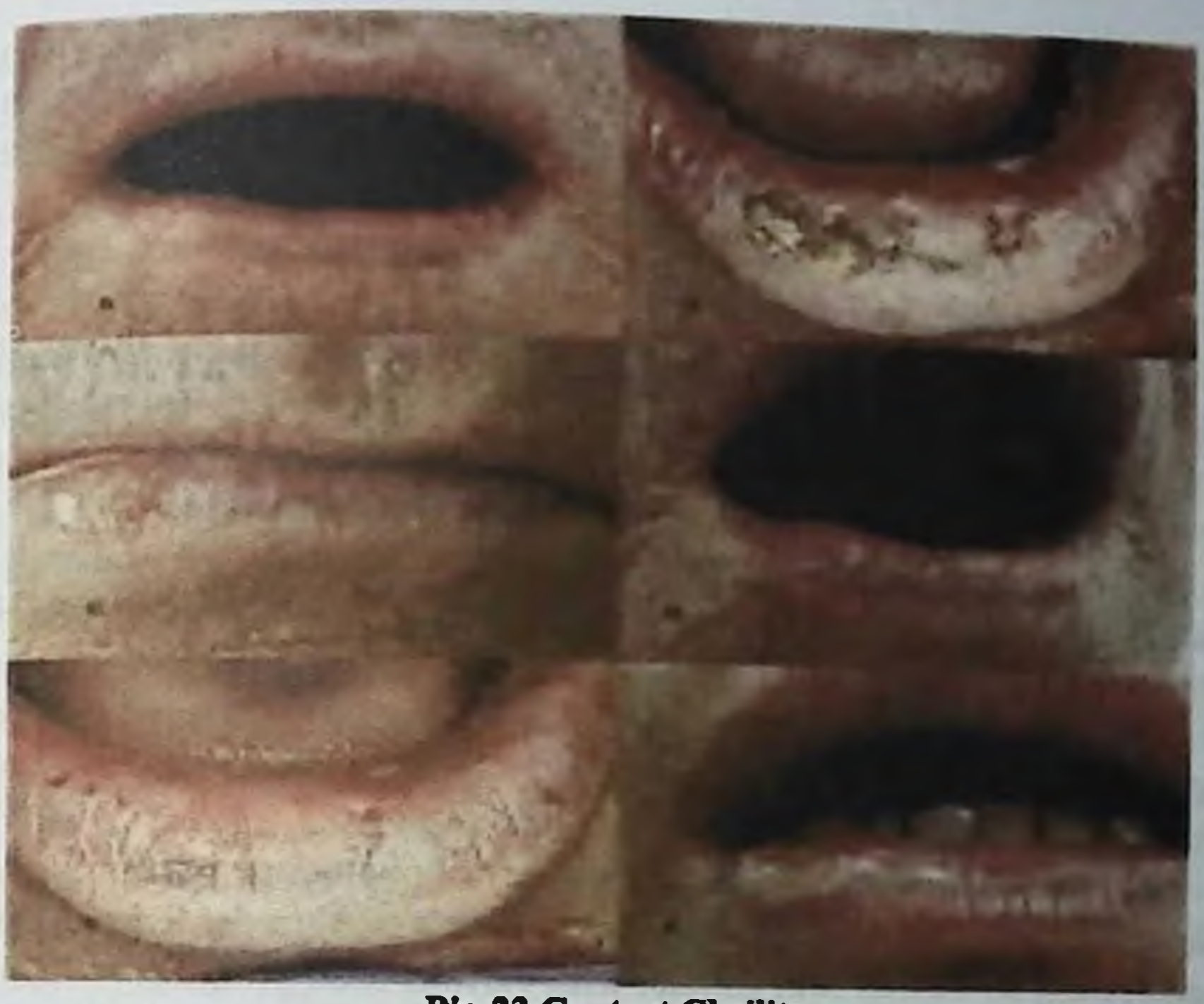
clinical picture. Erythema, vesicle, scab (necrosis) appear sequentially with clear boundaries of lesions, repeating the area of action of the damaging agent. The severity of clinical manifestations depends on the strength and duration of the damaging obligate factor. Patients complain of soreness and burning. In the case of simultaneous contact of the mucous membrane of the lips with a suprathreshold irritant, contact stomatitis may develop, which will be accompanied by erythema, erosive, ulcerative defect or the

formation of a scab against the background of a pronounced mucosal edema.

In this case, the clarity of the boundaries of the lesions can not be detected. A special case of simple contact cheilitis is meteorological cheilitis, which is caused by a long, repetitive combined effect of adverse meteorological factors: high or low humidity, wind, cold, air dust, insolation, etc.



Pic-22 Classification Cheilitis



Pic-23 Contact Cheilits

The flow is seasonal. The red border of the lower lip is usually affected. Erythema develops, slight infiltration, fine-lamellar peeling in the area of the red border of the lips. Patients are concerned about dryness, a feeling of tightening of the skin of the lips.

Diagnosis is based on anamnesis data, complaints, as well as the detection of characteristic rashes with clear contours.

Treatment. Establish the cause and stop the impact of the damaging factor. External anti- inflammatory therapy is indicated. With erythema and bladder, creams with topical glucocorticoids are prescribed for a period of 5-7 days. In the case of a secondary infection, combined preparations are indicated, including a corticosteroid and an antibacterial or disinfectant agent.

With a scab, disinfectant or antibacterial ointments are shown, with which you can carefully remove crusts and necrotic masses, in the future - ointments and gels that stimulate regenerative processes. In case of

meteorological cheilitis, oily and moisturizing lipsticks, wax ointment (contains cocoa butter, paraffin, lanolin, petroleum jelly), photoprotective creams, and lipsticks with a photoprotective effect (UVA and UVB filters) are recommended.

It is important to minimize the effect of the factors that cause the disease. With severe inflammation, short-term (for 1-2 days) use of a non-fluorinated corticosteroid cream is possible. Inside, B vitamins, nicotinic acid can be prescribed. **Allergic contact cheilitis** is an inflammatory skin disease of the red border of the lips that occurs in response to repeated (more than 2 times) contact with an optional irritant (allergen).

Etiology and pathogenesis. The disease develops as a result of sensitization of the red border of the lips to various chemicals and is a manifestation of an allergic reaction of the fourth type. The reason may be low molecular weight substances that easily penetrate tissues (haptens).Sensitization may develop over several days or years. Further cross- reactions are also possible.

Allergic cheilitis can be caused by the following agents: components of lipsticks, toothpastes and solutions, substances used in dentistry (epimin, eigenol, etc.), as well as skin contact with various food products (citrus and other exotic fruits), wind instrument mouthpieces, cigarettes, chewing gum, etc.

Clinical picture. At the site of contact with the allergen, edematous erythema develops with fairly clear boundaries. Against the background of erythema, the formation of small vesicles is possible, which quickly open with the formation of scale-crusts. With a long course, the red border of the lips becomes dry, multiple small grooves and cracks appear on it, and the focus can go beyond the red border. Then on the surrounding skin there is a fuzziness of its boundaries. Patients are concerned about itching and burning of the lips.

Diagnosis is based on the characteristic data of the anamnesis and the clinical picture. To clarify the allergen, skin-allergic tests are shown.

Treatment. After the obligatory elimination of contact with the allergen, lotions with astringents and anti-inflammatory agents (1% tannin, boric acid, etc.), non-halogenated corticosteroid creams (for 3-5 days) are recommended. When the process is torpid, systemically hyposensitizing

drugs can be prescribed. In the future - "basic" care, the selection of hypoallergenic lipstick or toothpaste.

Actinic cheilitis is an inflammatory disease of the skin of the red border of the lips, resulting from insolation and related to photoallergic reactions.

Etiology and pathogenesis. The disease is caused by acute or chronic ultraviolet exposure. Various substances can act as an exogenous photosensitizer (eosin, which is part of lipsticks, bergamot oil, etc.). clinical picture. Allocate **exudative** and **dry** forms of the disease. Both forms are considered precancerous conditions. The lower lip is most commonly affected. The disease is prone to exacerbation in spring and summer, in winter there may be a partial or complete remission.

The exudative form does not differ from the manifestations of allergic contact cheilitis. With prolonged existence, persistent cracks, erosion, ulcers can form.

In **the dry form**, the red border becomes bright red, covered with small silvery scales. A fuzzy border between the mucous membrane of the lip and the red border, as well as between the skin and the red border of the lips, is characteristic.

Gradually, against the background of chronic erythema, small whitish nodules may form, less often - warty growths.

Diagnosis of actinic cheilitis is based on a characteristic history of the disease, the seasonality of the course. To exclude malignancy, cytological and histological studies are carried out.

Treatment and prevention. It is recommended to avoid sun exposure and not to use lipsticks containing eosin. For photoprotection, sunscreens with a high sun protection factor (SPF) or sunscreen lipsticks are prescribed. Nicotinic acid preparations, antimalarial drugs, calcium preparations are shown. Externally, emollients and moisturizers are used for the lips. For patients with actinic cheilitis, it is necessary to establish dispensary observation.

EXFOLIATIVE CHEILITIS is an inflammatory disease of the red border of the lips with a genetic predisposition, belonging to the group of psychosomatic dermatoses.

Etiology and pathogenesis. Exfoliative cheilitis occurs in individuals with a tendency to constantly lick their lips, which in some cases is an obsessive action. There are familial

cases. Psychoemotional factors play an important role in pathogenesis.

Anxious (with exudative form) and **depressive** (with dry form) conditions are revealed. Most patients also have mild **thyrotoxicosis.**

clinical picture. In both forms of exfoliative cheilitis, the process is localized on half of the red border of the lips, adjacent to the mucous membrane.

In **the exudative form,** crusts of a grayish-yellow or yellowish-brown color appear in the indicated localization. After removing the crusts, a smooth red, slightly damp surface is exposed. The mucous membrane in the Klein zone is moderately hyperemic and edematous, it can be covered with an easily removed grayish-white coating. Patients are concerned about burning, soreness and gluing of the lips.

The dry form is characterized by thin, light yellow or pale gray, resin-like scales, firmly attached in the center. When removing the scales, a dry hyperemic red border of the lips isexposed.

Diagnosis is based on the typical clinical presentation and behavioral characteristics of patients. Examination of thyroid function and consultation of a psychoneurologist are necessary.

Treatment. Psychotherapeutic correction, treatment of thyrotoxicosis are shown. Fatty and moisturizing creams and ointments are used, as well as lipsticks with a similar effect.

Glandular chellitis is a chronic inflammatory disease of the red border of the lips, resulting from heterotopia, hyperplasia and hyperfunction of the small salivary glands or their ducts in the area of the red border and Klein's zone.

This anomaly of the salivary glands usually appears after the end of puberty. Etiology andpathogenesis. The disease provokes the pathology of the gums and dentition. It is possible to develop secondary glandular cheilitis against the background of a number of chronic dermatoses (**lupus erythematosus, lichen planus,** etc.

Clinical picture. In the area of the transition of the mucous membrane to the red border ofthe lips, sometimes on the red border of the lips, dilated mouths of the salivary glands are visible in the form of red dots, from which secrete droplets of saliva.This is clearly visible 10-20 s after the drying of the lesion.

With a long course, **leukoplakia** develops around the mouths of the salivary glands in the form of whitish rings.Frequent wetting of the red border of the lips with saliva contributes to dryness and leads to chronic cracks.There is also a complicated form glandular cheilitis

- Volkman's hashgyt.

At the same time, the lips increase significantly in size, their entire surface is covered with scales and crusts, when removed, dilated excretory ducts of the glands are visible, from which pus is exuded.Regional lymphadenitis is characteristic, there may be symptoms of intoxication.

Patients complain of increasing sensitivity and soreness. Abscesses of the lips may form,

followed by the formation of fistulous openings. In 20-30% of cases, Volkmann's cheilitis transforms into squamous cell carcinoma.

Diagnostics. A typical clinical picture and the release of drops of salivary gland secretion after drying the lesion are taken into account. If necessary, a histological examination is performed, which reveals the presence of heterotopic salivary glands.

Treatment. Heterotopic salivary glands are removed surgically or by electrocoagulation, laser destruction. With secondary glandular cheilitis, therapy of the underlying dermatosis is indicated. With Volkman's cheilitis, broad-spectrum antibacterial drugs are indicated; when abscesses form, they are opened.

Outwardly, ointments containing disinfectants or antibiotics are recommended. After the regression of the purulent process, it is necessary to remove the heterotopic salivary glands.

SYMPTOMATIC CHEILITS

Atonic cheilitis is one of the symptoms of atopic dermatitis and may be its only manifestation in remission. clinical picture. It is more common in children and adolescents. The red border of the lips and the surrounding skin are affected. Most often, the process is more pronounced in the corners of the mouth, and the part of the red border adjacent to the mucosa remains unaffected.

Erythema appears with fairly clear boundaries, moderate swelling of the skin and the red border of the lips is possible. In the future, as acute inflammation subsides, lichenification is formed. The red border is infiltrated, flaky, and multiple thin radial grooves form on its surface. Accompanied by itching.

After the exacerbation of the disease subsides, infiltration and small radial folds in the corners of the mouth may persist for a long time.

Diagnosis of atopic cheilitis is based on a characteristic atonic history, identification of other manifestations of atopic dermatitis.

Treatment. A complex therapy of atopic dermatitis is carried out in accordance with the age phase and the severity of the exacerbation. Outwardly, in the acute phase, corticosteroid creams or ointments are indicated. In the future - fattening and moisturizing creams, ointments and products for the care of the red border of the lips.

Drug-induced cheilitis is a collective group of various lesions of the red border of the lips that occur when taking medications. The most common manifestation of drug- induced cheilitis **is toxicoderma.**

In addition, it is also possible to change the skin of the red border as a result of the side effects of a number of drugs (for example, dry lips and cracks during therapy with synthetic retinoids).

clinical picture. Toxicoderma on the lips can manifest as giant or **ordinary urticaria, fixed erythema, bullous toxicoderma, polymorphic exudative erythema, Stevens-**

Johnson syndrome and Lyell's syndrome.

Diagnosis of drug-induced cheilitis, which is a manifestation of toxicoderma, is based on anamnesis data, combination with other manifestations of toxicoderma, positive vitral tests with allergens.

Treatment. Carry out complex therapy of toxicodermia. On the lips in the presence of erosive defects, aqueous solutions of aniline dyes, combined preparations, including glucocorticoids and antibacterial agents, agents that accelerate regeneration, are prescribed.

In the presence of crusts, ointments with disinfectants and antibacterial agents are recommended. The choice of any external preparations should be carried out with caution, taking into account individual tolerance.

INFECTIOUS CHEILITIS is a collective group of diseases with lesions of the red border of the lips with various infectious agents (streptococci, yeast-like fungi of the genus Candida, herpes simplex virus, pale treponema, mycobacterium tuberculosis). "cheilitis).

Streptococcal seizure (impetigo ftssurica, sea angulus infectiosus) is a type of **streptococcal impetigo**, characterized by the appearance of conflicts in the corner of the mouth, which quickly opens with the formation of erosion and a linear crack covered withyellowish-gray crusts.

In **primary syphilis** on the red border of the lips, the primary syphiloma occurs in the form of an erosion or ulcer with a densely elastic infiltrate at the base, often covered with a tightly seated crust, sometimes it hypertrophies.Often there are cracks with bleeding andsoreness.

When located in the corners of the mouth, the primary syphiloma has a slit-like character.Regional lymph nodes are enlarged, densely elastic consistency, painless on palpation, not soldered to the skin and surrounding tissues.

With secondary syphilis, papules can be localized on the lips, oral mucosa and in the corners of the mouth. The most common manifestation is syphilitic seizure: in the corner of the mouth, a slit-like erosion is formed with an infiltrate at the base, covered with a hemorrhagic crust.

With **candidal chellitis**, there is moderate swelling and cyanosis of the red border of the lips, as well as thin grayish lamellar scales with raised edges, thinning of the skin, radial grooves, and cracks. Subjectively worried about dryness, slight burning, sometimes soreness.

With candidal macrochellitis, the lips thicken significantly, thick crusts and bleeding cracks appear on their surface.

In patients with **candidal stomatitis,** the process often extends to the corners of the

mouth. Perhaps an isolated lesion of the corners of the mouth (candida, or yeast, jamming): limited erosion / cracks on a slightly infiltrated base, surrounded by a fringe of slightly raised, whitened epidermis. The appearance of jamming is promoted by maceration of the corners of the mouth, which occurs with an incorrect bite.

Herpes simplex (herpes symplex) on the lips is accompanied by their swelling, the appearance of grouped vesicles. After the opening of the vesicles, erosions with small- cyclic outlines are formed, covered with hemorrhagic, less often. Lip lesions in various dermatoses

In discoid and disseminated lupus erythematosus, four forms of lesions of the red border of the lips are distinguished: a typical form without clinically pronounced **atrophy and hyperkeratosis, an erosive-ulcerative, deep form of Kaposi-Irgang.**

The outflow of the focus beyond the red border of the lips is characteristic. With a typical form, erythema, hyperkeratosis and atrophy occur. Gradually, an area of atrophy is formed in the center of the focus, along the periphery of which there is an inflammatory roller.

A diffuse lesion is possible, in which the entire red border of the lip is saturated red, moderately infiltrated, covered with dense grayish scales. When scraping and removing scales, pain occurs (positive symptom of Besnier-Meshchersky).

The form of lesions of the red border of the lips without severe atrophy. Characterized by erythema and slight hyperkeratosis, occasionally - infiltration and telangiectasia. The defeat of the red border of the lips is often diffuse.

Erosive and ulcerative form. The lesion is localized on the lower lip. Erythema and edema are characteristic. In the future, cracks, erosions, ulcers are formed, covered with serous or serous-hemorrhagic crusts. Erosions are located on a moderately infiltrated base, hyperkeratosis and atrophy are revealed along the periphery of erosions.

Deep form of lupus erythematosus Kaposi-Irgang. A lesion in the form of a node protruding above the surface of the skin. On the surface of

erythema, there may be moderate peeling. The diagnosis is usually confirmed histologically.

Diagnosis is based on the identification and evaluation of the entire complex of clinical manifestations. It is mandatory to examine the patient to exclude the systemic nature of the process and for the presence of foci of focal infection.

Assign antimalarial drugs, preparations of nicotinic acid, ascorbic acid, calcium. Outwardly, only short courses of topical glucocorticoids are possible due to the risk of their atrophogenic effect, which can aggravate cicatricial atrophy after resolution oftypical lupus erythematosus lesions.

Adequate photoprotection (high SPF sunscreens or sunscreen lipsticks) is essential to prevent recurrence.

With **lichen planus**, five clinical forms are distinguished: **typical, hyperkeratotic,**

erosive-ulcerative, pigmentary, bullous.

Unlike the manifestations of lupus erythematosus, it is not typical for the lesions to go beyond the red border of the lips. For a typical form, the most characteristic is the appearance of small (up to 2 mm) nodules of a whitish-pearl color, which merge with each other, forming a bizarre pattern (fern leaf, mesh, etc.).

In patients with a **hyperkeratotic form**, keratinization foci with sharp boundaries may form.

With an **erosive-ulcerative form**, erosions appear, less often ulcers. Subjectively, patientsare concerned about soreness when taking food.

In the **pigmented form**, against the background of typical rashes, a characteristic persistent grayish-brown pigmentation is formed.

In the **bullous form**, along with typical rashes, small blisters are formed, opening with the formation of fairly rapidly epithelialized erosions.

The diagnosis is established on the basis of the detection of typical rashes on the smooth skin and oral mucosa, in some cases a histological examination is performed.

Treatment. Inside prescribe antimalarial drugs. Outwardly - short courses of external glucocorticosteroids, emollients, moisturizers for lip care.

Cheilitis in congenital and **ichthyosis vulgaris** always occurs against the background of other manifestations of these diseases. Peeling of the lips, smoothness of the contours of the red border, cracks are characteristic. In patients with congenital ichthyosis, after birth, numerous deep cracks are detected in the area of the corners of the mouth and on the red border of the lips, which leave cicatricial changes after healing, leading to eversion of the mouth opening.

Diagnosis is based on the detection of clinical signs of ichthyosis.

Treatment. Moisturizing and fattening lipsticks are recommended against the background of rational therapy for ichthyosis.

Test tasks

1 Microbial eczema is characterized by: 1. asymmetry of foci 2. clear boundaries 3. symmetry of foci

2. Frequent localization of microbial eczema: 1. face 2. trunk 3. lower limbs

3. In the treatment of eczema, they use: 1. antihistamines 2. desensitizing drugs 3.vitamins

4. Factors contributing to the development of occupational skin diseases: 1. resins 2.solvents 3. alcohol 4. lime

5. Varieties of occupational dermatoses caused by industrial allergens: 1. occupational eczema 2. occupational toxicoderma 3. occupational urticaria 4. pink lichen

6. Occupational dermatoses caused by infectious and parasitic agents: eryzepeloid 1. deeptrichophytosis 2. lichen planus 3. candidiasis

7. Industrial allergens can be: 1. synthetic glue 2. antibiotics 3. paints 4. epoxy resins

CHAPTER 4 SEXUALLY TRANSMITTED INFECTIONS. GENERAL COURSE OF SYPHILIS, MICROBIOLOGY AND EPIDEMIOLOGY OF SYPHILIS, ETIOPATOGENESIS. PRIMARY SYPHILIS. CLINICAL-LABORATORY AND SEROLOGICAL DIAGNOSIS.

GENERAL INFORMATION ABOUT SYPHILIS

The term "**venereal disease**" was first used by the French physician Jagues de Bethencourt in 1527. Speaking of syphilis "morbus venereus s. lues venera", he emphasized the predominantly sexual transmission of the disease. The sexual way of spreading syphilis was recognized by contemporaries very quickly.

French physician Jean Fernel (Jean Fernel, 1497-1559) wrote about "animal venereal poison" that penetrates first into the genitals, and then spreads throughout the body. However, the founder of the doctrine of syphilis is considered the Italian scientist, personal physician of Pope Paul III and friend of Copernicus - Hieronymus Fracastorius Veronensis (1478-1553), who in his poem "Syphilis, or the Gallic Disease" (Syphilis, sive morbus gallicus, 1530) described a swineherd named Siphilus (sus - pig, philos - addiction) who lived in a mythical country, punished by this disease by the gods: "This is a consequence of the crime of the shepherd Siphil, who erected an altar in the forbidden forest and made an inappropriate sacrifice to the gods."

The poem also describes methods of treatment, including sulfur and mercury-containing substances. Fracastorius (1546) emphasized that the causative agent of syphilis is transmitted mainly through sexual contact, although children can become infected from their mothers.

The name "syphilis", proposed by Thracastorius, quickly supplanted the diverse designations of this new disease for Europe, called either by country (Spanish, Portuguese, Venetian, Polish, French disease), or by the name of the patron saint of a hostile country (the disease of St. Job, St. Semant, St. Maine, St. Möbius, etc.).

There were more than 300 names that reflected the geographical course of the epidemic. Currently, in addition to the term syphilis, the name "**lues**" (from Latin - plague, plaque, infection, pestilence, death,

sexual plague, great pox), proposed in 1554 by the French physician Jean Fernel (lues venerea) is sometimes also used.

One of the first problems that interested venereologists was the study of the origin of syphilis. It should be noted that so far there has not been a consensus on this issue. Scientists-"Europeanists" believe that syphilis has always existed in Europe, that it is as "old as humanity itself", as evidenced by the mention of symptoms similar to syphilis in various written sources of antiquity, as well as the finding of syphilitic-like changes in the bones found during archaeological excavations.

In the works of the great scientists of antiquity Hippocrates, Galen, Celsus, Avicenna,

Plutarch, lesions of the skin and mucous membranes are described, resembling hard chancre, wide condylomas, papular syphilides, gummas. "Africanist" scientists T. Cockburn and E. Hudson claim that Africa is the birthplace of syphilis, where endemic non-venereal treponematoses (yaws, pint, bejel) are still common, the pathogens of which are morphologically close to the causative agent of syphilis and have existed on earth for thousands of years.

They believe that non-venereal treponematoses that came from Africa to Europe during the wars, the slave trade, took on a venereal character due to the influence of climate and other social and living conditions.

According to another hypothesis, expressed in 1525 by Oviedo, in 1492 the expedition of Christopher Columbus went in search of a sea route to India. Instead, they traveled to the Caribbean, from where they brought syphilis to Europe. Proponents of this theory suggest that syphilis originated from the sexual spirochetosis of South American llamas and, as a result of bestiality, passed to humans.

A syphilis epidemic broke out in Europe shortly after the return of the Columbus expedition on March 15, 1493. The doctor of the expedition of Christopher Columbus in his diary describes a new disease among sailors, which they contracted on the islands of Hispaniola (Haiti and the Dominican Republic), where, according to the natives, it has always existed.

An important factor in the spread of syphilis in Europe was the campaign against Naples of Charles VIII, in whose mercenary army there

were members of the expedition of Christopher Columbus. In the first years after the start of the syphilis epidemic in Europe, considerable attention was paid to the study of its clinical signs.

Lesions of the skin, mucous membranes, internal organs, and bones have been described. In the 16th, 17th and early 18th centuries all lesions of the genital organs were considered syphilitic. For about two centuries, the Unitarian doctrine of the identity of the "poisons" of gonorrhea, syphilis and chancre dominated.

In the middle of the XVIII century. there are works "in which the dogma of the unitarians was rejected: syphilis and gonorrhea are considered by dualists as different diseases. However, the English surgeon Hunter in 1786 instilled on the head of the penis and on the foreskin discharge from the urethra of the patient, as he thought, with gonorrhea, and he developed manifestations of syphilis.

Apparently, the patient-donor suffered from both syphilis and gonorrhea. Only in the 30s of the XIX century. the dispute between unitarians and dualists was finally resolved by Ricord. Infecting 1400 healthy people, he proved that syphilis and gonorrhea are different diseases. In 1879 Neisser discovered the causative agent of gonorrhea, and only in 1905 Sghaudin and Hoffmann discovered treponema pallidum. In 1903 I.I.

Mechnikov and E. Roux (Roux), experimenting on monkeys, received a positive

inoculation of syphilis in chimpanzees, and syphilis in monkeys arose and proceeded identically to human syphilis. In 1904 D.K. Zabolotny received experimental syphilis from another great ape, a baboon. In 1906, Wasserman, Neisser and Brook proposed a serological diagnosis of syphilis, based on the complement fixation reaction.

For a number of centuries, syphilis was treated with mercury preparations. In 1836, Wallace introduced iodine preparations into the practice of treating syphilis. In 1909, Erlich and Hata synthesized salvarsan, calling it "606" or "silver bullet", and in 1913 - neosalvarsan. In 1921, Levaditi and Sazerac began to use bismuth preparations, and in 1943 Mahoney, Arnold and Harris proved the high therapeutic efficacy of penicillin, the preparations of which are still used today.

The spread of syphilis in Russia dates back to the end of the 15th and the beginning of the 16th century. The socio-economic conditions of that time: serf labor, the servitude and powerless position of women, prostitution and other conditions of feudal society, as well as the lively trade relations between Russia and the cities of Western Europe, contributed to the rapid spread of syphilis in Russia.

Already in the first years of the existence of St. Petersburg, syphilis and gonorrhea, or, as they said then, "fracture", became so widespread that Peter I ordered the police to catch "walking girls" in the city and imprison them in the so-called "spinning houses", where they worked. spinning.

A great contribution to the study of syphilidology was made by the founders of Russian medicine: M.Ya. Mudrov, N.I. Pirogov, I.E. Dyadkovsky, G.A. Zakharyin, S.P. Botkin,

A. A. Ostroumov and others.

Scientific venereology in Russia originated within the walls of therapeutic and surgical clinics. The founder of the scientific school of venereology in Russia was V.M. Tarnovsky (1837-1906), who headed the first department of syphilidology at the Medico- Surgical Academy. On the initiative of V.M. Tarnovsky with the support of A.G. Polotebnov in St. Petersburg, the first special scientific society in Europe was founded: the Russian Syphilidological and Dermatological Society (now named after V.M. Tarnovsky).

The history of syphilis knows great victories and the bitterness of defeat, this infection claimed hundreds of thousands of lives, did not spare even small children. Modern advances in epidemiology, diagnosis, treatment and follow-up can save patients from severe, sometimes fatal outcomes.

Etiology. The causative agent of syphilis - **pale treponema (treponema pallidum)** - was discovered in 1905 by F. Shaudin and E. Hoffmann.

Pale treponema can be found in discharge from hard chancres, erosive papules, in lymph nodes, in the blood of patients, in all exudates in patients with active secondary syphilis,

in cerebrospinal fluid, milk of nursing mothers, tonsils and mucus of the cervical canal plug of patients with latent syphilis, in the head brain in patients with progressive paralysis and spinal cord in patients with tabes.

Urine, tears, sweat, saliva do not contain pale treponemas, but they can become infected in the excretory tract.

Pale treponema has three forms of existence: **spiral, encysted and L-shaped.**

In infectious forms of syphilis, the spiral form is dominant. The number of turns in pathogenic strains is usually 6-8. Each turn of pale treponema repeats the structure of the main morphological structures (fibrils, membranes, segments of the protoplasmic cylinder) of the previous turn.

The main way of reproduction of pale treponema in the stage of active growth is transverse division. The principle of morphological symmetry ensures the reproduction of the same type of spiral specimens during transverse division. Along with transverse division, spiral-shaped microorganisms have forms of sustainable reproduction - cysts, which allow pale treponema to survive in adverse conditions.

Cyst formation is not the only form of preservation of the pathogen in the body during the latent period or in case of unsuccessful treatment; in these cases, L-transformation of the microbial cell is possible.

With L-transformation, there is a partial or complete loss of the cell wall, a decrease in metabolism and a violation of cell division processes. They have negligible pathogenicity, do not cause obvious manifestations of syphilis, but may maintain serological resistance. Subsequently, when favorable conditions for their life appear, they reverse into their usual mobile forms and sometimes cause a relapse of the disease. Cysts have antigenic properties, contribute to the formation of various antibodies in the human body, which is detected by positive serological tests.

In L-forms, antigenic properties are slightly or absent, therefore, persons in whose body pale treponemas have L-forms are serologically negative or, with negative standard serological reactions, RIBT and RIF may be positive. The only reservoir of pale treponema in nature is a sick person. The resistance of pale treponema to external influences is low. It dies quickly when it dries out, but on wet linen it can live up to 11 hours.

Frozen pale treponemas retain viability and virulence for a long time. First, virulence disappears, and only then mobility. In the tissues of corpses, pale treponemas can remain mobile for up to 48 hours. The optimum temperature for it is 36.5-37 ° C. Treponema pallidum dies within 15 minutes at 55°C. It is very sensitive to various chemicals and oxygen (it is a facultative anaerobe).

General pathology of syphilis. To infect a person with syphilis, the penetration of pale treponema into the skin or mucous membrane with impaired integrity is necessary.

Occasionally, infection with syphilis can occur when pale treponema is introduced directly into the blood (in doctors with cuts and injections during surgery, in patients with blood transfusions).

The source of infection with syphilis is a sick person. Particularly contagious are patients who have the following manifestations of syphilis: erosive or ulcerative hard chancre, erosive papules on the skin and mucous membranes, hypertrophic papules (**condylomas lata**).

In pustular syphilides, there are much fewer pale treponemas, they are located more deeply. Gummas and tubercles of the tertiary period are practically non-infectious, pale treponemas in small quantities are found only in the marginal zone of the undecayed infiltrate.

Ways of transmission of the disease: sexual, household, transfusion and transplacental. In 99.8% of cases, the infection is transmitted sexually. Non-sexual infection is possible in the performance of professional duties (medical workers), through kisses, as well as indirect (general utensils, lipstick, smoking pipes, wind musical instruments, etc.).

Most often, the household transmission of syphilis is a poorly collected anamnesis. With the transfusion route of infection, treponema immediately enters the bloodstream, and clinical manifestations occur without primary syphiloma, **"headless syphilis" (syphilis d'emblee)** develops. After about 2 months, rashes of secondary fresh syphilis appear.

With congenital syphilis, infection of the fetus by a sick mother occurs transplacentally, usually not earlier than the 16th week of pregnancy, after the formation of the placenta. The incubation period for syphilis lasts an average of 3-4 weeks from the moment of infection until the first visible manifestations of syphilis appear.

However, in some patients it can reach 4-6 months due to the uncontrolled use of antibiotics for various diseases, while there may be a "headless course of syphilis." The shortening of the incubation period to 10-14 days is possible with the development of bipolar chancres.

The first clinical sign of the disease is a hard chancre that appears at the place where pale treponema has entered the body (according to the figurative expression of the French, "with syphilis, the place that sinned is the first to be punished"). From the time of the formation of a hard chancre until the appearance of a rash of secondary fresh syphilis, 6-7 weeks pass, corresponding to the primary period of syphilis, in the first half of which serological reactions are negative, in the second - positive (according to the Wasserman reaction).

The secondary period occurs on average 9-10 weeks after infection, i.e. 6-7 weeks after the appearance of a hard chancre and lasts 3-4 years.

Secondary syphilis is subdivided into secondary fresh syphilis, when abundant bright rashes appear on the skin and mucous membranes and at the same time, patients have a

hard chancre that has not yet healed or a fresh mark after it (pigmented spot or scar) and **regional scleradenitis**; secondary recurrent syphilis - the period of subsequent repeated rashes and secondary latent syphilis, in which there are no active manifestations on the skin and mucous membranes.

The more time has passed since the onset of the disease, the longer the latent stage. With **secondary recurrent syphilis**, the number of rashes with each subsequent relapse becomes smaller, and the elements themselves become larger, prone to grouping, merging, localized in folds and around natural openings. If the patient does not receive treatment or it is not enough, then after 3-4 years, the tertiary period of syphilis may occur.

This period is characterized by the formation of **tertiary syphilides (tubercles and gummas)**. Gummas can form on the skin and mucous membranes, in the subcutaneous tissue, bones, internal organs and the nervous system. The tertiary period of syphilis can last for many years.

In the occurrence of tertiary syphilides, great importance is attached to trauma in the broad sense of the word (physical, medical, mental),

factors that weaken the body's defenses (chronic infections, intoxications, including alcohol, severe somatic diseases).

Poorly or completely untreated patients may develop various forms of neurosyphilis (meningovascular syphilis, dorsal tabes, progressive paralysis), as well as syphilis of internal organs (mesoaortitis, aortic aneurysm, liver damage, etc.). The course of the disease depends on the characteristics of the reactivity of the body, concomitant diseases (tuberculosis, alcoholism, AIDS, etc.), the quality of the treatment, socio-economic conditions, etc. Immunity with syphilis.

With a syphilitic infection, non-sterile infectious immunity develops in the patient's body, which occurs as a response of the body to the presence of a pathogen in it and exists as long as there is a pale treponema in the body. In response to the presence of pale treponema (antigen), various antibodies are formed - immunoglobulins.

The humoral response to the introduction of pale treponema into the macroorganism begins with the production of specific IgM antibodies, later the content of IgG and IgA increases. The presence of specific IgM immunoglobulins is considered as a sign of the onset of the disease.

Detection of treponema-specific IgM makes it possible to diagnose an acquired infection as early as possible, since these immunoglobulins appear as early as 2 weeks after infection. Detection of IgM is of great importance in the incubation period and in the early stages of the development of a syphilitic infection.

Class G antibodies appear no earlier than 4 weeks after infection. The predominance of the level of IgG over the level of IgM occurs with massive hematogenous dissemination of pale treponema (**secondary fresh syphilis**).

Activation of a syphilitic infection, accompanied by a relapse of clinical manifestations (**secondary recurrent syphilis**), is characterized by a maximum level of IgG and a minimum of IgM. The latent state of infection, associated with the absence of any clinical manifestations and a decrease in the number of pale treponemas (early latent syphilis), causes suppression of antibody production.

Synthesis of IgG and IgM compared with secondary recurrent syphilis is reduced by 1.8 times. Early IgM antibodies and later IgG

antibodies are directed to the same antigens. As IgM antibody production declines, IgG production continues.

A joint study of antibodies represented by these classes is more informative. A correlation was established between the content and ratio of antitreponemal antibodies of the IgM and IgG classes with the clinical form of syphilis. As the duration of the disease increases, the patient's immunological reactivity changes, there is a gradual decrease in humoral immunity and an increase in cellular immunological reactivity.

This is confirmed by the fact that during the period of **tertiary syphilis, specific granulomas (tubercles and gummas)** develop - typical manifestations of delayed-type cellular reactions. During this period, classical serological reactions can often be negative. The possibility of superinfection (re-infection of an uncured patient when a new sexual contact is established with another patient with syphilis) is debatable, since there is no evidence of differences in the properties of the pathogen during primary infection and with suspected superinfection.

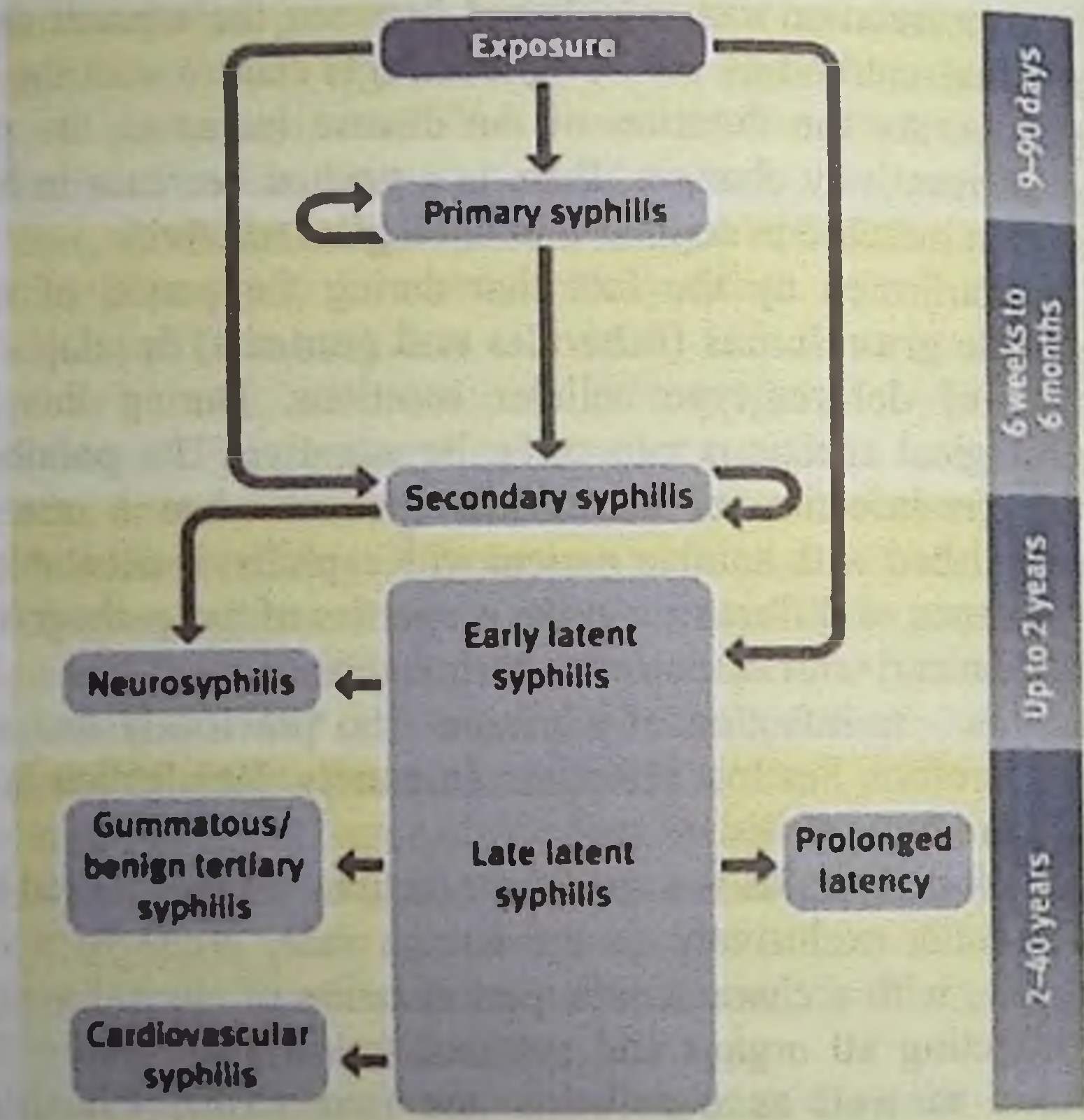
Reinfection - re-infection of a person who previously had syphilis (cured) and, therefore, has lost infectious immunity. Reinfection is one of the proofs of cure for syphilis.

Classification. **Syphilis** is a specific infectious disease caused by pale treponema, peculiar exclusively to the human race, prone to a chronic relapsing course, with a characteristic periodization of clinical symptoms, capable of affecting all organs and systems, transmitted mainly through sexual contact, as well as transfusion and intrauterine. Classification. According to M KB-10, syphilis was assigned to the group "certain infectious and parasitic diseases." According to this classification, syphilis was divided into congenital, early, late and unspecified. This classification is not entirely convenient for clinicians, since it is intended primarily for statistical processing of indicators.

For this reason, in this manual, we will use the previous classification of syphilis adopted in our country and based on its periodization.

Primary seronegative syphilis (syphilis I seronegativa). Primary seropositive (syphilis I seropositiva). **Secondary fresh syphilis** (syphilis II recens) **Secondary recurrent syphilis** (syphilis II recidiva). **Latent early syphilis** (syphilis latens parecox) lasting up to 2 years. **Sero-recurrent**

syphilis. Seroresistant syphilis. Tertiary syphilis (syphilis III). **Latent syphilis** (syphilis latens tarda). **Syphilis (acquired)** without clinical



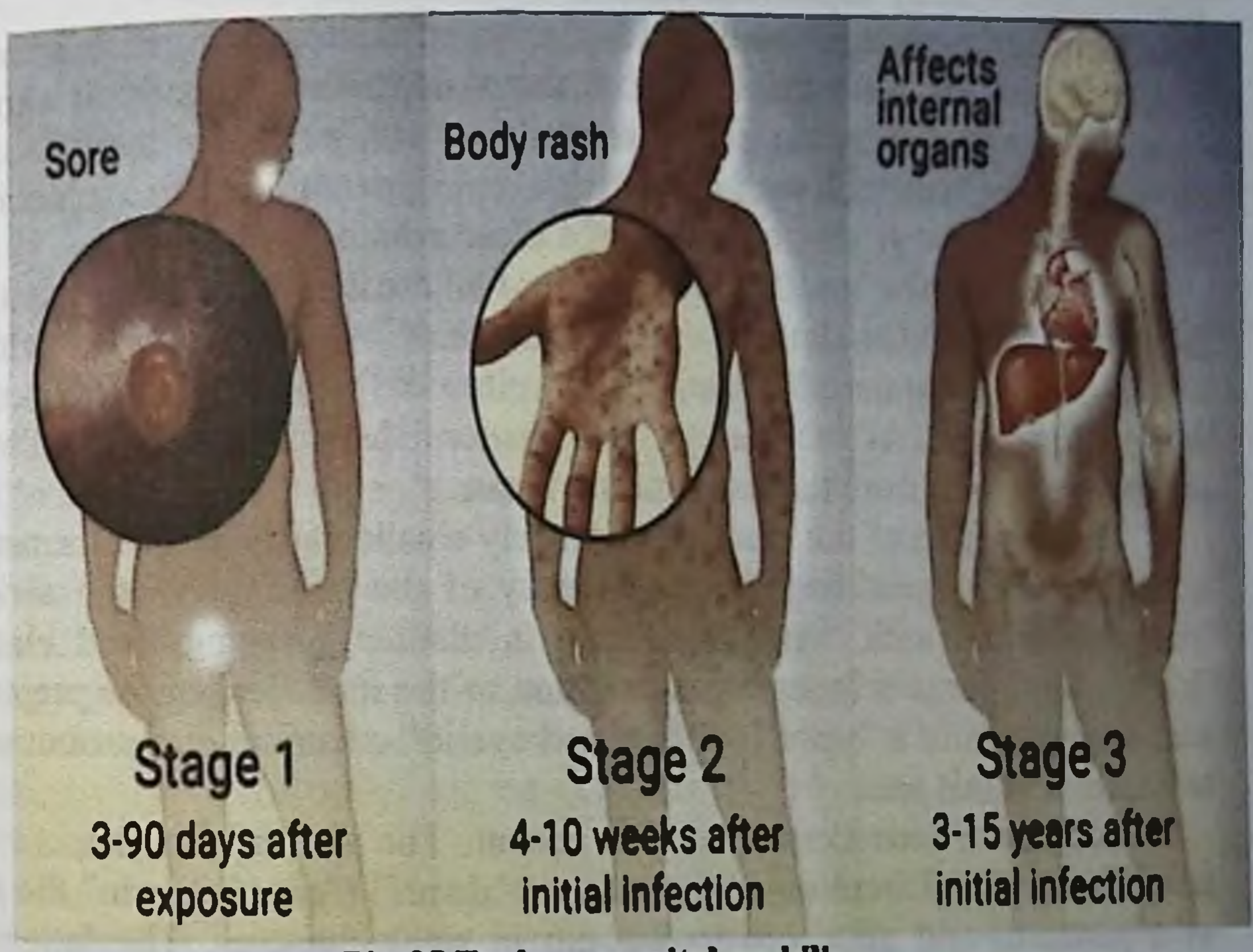
Pic-24 Syphilis

manifestations with a positive serological reaction 2 years or more from the moment of infection. Latent syphilis, unspecified.

Cases with a positive serological reaction to syphilis when it is impossible to establish the timing of infection. This group includes persons who started treatment at a previously undetermined stage of syphilis.

Early congenital syphilis (syphilis congenita parecox). Congenital syphilis of infancy (up to 1 year) and early childhood (up to 2 years) age. Late congenital syphilis (syphilis congenita tarda). Latent congenital syphilis (syphilis congenital latens). Syphilis of the nervous system: early

(**neurosyphilis praecox**) - with prescription of a syphilitic infection up to 2 years; late (**neurosyphilis tarda**) - with prescription of a syphilitic infection over 2 years. Dorsal tabes (**tabes dorsalis**). Progressive paralysis (paralysisprogressiva). Visceral syphilis (syphilis visceralis) indicating the affected organ.



Pic-25 Early congenital syphilis

ACQUIRED SYPHILIS PRIMARY SYPHILIS PERIOD (SYPHILIS PRIMARIA)

clinical picture. The beginning of the primary period of syphilis is characterized by the appearance of a primary affect ("hard chancre" - ulcus durum, from the French chancre - ulcer). The primary defect is far from always in the form of an ulcer, so the name of the defect of the skin or mucous membrane is more correct - **primary syphiloma**.A hard chancre corresponds to an ulcerative lesion.

Primary syphiloma appears on average 3-4 weeks after contact with a patient with an

infectious form of syphilis. It occurs at the site of primary introduction of pale treponema through damaged skin or mucous membranes. A chancre begins with erythema, which rapidly infiltrates to a papule, followed by superficial erosion or ulceration.

Primary syphiloma reaches its maximum development in 1-2 weeks and can heal in 4-6 weeks even without treatment. Usually it can be observed at the beginning of the secondary fresh period of syphilis. The erosive defect epithelizes, often with depigmentation, and ulcerative syphilomas scar with the formation of a clear, rounded scar.

A hard chancre has the appearance of an erosion or a superficial ulcer of a rounded saucer-shaped shape with clear even boundaries, with a smooth, shiny "varnished" meat- red or yellowish-pink bottom, with scanty serous discharge. At the base of the chancre there is a dense infiltrate, sharply delimited from the surrounding tissue.

On palpation of the chancre, a densely elastic cartilaginous formation is determined, resembling the consistency of the cartilage of the auricle. The compacted area, when squeezed, is distinctly springy and elastic. When a syphiloma is located in the region of the inner leaf of the preputial sac, a symptom of a "visor" or "inverted eyelid" occurs due to pronounced infiltration in this area.

Subjective sensations are often absent. The size of the chancre can be different (0.4-1.0cm in size); there are "dwarf" ("chancre nain" the size of a pinhead) and giant (up to 3-5 cm in size) chancres. The latter are localized mainly on the scrotum or on the pubis.

Color: two variants are typical for primary syphiloma - the red color of "live meat"; gray, dull, as if covered with a layer of "spoiled fat" (A. Fournier), due to protein **coagulation and superficial necrosis.** Sometimes there is a grayish-yellowish color in the center, and the periphery is red (chancre en cocarde).

The edges are sloping, as in a flat saucer, descend to the center, or lie flush with the surrounding skin. The discharge of **uncomplicated syphiloma** is scanty, translucent, slightly cloudy. The number of hard chancres is different. In most patients, they are single.

Primary syphiloma can be localized on any part of the skin or mucous membranes. For this reason, the highest frequency of localization of a hard chancre on the genitals, in the places most often occurring during sexual intercourse, microtraumas is natural. So, in men, a hard chancre is found in the area of \u200b\u200bthe inner sheet of the preputial sac, the coronal sulcus, on the anterior surface of the penis, its head, the external opening of the urethra, scrotum, pubis, and inguinal region.

In women, chancres are more often localized on the large and small labia, in the region of the posterior commissure, cervix, clitoris, and external opening of the urethra. On the large or small labia, the chancre has a characteristic shape with a seal; in the region of the posterior commissure - semilunar.

In the region of the coronal sulcus, syphiloma often takes the form of a tumor, similar to a"swallow's nest, twisted under the edge of the head." In the area of \u200b\u200bfolds on two contacting surfaces, in the presence of friction, moisture and maceration, "chancre- imprints" or "chancre-kisses" are found.

Extragenital chancre, the frequency of which is 1.5-10%, can often be localized in the mouth and anus, less often on the hands, forearms, thighs, abdomen, axillary region, on the mammary glands and face. In the anus, the chancre takes the form of a book leaf. Bipolar chancres occur in different parts of the body, can be extragenital and at the same time in the genital area.

The second sign of the primary period of syphilis is **regional lymphadenitis (scleradenitis, concomitant bubo)**, which necessarily accompanies each chancre. Characterizing him, Rikor wrote: "He is a faithful companion of the chancre, and he accompanies him invariably, fatally, he follows the chancre like a shadow.

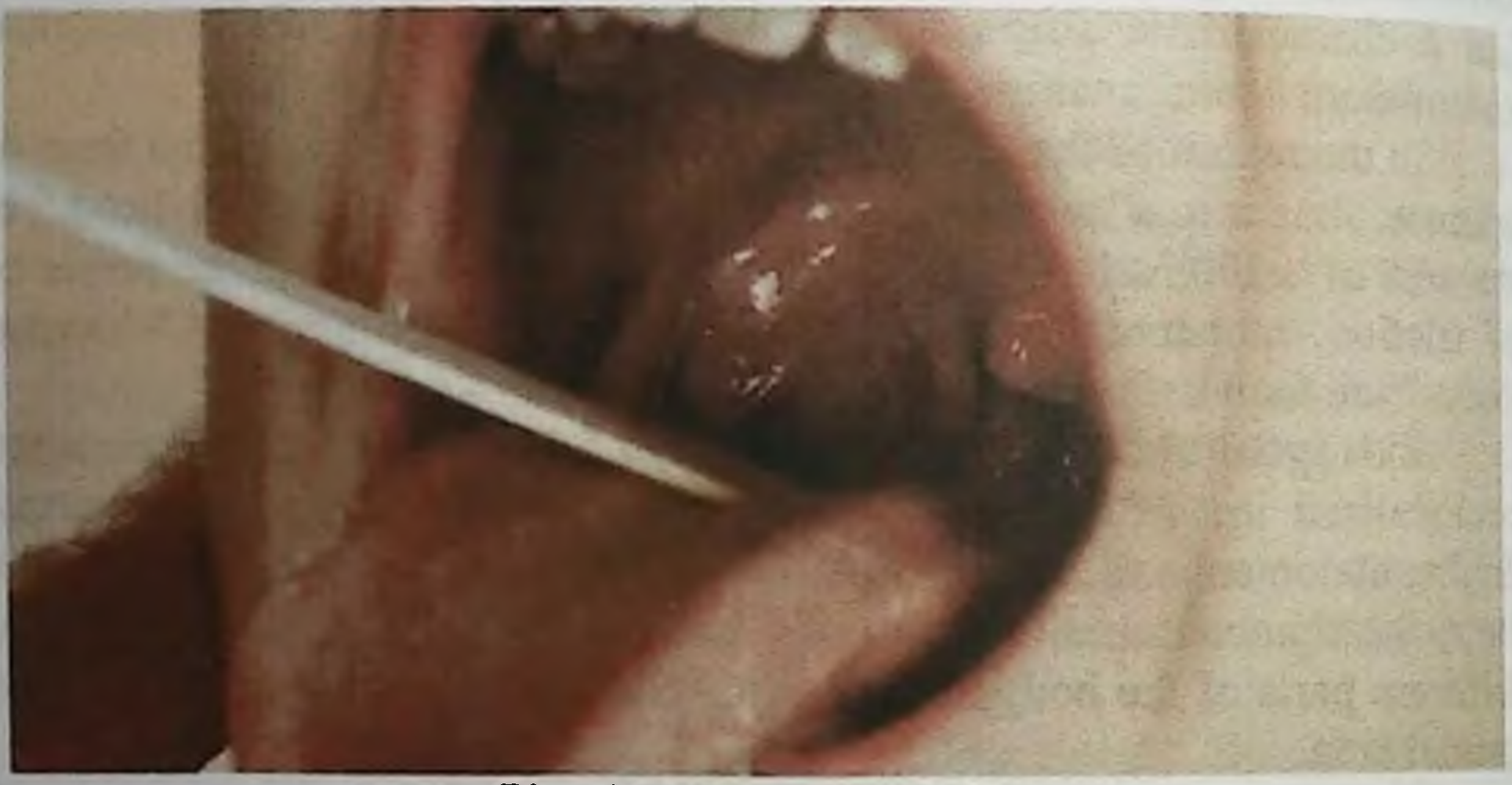
There is no hard chancre without bubo." In typical cases, regional lymph nodes are enlarged, dense, painless, not soldered either to each other or to surrounding tissues, mobile, the skin over them is not changed. They are found on the 6th - 10th day after the formation of primary syphiloma.

Sometimes, due to the decussation of the lymphatic tract, inguinal regional scleradenitis occurs on the other side. Most often there is bilateral

inguinal lymphadenitis, when several lymph nodes are enlarged, one in the center is larger than all the others - the **"Rikora pleiad"**.

The third sign of the clinical picture of primary syphilis is specific lymphangitis (inflammation of the lymphatic vessel from a hard chancre to regional lymph nodes). It manifests itself as a painless cord with separate "rosary", not soldered to the surrounding tissue, and is localized more often on the back of the penis (dorsal lymphangitis). It is quite rare in other areas.

Atypical chancres include: **chancre-amygdalite (tonsils), chancre-panaritium and indurated edema.**



Pic-26 Chancre-amygdalite

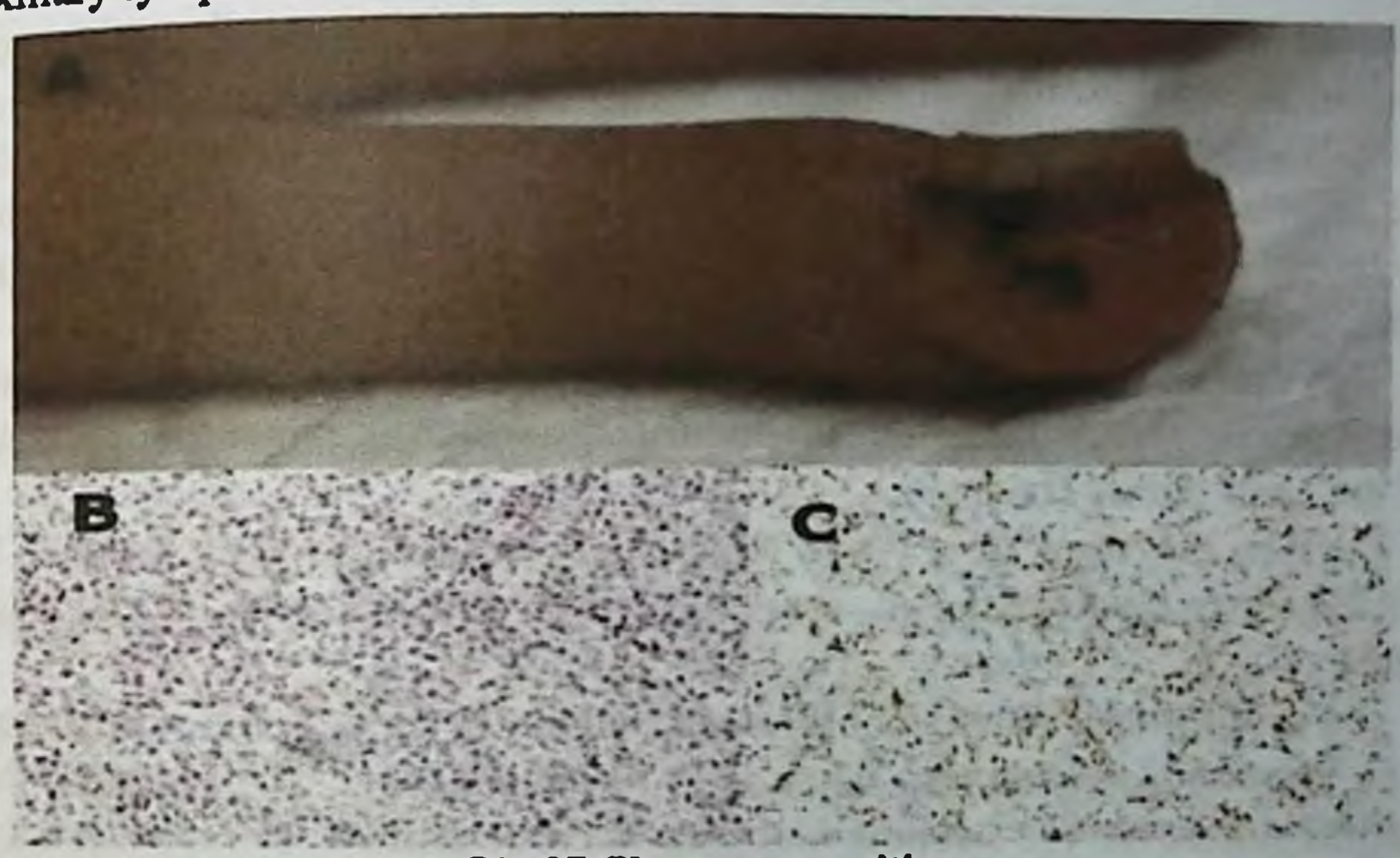
Chancre-amygdalite is characterized by a unilateral lesion, in which the tonsil increases in size, reddens, swells, thickens. On external examination, erosion or ulcers are not visible, but they can be found in the lacunae of the tonsils. Hyperemia is not spilled, unlike angina. One-sided enlargement of the cervical and submandibular lymph nodes is characteristic.

When swallowing, there is a slight soreness or discomfort. Symptoms of intoxication, fever, as a rule, are absent. The nasal voice is characteristic.

Chancre panaritium is located on the distal, nail phalanges of the fingers. Described and proposed the name Taylor (W. Taylor). It usually

occurs when infecting medical personnel working without gloves on the first three fingers of the right hand, more often on the index finger.

The process begins with erosion (or ulcers) and simultaneous compaction of tissues, most often in the region of the terminal phalanges of the fingers of the right hand. The phalanx swells and takes the form of a bulb or spherical swelling. The infiltrate is deep, dense, the skin over it acquires a dark red, stagnant-purple, "livid" color. Soon the elbow and axillary lymph nodes increase.



Pic-27 Chancre panaritium

The ulcer does not heal for a long time (for several weeks or months) and gives the impression of a **vulgar panaritium**. One of the features of the chancre-panaritium is its soreness. Indurative edema is usually observed when a hard chancre is located in the region of the large and small labia in women and in the region of the foreskin or scrotum in men.

The affected area becomes dense, hard, its size increases by 2-4 times, pain is not observed. When pressed, the recess is not formed. Skin color - dark red, sometimes with abluish tinge. The process is usually one-sided and lasts for several weeks. Complications of hard chancre include **impetiginization, erosive balanoposthitis and vulvovaginitis, phimosis,**

paraphimosis, gangrenization and phagedenism, which usually develop with secondary infection, irrational treatment and self-medication.

Impetiginization: when a secondary infection is attached, a hyperemic corolla appears along the periphery of the chancre, the tissues swell, the discharge becomes abundant, serous-purulent, pain appears in the area of syphiloma and in the regional lymph nodes.

When a hard chancre is located in the region of the glans penis, due to maceration and the addition of a secondary infection, the skin around the chancre turns red, swells, macerates with the formation of erosions of various sizes (**balanitis**).

With the transition of the inflammatory process to the inner leaf of the foreskin, **erosive balanoposthitis** occurs. In women, in the area of the large and small labia around the chancre, the phenomena of **vulvitis** and **vulvovaginitis** may develop.

Phimosis is characterized by edema and an increase in the volume of the foreskin, narrowing of the preputial ring, which prevents the opening of the glans penis. The penis

takes the form of a "bell tongue". A serous-purulent exudate is released from the preputial sac. Regional lymph nodes may be painful (Fig. 18.4).

Paraphimosis (stranglehold) occurs when the glans penis is infringed by a narrowed prepuce ring when it is forcibly pushed back beyond the coronal sulcus. Severe swelling and pain make it difficult to reposition the head, causing a violation of blood and lymph flow, which may require surgical intervention.

Gangrenous hard chancre is a rather rare complication that develops in weakened, emaciated people with reduced immunity. The necrotic process captures the entire surface of the hard chancre, a dense scab of a dirty gray or black color appears, after the rejection of which a deep ulcer is exposed. The defect is slowly filled with granulation tissue, followed by scar formation. Gangrenous chancre is accompanied by fever, chills, headache.

Phagedenic (Greek devouring) hard chancre is rare. Like gangrenous chancre, it develops in debilitated, emaciated patients and is characterized by tissue necrosis. It differs from gangrenous chancre in its

progressive * course, massive destruction of tissues and the spread of the process both along the periphery and in depth.

Diagnosis of phagedenic chancre before positive serological reactions is difficult, since the abundance of secondary microflora limits the possibility of detecting pale treponema. Diagnostics. The diagnosis of primary syphilis is made on the basis of a characteristic clinical picture, the detection of pale treponema in the discharge of primary syphiloma. If the chancre is complicated by phimosis or a secondary infection, a puncture of regional **scleradenitis** is performed. In the primary seropositive period, positive serologicalreactions are detected.

Anamnesis data and the results of examination of sexual partners are of great importancein making a diagnosis.

Tests for the assignment

1. The causative agent of syphilis: 1. Treponema pallidum 2. Treponema balanitidis

3. Treponema pertenue 4. Treponema caratea

2. Treponema pallidum was discovered: 1. in 1901 2. in 1889 3. in 1905 4. in 1926

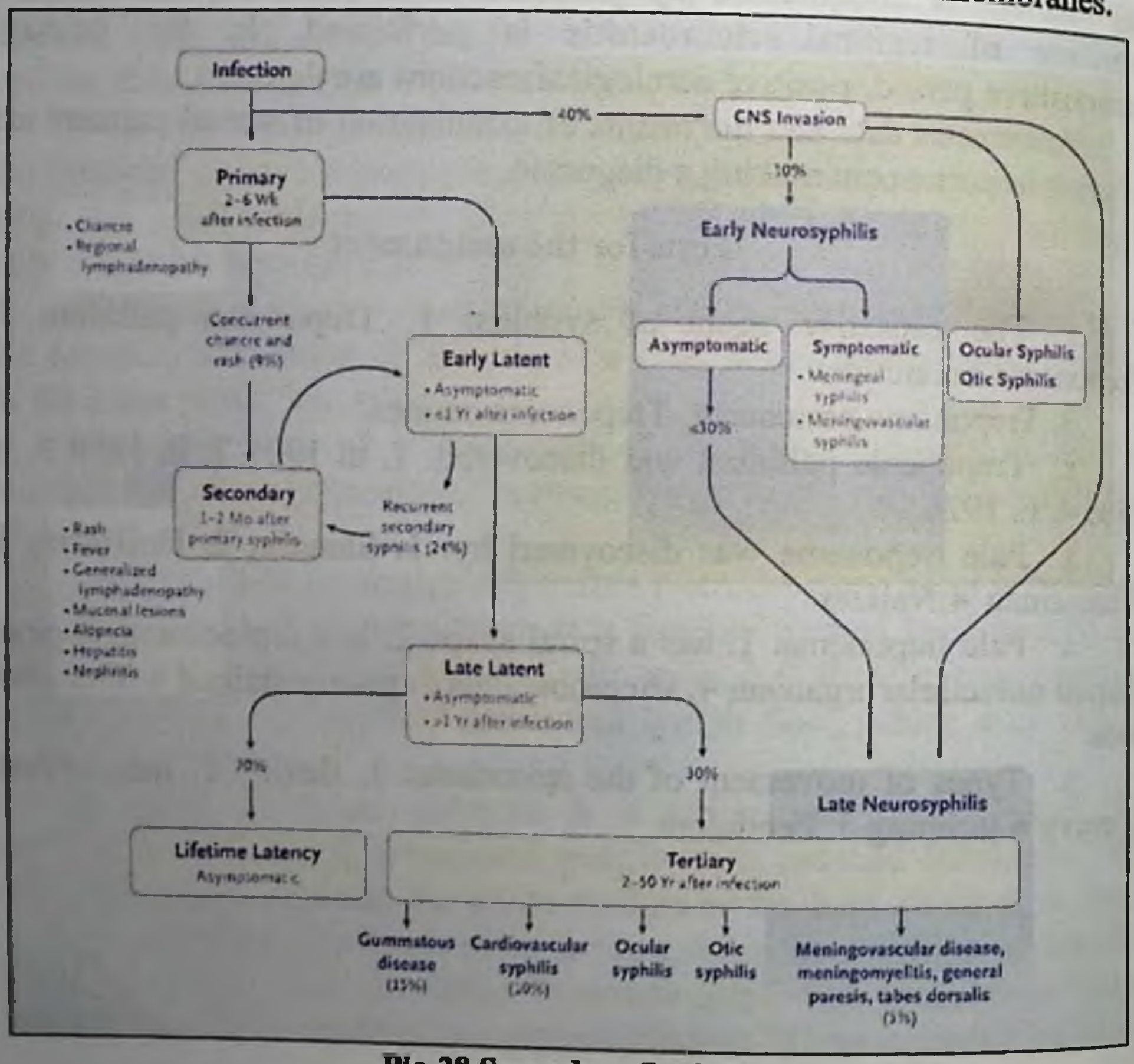
3. Pale treponema was discovered by: 1. Shaudin 2. Hoffmann 3. Wasserman 4.Neisser

4. Pale treponema: 1. has a spiral shape 2. is a diplococcus 3. pear-shaped unicellularorganism 4. streptobacillus 5. poorly stained with aniline dyes

5. Types of movement of the spirochete: 1. flexion 2. translational 3. wavy 4.jumping 5. Pendulum

CHAPTER 5 SECONDARY SYPHILIS. HIDDEN SYPHILIS. METHODS OF TREATMENT OF PATIENTS WITH SYPHILIS, DISPENSERIZATION. DETECTION AND SERODIAGNOSIS OF THE DISEASE.

Cinical picture. The secondary period of syphilis (**syrhilis secundaria**) occurs on average 6-7 weeks after the appearance of a hard chancre and is manifested by rashes on the skin and mucous membranes.



Pic-28 Secondary Syphilis

There are **secondary fresh syphilis** (syphilis II eecens), **latent** (syphilis II latens) and **recurrent** (syphilis II recidiva).

Clinically, the secondary period of syphilis is characterized by symmetrical rashes of spots, papules and pustules of pink or bluish-red color, leaving no scars (except for deep

pustules). Rashes are small, rounded, with clear boundaries, usually not accompanied by subjective sensations.

They suddenly appear, exist for a certain time and spontaneously disappear. In patients with fresh syphilis, **polyadenitis** is noted. All serological reactions in the secondary period of syphilis are sharply positive. The exacerbation reaction of Gerksheimer-Yarish- Lukashevich (Jarisch-Herxheimer) at the beginning of treatment is manifested by an increase in body temperature, a feeling of chills, myalgia, the appearance or intensification of a roseolous rash approximately 4 hours after the administration of penicillin due to the massive death of treponema and the release of endotoxin.

This reaction serves as a marker of ex juvantibus therapy (trial therapy) and a diagnostic criterion for primary and secondary fresh syphilis. With latent forms of syphilis, this exacerbation reaction does not appear.

Roseola is a pink spot, well demarcated, round, without peeling and subjective sensations, disappears during diascopy, does not rise above the skin. Roseola are arranged randomly, symmetrically, focally; with rounded outlines, peripheral growth is absent.

On the surface of roseola, the epidermis is not changed. A spotted rash usually appears on the 10th week after infection, the elements appear jerkily, and after 10 days the rash reaches its maximum development. After the introduction of penicillin, roseola becomes more pronounced.

When the body is cooled with air, roseola becomes brighter. Gradually, the color of the roseola darkens, and they disappear without a trace. The sizes of the elements are different - from dotted to 1 cm in diameter. If the patient is not treated, then the rash lasts for several weeks.

As a rule, roseola appears suddenly. The places of the most frequent localization of the roseolous rash (in order of gradual appearance) are the lateral surfaces of the trunk, chest, back, abdomen, thighs, etc., mucous membranes.

With secondary fresh syphilis, roseolas are bright, abundant, small, symmetrical, do not merge with each other. More often they are localized on the lateral surface of the body, on the flexor surface of the forearms, in the abdomen, on the hips. In secondary recurrent syphilis, roseolas are larger, their color is cyanotic or pale, the number of elements is insignificant, they are arranged asymmetrically, there is a tendency to group with the formation of figures.

Of the varieties of roseola, one should point out the towering **roseola (roseola elevata), granular (roseola granulata)** with an accentuated follicular pattern, **confluent (roseola confluens)** and **annular (roseola annularis)**, usually observed in recurrent syphilis.

Differential diagnosis of roseolous syphilis is carried out with the following dermatoses: **spotted toxidermia, pink lichen, pityriasis versicolor, rubella, measles and marbling of the skin.**

Cutis marmorata (marble skin) differs from syphilitic roseola in its bluish tint and looped pattern. Measles and rubella differ from syphilitic roseola with an acute onset, high fever, and obligatory lesions of the face, rear of the hands and feet, which are always free from the syphilis rash.

In addition, measles, unlike syphilis, is accompanied by catarrhal symptoms from the mucous membranes (photophobia, laryngitis, bronchitis) and is more common in childhood. Spotted (roseolous) rashes that develop with toxidermia are recognized by anamnesis, the absence of primary syphiloma or its traces and specific polyadenitis, the rich red color of the spots, their tendency to merge in the area of the flexor surfaces of the joints, as well as severe itching or burning.

With their resolution, peeling is often observed. With pityriasis, multi-colored lichen, a positive Balzer test is observed, a golden-yellow glow in the rays of a Wood's lamp, when scraping, the presence of small, pityriasis scales is revealed.

Pink lichen differs from syphilitic roseola by a variety of spots (small scaly spots and a large "**maternal plaque**") located along the lines of skin tension, peeling and the absence of **specific polyadenitis.**

Papular syphilis (syphilis papulosa) is the most frequent and varied rash in secondary syphilis. They do not cause subjective sensations, well

demarcated, dense, rounded or oval, brownish-red color, the color of ham, sometimes copper, with a "gloomy color".

At first, the surface of the papules is smooth, during the period of regression, peeling occurs, spreading from the center to the periphery, acquiring the form of a corolla **(Biett'scollar)**.

Without treatment, the elements are kept for 2-3 months. Disappearing, they leave behind temporary pigmentation. With specific treatment, papules quickly disappear and resolve, which is important for differential diagnosis as a "trial treatment" option.

There are the following types of papules in size: lenticular the size of a lentil, nummular up to 2-3 cm in diameter and less often miliary (1-2 mm in diameter). Papular elements can occur on any part of the skin and visible mucous membranes. Their characteristics are largely determined by localization.

The following options are distinguished: **corona Veneris** (on the border of the forehead and scalp), **seborrheic syphilide** in places where there are many sebaceous glands, **in the form of a cockade** (in the center there is a large papule, and on the periphery there is a rim of smaller ones).

In the folds, the rash easily merges and macerates, forming erosive and vegetative papules

- **wide warts** (condyloma lata). Usually they are located in the perineum and genitals and are much more common in recurrent syphilis.

Specific papular syphilides are papules on the palms and soles. They are usually lenticular papules with Biette's collarette on the periphery. Sometimes such elements are

characterized by severe hyperkeratosis. In the interdigital folds of the feet, papules are macerated, which often leads to an incorrect conclusion about the presence of mycosis of the feet in the patient.

A characteristic sign of papular syphilis is pain when pressed on its surface with a bellied probe **(hyperalgesia is a symptom of Yadasson)**.

Differential diagnosis of papular syphilis is carried out with the following dermatoses: teardrop-shaped parapsoriasis, psoriasis vulgaris, scabies, **papulonecrotic tuberculosis of the skin, lichen planus, genital warts, vulgar warts, toxidermia.**

Lichen planus - flat, dense, polygonal papules with a waxy sheen, umbilical depression in the center, purple; localized more often on the inner surface of the forearms, in the area of the flexor surfaces and mucous membranes of the oral cavity and genitals.

Teardrop-shaped parapsoriasis - soft, slightly raised above the skin, reddish-brown in color, covered with scales in the form of a wafer, removed entirely when scraped.

Psoriasis - papules of red and pink color, covered with silvery white scales; when scraping, the phenomena of the psoriatic triad are observed: stearin stain, terminal film, pinpoint bleeding ("Auspitz phenomenon"; elements are prone to peripheral growth with the formation of plaques of various sizes; the location is symmetrical, mainly on the extensor surfaces of the limbs, in the area of the elbow and knee joints, the scalp head. When scabies, papules are accompanied by severe itching, especially at night, which does not happen with papular syphilis.

Pustular syphilis (syphilis pustulosa) are much less common than spotty and papular. They occur in patients suffering from concomitant syphilis other infectious diseases, alcoholism, drug addiction. Often pustular syphilides are accompanied by signs of general intoxication, fever, malaise, pain in the joints

The following varieties are distinguished: **acne syphilis** (acne syphilitica) - elements the size of a pinhead, dense, red-brown, pointed, regular round shape; a yellowish or brown crust forms on top in 2-3 days, which soon falls off; without treatment, the rash lasts 1-1.5 months;

smallpox syphilis (varicella syphilitica) is rare, papules after 1-2 days turn into pustules with a central umbilical depression, then a thick crust slowly forms; rashes appear for 1-2 months;

impetiginous syphilis (impetigo syphilitica) the size of a pea and larger, superficial confluent pustules dry up into a continuous crust, at the base of which there is a slight infiltrate; after removing the crust, surface erosion remains, healing without scarring: **syphilitic ecthyma** (ecthyma syphilitica) usually develops not earlier than six months after infection; it is represented by an ulcer with sheer dense edges under a thick, layered crust; heals in a few months with a retracted round scar; the number of elements is limited; the most common localization is the shins;

syphilitic rupee (rupia syphilitica) - a formation similar to ecthyma, but deeper and larger, occurs on the trunk and limbs in debilitated, debilitated patients; extends deep into and around the periphery, can serpiginate; the crust is almost black, layered, as if floating in the ulcer; the bottom is covered with sluggish granulations; after healing, a deep, retracted scar remains.

Pustular syphilides differ from pyoderma (impetigo, ecthyma), acne, chickenpox by the absence of acute inflammatory phenomena, a bright erythematous rim, and the presenceof a bluish-red dense base.

Pigmentary syphilides - syphilitic leukoderma (leucoderma syphilitica, pigmented syphilide Pillo-Ardi) is characterized by a change in skin color on the back and sides of the neck, in the shoulder girdle, less often in other areas.

According to A. Fournier, "this is a patent for syphilis, written on the patient's neck." Syphilitic leukoderma occurs 4-6 months after infection, more often in women.

Leukoderma is usually subtle, represented by dyschromia (unsharply pronounced white spots against a background of moderate hyperpigmentation), giving the impression of a "dirty neck". The diameter of all elements is the same - from 0.5-2.0 cm, they do not merge with each other, there is no peripheral growth, peeling, itching and pain.

Sometimes areas of depigmentation evenly occupy the entire neck in the form of a **"necklace of Venus"** (collier de Venus). Sometimes leukoderma can occur on the chest, in the axillary regions, on the trunk and extremities. The face is usually not affected.

Leukoderma is noticeable better with side lighting. There are three forms: spotted - white spots surrounded by wide layers of hyperpigmented skin; lace or mesh - a picture of lace, in the loops of which white round or oval spots are laid; marble - the contrast ofdepigmentation is insignificant, the impression of dirty skin.

Pigmentary syphilide without treatment may not disappear for up to two years. In patients with leucoderma symptoms, the nervous system is often affected.

Syphilitic leukoderma should be differentiated from vitiligo and secondary leukoderma (with **psoriasis, versicolor**, etc.).

Vitiligo is characterized by the complete absence of pigment in the lesions, the larger size of the foci of depigmentation, the tendency to peripheral growth and fusion.

Secondary leukoderma, which arose at the site of multi-colored (**pityriasis**) lichen, is characterized by depigmented spots of various shapes and sizes, prone to merging and having scalloped outlines of foci, as well as the presence of scaly elements of a milky- coffee color, detected by a test with iodine tincture (Balzer test).

Test for latent peeling "strike with a Besnier's nail" is negative for syphilis.

Secondary leukoderma after **psoriasis, parapsoriasis, eczema, atopic dermatitis** has a larger size, different localization.

Anamnestic data and negative serological tests for syphilis will help in the diagnosis.

Syphilitic alopecia - baldness (alopecia syphilitica) develops gradually at 4-6 months after infection and is often combined with leukoderma. The scalp, mustache (especially under the nose and on the tips of the mustache), sideburns, beard, eyebrows, eyelashes, pubic area (mons Veneris) and labia majora are affected.

Distinguish diffuse hair loss throughout the scalp, as in some acute infectious diseases. This is a small-focal loss in the form of small foci, clearings resembling **"moth-eaten fur"**, and **mixed hair loss** - a combination of diffuse and small-focal processes.

Affected eyelashes due to partial loss and subsequent regrowth are of different lengths and spindle-shaped - stepped eyelashes - a sign of **Felix Pinkus** (Felix Pinkus). Eyebrow prolapse in small islands with discontinuity of the arc or rarefaction of the outer third is called by the apt expression of A. Fournier and P.S. Grigoriev "omnibus" or tram symptom.

Eyebrow changes are so characteristic that even in a tram, at first glance, you can diagnose syphilis. The eyebrow takes on a bristly appearance, resembling a bush. Hair sticks out straight or leans in different directions. The course of alopecia is usually slow. However, sudden hair loss is also possible.

Alopecia affects both sexes, but more often in women. The remaining hair often becomes dull, dry, can curl like vellus hair, and a "wig

symptom" occurs. Alopecia is always only temporary. In the future, the hair is completely restored. "Syphilis never makes you bald." Differential diagnosis. With alopecia areata, large rounded foci appear, more often single, sharply demarcated, having a shiny, glossy surface with a complete absence of hair (like a **"billiard ball"**), a zone of loose hair along the periphery.

With **microsporia and trichophytosis** of the scalp in the lesions, there is peeling and hyperemia, the remains of broken hair. Laboratory spores of the fungus are found in the affected hair.

With **seborrheic alopecia**, the hair is oily or dry, thin, brittle, the process progresses slowly, patients are worried about itching. Hair loss is usually localized in the frontal and parietal regions.

Premature baldness is hereditary, develops gradually, slowly, hair falls out primarily in the frontal and parietal regions. Serological reactions to syphilis are negative. Hair loss after acute infectious diseases (typhoid and typhus, influenza) is established on the basis of anamnesis data, diffuse pattern of alopecia, absence of signs of syphilis, negative serological reactions to syphilis.

Test tasks

1. Secondary syphilides of mucous membranes: 1. Rare 2. Observed in the form of papular and spotted rashes 3. There are fuzzy borders 4. Slightly contagious

2. After the appearance of a hard chancre, the secondary period of syphilis occurs, onaverage, after: 1. 5-6 weeks 2. 6-7 weeks 3. 6-8 weeks 4. 8-9 weeks

3. The secondary period is characterized by rashes: 1. nodes 2. roseol 3. papules 4. pustules 5. vesicles

4. Secondary fresh syphilis is characterized by: 1. scarcity of rashes 2. remnants of a hard chancre 3. pronounced Yarish-Lukashevich-Herxheimer reaction 4. papular rash 5. roseolous rash

5. Varieties of syphilitic roseola: 1. miliary 2. elevating 3. hemorrhagic 4. confluent

5. follicular

6. Syphilitic roseola must be differentiated from: 1. rubella 2. typhoid fever 3. toxidermia 4. psoriasis 5. pityriasis rosea

7. Specify the varieties of papular syphilides: 1. psoriasiform 2. acne-like 3. palmar- plantar 4. nummular 5. vegetative (general condylomas)

8. Differential diagnosis of papular syphilis should be carried out with all of the listed diseases, except: 1. lichen planus 2. guttate parapsoriasis 3. pemphigus foliaceus 4. Psoriasis

CHAPTER 6 THE TERTIARY PERIOD OF SYPHILIS. HATED SYPHILIS. INFLUENCE OF SYPHILIS ON INTERNAL ORGON AND NERVOUS SYSTEM.

TERTIARY SYPHILIS

clinical picture. In the absence of treatment and a decrease in the reactivity of the organism, 2-4 years after infection, and sometimes much later, manifestations of the tertiary period of syphilis may occur (recently it has been rare).

"The Tertiary period is a big "departure", or, better to say, the most "ill-fated station" in the course of syphilis, the station at which all the most important and most severe manifestations of the disease collide," A. Fournier defines this stage of the disease. Lesions during this period are manifested by tubercles (**syphilis III tuberculosa**) and gums (**syphilis II gumma**), they are destructive and end with scarring.

Elements of tertiary syphilis appear suddenly. They are represented by **tubercles** or **nodes** (gums). Rashes are few, asymmetrically located, tend to cluster. This period is also characterized by damage to the visceral organs and the nervous system. Patients with tertiary syphilis are non-contagious.

There are active, or manifest, tertiary syphilis (syphilis 111 activa) and latent (syphilis III latens).

Tubercular syphilis. Elements develop in the thickness of the dermis; have a rounded shape ranging in size from a lentil to a pea, protrude slightly above the level of the skin, dense, painless, red-cyanotic or brownish-red in color.

With diascopy, a pigmented spot remains. The elements are simultaneously in different stages of development. After the resolution of the tubercles, hyper- and then **hypochromic cicatricial atrophy** is first formed.

Clinical variants of tuberculous syphilides:

- **grouped** - located focally, often have the form of arcs, garlands;
- **serpiginating** - characterized by peripheral growth and fusion of tubercles; new elements appear on one side and, thus, the pathological process "creeps" in a certain direction;

- **drain** (platform) - are merged tubercles, while individual elements are not visible;
- **dwarf** - small, the size of a millet grain tubercles of pale yellow color, do not ulcerate and leave behind a barely noticeable atrophy.

Differential diagnosis of tubercular syphilis is carried out with tuberculosis and leprosy. **Hummous syphilis**. Gummas more often affect the skin, cardiovascular system, nervoussystem and bone apparatus. They develop in the hypodermis or internal organs and arenodules ranging in size from a pea to a hazelnut, and sometimes larger. Gummas are rounded, painless, at first dense, then soften, solder with the skin, which turns red, becomes red-violet, tense, and then gradually becomes thinner and breaks through.

From the newly opened gum, through a narrow fistula at the beginning, a little viscous,

transparent liquid is released, resembling glue - gum arabic (hence the name - gum). The edges of the ulcer are dense, roll-like, sheer down to the bottom, covered with a densely attached dirty-gray "gummous rod" - dead tissue, which departs after the development of demarcation.

Gummas are usually solitary, rarely found at the same time 2-3. Sometimes they merge and form diffuse gummous infiltrates. The most common location of gummas is the shins, head, nasal septum, palate, posterior pharyngeal wall, vocal cords, etc. **Hummous lesions** are accompanied by characteristic nocturnal pains. On the x-ray of the bones with gummas, a typical combination of foci of osteoporosis and osteosclerosis is revealed. It is extremely rare that the so-called tertiary roseola is observed on the trunk and limbs in the form of ring-shaped spotty foci of large sizes of red color of different shades, which exists for a long time, for several months.

Gummy syphilide until the moment of its disintegration is differentiated from lupoma, leproma, Bazin's compacted erythema, nodular allergic vasculitis, leishmaniasis. In rare cases, it is necessary to differentiate syphilitic gummas from deep mycoses.

SYPHILIS OF THE MUCOUS MEMBRANES The defeat of the mucous membranes occurs in any period of syphilis.

In primary syphilis, hard chancres are usually found on the mucous membrane of the genital organs (see "Primary syphilis"), but there is also an extragenital localization of primary syphiloma. In some cases, the lesion of the mucous membranes proceeds atypically. So, a chancre on the lip usually manifests itself in the form of erosion or an ulcer covered with a dense brownish crust.

Syphilis of the mucous membranes of the secondary period eruptions in the oral cavity, in the area of the pharynx, larynx, and genital organs are of great epidemiological significance, as they are often a source of infection.

On the mucous membranes, they appear as **spotted** (erythematous), **papular, erosive and ulcerative syphilides**.

Spotted syphilis is represented by rounded erythematous, clearly delimited spots, with a smooth surface, without subjective sensations. They often merge, forming areas of various shapes and sizes with clear scalloped edges. Against the background of a bright mucous membrane, the spots are often poorly distinguishable. Roseola is best seen on the hard palate. They last for a long time, disappear without a trace.

Papular syphilis is clearly delimited, dense, has a different shape and size due to the fusion of individual papules. The color is gray or bluish-white (opal plaques) (plagues opalines). Some elements can erode, ulcerate and cause minor pain.

Places of localization - lips, cheeks, especially along the line of closing of teeth, gums, hard and soft palate, tongue, tonsils and larynx, less often - nasal mucosa, conjunctiva, posterior pharyngeal wall, vagina and cervix. In the corners of the mouth and on the

genitals, often one half of the papule is located on the mucous membrane, and the other on the adjacent skin area.

Papules located on the mucous membrane of the larynx cause **hoarseness**, which disappears over time, and when the rash is localized near the opening of the Eustachian tube, tinnitus and hearing loss appear.

Papules at the corners of the mouth often develop painful cracks. On the back of the tongue, **erosive syphilis** may have a red or whitish tint, with a polished, smooth, shiny surface ("glossy" papules), when merged, they form foci of irregular or oval outlines and are more often located

below the level of the surrounding mucosa in the form of "**mowed meadow**" plaques behind due to the smoothness of the papillae of the tongue, i.e., erosivepapules are, as it were, "shaved".

Sometimes they are similar to "geographical" or "scrotal" language. Papules without erosion due to strong infiltration of the filiform papillae look like "cakes sprinkled with grains of sugar." Syphilis of the mucous membranes of the tertiary period. Such syphilides are observed in approximately 30% of patients with tertiary syphilis, mainly in the oral cavity, pharynx, nose, pharynx and larynx, produce great destruction and cause severe functional impairment.

Tubercular syphilis usually affects the palate and the palatine curtain at the same time; between groups of tubercles of a rich red color, areas of healthy tissue are preserved. Gummy lesions are expressed either by diffuse infiltration, or by isolated gumma, which acts as a dense knot, during the decay of which a perforation is formed.

The same single or multiple perforations are formed as a result of the disintegration of the gummous infiltration of the soft palate. Perforation causes slurred and nasal speech, the penetration of liquid food into the nasal cavity. In the hard palate, perforation occurs after bone necrosis.

Isolated gummas or gummous infiltrations in the nose are usually localized on the nasal septum. Their decay is accompanied by purulent discharge with an unpleasant odor, the formation of crusts, the forcible removal of which causes bleeding. The bone and cartilage of the septum are necrotic, forming a perforation, sequestration, followed by deformation, retraction of the back of the nose ("saddle-shaped" flattened nose).

Syphilis of the musculoskeletal system

In the secondary period of syphilis, bones and joints may be affected, although this is not always diagnosed. Subjectively, this is manifested by night pains of varying intensity in the bones of the skull, sternum, long bones of the limbs (**dolores osteocopy noctuml**), and objective changes are most often absent.

On the tibia can be found small dense spindle-shaped swelling - **periostitis**, also accompanied by nocturnal pain. In the future, this defeat disappears without a trace. In the tertiary period of syphilis, the bones of

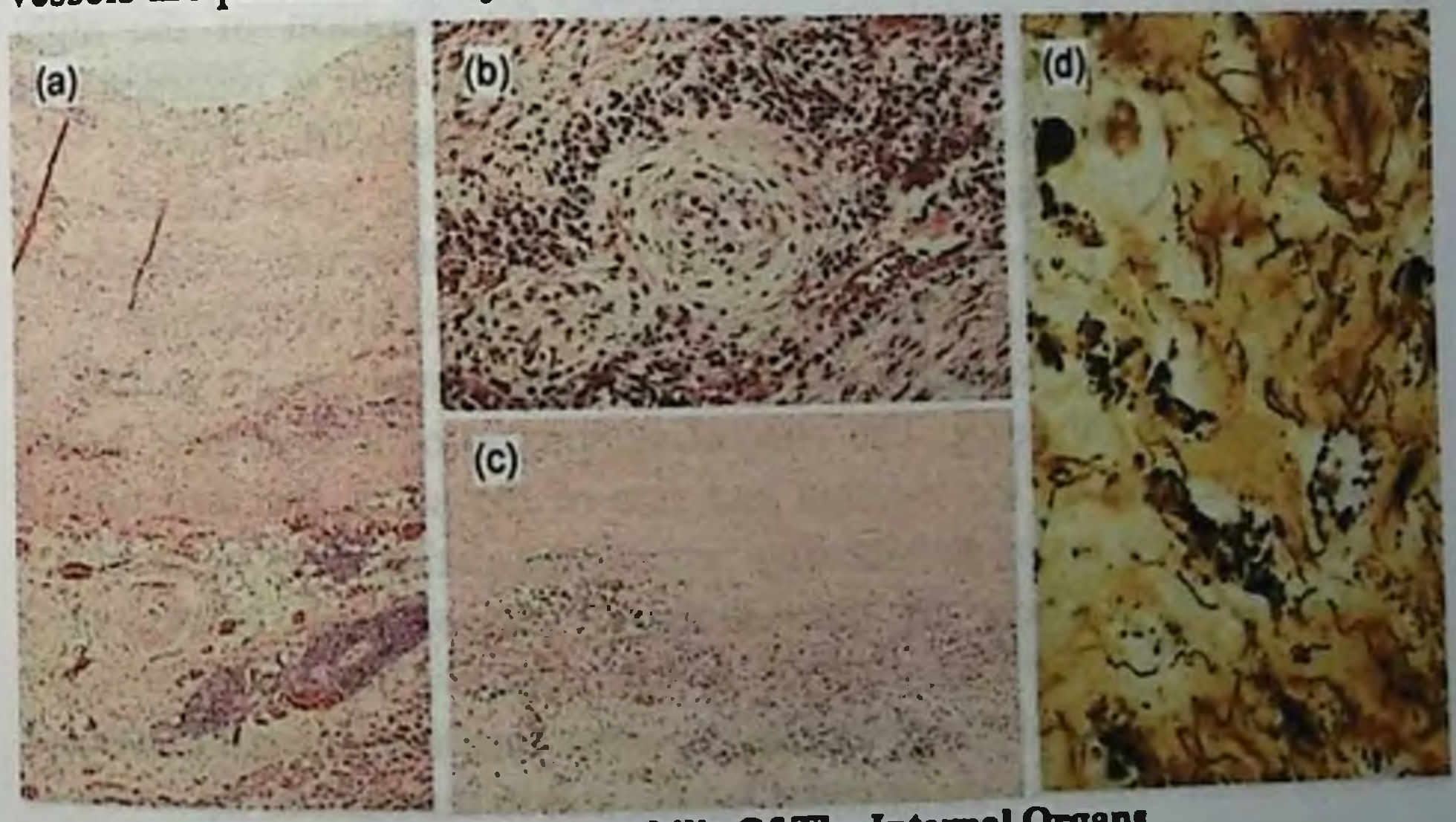
the legs, skull, sternum, clavicle, ulna, and nasal bones often suffer, where combined gummous lesions of the periosteum and bone develop.

SYPHILIS OF THE INTERNAL ORGANS

Syphilitic damage to visceral organs is a manifestation of a general syphilitic infection in one or more organs or systems of the body of a sick person. Lesions developing in the secondary period of syphilis, proliferative in nature, proceed much more easily than destructive ones observed in tertiary syphilis.

Most often, the cardiovascular system and liver are affected, the respiratory organs, stomach and kidneys are much less often affected.

Cardiovascular syphilis. Syphilis is a vasotropic infection, in which the aorta occupies the first place in terms of the frequency of lesions, with the development of **syphilitic aortitis** and its complications; myocardial damage (**syphilitic myocarditis**) is relatively rare.The aorta and coronary vessels are predominantly affected.



Pic-28 Syphilis Of The Internal Organs

There are two forms of syphilitic aortitis: **uncomplicated** and **complicated. Uncomplicated aortitis** is the initial form of syphilitic

lesions of the aorta, which is often asymptomatic and is recognized only on section.

With **complicated aortitis**, the patient develops stenosis of the mouths of the coronary arteries, insufficiency of the aortic valves and aortic aneurysm, the rupture of which can lead to death.

Diagnosis of syphilitic lesions of the heart is difficult.

The clinical picture of syphilitic myocarditis is very similar to the manifestations of other lesions of the heart muscle of infectious origin.

To clarify the etiology of heart lesions, anamnesis data and the results of a serological

examination are important. On the ECG, there may be violations of automatism, an increase in electrical systole, tooth pathology, signs of atrial overload. The frequency and severity of pathological changes in the heart is more noticeable in patients with secondary fresh and recurrent syphilis in comparison with patients with primary and latent syphilis. **Phonocardiography** reveals an increase in the duration of the high-frequency part of the second tone above the aorta, which is regarded as a sign of an increase in aortic stiffness as a result of its specific lesion.

In order to study intracardiac hemodynamics, the authors use polycardiography. Myocardial damage and preclinical aortitis are observed in the early stages of the disease, despite apparent clinical well-being.

Aortitis develops 10-20 years after infection and occurs mainly in patients aged 40-60 years. The clinical symptoms of a **syphilitic aortic aneurysm** depend on the location and size of the aorta, the direction of growth, compression of surrounding organs, the presence of concomitant damage to the orifices of the coronary arteries, and mitral valve insufficiency.

The main signs of a syphilitic aortic aneurysm are: pulsating areas of the chest in the region of the sternum handle and to the right of it;

percussion increase in the contours of the aorta in one or both sides of the sternum handle; difference in pulse - less filling and delay of the pulse wave on the side of the aneurysm; a kind of blowing systolic murmur in the area of vascular dullness (sometimes heard by the patient himself at night);

symptoms of compression by an aneurysm of neighboring organs and tissues: superior vena cava, trachea, bronchi, esophagus, nerve trunks;

symptoms of an aneurysm breakthrough into neighboring organs (trachea, bronchi, lungs, pleural cavity, esophagus, mediastinum, pericardial cavity);

radiologically - expansion and distinct pulsation of the vascular shadow, its sharply defined and even edges, the inseparability of the bulging shadow from the aorta when transilluminated in various directions.

Syphilis of the liver. There are early acute parenchymal hepatitis, acute liver dystrophy and late liver syphilis. Early acute parenchymal hepatitis in the secondary period of syphilis is observed relatively rarely, proceeds benignly, sometimes without jaundice, and passes quite quickly during treatment; much less often, its course becomes malignant.

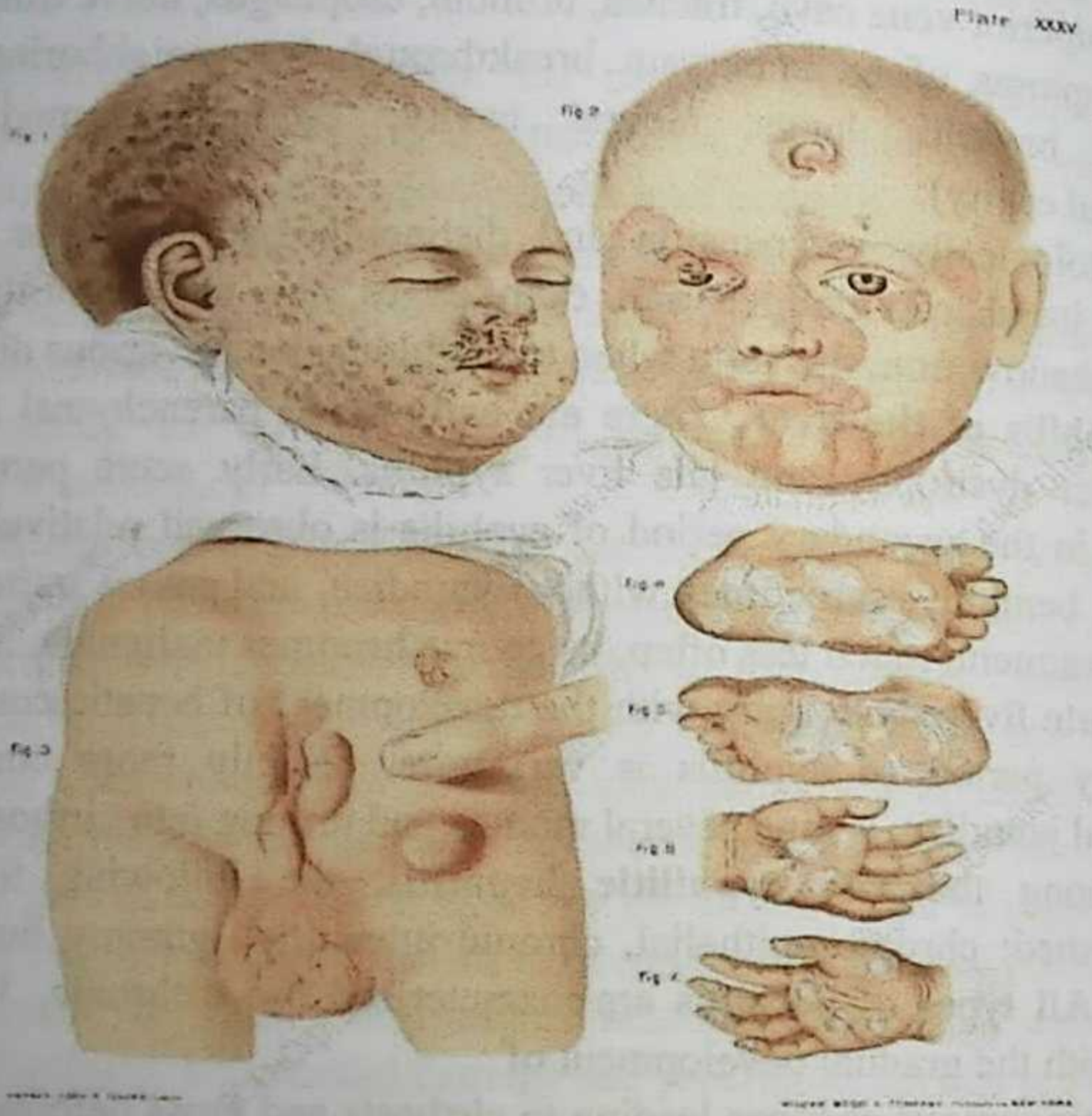
Acute liver dystrophy with the development of hepatic coma in the secondary period of syphilis is very rare. Slightly more common is prolonged jaundice, lasting several months and turning into cirrhosis.

Among the **late syphilitic hepatitis**, the following forms are distinguished: chronic epithelial, chronic interstitial, gummy limited and miliary. All types of hepatitis are characterized by a chronic, long-term course with the gradual development of

sclerogumous changes leading to cirrhosis and gross deformity of the liver.

CONGENITAL SYPHILIS

Congenital syphilis (**syphilis congenita**) is an intrauterine infection transmitted transplacentally from a mother with syphilis to the fetus and is characterized by a peculiar clinical picture and age-related periodicity. Epidemiology. Recently, congenital syphilis has been quite rare. According to our data, 3-5 cases of the disease per year have been registered in St. Petersburg in recent years. In this regard, its diagnosis is currently difficult, due to the absence of classical manifestations or an oligosymptomatic clinical picture.



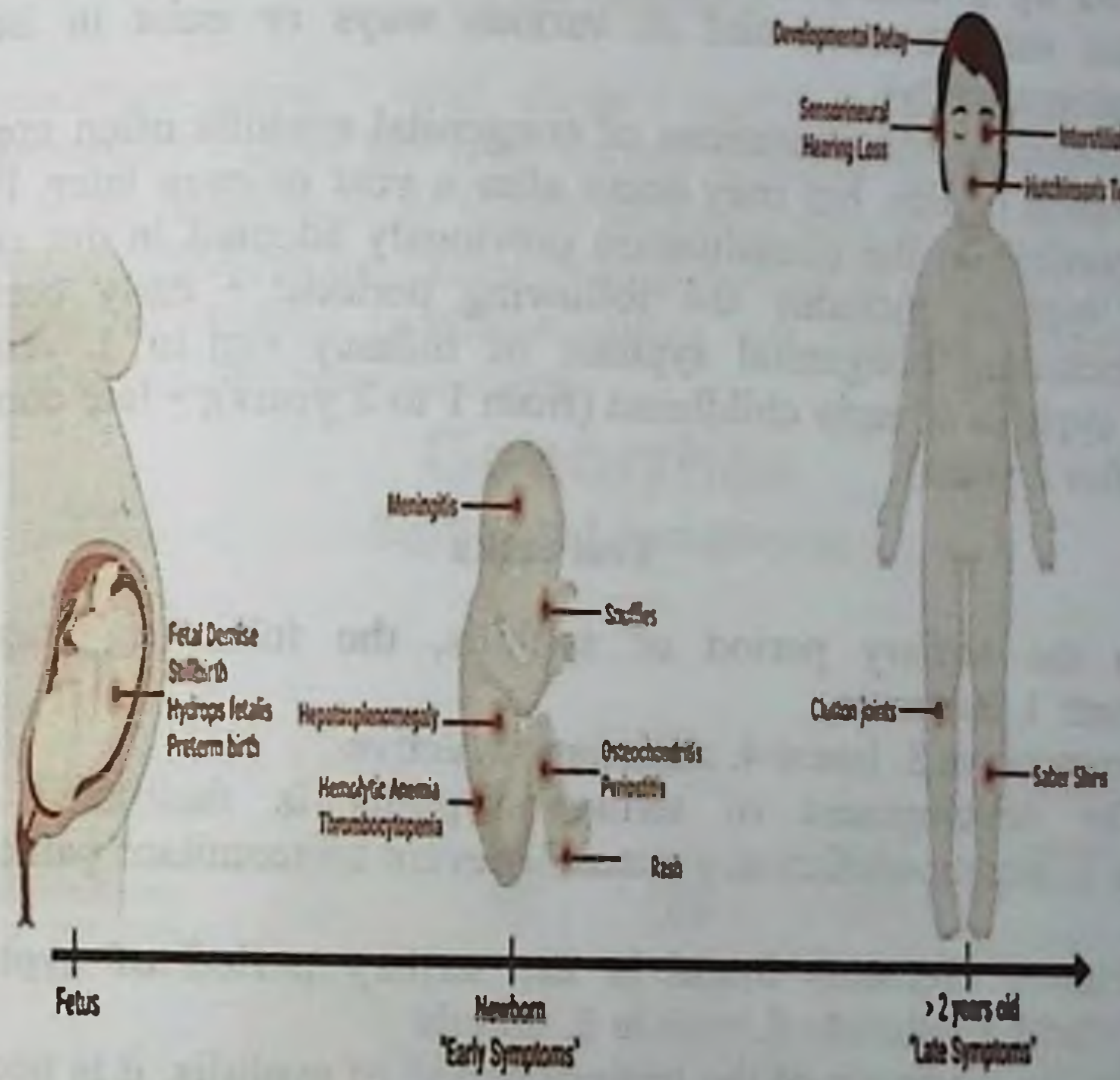
Pic-29 Congenital Syphilis

Etiology and pathogenesis. Infection of the fetus with syphilis occurs transplacentally starting from the 4th month of pregnancy. With untreated syphilis, a mother can infect herchildren throughout the entire childbearing period, but the most dangerous for offspring are the first 3 years from the moment of her infection, which corresponds to the primary, secondary and early latent period of acquired syphilis.

Treponema pallidum enters the fetus through the umbilical vein, lymphatic clefts of the umbilical vessels and through damaged vessels of the placenta.

Fetal syphilis. A stillborn fetus usually has a low body weight that does not correspond togestational age, or is born edematous and often with maceration of the skin. Autopsy reveals specific changes in tissues

characterized by inflammatory phenomena in the form of small cell infiltration, with a predominance of lymphocytes and plasma cells, followed by proliferation of connective tissue with the formation of miliary or solitary gums.



Pic-29 Congenital Syphilis

Most often, the liver and spleen are affected, which increase in size and thicken. In the kidneys, the development of tubules and glomeruli is disrupted, cysts are formed.

In the lungs, proliferation of connective tissue is found in the interalveolar spaces, the affected areas of the lungs are airless, compacted, have a whitish-gray color, which is called **"white pneumonia"**. Such lung damage may be incompatible with life. At 6-7 months of intrauterine life, long tubular bones are affected.

On radiographs in these cases, specific osteochondritis and periostitis can be detected. Changes on the part of the central nervous system are manifested by productive leptomeningitis with severe vascular sclerosis, meningoencephalitis, choroiditis, less often - gummous formations.

The cardiovascular system and gastrointestinal tract in fetuses are affected relatively rarely. If there is no pre- or intranatal death of the fetus, the newborn may develop a picture of active congenital syphilis, characterized by a variety of manifestations affecting many organs and systems that can be combined in various ways or exist in isolation (monosymptomatically).

Classification. Manifestations of congenital syphilis often appear in the first months of life, but may occur after a year or even later. For this reason, according to the classification previously adopted in our country, congenital syphilis includes the following periods: • early congenital syphilis, including congenital syphilis of infancy (up to 1 year) and congenital syphilis of early childhood (from 1 to 2 years); • late congenital syphilis (after 2 years).

Test tasks

1. In the tertiary period of syphilis, the following stages are distinguished: 1. subacute

2. progressive 3. latent 4. stationary 5. active

2. The development of tertiary syphilis is facilitated by: 1. alcoholism 2. immunodeficiency states 3. severe concomitant pathology 4. all right

3. Primary elements found in the tertiary period of syphilis: 1. roseola 2. wheal 3. tubercle 4. vesicle 5. nodule

4. For the diagnosis of the tertiary period of syphilis, it is necessary: 1. REEF 2. microscopic examination of eruptive elements 3. RIBT 4. cultural examination 5. histological examination

5. Syphilitic tubercles are characterized by: 1. positive symptom of Besnier- Meshchersky 2. dense consistency, copper-red color 3. pain on palpation 4. after ulceration - "mosaic scars" 5. the possibility of recurrence on the scar

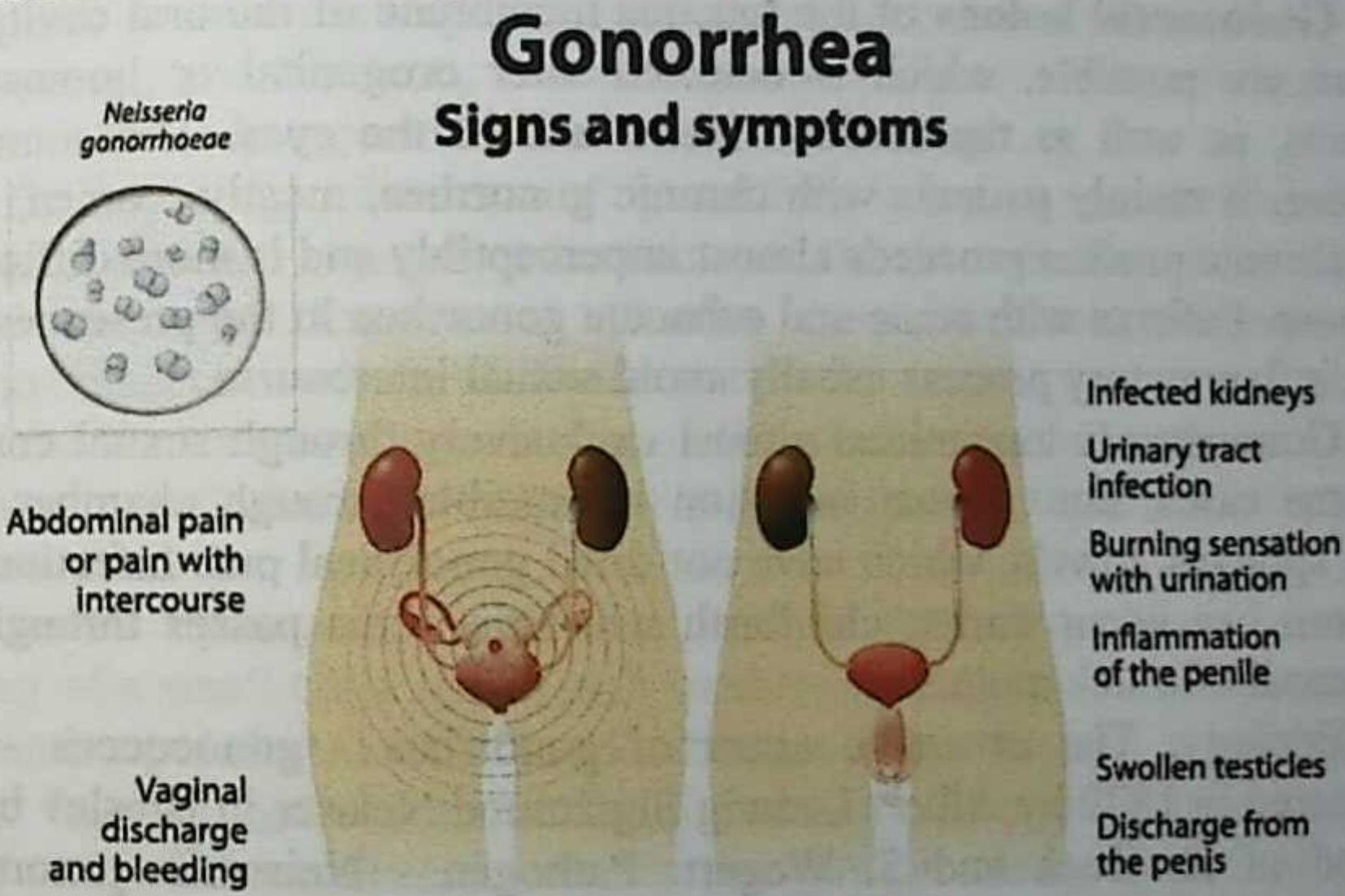
6. Gummy syphilide is characterized by: 1. low contagiousness 2. high contagiousness 3. densely elastic consistency 4. the bottom of the ulcer bleeds 5. the presence of a gummous rod

7. Scars with tuberculous syphilis are: 1. atrophic 2. mosaic 3. bridge-like 4. stamped.

CHAPTER 7 GONOCOCCA AND NON-GONOCOCCA URETHRITIS. COMPLICATED AND UNCOMPLICATED GONORRHEA. TRICHOMONIASIS. CHLAMYDIOSIS.

GONORRHEA

Gonorrhea (gono - seed, rhoea - outflow; syn. gonorrhea - from German tropfeln - drip) has been known to mankind since ancient times. Already in the third book of Moses on the Levites, in chapters 15 and 16, there are indications of the existence of gonorrhea at that time. In the oldest monuments of Indian medicine, there are detailed descriptions of gonorrhea with all its complications.



Pic-30 Gonorrhea

Descriptions of the clinical manifestations of gonorrhea are found in the ancient writings of China, Egypt, the Roman Empire, Greece. Hippocrates (460-350 BC) described gonorrhea as painful and difficult urination, affecting mainly young and middle-aged people "due to excessive indulgence in the pleasures of Venus." Gonococcal infection is one of the possible causes of diseases of the genital tract in young girls.

The issue of childhood vulvovaginitis is not new. Already in the first half of the XVIII century. in the medical literature there is a completely clear and accurate description of the symptoms of this disease.

Two cases relating to 1732 and beautifully described by Yog. Storch, illustrate a typical "familial" gonorrhea. The author erroneously connects the emergence of the disease in a non-sexual way with the hereditary constitution, since "the mother of these children and

even their grandmother are obsessed with the same disease."

Gonorrhea is an infectious disease caused by a specific pathogen, gonococcus, transmitted mainly through sexual contact and characterized mainly by lesions of the mucous membranes of the genitourinary organs.

Gonococcal lesions of the mucous membrane of the oral cavity and rectum are possible, which is detected after orogenital or homosexual contacts, as well as the mucous membrane of the eyes. The source of infection is mainly patients with chronic gonorrhea, mostly women, since their chronic process proceeds almost imperceptibly and is more difficult to diagnose. Patients with acute and subacute gonorrhea in the presence of an acute inflammatory process usually avoid sexual intercourse.

Gonorrhea is transmitted almost exclusively through sexual contact. In some cases, non- sexual infection is possible through chamber pots, linen, sponges, towels, which have not dried gonorrheal pus. Infection of a newborn can occur during childbirth when the fetus passes through the birth canal of a sick mother.

Etiology. The causative agent of gonorrhea - gonococcus - was discovered in 1879 by Albert Ludwig Sigizmund Neisser in Breslav by the method of R. Koch and S. Wegert. Pathogen - Neisseria gonorrhoea gonococcus - gram-negative diplococcus, shaped like coffee beans, 1.25 to 1.6 microns long and 0.7-0.8 microns in diameter, facing each other with their concave surface. Gonococcus belongs to the Neisseriaceae family, genus Neisseria. Gonococci stain well with aniline or Gram stains. Gonococci are usually located intracellularly in the protoplasm of leukocytes, usually in groups, but extracellular gonococci can sometimes be seen.

Studies of gonococci in recent years indicate changes in their biological properties (the presence of capsules, phagosomes, |3-lactamase,

reduced sensitivity to antibiotics, the appearance of L-forms). Gonococci mainly affect the mucous membrane of the urogenital tract, lined with cylindrical epithelium (urethra, cervical canal), less often - mucous membranes lined with stratified squamous epithelium (vulva, vagina). Vulvovaginitis occurs in girls, pregnant women and postmenopausal women.

Possible damage to the mucous membrane of the rectum, conjunctiva, nasopharynx, rarely skin. In men, the process can spread to the prostate gland, seminal vesicles, epididymis, testis, vas deferens, and in women - to the uterus, ovaries, fallopian tubes. Spreading through the bloodstream, gonococci can sometimes cause gonococcal sepsis and metastases to various organs. Gonococcal bacteremia affects the joints, eyes, pleura, endocardium, muscles, bones, and nerves. In newborns, the eyes are affected,conjunctivitis and keratitis develop.

Epidemiology. Sources of gonorrhea infection are patients with asymptomatic and asymptomatic forms of the disease. The main route of infection is sexual. Extrasexual infection is possible through direct contact (due to contact or leakage of discharge into the
eyes, on the mucous membrane of the oral cavity and rectum; when passing through the birth canal; the possibility of intrauterine infection is discussed).

Indirect non-sexual infection is rare with very close household contact of a small child with a sick mother, more often girls through a bed or chamber pot shared with the mother,as well as through common toilet items. In men, gonococcal infection causes inflammation of the mucous membrane of the urethra, in women the cervical canal, urethra, glands of the vestibule of the vagina and the rectum are affected, in girls * vulvovaginitis develops, they may develop proctitis due to leakage of purulent discharge from the vagina; in men - passive homosexuals develop proctitis.

With oral-genital contact, the pharynx and tonsils may be affected. The contact of gonococci on the conjunctiva of the eyes causes the development of gonorrheal conjunctivitis (gonoblennorrhea).

Cases of gonococcal sepsis, meningitis and arthritis in newborns are described, while it is assumed that infection could occur by the

hematogenous route or through infected amniotic fluid. An intranatal route of infection is possible.

Classification. In accordance with M K B -10, the following forms of gonococcal infection are distinguished.

Gonococcal infection of the lower urogenital tract without abscessing of the paraurethral and adnexal glands (includes: **urethritis, cystitis, vulvovaginitis, cervicitis**).

Gonococcal infection of the lower genitourinary tract with abscess formation of the paraurethral and adnexal glands (includes: **gonococcal abscess of the large vestibular glands**).

Gonococcal **pelvioperitonitis** and other gonococcal infections of the genitourinary organs (includes: **epididymitis, orchitis, prostatitis**, inflammatory diseases of the pelvic organs in women).

Gonococcal eye infection (includes: **conjunctivitis, iridocyclitis, neonatal ophthalmia gonococcus**).

Gonococcal infection of the musculoskeletal system (includes: **arthritis, bursitis, osteomyelitis, synovitis, tenosynovitis**).

Gonococcal pharyngitis.

Gonococcal infection of the anorectal region.

Other gonococcal infections (includes: **brain abscess, endocarditis, meningitis, myocarditis, pericarditis, peritonitis, pneumonia, sepsis, skin lesions**).

In Russia, a range of diagnostic and therapeutic interventions for patients with gonorrhea has been defined, united by the following models:

- localized gonococcal infection;
- gonococcal infection with systemic manifestations.

The clinical classification of gonorrhea is based on the duration of the disease: fresh (up

to 2 months) and chronic (over 2 months). Depending on the manifestation of manifestations, fresh gonorrhea is isolated, which is divided into acute, subacute and torpid. Chronic gonorrhea, as a rule, has a torpid course with exacerbations.

Epidemiologically isolated latent gonorrhea (gonococcal), which is more common in women. Clinical manifestations and inflammatory reaction are absent.

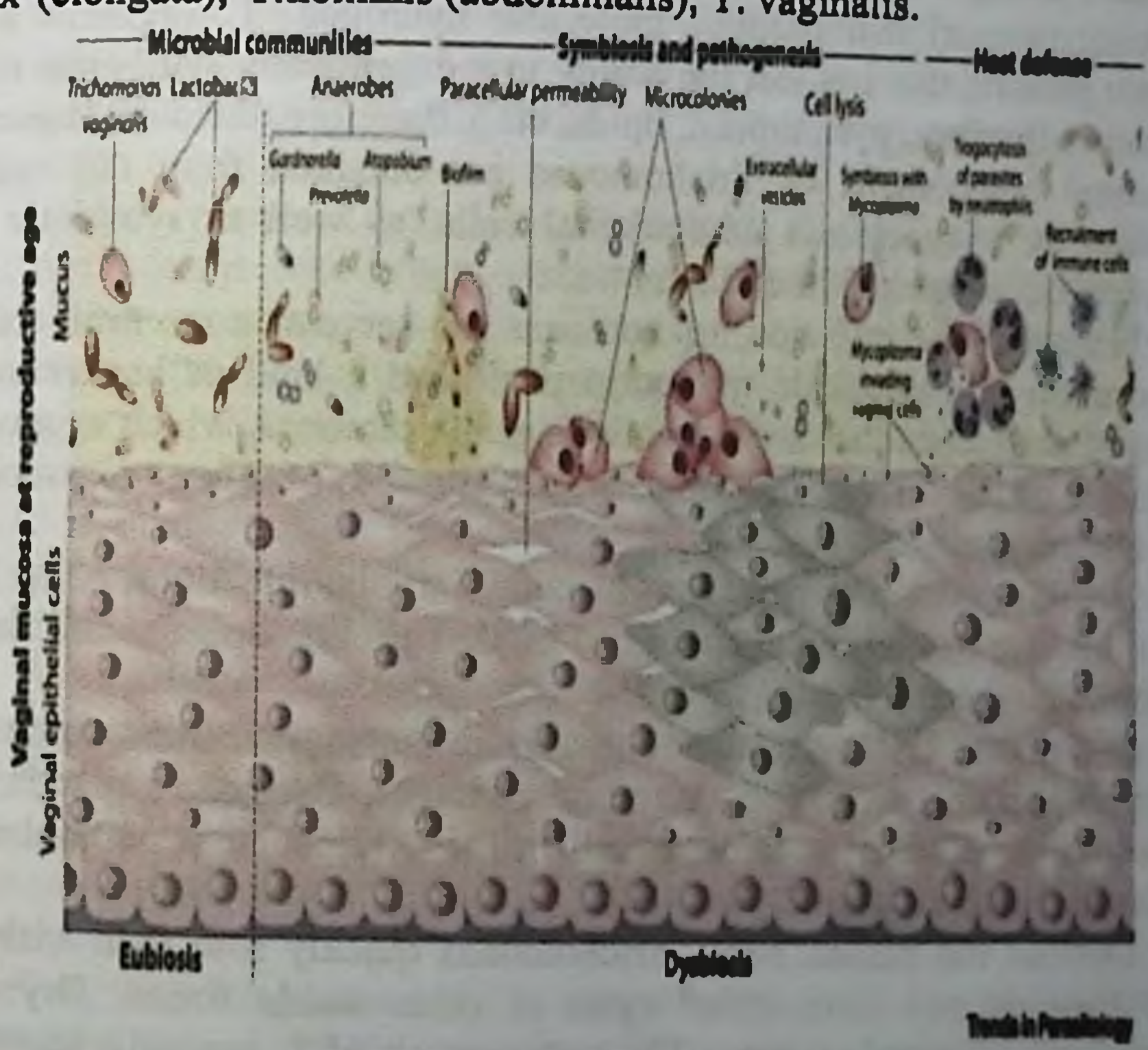
UROGENITAL TRICHOMONIASIS

Urogenital trichomoniasis (UGT) is a disease of the genitourinary system caused by theprotozoan unicellular parasite Trichomonas vaginalis.

The etiology of Trichomonas vaginalis belongs to the kingdom of higher protozoa(protists)

- Protozoa, flagella class
- Flagella, family Trichomonadidae, genus Trichomonas.

Three types of Trichomonas can parasitize in the human body: T. tenax (elongata), T.hominis (abdominalis), T. vaginalis.



Pic-31 Urogenital Trichomoniasis

Trichomonas tenax lives in the oral cavity. In dyspeptic disorders, as a rule, in children and less often in adults, the commensal of the large intestine is isolated - **Trichomonas hominis**. T. vaginalis is a single-celled protozoan 10 μm long and 7 μm wide, with five flagella. Trichomonas can

be pear-shaped, amoeboid or spherical in shape. The appearance of the cell varies depending on the growth and physico-chemical conditions of the environment. Unfavorable conditions for the growth of **T. vaginalis** contribute to the transformation of the amoeboid into an oval, so-called **non-flagellated (amastigote)** form, somewhat reminiscent of pseudocysts.

The nucleus of T. vaginalis is located in front of the cell and is surrounded by a porous

nuclear membrane.

In living Trichomonas, light microscopy reveals granules (hydrogenosomes) that produce molecular hydrogen. T. vaginalis is an obligate parasite that has lost the ability to independently synthesize many substances (purines, pyrimidines, lipids, etc.), therefore, the most important nutritional components of Trichomonas are obtained from the vaginal secretion by phagocytosis of epithelial cells, as well as symbiotic and opportunistic bacteria of the urinary tract.

With a mixed infection, trichomonas can serve as a reservoir for the preservation of pathogenic microorganisms and a kind of barrier to the action of antibiotics that are not active against protozoa, which aggravates the course of the disease and can cause a recurrence of infection associated with **trichomoniasis.**

In this regard, with mixed protozoan-microbial infections, either both infections are treated simultaneously, or first trichomoniasis, and then a concomitant disease.

In women, Trichomonas live mainly in the vagina, from where they penetrate into the urethra, paraurethral passages, Bartholin's glands, less often into the bladder, cervical canal. In men, Trichomonas colonize the urethra and prostate gland.

Outside the human body, Trichomonas quickly lose their viability, since they do not form either cysts or other stable forms. Drying is especially detrimental to them. The pathogenesis of T. vaginalis secretes a cellular uncoupling factor (CRF), with the help of which the pathogen penetrates into the intercellular space and, loosening the tissue, promotes the penetration of bacteria and the formation of an inflammation focus.

The most important link in the pathogenesis of the disease is the ability of Trichomonas to avoid the lytic action of complement and cell-

mediated reactions of the host's immune response. It is known that iron regulates the expression of proteases that contribute to the destruction of the C3 component of complement on the surface of the pathogen, and this allows the parasite to avoid complement-dependent neutralization.

In addition, Trichomonas vaginalis, like other parasites, secrete highly immunogenic antigens that are able to neutralize antibodies or cytotoxic T-lymphocytes.

Thus, trichomonas infection does not lead to the development of a pronounced immune response. Serum and secretory antibodies detected in patients with or recovered from trichomoniasis are only witnesses of an existing or past infection, but are not able to provide lasting immunity.

Reinfection with **T. vaginalis** in humans does not elicit immune protection.

Clinical picture Clinical manifestations of UGT are very diverse: from acute forms with bright, pronounced symptoms of inflammation to mild and asymptomatic course. There are no pathognomonic clinical (subjective and objective) signs of **trichomoniasis**, and no specific morphological changes were found in the affected organs and tissues. A decisive role in the development of clinical symptoms is played by the formation of various associations of **Trichomonas vaginalis** with pathogenic and conditionally pathogenic microorganisms of the urogenital tract and the type of response of the host organism.

As is known, in associations, the pathogenicity of each "participant" undergoes certain changes and in most cases increases. In addition, the role of one or another associate member in the chronic course of the disease is almost impossible to determine, therefore, with a mixed or combined infection, a wide variety of variants of the clinical course of the disease are observed.

Diagnosis Diagnosis of UGT is based on the identification of clinical signs of the disease and the detection of T. vaginalis in the test material.

Clinical signs of Trichomonas infection in the "classic" course of the disease are quite pathognomonic and include **yellow-green frothy discharge, itching, dysuria, dyspareunia, and "strawberry"** appearance **(pinpoint hemorrhages)** of the cervix and vagina.

However, the diagnosis cannot be made solely on the basis of the clinical picture for the following reasons:

- the listed symptoms may also be manifestations of other infections of the urogenital tract;
- characteristic for trichomoniasis "strawberry" symptom occurs only in 2% of patients;

Foamy discharge, which can be explained by the active growth of Trichomonas vaginalis, is observed in approximately 12% of infected women.

Due to the fact that the clinical symptoms of trichomonas infection often do not reflect the real picture of the disease, it is imperative to use laboratory diagnostic methods. Currently, in Russia and abroad, four laboratory methods for determining T. vaginalis are used: microscopic, cultural, immunological, and gene diagnostics.

Treatment regimens With the established diagnosis of trichomonas infection, all sexual partners are subject to treatment, even if the latter do not have clinical and laboratory signs of the disease.

In accordance with the methodological materials for the diagnosis and treatment of STIs and skin diseases, TsNIKVI (2003), the following treatment regimens for UGT are presented.

With common trichomoniasis: • metronidazole 2.0 g orally once; • or ornidazole 1.5 g orally once; • or tinidazole 2.0 g orally once. Alternative regimen: • ornidazole 500 mg orally every 12 hours for 5 days; • or metronidazole 500 mg orally every 12 hours for 7 days; • or nimorazole 2.0 g orally once.

For complicated or recurrent trichomoniasis: • metronidazole 500 mg orally 3 times a day for 7 days; • or tinidazole 2.0 g orally once a day for 3 days; • or metronidazole 2.0 g orally once a day for 3 days; • or ornidazole 500 mg orally every 12 hours for 10 days.

Perhaps the simultaneous use of local protistocidal and anti-inflammatory drugs and

immunomodulators. Trichomoniasis in pregnant women (treatment begins no earlier than the second trimester): • ornidazole 1.5 g orally once at bedtime.

Alternative regimen: • metronidazole 2.0 g orally once at bedtime. In the presence of symptoms in the early stages of pregnancy, local treatment with pimafucin in suppositories is prescribed. In urogenital trichomoniasis in children: • Ornidazole 25 mg/kg of body weight, the daily dose is prescribed in 1 dose at night.

Alternative scheme: • metronidazole for 7 days, the dose depends on age: ✧ from 1 year to 6 years - 1/3 tablet orally 2-3 times a day; ✧ from 6 to 10 years - 125 mg orally 2 timesa day; ✧ from 11 to 15 years - 250 mg orally 2 times a day. Precautions 1. To avoid the development of severe adverse reactions, patients are warned to avoid the use of alcohol and products containing it while taking metronidazole and tinidazole, as well as within 24hours after discontinuation of the drug.

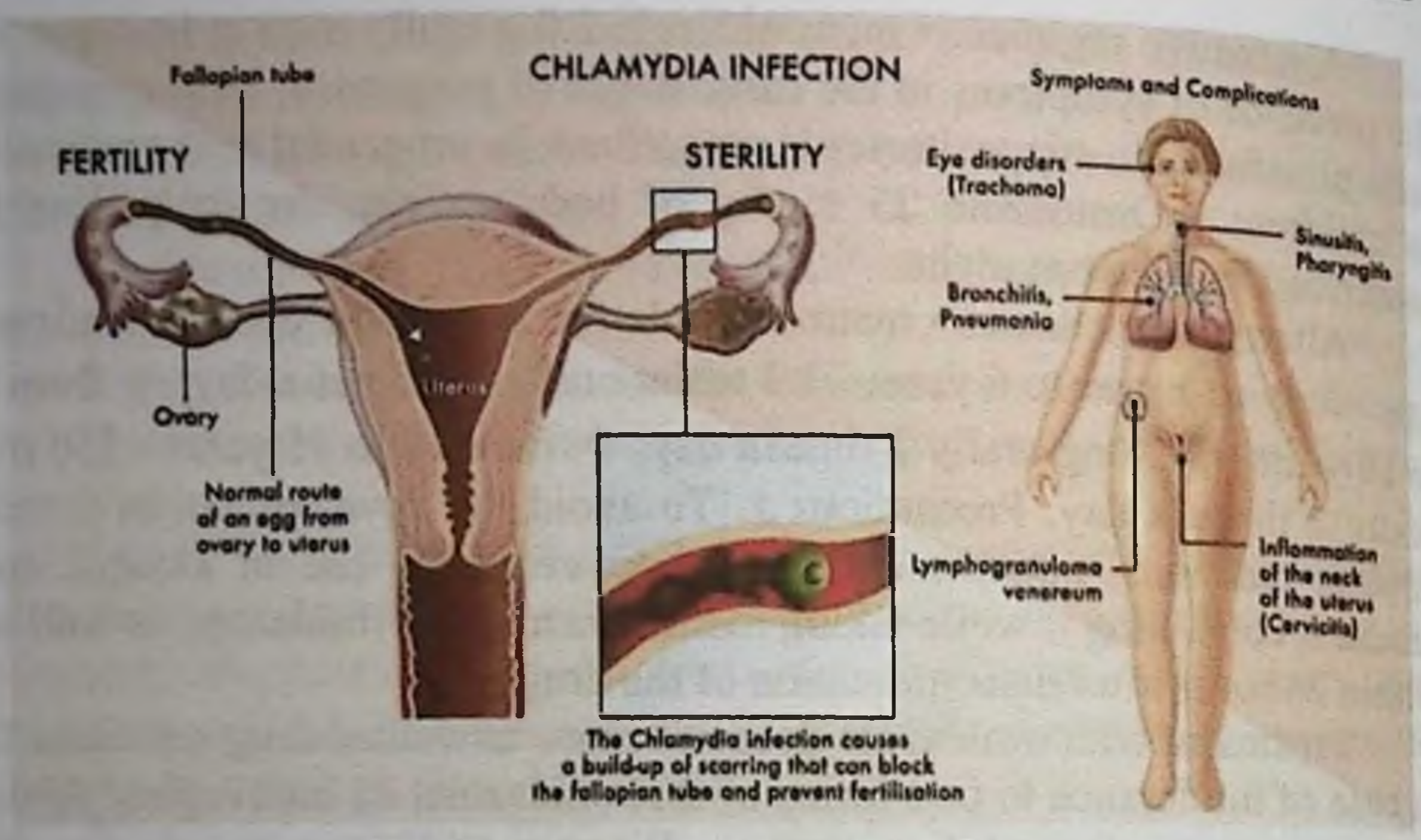
Treatment with ornidazole does not cause unwanted drug reactions. 2. In case of intolerance to oral forms of metronidazole, its intravaginal forms are also contraindicated. Cure criteria When establishing criteria for the cure of trichomoniasis, it is necessary to distinguish between etiological and clinical recovery. Under the etiological recovery is meant the persistent disappearance of T. vaginalis from the urogenital tract of the patient after treatment, which is confirmed by microscopic examination, as well as by culture andPCR.

UROGENITAL CHLAMYDIA INFECTION

Chlamydial infection is a sexually transmitted infection caused by Chlamydiatrachomatis (DK serotypes).

Etiology Chlamydia are classified as obligate intracellular parasites, and their reproduction cycle is realized when interacting with a sensitive host cell.

Pathogenesis In the modern view, the development of pathology in urogenital chlamydial infections is a consequence of tissue damage as a result of intracellular reproduction of **C. trachomatis** and the subsequent inflammatory reaction of the body.



Pic-32 Urogenital Chlamydia Infection

Heat shock protein (Hsp60) is of great importance in the formation of the immune response. Its expression under unfavorable conditions, in particular in response to the action of certain antibiotics, leads to the production of pro-inflammatory cytokines, suppression of the synthesis of the main outer membrane protein (MOMP) and lipopolysaccharides.

The course of urogenital chlamydial infections is influenced by disorders of the patient's immune system, features of the histocompatibility complex, and a decrease in the level of unsaturated fatty acids. The latter leads to a change in the structure of target cell membranes and becomes a favorable background for adhesion and reception of chlamydial elementary bodies on the cell surface.

Clinical manifestations General information Chlamydial infection has no specific signs by which it can be distinguished from inflammatory diseases of other etiologies. The inflammatory process in the genitourinary tract begins with damage to the mucous membrane of the cervical canal, urethra and rectum, so the most common clinical manifestations of CCI are signs of **urethritis** in men and **cervicitis** in women.

In this case, the severity of clinical symptoms can vary from an active inflammatory reaction, accompanied by copious discharge from the urethra

and / or cervical canal, to scanty mucous secretions without obvious signs of inflammation. During the development of infection, nearby organs and tissues are involved in the inflammatory process, thereby complicating the course of CCI.

Diagnosis Clinical picture: signs and symptoms due to cervicitis and urethritis, as well as

complications of the disease.

Laboratory diagnostics The cultural method is based on the isolation of the pathogen in a culture of McCoy, L-929 or HeLa cells. It has the highest specificity, but its use in practical healthcare is limited by high cost and labor intensity.

Nucleic acid amplification methods. PCR is a repeatedly repeated cycles of synthesis (amplification) of a specific nucleotide sequence of the target gene; the number of molecules in a short time can reach millions of copies.

The sensitivity of PCR is 98–100%, the specificity is 82–100%. PCR is the main method for diagnosing CCI.

Serological methods. Based on the determination of specific antibodies to chlamydia (IgA, IgG) in the blood serum. The test is justified in the localization of pathological processes in the uterus and its appendages, generalized chlamydial infection, but is used as a screening test (must be confirmed by detection of the pathogen by PCR).

Differential diagnosis It is carried out with gonococcal, trichomonas, mycoplasmal infections.

Treatment The protocol for the management of patients with CCI has not yet beenapproved.

The legal document that determines the tactics of a doctor in treatment is the Federal Guidelines for the Use of Medicines (formular system), issues VIII and subsequent, approved by the Ministry of Health and Social Development of the Russian Federation as a regulatory document for doctors on rational pharmacotherapy of the most common diseases.

Federal guidelines recommend the following treatment regimens for urogenital chlamydial infections. Drugs of choice (for adults and children over 12 years of age) • In acute course, azithromycin 1.0 g once.

With chlamydia of the upper parts of the genitourinary system, pelvic organs, 1.0 g once aweek, for 3 weeks. • Doxycycline orally 0.1 g every 12 hours for 7-10 days in acute and subacute course, with lesions of the upper genitourinary system, pelvic organs and other organs - 0.1 g every 12 hours for 14-21 day Alternative drugs • Oral ofloxacin 0.4 g every 12 hours for 7-10 days.

• Roxithromycin inside 0.15 g every 12 hours for 10 days. • Erythromycin inside 0.5 g every 6 hours for 10 days.

The Russian Society of Dermatovenereologists additionally recommends josamycin 0.5 gorally 3 times a day for 7 days as the drug of choice. In CSI of the upper genitourinary system, pelvic organs and other organs, the duration of the course of treatment with alternative drugs should be 14–21 days.

For the treatment of pregnant women • Spiramycin1 3 million IU 3 times a day for 7-10 days. • Josamycin 0.5 g 3 times a day for 7 days. • Azithromycin 1.0 g orally once. •

Erythromycin 0.5 g orally 4 times a day for 7 days. • Amoxicillin 0.5 g 3 times a day for 7days.

In the Russian Federation, a manual for doctors "Infections in obstetrics and gynecology: diagnostics and antimicrobial chemotherapy" was published, approved by order of the Ministry of Health and Social Development of the Russian Federation dated May 19, 2006 No. 16/171-16-3, which presents treatment regimens for CCI in pregnant women.

Treatment of children weighing less than 45 kg and under the age of 8 years • Josamycin 50 mg/kg body weight in 3 divided doses for 10 days. • Azithromycin at a daily dose of10 mg/kg of body weight 1 time per day for 3 days. • Erythromycin 50 mg/kg body weight in 4 oral doses for 10-14 days. Recommended regimens • Azithromycin 1 g once orally. • Amoxicillin 500 mg 3 times a day for 7 days. • Erythromycin (base) 500 mg 4 times a day for 7 days.

Alternative regimens (designed for 7 days) • Josamycin 750 mg 2 times a day. • Erythromycin (base) 250 mg 4 times a day. Treatment of clinical complications caused by urogenital chlamydial or mixed infection (**epididymitis, prostatitis, salpingo-oophoritis, endometritis, infertility**)

is carried out taking into account the diagnosis and individual characteristics of the patient's body.

It is advisable to use azithromycin as an intravenous infusion, drip 500 mg 1 time per day for 2 days, then orally at a dose of 250 mg to complete the 7-day general course of therapy.

Perhaps its combination with metronidazole. The degree of cure of urogenital chlamydialinfection should be assessed 1 month after the end of etiological therapy by PCR. Prevention Primary prevention consists of avoiding sex with unknown partners or using barrier methods of contraception. Antenatal screening of pregnant women for C.trachomatis may prevent the development of chlamydial infection in newborns.

Secondary prevention consists in the simultaneous treatment of the patient and sexual partners (who had contacts for 60 days - according to the same schemes). During the period of treatment and dispensary observation, it is recommended to refrain from sexual intercourse or use barrier methods of contraception until the criterion of cure is established.

Tests for knowledge control

1. Tests for knowledge control 1. Which of the following pathogens causes dyshidrotic epidermophytosis of the feet? a) red trichophyton; b) Trichophyton mentagrophytes ; c) Epidermophyton floccosum ; d) trichophyton purple; e) Trichophyton warty.

2. Anthropophilic fungi that cause trichomycosis include all of the following, except: a) red trichophyton; b) fluffy microsporum; c) Shenlein's trichophyton; d) mentagrophytes trichophyton; e) rusty microsporum.

3. Which of the mycoses is most characterized by the formation of pronounced hyperkeratosis, cystic and annular peeling on the feet? a) athlete's foot; b) inguinal epidermophytosis with damage to the foot; c) rubrophytosis of the foot; d) anthropophilic trichophytosis.

4. Which of the following drugs is not effective in the treatment of dermatomycosis? a) terbinafine; b) itraconazole; c) ketoconazole; d) fluconazole; e) norsulfazole.

5. Superficial trichophytosis of the scalp is characterized by: a) the presence of small multiple foci with unchanged hair; b) large and small foci with black dots; c) clear outlines of foci; d) thinning of hair in the frontal

and temporal regions; e) the presence of a white muff at the base of the hair.

6. Hair fragility occurs at a height of 5-8 mm: a) with infiltrative-suppurative trichophytosis;

b) favus; c) microsporia; d) superficial trichophytosis; e) focal alopecia.

7. What is the dosage of terbinafine for the treatment of onychomycosis of the feet? a) 50 mg/day; b) 100 mg/day; c) 250 mg/day; d) 150 mg/day; e) 500 mg/day.

Answer standards: 1B; 2G ; _ 3C; 4D ; _ 5G ; _ 6B; 7B.

1. What dermatological diseases are caused by herpes viruses? a) warts; b) shingles; c) toxic epidermal necrolysis; d) lichen planus; e) molluscum contagiosum.

2. Name the causative agent of herpes zoster: a) herpes simplex virus; b) smallpox vaccine virus; c) chickenpox virus; d) shingles virus; e) varicella-zoster virus and herpes zoster.

3. What is the name of the mechanism of formation of vesicles in herpes? a) spongiosis; b) balloon degeneration; c) acantholysis; d) epidermolysis; e) destruction of the basement membrane.

4. What complications are not observed in herpes zoster? a) secondary infection; b) postherpetic neuralgia; c) cachexia; d) scarring of the skin; e) dysfunction of the gastrointestinal tract.

5. What laboratory methods are not used in the diagnosis of herpes simplex? a) isolation of the pathogen in cell cultures; b) infection of chicken embryos; c) serological methods; d) biochemical blood test; e) electron microscopy.

6. The reservoir of papillomavirus (wart virus) is: a) a sick person; b) cattle; c) rodents; d) amphibians (toads, frogs); e) blood-sucking insects.

7. Infection with warts occurs: a) by direct contact through skin damage; b) alimentary way;

c) inhalation; d) blood transfusion; e) any of the above methods.

8. The most frequent localization of warts vulgaris: a) external genitalia; b) hands; c) the scalp; d) mucous membranes; d) face.

9. What diseases should be differentiated from genital warts in the perianal region? a) limited neurodermatitis; b) candidiasis; c) primary syphilis; d) secondary syphilis; e) nonspecific ulcerative colitis.

10. Which of the following ointments do not have an antiviral effect? a) helepic acid; b) acycloviric acid; c) sulfuric acid; d) oxolinic acid; e) gossypol.

Answer standards: 1B; 2D ; _ 3b; 4b; 5G ; _ 6A; 7a; 8B; 9g; 10V.

Task #1

A 40-year-old man addressed a dermatologist with complaints of facial skin lesions for 2 years. The process is persistent chronic, poorly treatable. On examination, multiple folliculitis and ostiofolliculitis are visible on the face.

Tasks:

1. Make and justify the diagnosis.
2. Make an examination plan.
3. Name the specialist whose consultation the patient needs.

5.Prescribe a treatment. Standard for task number 1

1. Staphylococcal sycosis (chronic course, typical clinical picture).
2. General clinical methods (UAC, OAM), bacteriological method, antibiogram,
4. Consultation with an immunologist.
5. Antibiotics in accordance with the antibiogram, immunocorrection. Locally - aniline dyes, baneocin ointment, fucidin, suberythemal doses of UVR, spa treatment during remission.

Task #2

A 26-year-old patient, a music teacher, consulted a doctor with complaints of rashes in the beard and mustache area, accompanied by a burning sensation, slight itching, and tingling.

Considers himself ill for 2.5 years. Initially, separate vesicles with purulent contents appeared on the chin, pierced in the center by hair. The pustules dried up with the formation of crusts, then new rashes appeared in the same place. The skin in these places became dense, bluish in color. The process is undulating. With each exacerbation, the lesion becomes larger. I went to the doctor at the place of residence, disinfectant solutions and ointments were prescribed, there was no effect from the treatment. The

disease has a depressing effect on the mental state of the patient. From associated diseases notes chronic sinusitis, chronic rhinitis.

On examination: the process is located on the skin of the chin and nasolabial triangle. The skin on palpation is dense, infiltrated, bluish-red. Against this background, a large number of pustular elements, many of which are penetrated by hair. Multiple dirty yellow crusts.

Tasks:

1. Make and justify the diagnosis.
2. ake an examination plan.
3. Perform differential diagnostics.
4. Give treatment.

Standard for task number 2

1. Staphylococcal sycosis.
2. General clinical method, blood for RMP, RPHA, ELISA, HIV, bacteriological culture withantibiogram.
3. Impetigo vulgaris, infiltrative suppurative trichophytosis.
4. Epilation, immunocorrection, antibiotic therapy.

Task #3

A 38-year-old patient came to see a general practitioner with complaints of a painful red induration above the upper lip on the left. Restless chills and feeling unwell. Sick for 3 days. The onset of the disease is associated with the fact that she tried to squeeze out the acne element above the lip. At this point, a painful seal formed, which quickly increased in size, the skin over it turned red. Body temperature rose to 37.5°C, headache, general malaise appeared.

On examination: on the skin above the upper lip there is a node up to 1.5 cm in diameter, painful on palpation, located deep in the skin. The skin above the node is edematous, bluish- cherry color. Submandibular lymph nodes are enlarged, painful.

Tasks:

1. Make and justify the diagnosis.
2. Make an examination plan.
3. Specify possible complications of the disease.
4. Prescribe the treatment for this patient.
5. Specify measures to prevent the disease.

Standard for task number 3

1. Furuncle in the area of the nasolabial triangle.
2. General clinical methods, surgeon's consultation.
3. A complication of boils in the face (nasolabial triangle) is - purulent meningitis, vascular thrombosis, sepsis.
4. Treatment plan: hospitalization in the surgical department; antibiotics (kefzol, ceftriaxone, sumamed, etc.), vitamins B1, B6, B12, ascorutin, telfast 180 mg 1t. 1 time per day, licopid 10 mg 1 time for 10 days.

External therapy: opening a boil; on the first day - hypertonic solution, then "Levomekol" ointment, followed by the transition to fucidin cream 1% 2 times a day until complete healing.

5. Primary prevention of pyoderma - compliance with the rules of personal hygiene, timely antiseptic treatment of microtraumas, cracks, wound surfaces, etc. Secondary prevention - preventive medical examinations of children's groups and persons of decreed groups.

Task #4

A 32-year-old patient addressed a dermatologist with complaints of a painful red induration inthe forehead. Restless chills and feeling unwell.

Sick for 5 days. The disease is associated with the fact that she tried to squeeze out an abscessin the forehead. A day later, a painful induration a little larger than a pea formed. Two days later, the infiltrate increased, the skin over it turned red. The temperature was 38.4 C. The

patient began to suffer from headaches and general malaise.

local status. In the forehead area, a knot the size of a cherry, painful on palpation. The skin above the knot is bluish-red. Regional lymph nodes are enlarged, painful.

Tasks:

1. Make and justify the diagnosis.
2. Outline a plan of treatment and preventive measures for this patient.
3. Specify possible complications.
4. Give recommendations to the patient after treatment.

Standard for task number 4

1. Furuncle in the forehead.

2. Antibiotics, topically - pure ichthyol, UHF.
3. Thrombosis of cerebral vessels, meningitis, sepsis.
4. Exclude traumatization of rashes on the face, self-treatment. Avoid hypothermia.

Task number 5

A teenager consulted a dermatologist with complaints of a painful red induration above the upper lip on the left. Restless chills and feeling unwell.

Sick for 5 days. An abscess appeared above the upper lip. The patient tried to squeeze it out. A day later, a painful induration a little larger than a pea formed. Two days later, the infiltrate

increased, the skin over it turned red. Body temperature was 37.8 malaise appeared.

C, headache, general

local status. On the skin of the nasolabial triangle on the left, a knot the size of a cherry, painful on palpation. The skin above the knot is bluish-red. Submandibular lymph nodes are enlarged, painful.

Tasks:

1. Make and justify the diagnosis.
2. Specify possible complications of this disease.
3. Make a plan for treating the patient.
4. Consult the patient after treatment. Standard for task number 5

1. Furuncle in the area of the nasolabial triangle.
2. Complications of this disease:

A complication of a boil in the face (nasolabial triangle) is thrombosis, abscess, sepsis.

3. Treatment of this disease.

Hospitalization in the surgical dental department.

Appointment of β-lactamase-resistant antibiotics (kefzol, ceftriaxone, sumamed, etc.); vitamins B1, B6, B12, ascorutin; Lactofiltrum, Telfast, Likopid.

External therapy: surgical treatment (without tissue section). First treated with 3% hydrogen peroxide solution and aniline dye. Then pure ichthyol is applied, followed by the application of ointment dressings with

a hypertonic solution, later - fucidin cream. In case of abscess formation, surgical opening and drainage.

4. Exclusion of mechanical removal (extrusion) of any elements on the face.

Task number 6

A 40-year-old patient addressed a dermatovenereologist with complaints of rashes on the skin of her back, thighs, buttocks, fever up to 38C, general weakness, malaise.

He considers himself ill for three years, when there were single painful nodes in the back and chest, accompanied by fever, general weakness. She turned to a surgeon for medical help. Boils were diagnosed. Antibacterial therapy was prescribed. Since that time, she periodically noted the appearance of boils on the skin of the trunk and extremities. Taking antibiotics gives a temporary effect.

local status. The process is disseminated. Rashes are localized on the trunk and extremities, represented by 5 limited dense nodes of purple-red color, protruding above the surface of the skin, painful on palpation. Separate nodes in the center have a pustule, in two nodes in the center a necrotic core with purulent discharge is clearly visible.

Tasks:

1. Make and justify the diagnosis.
2. Make a plan for examining the patient.
3. Prescribe a treatment.
4. Give recommendations to the patient after the course of therapy.

Standard for task number 6

1. Furunculosis.
2. OAK. Immunogram, finding out the causes of immunodeficiency. Blood sugar level, blood for HIV, hepatitis.
3. Antibiotics, staphylococcal bacteriophage, gamma globulin, toxoid; multivitamins. Outwardly - ichthyol, aniline dyes, UHF, UFO.
4. Examination for somatic pathology. Treatment of identified diseases, immunocorrection.

Task number 7

A 38-year-old patient addressed a dermatovenereologist with complaints of rashes in the beard and mustache area. Considers himself ill

for a year. Constantly on the face in the area of the beard and mustache appear pustules, crusts. The skin in these places became dense, bluish in color. The process is chronic relapsing in nature, without complete remission. With each exacerbation, the lesion becomes larger. The disease has a depressing effect on the mental state of the patient. Of the comorbidities noted chronic dental caries, sinusitis.

local status. The process is located in the area of the beard and mustache. The skin on palpation is dense, infiltrated, bluish-red. Against this background, a large number of pustular elements penetrated by hair. Multiple dirty yellow crusts.

Tasks:

1. Make and justify the diagnosis.
3. Make an examination plan.
4. Perform differential diagnostics.
5. Specify the methods of treatment and prevention.

Standard for task number 7

1. Staphylococcal sycosis.
2. General clinical, biochemical blood test, blood sugar, antibiogram.
3. Seborrheic dermatitis, impetigo vulgaris.
4. Antibiotics taking into account the antibiogram; staphylococcal bacteriophage, gamma globulin, toxoid; immunocorrectors as prescribed by an immunologist; externally - ichthyol, aniline dyes, antibiotic ointments; UHF, UFO. Permanent skin care, When shaving, use cleansing gel Sebium-mousse (Bioderma).

streptoderma Task number 8

A 75-year-old patient addressed a doctor with complaints of rashes and cracks in the corners of his mouth. Worried about itching, salivation, soreness when eating. He considers himself ill for a month, when painful cracks appeared in the corners of his mouth. He was treated on his own - he rinsed his mouth with a solution of furacilin and lubricated the cracks with iodine. She has a history of diabetes mellitus and has dentures.

On examination: in the area of the corners of the mouth there are linear cracks covered with honey-yellow crusts, upon removal of which an erosive surface is revealed. The patient has dentures and carious teeth.

Tasks:

1. Make and justify the diagnosis.
2. Indicate the etiology and predisposing factors of the disease.
3. Conduct a differential diagnosis of this disease.
4. Make an examination plan.
5. Indicate the tactics of treating this patient.

Standard for task number 81.Slit-like impetigo.

2. The causative agent is streptococcus. Predisposing factors: dentures and carious teeth,immunodeficiency in old age.

3. Candidiasis of the corner of the mouth (as one of the manifestations of candidal stomatitis), syphilitic seizure (papule - as a manifestation of secondary syphilis), ariboflavinous seizure (with hypovitaminosis of B vitamins).

4. UAC, OAM, biochemical blood test (total protein, total bilirubin, ALT, AST, alkaline phosphatase, creatinine, urea, glucose), immunity test (if necessary), culture with an antibiogram.

5. Antibiotic therapy (azithromycin 6-10 days). Local treatment: aniline dyes (methylene blue, fukortsin); cream "Fucidin" on the affected areas 2 times a day, 6-10 days.

According to indications, antifungal drugs (diflucan) for the prevention of candidiasis;immunotherapy (with severe immunodeficiency).

Task number 9

A mother with a 4-year-old child came to the doctor with complaints of rashes on the skin of the face and cracks in the corners of the mouth, accompanied by itching, salivation and pain when eating. The child fell ill 1 week ago, when rashes appeared on the skin of the face, cracks in the corners of the mouth. The child attends kindergarten. Two children in the kindergarten group have similar rashes.

local status. The process is localized on the skin of the cheeks, in the corners of the mouth. On the skin of the cheeks there are superficial pustules, from lentils to peas, the tire is sluggish, serous-purulent crusts, erosion. On the periphery of the pustules there is an edematous-hyperemic corolla. There are cracks in the corners of the mouth with fragments of the epidermis along the periphery.

Tasks:

1. Put a preliminary and justify.

2. Specify the factors for the development of this disease.
3. Differential diagnosis of this disease with other dermatoses.
4. Make a plan for treating the patient.
5. Disease prevention in kindergarten.

Standard for task number 9

1. Angular stomatitis (jam), streptococcal impetigo.
2. The occurrence of impetigo is facilitated by: poor hygienic skin care, trauma, skin maceration, decreased immunity, adenoids, diabetes mellitus, hypothermia, dryness and violation of the integrity of the epidermis, overheating.
3. With herpes infection (herpes simplex and herpes zoster), eczema (microbial, true), atopic dermatitis.
4. Treatment plan. Diet with the exception of carbohydrates. Antibiotics (Sumamed - 6 days). Antihistamines (Zyrtec). Treat the affected skin with brilliant green, treat erosion with aqueous solutions of aniline dyes (fucorcin, methylene blue), temporarily exclude water procedures and massage. Floradofilus 1 caps per day (pro- and prebiotic).
5. Examination of children and kindergarten staff.

Task number 10

A 50-year-old patient addressed a doctor with complaints of rashes and cracks in the corners of the mouth, a rash on the face. Worried about itching, salivation, soreness when eating.

He considers himself ill for 2 months, when cracks first appeared in the corners of the mouth, then rashes on the face. During this time, deterioration was followed by periods of improvement. He was treated on his own - he rinsed his mouth with a solution of furacillin and lubricated the cracks with sea buckthorn oil. Concomitant diseases include conjunctivitis, chronic colitis. Suffering from chronic sinusitis, periodontal disease.

local status. The process is localized in the corners of the mouth and on the skin of the face. There are linear slit-like cracks in the corners of the mouth. There are flaccid pustules on the skin of the face, honey-yellow crusts, when removed, the erosive surface is exposed.

Tasks:

1. Make a diagnosis and justify it.
2. List the diseases with which it is necessary to differentiate.
3. Outline a plan of treatment and preventive measures.

4. Give recommendations to the patient after clinical recovery.

Standard for task number 101.Slit-like impetigo.

2. Candidiasis, syphilitic papules.

3. Outwardly - aniline dyes, antibacterial ointments, UVI.

4. Treatment of pathology of the gastrointestinal tract, sanitation of the oral cavity, nasopharynx. Examination for other somatic pathology (diabetes mellitus, oncopathology,etc.).

FUNGAL DISEASES OF THE SKIN - MYCOSIS

Task number 11

A 70-year-old man has been suffering from skin lesions for a year.

On examination: erythema-squamous foci of various sizes with scalloped outlines are visible in the trunk area. There is obesity 2 degrees.

Tasks:

1. Make and justify the diagnosis.
2. Make an examination plan.
3. Name the specialists whose consultation the patient needs.

5.Prescribe treatment to the patient.Standard for task number 11

1. Generalized mycosis of smooth skin.
2. General clinical laboratory tests, blood for HIV, hepatitis, sugar.
3. Consultations of an immunologist, an endocrinologist.

5. Fungicidal preparations (lamizil, itraconazole), immunocorrection as prescribed by an immunologist. Locally - lamisil ointment, clotrimazole, zalain.

Task number 12

A mother with an 8-year-old child came to the doctor with complaints of slight itching and rashes on the skin of the face, a focus of baldness on the scalp.

The disease arose a month ago, when parents noticed the appearance of spots on the skin of the face, and then a focus of baldness on the scalp. Shortly before the onset of the disease, the child brought home a kitten from the street. Of the past diseases, the mother notes scarlet fever, rarely ARVI.

On examination: on the smooth skin of the face there are several erythematous foci of a rounded shape, with a periphery ridge of merged vesicles, crusts, papules, in the center the foci are covered with grayish scales. On the scalp, in the occipital region, a rounded baldness center 4 cm

in diameter, covered with gray scales. The hair in the focus is broken off at the level of 6-8 mm and has a whitish cap.

Tasks:

1. Make and justify the diagnosis.
2. Make an examination plan.
3. Name with what diseases it is necessary to differentiate.
4. Indicate the etiology and pathogenesis of this disease.
5. Outline a plan for therapeutic and preventive measures.

Standard for problem number 12

1. Microsporia of smooth skin and scalp.
2. Luminescent, microscopic and cultural diagnostics.
3. Syphilitic alopecia, other types of fungal infections of smooth skin and scalp.
4. The causative agent is fungi of the genus Microsporum feline, rusty. Affects skin, hair,nails.
5. Local and systemic antimycotics. Work in the epidemiological center. Clinical andmicroscopic control.

Task number 13

An 18-year-old patient consulted a dermatologist with complaints of spots on the skin of the neck and chest. Considers himself ill for a year. Noticed small brown spots on the skin of the chest. At first, the spots were single and did not bother. Over time, the spots became more, they increased in size. After tanning, white spots remained in their place. The dermatologist prescribed salicylic alcohol externally. After treatment, there was an improvement, but then the spots reappeared. Of the concomitant diseases notes vegetative neurosis, excessive sweating.

local status. On the skin of the upper half of the body and neck, there are scanty yellowish- brown spots covered with pityriasis scales. There are single depigmented spots on the neck.

Tasks:

1. Make and justify the diagnosis.
2. Specify the etiology and pathogenesis of the disease.
3. Make an examination plan.
4. Conduct a differential diagnosis of this disease.
5. Prescribe a treatment. Standard for problem number 13

1. Multicolored lichen (syn. pityriasis versicolor).

2. Ringworm is caused by the fungus Malassezia furfur. The fungus lives only on human skin in a saprophytic or pathogenic form. The disease is slightly contagious. Close contact is needed to transmit the disease, so these diseases are more likely to run in families. It predisposes to its occurrence a deficiency of immunity, high humidity of the skin.

3. Diagnosis of this disease. When illuminated by a Wood's lamp, the spots glow golden yellow; positive test with 5% tincture of iodine (Balzer's test); the phenomenon of Besnier - the phenomenon of "chips"; microscopic examination of scales for fungi.

4. Differential diagnosis: with pink lichen, leucoderma in secondary syphilis, vitiligo.

5. Keratolytic, antimycotic ointments, Lamisil spray. Outwardly - body scrub 1 month.

Task number 14

A mother with a 6-year-old child came to see a doctor. Complaints about the appearance of foci of baldness on the head and spots on the skin of the hands, slight itching. Two weeks ago, the child developed foci of baldness on the scalp and red spots on the skin of the hands. Similar rashes are noted in a 4-year-old sister. There are pets - a cat and a dog. The child attends kindergarten.

local status. On the skin of the forearms, there are several erythematous foci of a rounded shape, with clear boundaries, up to 2 cm in diameter. In the center, the foci are covered with grayish scales, along the periphery - a roller of merged crusts, papules, vesicles. On the scalp, in the parietal and occipital region, there are two foci of alopecia up to 2.5 cm in diameter, covered with gray asbestos-like scales. The hair in the foci is broken off at the level of 4-6 mm, surrounded by a whitish cap.

Tasks:

1. Make and justify the diagnosis.
2. Make an examination plan.
3. Conduct a differential diagnosis of this dermatosis.
4. Prescribe a treatment.
5. Specify what kind of work neo.

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