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DERMATOVENEROLOGY

CHAPTER-1

MINISTRY OF HEALTH OF THE REPUBLIC UZBEKISTAN
SAMARKAND STATE MEDICAL UNIVERSITY

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DERMATOVENEROLOGY
Chapter-1

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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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LIST OF ABBREVIATIONS AND SYMBOLS

♠ - trade name of the medicinal product
℘ — medicinal product is not registered
⊗ — canceled medicinal productAG - antigen
AGLS - antihistamine drugsAD - atopic dermatitis
ACTH - adenocorticotropic hormoneANF - antinuclear factor
ASD - Dorogov's antiseptic stimulatorAT - 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 drugsGLP - glycoprotein
DM - dermatomyositis
DNA - deoxyribonucleic acid GIT - gastrointestinal tract IB - immunoblotting
IR - immune complexIL - interleukin
PI - protease inhibitor PPI - drug intake index
ELISA - enzyme immunoassayIFN - 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 beenexpanded.

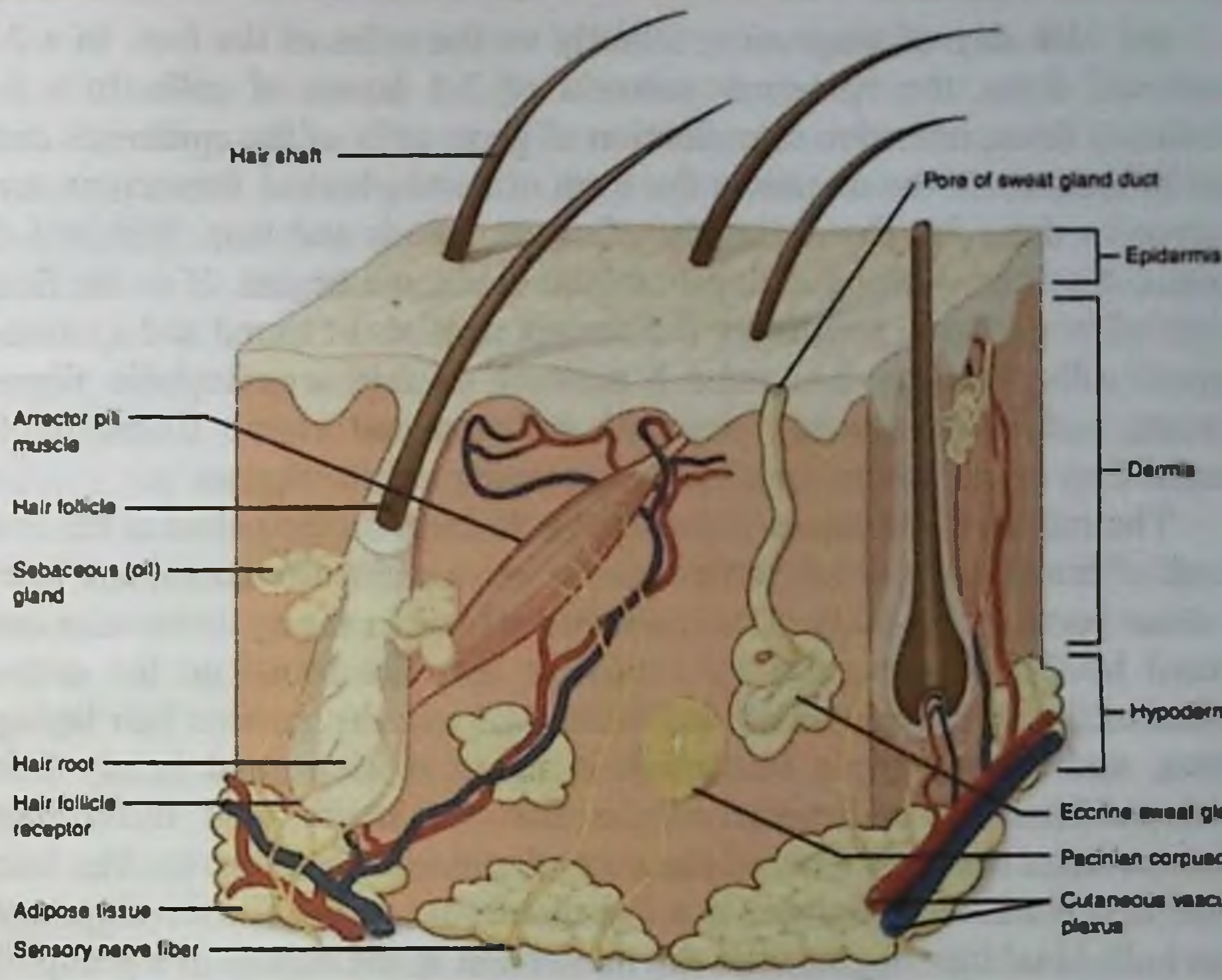
CHAPTER 1. GENERAL CLINICAL PICTURE AND PATHOMORPHOLOGY OF DERMATOSES. ANATOMY AND HISTOLOGY OF THE SKIN AND MUCOUS MEMBRANES. METHODS OF EXAMINATION OF PATIENTS WITH SKIN DISEASES AND STDS. ENDOGENOUS AND EXOGENOUS FACTORS IN THE DEVELOPMENT OF DERMATOSES. PRIMARY AND SECONDARY MORPHOLOGICAL LESIONS. PRINCIPLES OF DIAGNOSTICS, GENERAL AND LOCAL TREATMENT OF SKIN DISEASES. ANATOMY AND HISTOLOGY OF THE SKIN AND SKIN DERIVATIVES IN CHILDREN.

STRUCTURE OF THE SKIN: The skin is a barrier organ that separates the internal environment of the body from the outside world. Having a complex structure, the skin is closely connected with all the systems and internal organs of the human body.

Even a minor pathology of the internal organs or the nervous system, hematopoiesis and the circulatory system is accompanied by a variety of skin rashes, changes in its relief, elasticity, color. Skin color depends on the content of the following pigments: melanin (black or dark brown pigment produced by melanocytes), melanoid (dark pigment - a product of keratin in the stratum corneum of the epidermis), carotene (yellow pigment - provitamin A), oxyhemoglobin (pink pigment), reduced hemoglobin (dark blue pigment, its content increases with acrocyanosis).

The skin plays an essential role in maintaining both homeostasis (maintaining a constant internal environment) and homeokinesis, the latter reflecting the dynamic regulation of the internal environment. It has long been noted that the skin of a person's face, in particular, reflects his gender, age, emotionality and, to some extent, intelligence, i.e. each person has his own facial expressions and so on, the so-called facial expressions.

The skin consists of 3 layers: epidermis, dermis and subcutaneous tissue (hypoderm). Hair, nails, sebaceous and sweat glands are appendages of the skin. Knowing a person's weight, breast size and height, you can determine the area of \u200b\u200bhis skin. There are several formulas for calculating skin area:



Pic-1 Structure Of The Skin

1. Alain's formula: skin area in cm2 = weight in kg × 0.425 × height in cm × 0.725 × 71.81. 2. Mi formula: S = 12.33 P 2 for adults and S = 11.93 P 2 for children, where P is the breast volume, it can be replaced by the patient's weight.3. Turovsky's formula: 1 kg of an adult's weight falls on 221 cm2 of the skin surface, and in adolescents - on 378 cm2. Sometimes the dose of a drug is calculated per unit area of the body (for example, in oncohematology). The skin area of an adult is 1.5-2 m2, the skin mass (epidermis + dermis) is about 5% of the total body weight, and with subcutaneous fat - 13-18%. The total thickness of the epidermis and dermis ranges from 0.5 to 5 mm.

skin embryogenesis. Human skin develops from two embryonic anlages. From the ectoderm, the epidermis and skin appendages develop: skin glands, hair and nails. The mesoderm is the source of the formation of the dermis and hypodermis with vessels located in them.

The epidermal layer of cells is formed by the 14th day of embryogenesis, it is separated from the underlying mesenchyme by a thin basement membrane. The bilayered epidermis first appears on

the 35th day of pregnancy, initially on the soles of the feet. In a 2-month-old fetus, the epidermis consists of 2-5 layers of cells. In a 3-month-old fetus, intensive reproduction of germ cells of the epidermis and their immersion in the dermis in the form of hemispherical formations are observed - these are the rudiments of sweat glands and hair. Within 4-6 months, the formation of all layers of the epidermis begins. If in the first month of intrauterine pregnancy the dermis consists of round and spindle-shaped cells, then by 2 months a network of thin argentophilic fibers appears, and by 3 months - separate collagen and elastic fibers, by 6 months they form intertwining bundles. considerable thickness.

The rudiments of sweat glands begin to form on the palms at the 3rd month of intrauterine development, on the soles at the 4th month, and later on other parts of the body. The rudiments of hair in the eyebrow area are formed by 2.5 months, and by 3 months they are found on the entire surface of the face, scalp, chest and back. At 5 months, another hair laying occurs, and later a triple bookmark is possible. Long and bristly hair develops from the rudiments of the first bookmark, and fluffy hair develops from the rudiments of the second and third bookmarks. The hair of the first bookmark changes in a 7-8-month-old fetus. The development of an individual hair begins with the immersion in the dermis of a group of rapidly multiplying epidermal cells. Gradually, the hair germ takes the form of a long thread with a rounded and thickened end with a recess into which the connective tissue papilla grows with vessels and nerve endings.

Epithelial cells located peripherally from the center of the papilla form the inner epithelial membrane of the hair, which is the channel along which the growing hair moves. Epithelial protrusions appear on the side of the epithelial hair bud. Then the muscle that raises the hair joins the lower one. The sebaceous gland develops from the middle protrusion, and the apocrine sweat gland develops from the upper protrusion.

At the 3rd month of development, the rudiments of nails appear in the fetus. First, there is a thickened layer of epithelium called the primary nail bed. It passes to the fingertip and is surrounded on the proximal and lateral sides by epithelial folds. These folds then form the nail folds. A real nail is formed from the epithelium located under the nail fold and in the proximal part of the nail. in the area of the matrix. By the end of the embryonic period, it reaches the end of the finger. The division of the connective tissue part of the skin into the dermis and hypodermis occurs by the end of the 3rd month of embryonic development. At 5 months, there is an ingrowth of layers of fibrous tissue into the subcutaneous fat layer

and its division into lobes. The latter form fat cells and an abundance of capillaries. By 7 months of fetal development, all layers of the epidermis are fully formed in the fetus with keratinization of cells on the palms and soles, a clearly formed network of collagen and elastic fibers of the dermis with a significant number of fibroblasts and macrophages, as well as undifferentiated cells. In the last trimester of pregnancy, all layers of the skin, the basement membrane structurally form a single morphological organ, consisting of the epidermis, dermis and subcutaneous fat. The epidermis is a stratified squamous keratinized epithelium. The epidermis covers the entire outer surface of the body, passing into the mucous membrane in the area of natural openings (eyes, mouth, genitals).

In an adult, with a total epidermis weight of about 0.5 kg, its thickness has clear topographic differences: the thickest on the skin of the palms and soles, and the thinnest in the periorbicular region. On the surface of the epidermis, grooves and folds are visible, forming a characteristic and permanent pattern for a given person, called dermatoglyphics, which persists throughout life. The epidermis determines skin color, which depends on race, sex, age and is determined by the content of melanin, melanoids, carotene, hemoglobin and its metabolic products. The epidermis contains the following cells: keratinocytes, melanocytes, Langerhans, Greenstein, Merkel and Thorn cells. In the epidermis, processes such as cell proliferation (mitosis), cell emergence to the surface, keratinization and exfoliation are constantly taking place.

From the moment of mitosis of the basal keratinocyte to the detachment of the stratum corneum, an average of 21-30 days, sometimes 65 days, passes (in women, the rate of renewal of the uterine endothelium is usually identical to the rate of renewal of the endothelium of the uterus). epidermis, for example, on the back of the hand, i.e. equal to the duration of the intermenstrual period). Since all keratinocytes in the process of moving up after mitosis undergo the same type of changes, microscopic assessment of the structure of the epidermis is usually divided into 5 layers, reflecting the different stages of differentiation of these cells: basal, spiny, granular, shiny, and excited. The epidermis is separated from the dermis by a three-layer basement membrane.

Recently, the epidermis has been increasingly divided into 2 zones: the first is represented by living cells and is called the Malpighian layer, which includes the basal, prickly, granular layers, and in some places the hyaline or shiny layer, and the second - the cells of the stratum corneum are devoid of a nucleus. Basal or germinal layer (stratum basale) is

represented by a single row of cylindrical cells with basophilic (due to the high content of RNA) cytoplasm and a large nucleus with 1-2 nucleoli. These cells are characterized by high mitotic activity, are connected to each other by desmosomes, and to the basement membrane by hemidesmosomes. Hemidesmosomes contain a specific protein (pemphigoid antigen) that binds to the anchor filaments of the lamina lucida of the basement membrane.

The mitotic activity of basal keratinocytes is stimulated by epidermal growth factor, individual interleukins, and the division of these cells is slowed down by chalons, tumor necrosis factor- alpha, interferons, and locally applied glucocorticoids.

In the basal layer, in addition to keratinocytes, there are melanocytes, cells of the Langerhans process, Greenstein cells, tactile Merkel cells. In addition to stem epidermocytes, there are also dividing precursors in the basal layer, which, after division, fall into the overlying spinous layer.

Spiny layer (stratum spinosum) consists of 5-8 rows of cubic epidermocytes with cytoplasmic outgrowths, which, in contact with similar outgrowths of neighboring cells, form a network of intercellular channels through which diffusion nutrition of the epidermis is carried out (there are no vessels in the epidermis). epidermis!). The cells of the spinous layer are weakly basophilic. In the upper rows of the spiny layer, the cells are more flattened, the cytoplasm contains numerous

mitochondria, polyribosomes, the Golgi complex, the endoplasmic reticulum, microtubules, and a developed network of keratin filaments (tonofibrils). Along with keratinocytes, Langerhans cells are also found in this layer.

Granular layer (stratum granulosum) consists of 1-4 rows of flattened rhomboid cells, in the cytoplasm of which there is an abundance of basophilic keratohyalin granules between bundles of keratin filaments. These cells contain lamellar bodies (keratinosomes, or Odland bodies), which are accumulations of lipid membrane-like structures (glycolipids, glycoproteins, free sterols) and a set of hydrolytic enzymes. Keratohyalin granules contain filaggrin protein (from English - a protein that aggregates filaments), which promotes the aggregation of tonofilaments that make up the cytoskeleton of epidermocytes into a single complex, contributing to the transformation of this cell into a horny scale. In the cells of the granular layer, keratolinin is also biosynthesized, a protein that accumulates above the cytolemma. cells, which leads to its thickening. All of the above proves that the cells of the granular layer have sufficient

functional activity and cannot be considered a degradation product of keratinocytes.

lustrous layer (stratum lucidum) is not expressed on the whole skin, but is well represented on the skin of the palms and soles. It consists of l-4 layers of flat cells with poorly distinguishable borders. The cells contain the protein eleidin, which is considered a marker of this layer. The stratum corneum consists of 15-20 or more layers of horny tile-like scales connected by reduced desmosomes. Under the microscope, the scales are polygons with a diameter of 30 microns and a thickness of 0.5-0.8 microns. This shape allows them to fit more tightly to each other. In different parts of the body, the scales of the stratum corneum may have a columnar or columnless organization. Non-columnar organization is characteristic of skin with a thick stratum corneum and high mitotic activity of the epidermis.

Horny scales are composed mainly of keratin. On the surface of the stratum corneum there is a water-lipid film with a pH of about 5.5. Melanocytes are dendritic light cells located between the epidermocytes of the basal layer.

Melanocytes are derived from the melanoblasts of the embryonic neural crest. The main function of melanocytes is the production of the main skin pigment - melanin - from the amino acid tyrosine, which, under the action of copper-bound tyrosinase and ultraviolet rays, is converted into dihydroxyphenylalin (DOPA), and then with the help of DOPA oxidase through the stages of DOPA-quinone, DOPA-chromium to melanin. Melanocytes have dendritic processes and special melanosome organelles that accumulate and spatially orient the synthesized melanin. Melanosomes have a high-contrast fine-grained structure and are surrounded by an elementary membrane. They are assembled into melanosome complexes that form a screen over the nucleus of basal keratinocytes, protecting the genetic apparatus of dividing cells from the mutagenic effects of UVB. The process of melanogenesis in the skin depends on the state of the endocrine organs, the availability of vitamins and the activity of enzyme systems. It is known that the lackof B vitamins inhibits melanogenesis, and the lack of vitamins A, C and nicotinic acid stimulates

melanogenesis. Melanocyte-stimulating, adrenocorticotropic and sex hormones stimulate the synthesis of melanin, with an excess of which develops hyperpigmentation. The synthesis of melanin is under the control of genetic control, which determines the distribution of melanosomes in

melanocytes and keratinocytes, and in blacks, melanosomes are distributed not only in keratinocytes of the basal, but also in the spiny and even granular layer. One of the factors stimulating melanogenesis is ultraviolet radiation. Sunburn is a protective reaction of the skin against the damaging effects of ultraviolet rays on the nuclei of the cells of the epidermis and dermis. Normally, there are 34 keratinocytes per melanocyte, into which it secretes melanin granules (this is the so-called melanin epidermal unit). The transfer of melanin, most likely, is carried out by phagocytosis of keratinocytes of individual granules or groups of them, together with a part of the melanocytic process. At least three groups of diseases are associated with the pathology of melanocytes: melanoma (malignant degeneration of melanocytes), albinism (lack of tyrosinase or its blocking in melanocytes leads to hypo- or depigmentation of the skin) and vitiligo (appearance of areas without pigment and melanocytes). Processed cells of Langerhans, or intraepidermal macrophages, are found in the basal and more often spiny layer of the epidermis. Their number ranges from 300 to 100.0/mm2 and decreases under the influence of UV radiation, laser irradiation and general deep cooling. They differ from keratinocytes by the presence of processes, the absence of tonofibrils and desmosomes, and from melanocytes by the absence of melanosomes. They are characterized by the presence of Bierbeck granules (tennis racket-shaped granules and rod-like structures). Langerhans cells have been shown to be an important component of the skin's immune system and are involved in the immune response. They play a key role in the hypersensitivity reaction in the skin, are able to migrate and deliver antigens to T-lymphocytes not only in the epidermis, but also in the dermis, lymphatic vessels and in the paracortical zone of the lymph nodes. Upon repeated antigen exposure, Langerhans cells are often targeted by cytotoxic T-lymphocytes, leading to destruction of Langerhans cells with the release of prostaglandins, mesosomal enzymes, and other inflammatory mediators. Langerhans cells play an important role in protecting against skin tumors. A decrease in the number of Langerhans cells in the early stages of carcinogenesis may allow neoplastically transformed cells to activate T-suppressors and avoid immune damage. destruction. In malignant tumors of the skin, the number of Langerhans cells decreases against the background of a general decrease in antitumor protection. Langerhans cells bind their own epidermal antigens in autoimmune diseases (pemphigus, lupus erythematosus) and become targets for autoreactive T- lymphocytes, while the number of Langerhans cells decreases. It is known that Langerhans cells, together

with keratinocytes and lymphocytes, are involved in the formation of a specific granuloma. The possible role of Langerhans cells in the pathogenesis of vitiligo is under discussion. in the foci of this pathology, against the background of the complete absence of melanocytes, an increased number of these cells is noted. The population of Greenstein cells is 1- 3% of all epidermal cells, and their number ranges from 5 to 600 cells/mm2. There are two points for assessing their possible function: - these are natural killers that carry out the lysis of dying keratinocytes; – they are antigen-presenting cells for T-suppressors penetrating the epidermis.

According to O. D. Myadelts and V. P. Adaskevich, in the epidermis there is a peculiar system of regulation of immune reactions, acting according to the "plus-minus" type: one advantage of this system is Langerhans cells and T-helpers, the second is Greenstein cells and T - suppressors. Merkel cells are found in the basal layer of the epidermis, hair follicles, and oral mucosa and are structurally associated with nerve fibers. They develop from the neuroectoderm, their number is 150 cells per 1 cm 2, and much more at the fingertips. In an adult, Merkel cells accumulate 2-3, and in the elderly - 3-4 cells, although the total number of cells in them decreases, as in chronic inflammation of the skin. Merkel cells are sensitive nerve endings of the musculoskeletal system. Merkel cells contain methencephalin, vasointestinal polypeptide, neuropeptides, nerve growth factor, skin endorphin, bombesin, etc.

All this gives the right to consider Merkel cells as neuroendocrine cells of the skin. Merkel cell carcinoma has been described. Toker cells are epithelial cells with a light cytoplasm, localized only in the area of the nipple of a normal mammary gland. Their nature and functions are not yet clear, but it is with their rebirth that the origin of Paget's disease is associated. The basement membrane separates the epidermis from the dermis. It has a thickness of 40-50 nm. An electron microscopic examination of the basement membrane revealed its heterogeneity, it consists of 4 complex components: the plasmolemma of the basal cells, light and dark plates, and the subepidermal plexus. The basement membrane is connected to the epidermis by hemidesmosomes, anchoring filaments, and adhesion molecules, and to the dermis by anchored fibers and bundles of dermal microfilaments. The functions of the basement membrane are diverse, of which the most important are: supporting (binding cells of various origins), trophic (regulating the penetration of substances into keratinocytes), barrier (limiting the formation of cells of

various origins). penetration of antigens, antibodies and CEC), a regulatory role in development and morphogenesis.

The dermis consists of two layers: papillary (stratum papillaire) and reticular (stratum reticulaire).

The papillary layer is located under the epidermis and is represented by loose fibrous connective tissue. The presence of epidermal and connective tissue papillae increases the metabolic zone between the epidermis and the papillary layer. The papillary layer contains the main substance, an abundance of thin collagen and elastic fibers. The ground substance contains glycoproteins, proteoglycans and glycosaminoglycans.

The reticular layer The dermis is formed by dense, unformed fibrous connective tissue, which contains fibrous structures: collagen, elastic, and reticular (reticulin, argyrophilic) fibers arranged in bundles. In the reticular layer, which forms the stroma of the dermis, there are fewer cellular elements than in the papillary layer. In both layers of the dermis, in addition to collagen, elastic and argyrophilic fibers, there are many cells: fibroblasts, macrophages, histiocytes, mast cells, leukocytes.

Arterial and venous vessels, as well as lymphatic vessels, pass through the dermis; during vascularization, the skin has its own topographical features. So, in the skin of the abdomen there are 5 vascular networks: deep fascial, superficial fascial, deep cutaneous, suckling, papillary. The deep dermal network supplies blood to fat lobules, sweat glands, and hair follicles. The sucking network nourishes the sebaceous glands and hair roots. From the terminal arterioles in the direction of the skin papillae, 3-4 capillaries depart, having the shape of a loop and a head hairpin. The capillaries empty into the superficial venous network just below the papillae. This is followed by the second subcapillary venular network, then the deep dermal venous network and subcutaneous venous plexuses. There is a superficial network of lymphatic vessels under the subcutaneous venous plexuses and a deep network in the hypodermis. The lymphatic vessels of the skin are a kind of drainage system involved in the recycling of blood plasma proteins from the bloodstream to the tissues and vice versa, which removes metabolites and catabolites.

The skin is innervated by the central and autonomic nervous systems. The nerves of the skin also form several plexuses: deep in the reticular layer and superficial in the papillary layer. Thick myelin fibers innervate mechanoreceptors, while thin myelin fibers innervate pain and itching receptors both in the dermis and epidermis. Nerve endings may be free or encapsulated. In the epidermis, neuroreceptors are located in the form of

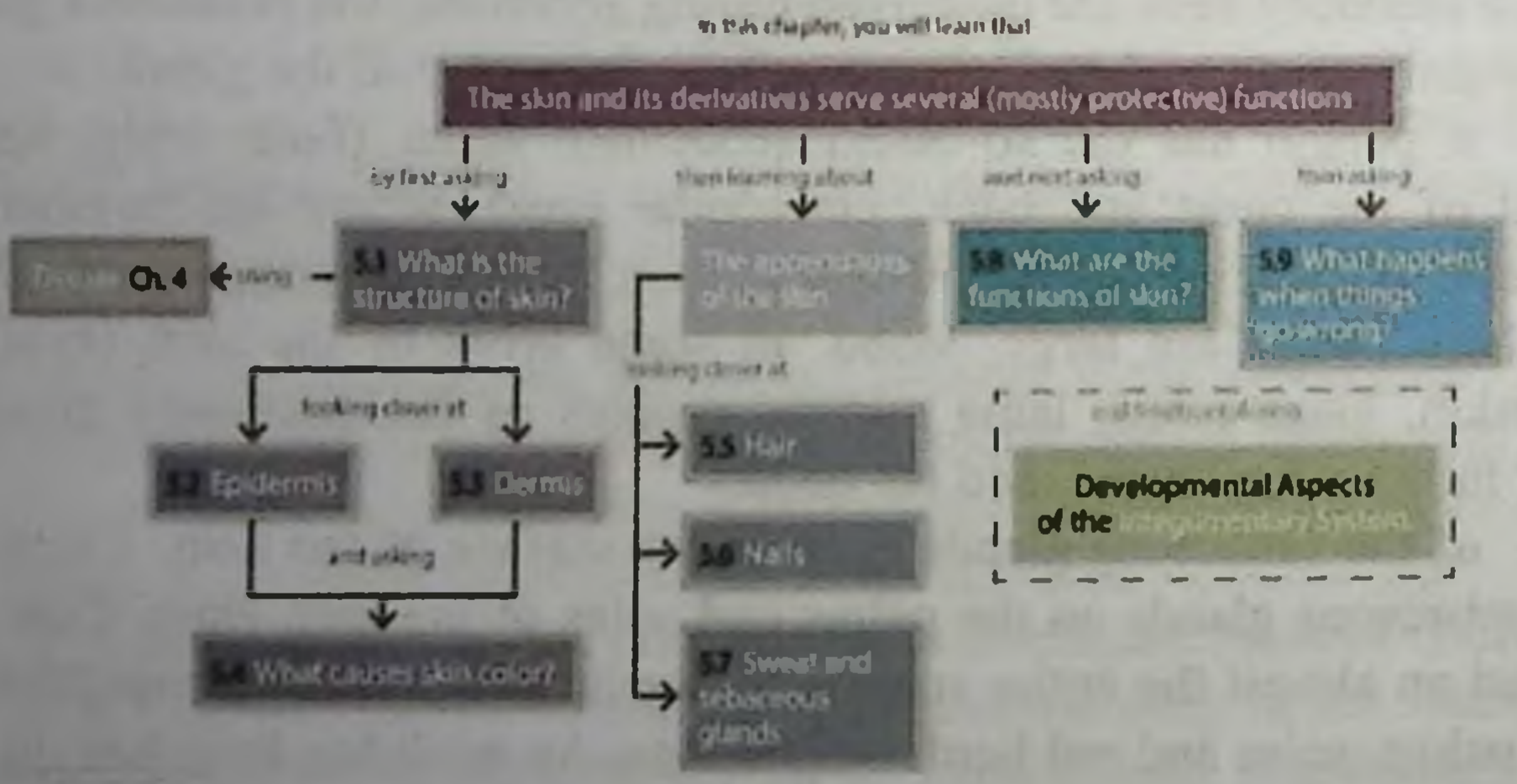
Merkel cells; in the dermis there are tactile Meissner bodies and temperature receptors - Krause flasks (cold) and Ruffini bodies (heat), in the subcutaneous tissue - Vater-Pacini bodies that perceive pressure and vibration. Human skin appendages include sweat and sebaceous glands, hair and nails. The sweat glands are completely differentiated at the 8th month of intrauterine development. According to the type of secretion, sweat glands are divided into eccrine (merocrine), secreting a secret by exocytosis without destroying the secretory element of the gland, and apocrine, the secretion of which is carried out with detachment of the cell tip, i.e., with partial destruction of the glandular tissue. Eccrine glands are found on 99% of the body surface, and apocrine glands are found in the armpits, perineum, and areolas of the nipples. The number of glands per 1 cm 2 of the surface ranges from 64 to 700 in different parts of the body. Apocrine glands begin to function during puberty. Sweating is regulated by the hypothalamus. The sebaceous glands in most cases have a complex alveolar structure, the type of secretion is holocrine. The sebaceous glands surround the hair follicle (in the amount of 4-8) and open in its upper third. They produce sebum - a kind of lubricant for hair and skin. The released sebum mixes on the surface of the skin with the secretion of the sweat glands, forming a kind of Marchionini hydro-fatty acid mantle, which has a slightly acidic reaction (pH 4.5-5.5) and has protective properties against microbes and fungi.. Since secretory cells are destroyed during secretion, the sebaceous gland constantly grows and pushes new cells to the center of the gland.

A person has the so-called seborrheic zones (face, scalp, upper third of the chest and back, perigenital area), where the number of sebaceous glands reaches 750-900 per cm 2, and the glands themselves in these places are large, lobed. In some areas of the skin (eyelids, foreskin, glans penis, labia minora, nipples of the mammary glands), hair follicles are absent, and the ducts

of the sebaceous glands open to the surface of the skin. There are no sebaceous glands on the palms and soles of the feet. Hair. They are found on almost the entire surface of human skin, with the exception of the palms, soles and red border of the lips. An adult has long hair on the head, beard, mustache, bristly hair on the eyebrows and eyelashes, pubic hair and armpits. In men, up to 90% of the hair on the chest, torso, shoulders, arms and legs, and in women 35% of the hair are terminal;

Samdtu
axborot resurs markazi

respectively, 10 and 65% remain downy. In hair, there is a distinction between shaft and root. Under a microscope, the brain, cortex and cuticle are found in the hair. Melanosomes, containing pigment produced by melanocytes of the hair follicle, they are located in the cortex and medulla of the hair, determining their color. Hair color depends on the content of two pigments: yellow-red pheomelanin and black-brown eumelanin. The hair root is surrounded by an inner and outer epithelium, as well as a skin sheath. The hair root is enclosed in a hair follicle. A person has about 5 million hair follicles, of which about 1 million are located on the scalp. A person has a constant change of hairstyle. The hair cycle consists of an active growth stage (anagen) that lasts 2-6 years, a regression stage (catagen) of 2-4 weeks, and an active growth stage (anagen). rest (telogen) - several months. Each hair follicle sheds its hair an average of 25- 30 times and gives rise to a new hair (with a lifespan of 75-80 years). Nails are horny plates lying on the nail bed. The nail bed is limited by the nail fold connected to the lamina by the skin (eponychium). The nail plate is divided into root, body and edge. The root of the nail is called the nail matrix. The growth of the body of the nail is carried out due to the constant process of reproduction of matrix cells. A complete change of the nail takes an average of 5-6 months.



Pic-2 Funvtion Of The Skin

SKIN FUNCTIONS :

1. The protective function includes mechanical protection of the skin from external influences, as well as protection from ultraviolet radiation, chemical irritants, pathogenic bacteria, fungi, viruses. Mechanical protection against bruises, pressure, tears and sprains is due to the properties of the epidermis (its strength, elasticity, ability to recover), elasticity and mechanical resistance of the fibrous structures of the dermis to pressure from a blunt object (tensile elasticity of the skin is associated with the straightening of collagen fibers along the axis of tension, and return to its original state is provided by elastic fibers), buffer properties of subcutaneous fat. Protection of the skin from ultraviolet radiation is provided primarily by the stratum corneum, which completely traps infrared rays and partially - ultraviolet. Depending on the wavelength, three fractions of ultraviolet rays are distinguished: UV-A (320-400 nm), UV-B (290-320 nm) and UV- C (200-290 nm). UV-A and UV-B fractions have the greatest biological effect on the body. UV- A can penetrate deep into the dermis, can cause increased sensitivity to the sun and accelerate skin aging. UV-B affects the epidermis and can cause burns, premature skin aging, precancerous skin conditions, and skin cancer. Protecting the skin from UV radiation also It is provided by the presence of a protein barrier in the stratum corneum and a melanin barrier in the epidermis. Prolonged exposure to the sun can lead to thickening of the epidermis, the development of solar elastosis and keratosis, and neoplasms in the skin. The keratin of the intact stratum corneum provides protection against chemical irritants. Intact skin protects the body from the harmful effects of the external environment, from the introduction of microorganisms. The bactericidal properties of the upper layers of the skin are due to the chemical composition of sebum and sweat, the acidic reaction of keratin, the presence of a water-fat layer on the surface of the stratum corneum. mantles with acidic pH (pH = 4.5-5.5). The composition of the water-lipid mantle of the skin includes low molecular weight fatty acids with a bacteriostatic effect. Physiological peeling also helps prevent bacterial growth. For 1 cm 2 of a healthy person's skin, there are from 40,600 to several million different microorganisms involved in the antimicrobial protection of the skin and mucous membranes from pathogenic microbes. Any damage to the skin reduces the effectiveness of antimicrobial protection. The skin has a

significant electrical resistance, photo- and radioprotective action. The skin is able to withstand significant temperature changes, minor injuries.

2. The thermoregulatory function is based on the processes of heat generation, heat conduction and heat transfer. This function of the skin is ensured by the low thermal conductivity of the stratum corneum, subcutaneous fat, the state of blood and lymph circulation, and the excretory activity of the sweat and sebaceous glands. The skin has a powerful vascular network, and an increase in ambient temperature leads to the expansion of skin vessels, increased blood flow and heat transfer. When the skin becomes colder, the blood vessels in the skin constrict and heat transfer decreases sharply. An important role in thermoregulation is played by arteriovenous shunts, which are abundant in the skin of acral regions (nose, lips, ears, hands, and feet). Their activity is regulated by noradrenergic sympathetic nerves. With a decrease in sympathetic tone,

the skin vessels expand, the skin becomes warmer than the surrounding air and heat transfer increases due to cooling. Heat transfer by radiation and radiation is called "dry heat transfer" (Ivanov O.L., 2002), which accounts for up to 25% of heat transfer. Evaporation of sweat is a very efficient way to release heat. The regulation of sweating is carried out by the central nervous system and cholinergic sympathetic fibers (therefore, pilocarpine and acetylcholine increase, and atropine inhibits sweating). Examples of diseases to which they are susceptible violation of heat transfer of the skin, are psoriasis, toxidermia, fungal mycoses, Caesar's syndrome, etc.

3. The immune function is provided by a complex immunoreactive system of the epidermis, dermis, subcutaneous tissue and basement membrane, which prevents the penetration and spread of foreign antigens in the body. The main elements of the immune system of the skin are keratinocytes, Langerhans and Greenstein cells, epidermal T-lymphocytes. T-lymphocytes of the skin are located mainly in the dermis, in the epidermis they are less than 10%. Recognition of exogenous and endogenous antigens is carried out by T-lymphocytes after their presentation by antigen-presenting cells (Langerhans or Greenstein cells), O.L. Ivanov (2002) points out that for recognition by T-helper lymphocytes (CD 4+), the antigen must be presented in combination with class II GCS (HLA - DR, DP, DQ), and most T-

suppressor lymphocytes (CD 8+) recognize antigen in association with HLA molecules Class I (HLA - A, B, C). The antigen- specific T-cell response consists in the formation of blast forms of T-lymphocytes, which, after receiving the antigen-MHC complex from antigen-presenting cells in the lymph nodes, return to the skin areas containing the antigen.

4. Secretory function. The skin synthesizes keratin, vitamin D from ergosterol and vitamin A from carotene. The sebaceous glands secrete sebum, which is the lubricant for the upper layers of the skin. The composition of sebum includes free higher and lower, as well as associated fatty acids in the form of cholesterol esters and other sterols, free cholesterol, a small amount of hydrocarbons, traces of nitrogenous and phosphorus compounds. Partial synthesis of sex hormones also occurs in the skin.

5. Excretory function. Mineral oil products, carbohydrate, protein and fat metabolism are excreted from the body with sweat and sebum. With sweat, a significant amount of water and a number of harmful metabolic products (urea, ammonia) are lost, and vitamins, hormones, trace elements, and drugs are also released.

6. Suction function. The skin is able to absorb substances soluble in water and fats. The absorption of chemicals and toxic decomposition products increases with parakeratosis and inflammatory reactions in large areas of the skin, especially with the destruction of the stratum corneum. The molecular weight of a substance is critical for absorption. Hot compresses and steam baths increase the skin's ability to absorb substances dissolved in water or finely dispersed in fats.

7. Respiratory function. The skin carries out about 2% of the body's gas exchange, helping the

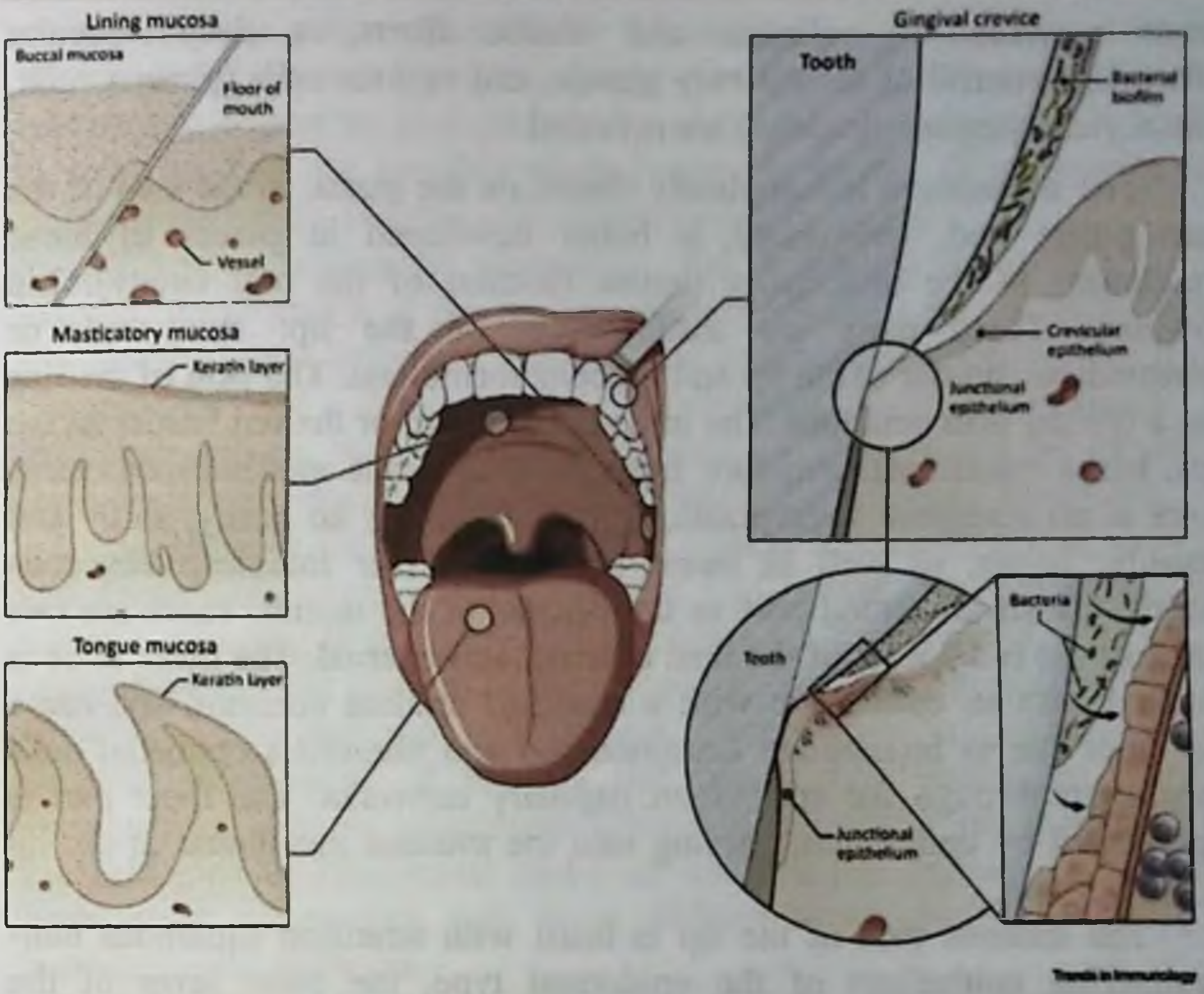
lungs. Skin respiration increases with increasing humidity. ambient and body temperature, during physical exertion, during acute inflammatory processes in the skin.

8. Receptor function. The skin carries out all types of reception: temperature (heat, cold), pain, tactile (touch, pressure, vibration). The receptor function of the skin is provided by Merkel cells, efferent and afferent nerve fibers, encapsulated and free nerve endings. There are active points on the skin that project the functional state of the internal organs, endocrine, immune and nervous systems. The functional

connection of the skin with the above organs and systems is carried out with the help of myelinated (A-fibers) and non-myelinated (C-fibers) sensory nerves. Among the mechanoreceptors that perceive touch are hair follicle receptors (on skin covered with hair), and on skin devoid of hair, fast-responsive tactile Meissner bodies and slow-responsive Merkel receptors; in the dermis and subcutaneous tissue - Ruffini bodies. Heat and cold are perceived by thermoreceptors, more often encapsulated (Krause cold flasks are activated at a temperature of 1- 20°C below normal skin temperature (34°C); Ruffini heat bodies are activated at a temperature of 32 to 35°C, and nociceptors are activated at a temperature above 45°C). The position of the body in space and the feeling of pressure are perceived by the lamellar bodies of Vater-Pacini. Itching - a modified sensation of pain - is carried out along unmyelinated C-fibers extending from the upper part of the dermis, both skin and mucous membranes. Itching of the skin is a cortical process and passes through three sections: peripheral, interspersed in the skin, central - in the upper sections of the central nervous system, and conductive, connecting both of these sections.

9. Exchange function. The skin is involved in all types of metabolism. Thanks to the hydrophilic structures of the dermis and subcutaneous tissue, the skin deposits and catabolizes proteins and carbohydrates, amino acids and cholesterol, products of lipid peroxidation. The skin has a complete set of necessary enzymes, including hydrolases, transferases, lipases, oxidoreductases, isomerases, synthetases, as well as a sufficient amount of B, C, E and A vitamins to ensure the normal course of keratinization, carbohydrate and protein metabolism and antimicrobial protection.

STRUCTURE OF THE MUCOSA OF THE MOUTH : In the area of natural openings (mouth, nasal passages, external opening of the urethra, vestibule of the vagina, anus), the skin passes into the mucous membranes. This transition zone has no subcutaneous fat, according to skin, sebaceous glands and hair (red border of the lips, inner layer of the foreskin, glans penis, inner labia, clitoris).



Pic-3 Structure Of The Mucosa Of The Mouth

The mucous membrane consists of three layers: epithelial, connective tissue layer of the mucous membrane and submucosa. The epithelium of the oral mucosa is represented by the basal and prickly layers. Cylindrical cells of the basal layer are located on the basal membrane, the mitotic activity of these cells is 3-4 times higher than in keratinocytes of the basal layer of the skin. Langerhans cells, melanocytes and leukocytes are also found in the basal layer. The spiny layer consists of 3-5 rows, and the cells of the upper rows are flattened, accumulate alpha-keratin and become keratinized with the preservation of nuclei (physiological parakeratosis). In the area ofthe hard palate and tongue, keratinization of epithelial cells with prolapse of nuclei can occur. When foci with keratinizing cells appear in other areas of the oral cavity, it is necessary to exclude some diseases: lichen planus, lupus erythematosus, leukoplakia. The basement membrane of the oral mucosa consists of elastic, collagen and pre-collagen fibers that form the basis of the oral mucosa. In the connective tissue part of the mucous membrane there is an abundance of blood and lymphatic vessels, nerve fibers and endings, and small salivary glands. In the submucosa, a

dense network of collagen and elastic fibers, a deep vascular network,glomeruli of the salivary glands, and various cells (plasma cells, histiocytes, lymphocytes, etc.) are revealed.

The submucosa is completely absent on the gums, in the area of the hard palate and, conversely, is better developed in places of loose attachment to the underlying tissues (bottom of the oral cavity). Lip structure. Three parts are distinguished in the lip: skin, red, or intermediate, border of the lip and mucous membrane. The skin of the lips has a typical skin structure. The intermediate part, or the red border of the lips, has a transitional structure from the skin to the mucous membrane, there is no complete keratinization on it, there are no horny, shiny and granular layers, as well as sweat glands and hair follicles, sebaceous glands. glands are found only in the corners of the mouth. There are two zones of the red border of the lips: external and internal. The outer zone is covered with an epithelium with a modified stratum corneum and has a red color due to incomplete keratinization and numerous epithelial cells translucent through the epithelium. capillary networks. The inner part is represented by epithelium, passing into the mucous membrane of the lip (Klein's zone).

The mucous part of the lip is lined with stratified squamous non-keratinizing epithelium of the epidermal type, the outer layer of the epithelium contains nuclei, elongated dermal papillae are characteristic. The innervation of the lips, oral mucosa, gums and tongue is provided by the trigeminal, facial, lingual and hypoglossal nerves; the first three nerves provide sensory innervation, and the motor nerve provides the hypoglossal. Throughout the mucous membranesof the mouth, tongue and red border of the lips, the circulatory and lymphatic systems are represented by capillaries, venules and the lymphatic network. The large and small salivary glands are located in the oral cavity. The major salivary glands (parotid, submandibular, and sublingual) are located outside the oral cavity and have long excretory ducts. Minor salivary glands are divided into labial, buccal, palatine and lingual. The salivary glands are innervated by both sympathetic and parasympathetic nerve fibers, and the bulbar salivary centers are controlled by the central nervous system.

PRINCIPLES OF DIAGNOSTICS OF SKIN DISEASES METHODS OF EXAMINATION OF A SKIN PATIENT AND DESCRIPTION OF SKIN RASHES

The clinical picture of any dermatosis is based on subjective and objective symptoms. Subjective symptoms: most often itching, burning,

soreness, sharp pain or paresthesia - "crawling", hypoesthesia, complete anesthesia. When making a diagnosis, it is necessary to study the patient's complaints, collect an in-depth history of the disease and the patient's life in order to find out possible etiopathogenetic factors of exogenous and endogenous nature, the role of hereditary predisposition.

When collecting an anamnesis, the following information is of interest: the duration of the present disease; possible factors that caused the disease; is it a primary manifestation or recurrence; Of interest is the history of the disease, the relationship with hereditary or infectiousgenesis, the presence of seasonality, finding out the causes of exacerbations of the disease, the effectiveness of previous treatment.

Attention is drawn to gender, age, marital and social status, working and living conditions, the presence of bad habits, past infectious and somatic diseases. It is mandatory to conduct a study of the state of the internal organs and systems of the patient at the time of treatment and the condition of the skin and oral mucosa. Skin disease is manifested by a different combination of primary and secondary elements on the skin (exanthema), mucous membranes (enanthema), which are formed under the influence of exogenous and / or endogenous factors. In fact, the essence of diagnostics lies in a differentiated clinical analysis of morphological elements.

TECHNIQUE AND METHODS OF DERMATOLOGICAL EXAMINATION

Inspection of the skin is carried out in diffused daylight and a temperature of 20-25°C. It is necessary to examine the entire skin, i.e. the patient must completely undress. At the same time, skin color, dermography, humidity, the nature of the skin rash (inflammatory or non-inflammatory), localization, size, color, shape, outline of the elements of the rash, etc. areevaluated.

Inspection of the oral mucosa is preferably carried out using a bright directional electric light. Examination of skin lesions with a magnifying glass allows you to clarify the nature of the scales, the presence of scabies rollers, rollers, depressions, depressions, for example, an depression in the center of the elements with lichen planus, erythema multiforme. Palpation is used to assess the state of turgor (decreased with skin aging) and skin elasticity (excessive extensibility of the skin in the Chernogubov-Ehlers-Danlos syndrome), to determine the temperature difference in the lesions of the skin compared to areas of unchanged skin. It is by feeling (palpation) that the depth of the elements of the rash (for example,

papules, tubercles, nodes), the size of the elements of the rash, their consistency, soreness, and connection with surrounding tissues are determined. The size of the rash is best described in the metric system (for example, a knot with a diameter of 2 cm). The location (depth of occurrence) of individual elements of the rash is best described in relation to the skin layers: epidermal, epidermal-dermal, dermal, subdermal location. Scraping the surface of loose elements is carried out with a scalpel, glass slide or fingernail (for example, with psoriasis).

When scraping, specify the nature of the peeling (small or large-lamellar, pityriasis, cystic), the strength of the scales (easily scraped off with psoriasis and pityriasis, and, conversely, firmly held with lupus erythematosus, ichthyosis vulgaris). Dermatoscopy, video dermatoscopy (epiluminescent microscopy, microscopy of the skin surface) is a non-invasive method for diagnosing visual assessment of skin formations. It allows you to recognize morphological structures that are invisible to the naked eye, and to evaluate clinical and morphological changes in the skin in a new way, especially with pigmented lesions, in particular, with melanoma. The method consists in examining a skin area using a dermatoscope, stereo microscope, camera or digital recording system. The magnification range varies from 6 to 100 times, more often - 10 times.

The dermatoscope can be used with or without immersion oil. Using a video dermatoscope allows you to observe pigmented formations on the monitor, convert them into digital form and enter them into the memory of a computer connected to the system. Video dermatoscopy is used in the diagnosis of pigmented formations, infectious-parasitic, viral dermatosis in the early stages of development. Diascopy or vitropressure. A test area of the skin is pressed with a glass slide or a transparent plastic spatula to determine the vascular nature of the rash element (erythema disappears when pressed, but there are no hemorrhagic and pigmented elements). Diascopy is used to detect the phenomenon of "apple jelly" in lupus erythematosus and the phenomenon of "mote" in skin sarcoidosis. Probe symptom (Pospelov's symptom) - when lightly pressed with a bellied probe on the surface of the tubercle in case of skin tuberculosis, the probe easily sinks into the depth of the tissue and leaves a slowly absorbable depression.

Carrying out special laboratory and hardware studies:

a) luminescent diagnostics using a Wood's lamp allows you to assess the nature of the glow of the affected hair and scales of skin diseases with microsporia, pityriasis versicolor, lupus erythematosus;

b) research: - on leukocytes (with lupus erythematosus); - acantholytic cells smear-imprints with erosions in acantholytic pemphigus; - pale treponema, for Streptobacillus Petersen - Ducray separated erosive, ulcerative and weeping elements in syphilis, soft chancre; - scabies mite (for scabies) or Demodex folliculorum (for demodicosis); - microscopy and inoculation of affected scales and hair, nails for fungi - - microscopy and inoculation of material from foci of infection for flora and determination of the sensitivity of microorganisms to antibiotics;

c) a histological examination of a skin biopsy is used to clarify the clinical diagnosis, primarily with skin lymphoma, parapsoriasis, skin tumors, skin tuberculosis, bullous pemphigoid, tertiary syphilis, leprosy, deep mycoses, and rare dermatoses;

d) carrying out skin-allergic tests: application, scarification, intradermal or in vitro tests (leukocyte agglomeration test, basophil degranulation test, mast cell degranulation test, lymphocyte blast transformation test, etc.

e) immunodiagnostics (level I and II reactions) to determine the immune status in severe chronic dermatoses (pustular and arthropathic psoriasis, pemphigus, skin lymphomas, atopic dermatitis, etc.).

PRIMARY AND SECONDARY MORPHOLOGICAL ELEMENTS

Primary morphological elements are divided into proliferative and exudative. Proliferative features include macules, papules, bumps, or nodules, and exudative features include vesicles, vesicles, pustules, and wheals. Another classification of the primary elements of the rash is often used: they are divided into asexual (spot, papule, tubercle, node, blister) and cavity (bladder, blister, abscess). If the patient has 2 or more primary elements on the skin, they speak of true polymorphism. The evolutionary development of one primary element, such as a bubble, and the appearance of secondary elements masquerading as primary elements, indicates a false, or evolutionary, polymorphism. Spot (macula) - asexual primary element, manifested by a change in skin color in a limited area. There are islet-inflammatory spots (roseola, erythema), varying in size; vegetative-vascular non-inflammatory spots (erythema of shame); spots as anomalies in the development or location of blood vessels (hemangiomas,

telangiectasias). From specifically allocate hemorrhagic spots resulting from an increase in the permeability of the vascular wall or rupture of the vessel (petechiae, purpura, ecchymosis, vibricide, hematoma). Age spots can be congenital (nevi), spontaneous (freckles, chloasma, lentigo), formed under the influence of drugs or other factors (toxic melasma) and artificial (tattoos, permanent makeup). With a decrease in the amount of pigment in the skin, they speak of hypo- and depigmented spots, which can be either congenital (albinism) or acquired (vitiligo). Vascular and dyschromic spots may be secondary. A nodule (papule) is an asexual primary element, which is a superficial compact element of an inflammatory or non-inflammatory nature, protruding above the skin surface, of a dense elastic consistency, which does not leave a scar after resorption. Papules can be the result of: - proliferative processes in the epidermis, which are the result of three pathomorphological processes: acanthosis (due to thickening of the spinous layer in eczema), hypergranulosis (an increase in the number of rows of the granular layer - in lichen planus), hyperkeratosis (thickening of the stratum corneum - in hyperkeratosis of the palms and soles) - infiltrates in the dermis are the result of the formation of infiltrates of inflammatory (secondary syphilis) and non- inflammatory (more often a tumor) origin in the dermis or the growth of various structures of the dermis (for example, blood and lymphatic vessels) - deposits of metabolic products (amyloid, most are epidermal-dermal papules that occur in psoriasis, lichen planus, atopic dermatitis, eczema, lupus erythematosus, Dühring's dermatitis, leprosy, etc. Epidermal-dermal and dermal papules have an inflammatory genesis. In terms of size, miliary papules (up to 2 mm in diameter), lenticular papules (4-8 mm in diameter), there are nummular papules (i.e. e. the size of a coin - 2-3 cm in diameter) and plaques (more than 5 cm in diameter). The shape distinguishes between flat (epidermal, epidermodal), hemispherical (dermal) and pointed (follicular) papules. The color of the papules depends on the nature: papules of inflammatory origin have various shades of red, and nodules of non-inflammatory origin are either hyperpigmented or have the color of normal skin. The surface, shape and consistency of papules can be very diverse. Tuderculum is an asexual rounded non-inflammatory element located in a reticular layer, approximately the size of a cherry tree. it has a hollow (with a diameter of 2 to 7 mm), hemispherical or flat shape, dense elastic or pasty consistency, rising above the surface of the skin. The formation of a tubercle is

based on productive inflammation in the dermis with the formation of an infectious granuloma due to infiltration by lymphocytes, epithelioid cells, Langhans giant cells (with leprosy, skin tuberculosis, tertiary syphilis). evolution of the tubercle often ends with disintegration and subsequent scarring. The reverse course of its development is possible in a dry way without disintegration with the formation of cicatricial atrophy. The color of the tubercles varies from reddish brown to reddish-cyanotic.

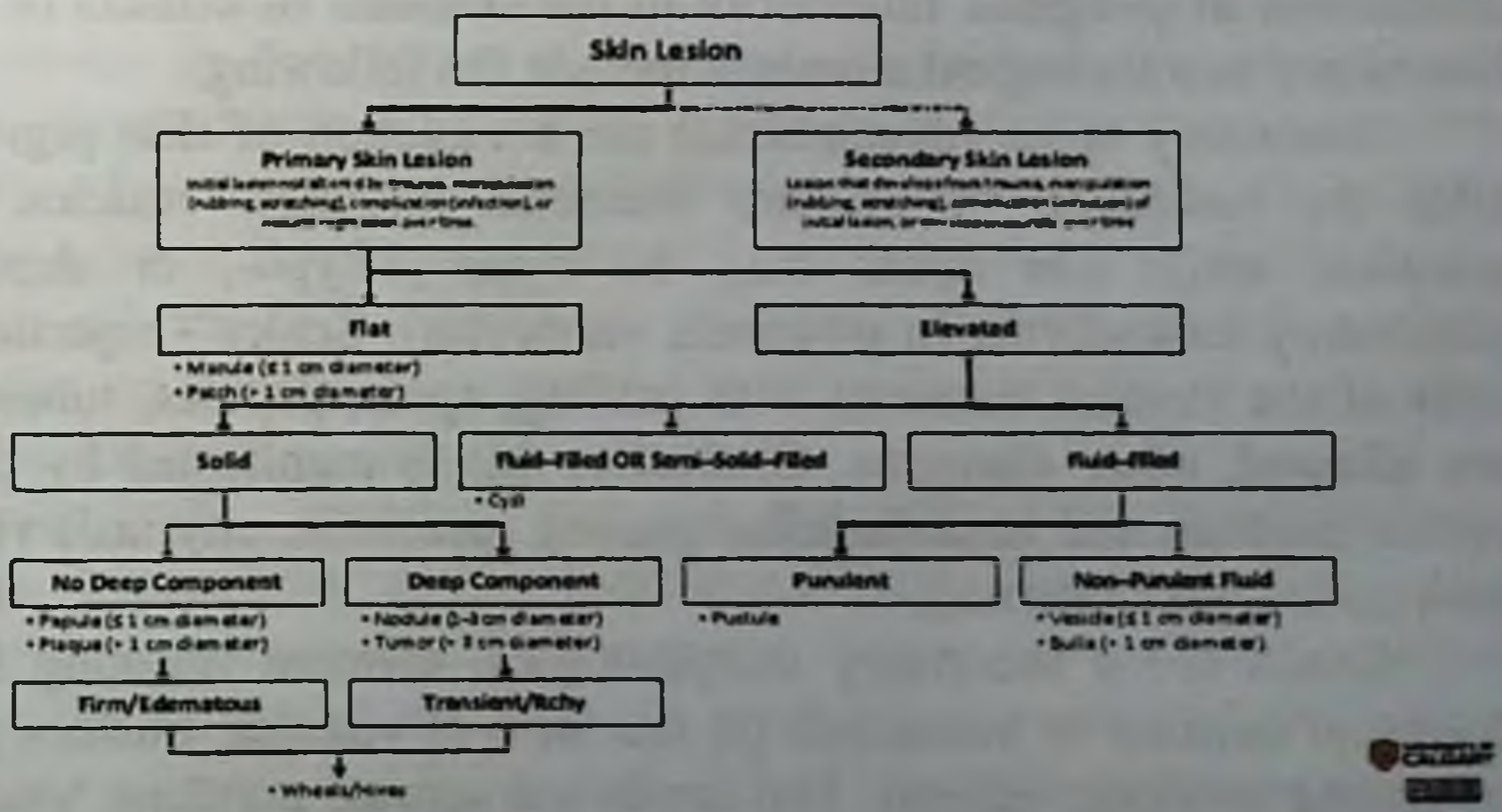
A node (nodus) is a large asexual infiltrated element, densely elastic in consistency, located in the subcutaneous fat and in the deep layers of the dermis, round in shape, rising above the surface of the skin.

Knot sizes range from hazelnut to chicken egg and larger. Nodules can be inflammatory or non- inflammatory. Non-inflammatory nodules are most often tumors (lipomas). Most of the nodes resulting from specific inflammation end in decay, ulceration and scarring (gum, scrofuloderma, leproma).

Blister (urtica) - a limited, itchy, asexual primary element of an islet-inflammatory nature, whitish-red in color with a smooth surface, dense consistency, rising above the surface of theskin, which is based on swelling of the papillary dermis (urticaria, Dühring's dermatitis). After afew minutes or a few hours, the blister resolves without leaving any traces. Sometimes, due to diffuse edema of the subcutaneous tissue, a giant blister (Quincke's edema) develops.

Morphology of Skin Lesions

Primary Skin Lesions



Pic-4 Morphology of skin lesions

The Vesicle (vesicula) - a hemispherical cavity 1-9 mm in size, located subcorneally or intraepidermally, filled with serous fluid. Histologically, the vesicle is the product of spongiosis (eczema), ballooning degeneration (herpes), or vacuolar dystrophy (dyshidrotic eczema). The evolution of a vesicle can be twofold: it either opens, forming an erosion, which then becomes covered with a crust and epithelializes, or its contents dry out into a crust, and after its falling off, a hyperpigmented spot remains for some time.Bladder (bulla) is a cavity formation containing serous fluid, blood or pus, 6-10 mm or more in size, rounded in shape, protruding above the level of the skin (pemphigus vulgaris, Dühring's herpetiform dermatitis). Blisters can be caused by exogenous factors (friction, burns, strong acids and alkalis) or endogenous factors (circulating immune complexes causing acantholysis, epidermolysis). Immune complexes have an immunopathological effect on epidermocyte desmosomes (acantholysis in pemphigus), basement membrane (epidermolysis in bullous pemphigoid), or on the connective tissue of the tips of the papillae (in Dühring's dermatitis).

A pustule is a cavity element ranging in size from 1 to 10 mm, filled with purulent contents, hemispherical or flat. Pustules are located in the epidermis or dermis. The formation of an abscess is based on necrosis of epidermal cells under the influence of waste products of pyogenic microbes with the formation of a purulent cavity. Pustules can be follicular (osteofolliculitis, folliculitis, boils) and non-follicular (conflicts in streptococcal and impetigo vulgaris). Secondary pustulosis is the attachment of pyogenic microflora to the contents of blisters or vesicles. Secondary morphological elements include the following.

Secondary spots (dyschromia) are a violation of skin pigmentation after the resolution of primary elements (papules, vesicles, blisters, pustules, etc.). The spots may be hyper-, hypo-, or depigmented (secondary leukoderma in pityriasis versicolor). Scales - rejection of the cells of the stratum corneum with peeling, spots, papules, tubercles, etc. are allowed. other elements. Clinically, this is manifested by pityriasis, cystic, medium and large-lamellar peeling (psoriasis, pityriasis versicolor, pink lichen, toxicoderma).

Crusts are a secondary morphological element resulting from the drying of exudate or transudate (in the form of vesicles, blisters, pustules, separating erosions, ulcers). The crusts are serous, purulent, bloody, also flat, thick-layered, dense, oyster-like. Abrasions, excoriations - excoriatio)

- skin damage resulting from mechanical trauma, scratching. Abrasions can be superficial (scabies) or deep (nodular itching, pathomymia).

Fissure (fissure) - linear damage to the skin due to loss of elasticity or inflammation. Cracks can be superficial (within the epidermis), which disappear without a trace (eczema of the hands and feet), and deep (within the dermis), leaving scars (papular infiltration of the Hochsinger skin,

leaving Paro-Robinson-Fournier radial scars). around the mouth). Erosion (erosio) is a superficial violation of the integrity of the skin within the epidermis. Erosions appear at the site of open blisters, blisters, serous papules (true eczema, dyshidrosis, true pemphigus, diffuse streptoderma).

An ulcer (uleus) is a deep skin lesion within the epidermis, dermis, and often hypodermis due to necrotic processes. Ulcers are formed during the opening of boils, tuberculous tubercles, with leprosy. skin tumors disintegrate, trophic disorders develop. The ulcer leaves scars. Scarring (scar) is the result of the replacement of a skin defect with coarse fibrous connective tissue. Scars are formed after cuts, burns, ulcerations, deep pustules, tubercles, knots, cracks.

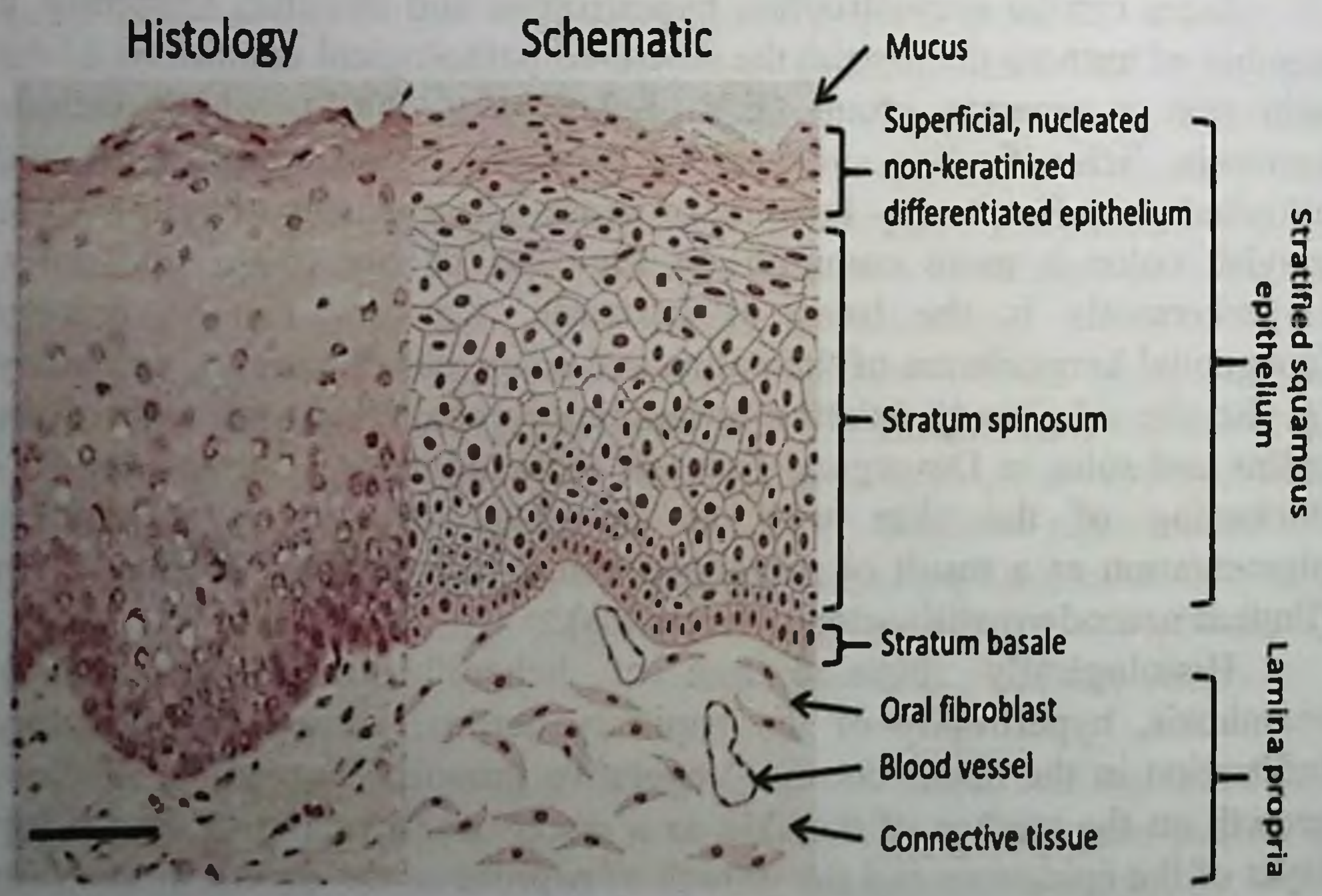
Scars can be normotrophic, hypertrophic and atrophic. Currently, a number of authors distinguish the so-called pathological conditions of the skin into a separate group (E.V. Sokolovsky, 2005), which include keratosis, lichenification, vegetation, dermatosclerosis, anetoderma and atrophoderma. Keratosis - layering of dense horny masses of yellowish or grayish color is more common on the skin of the palms and soles. Hyperkeratosis is the basis of keratosis. Keratosis can be primary (congenital keratoderma of the palms and soles, petrifaction) or secondary (at the site of already existing inflammatory changes in the skin of the palms and soles in Devergie's disease). Lichenification (licnenificatio) - thickening of the skin with an enhanced pattern, rough surface, pigmentation as a result of prolonged irritation (combing) of these areas (limited neurodermatitis, atopic dermatitis).

Histologically revealed foci of lichenification: hyperkeratosis, acanthosis, hypertrophy of the papillary dermis, chronic inflammatory infiltration in the upper dermis. Vegetative growth (vegetatio) - villous growth on the surface of the skin as a result of thickening of the prickly layer of the epidermis and the growth of papillae of the dermis in the form of cauliflower (spiky papilloma), in the form of horny layers (vulgar warts) or the growth of papillae of the dermis in the area of inflammation in the

folds (wide warts in secondary syphilis, eroded papules in vegetative pemphigus). The histological basis of vegetation is papillomatosis. Anetoderma (anetoderma) - the presence of areas of atrophy of the skin itself with a herniated protruding surface. These elements of the rash are characterized by the "bell button" symptom. It is based on atrophy of the elastic fibers of the dermis. There are primary and secondary anetoderma. An example of a primary anetoderma is the Jadassohn-type anetoderma, and a secondary anetoderma may form at the site of some itchy rashes. Atrophoderma - retraction of the skin of various depths over areas of atrophy of the subcutaneous fat, while the skin pattern in the retraction area does not change. An example of primary atrophoderma is Passini-Pierini atrophoderma, and secondary atrophoderma is atrophic changes at the site of resolved panniculitis nodes or as a complication of lipoaspiration.

HISTOLOGICAL CHANGES IN THE SKIN AND MUCOSA OF THE MOUTH

Pathological processes in the skin may have a proliferative or exudative nature of inflammation and be located in the epidermis or dermis.

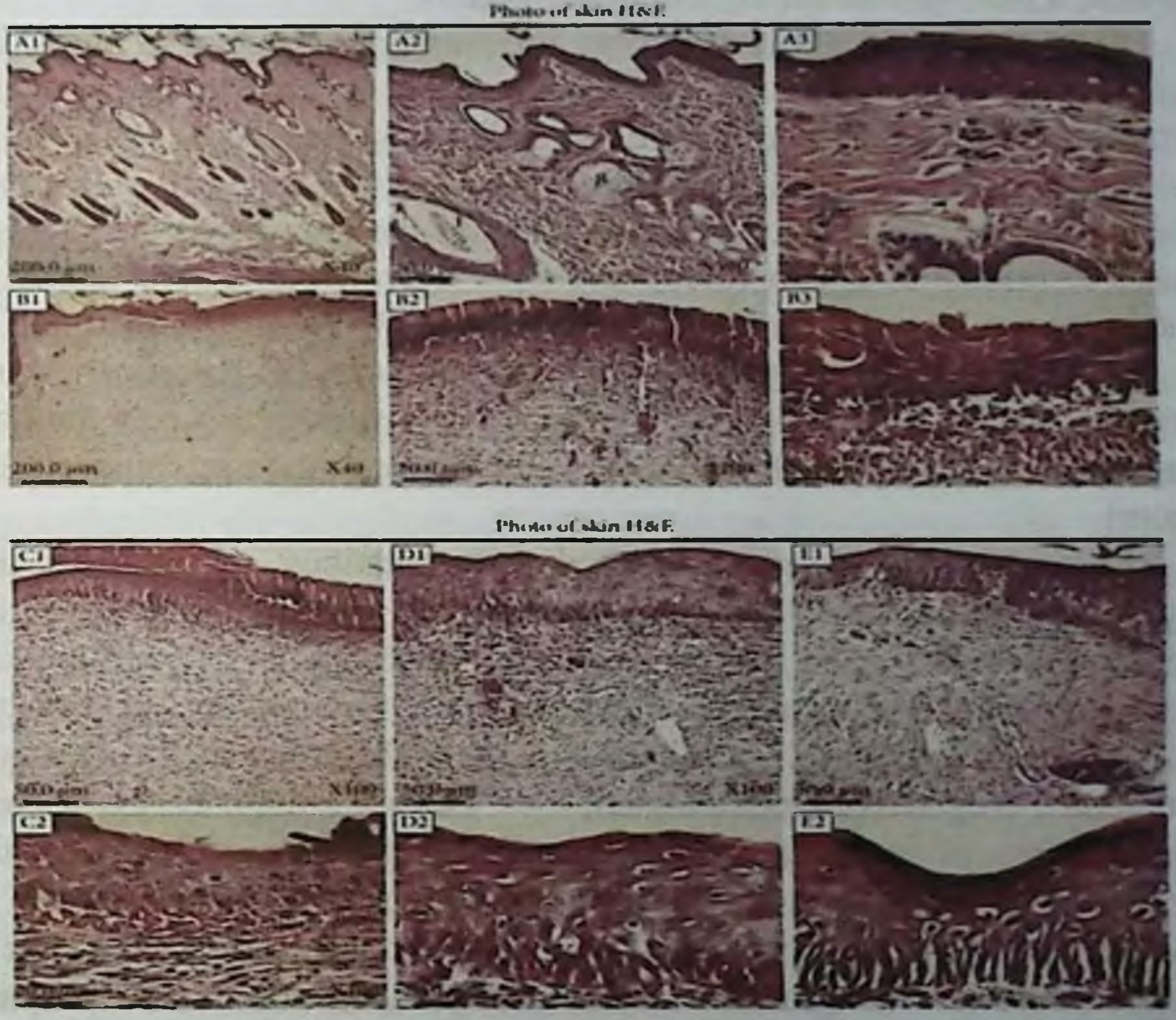


Pic-5 Histological Changes In The Skin And Mucosa Of The Mouth

PROLIFERATIVE HISTOPATOMORPHOLOGICAL CHANGES IN THE EPIDERMIS AND DERM

Acanthosis - proliferation of cells of the spinous layer, an increase in the number of their rows, the introduction of epithelial papillae into the papillary layer of the dermis. It is determined for psoriasis, eczema, neurodermatitis, warty tuberculosis.

Anaplasia - atypia of the nuclei of epithelial cells, characterized by an increase in their size, hyperchromatosis and the presence of atypical mitotic figures. Occurs in malignant tumors of the skin.



Pic-6 Proliferative Histopatomorphological Changes In The Epidermis And Derm

Hyperkeratosis - excessive keratinization of the epidermis, in which hyperplasia of the stratum corneum is associated with an excess amount of altered keratin. There is a thickening of the stratum corneum as a result of excessive formation of horns (psoriasis) and / or delayed exfoliation of horny scales (ichthyosis). According to the mechanism of development, there are physiological (palms, soles); acquired (corns); hereditary

(keratoderma, ichthyosis) hyperkeratosis. There are proliferative and retention hyperkeratosis. Proliferative hyperkeratosis occurs with lichen planus and neurodermatitis, is a consequence of increased activity of epidermal cells and occurs against the background of thickening of the granular and prickly layers. With retention hyperkeratosis, there is a delay in the rejection of horny cells and thinning of the granular and prickly layers, which is observed with ichthyosis vulgaris.

Granulosis - hypertrophy of cells of the granular layer of the epidermis and an increase in the number of rows of granular cells up to 4-5 or more, a pathognomonic sign of lichen planus.

Granuloma is a limited chronic inflammatory reaction of the skin, which is characterized by the accumulation of mononuclear cells (lymphocytes) with an admixture of epithelioid, multinucleated giant cells, histiocytes, macrophages. Granuloma may be the result of a chronic proliferative process or occurs in response to the introduction of foreign bodies or infectious agents (oleogranuloma, silicone granuloma, tubercles of skin tuberculosis, leprosy, tertiary syphilis, etc.).

Dyskeratosis is a violation of the process of cornification of keratinocytes. Dyskeratosis is of two types: acantholytic (Daria's disease, warts ci) and neoplastic (keratoacanthoma, Bowen's disease, spinocellular cancer).

Microabscesses - small limited accumulations of cells in the epidermis and subepidermal papillae: - Munro neutrophilic microabscesses in the stratum corneum (psoriasis); - neutrophilic-eosinophilic microabscesses on the tops of the papillae (Dühring's dermatitis herpetiformis); - eosinophilic microabscesses in the growing epidermis (vegetative pemphigus); – Potry's microabscesses are accumulations of immature lymphocytes in the epidermis. Characteristic of T-cell lymphomas of the skin, pemphigoid. Parakeratosis - characterized by impaired keratinization, manifested by partial or complete disappearance of the granular layer, the presence of a non-compact stratum corneum with reduced nuclei and air spaces. It occurs in psoriasis, parapsoriasis, lichen planus, atopic dermatitis and other inflammatory dermatoses.

Papillomatosis - proliferation of cells of the papillary layer of the dermis, their elongation and deep ingrowth into the epidermis. It forms the morphological basis of vegetation, a secondary element of a skin rash. It is observed in psoriasis (provides the third component of the psoriatic triad - spot bleeding). Colloidal bodies (Civatt bodies) - the presence of rounded

formations with a diameter of up to 10 microns in the lower parts of the Malpighian layer. Such destructively altered epidermal cells are observed in lupus erythematosus and lichen planus. Exocytosis - the penetration of the pathological process from the dermis into the epidermis, is observed in skin lymphomas, lichen planus.

EXUDATIVE-HISTOPATOLOGICAL CHANGES IN THE EPIDERMIS AND DERM

Acantholysis. The process is characterized by the destruction of intercellular bridges in the cells of the spinous layer as a result of autoallergic and complex enzymatic reactions, which leads to the formation of intraepidermal cavities in which acantholytic cells are found. Acantholytic cells are degenerative cells of the prickly layer with a large hyperchromic nucleus, a narrow band of cytoplasm, and the cell itself is small. Acantholysis is characteristic of all forms of acantholytic pemphigus. balloon degeneration. The resulting intraepidermal cavities are the result of a powerful intercellular edema, reticular dystrophy with a break in the bonds between the cells of

the epidermis. They they are the basis for the appearance of small bubbles on a swollen background, which is typical for herpes.

Vacuolar degeneration - the formation of one or more cavities (vacuolitis) in the cytoplasm of edematous epidermocytes. The vacuoles are filled with fluid and may form a crescent shape around the nucleus. Vacuolar dystrophy of epidermocytes occurs with flat warts, allergic dermatitis. Reticular dystrophy. In the epidermis, as a result of a sharp intracellular edema and rupture of intercellular bridges, multi-chamber intraepidermal cavities are formed. This process is observed in acute dermatitis, herpes.

Spongiosis - intercellular edema in the prickly layer with expansion of the intercellular spaces. Occurs with eczema, acute dermatitis.

PRINCIPLES OF TREATMENT OF SKIN DISEASES

The authority of a dermatovenereologist directly depends on the depth of his clinical thinking, the ability to look and see, listen and understand, inspect and analyze, diagnose and treat, the ability to select an individual treatment in each case, adequate to the form, stage and prevalence of the process, gender and age of the patient, concomitant pathology, household and professional features of the case based on the results of previous treatment. In most cases, the treatment of patients with one or another dermatosis is aimed either at eliminating the cause of the disease (etiological treatment), or at correcting certain aspects of the

pathological process (pathogenetic therapy), or at eliminating individual symptoms of the disease when its etiopathogenesis is not clear (symptomatic therapy). Comprehensive treatment of a patient with dermatosis includes patient compliance with a preventive treatment regimen, organization of therapeutic nutrition, general and external (local) drug therapy, physiotherapy, non-drug treatment, including psychotherapy, spa treatment, and, if necessary, surgical treatment. care.

THERAPEUTIC AND PREVENTIVE REGIME

1. Washing. General washing in the shower or in the bath is not indicated for pustular skin lesions (streptococcal and vulgar impetigo, acute furunculosis, hidradenitis); in the first weeks of treatment of microsporia, favus and trichophytosis in order to avoid dissemination of infection; with pink lichen, washing with soap and a washcloth is prohibited due to the possible spread of the process; in acute allergic inflammatory dermatoses with weeping phenomena. In some cases, alcohol wiping of unaffected areas is allowed up to 2 times a day according to the principle "from the periphery to the center" using 1% salicylic acid, 3% boric acid or pure 40-70% ethyl alcohol. Gentle washing without soap and washcloths, preferably using a shower gel without rubbing with a towel, is indicated for patients with progressive psoriasis, lichen planus, atopic dermatitis.

2. Clothes. In case of allergic, inflammatory and itchy dermatoses, the patient should wear underwear only made of cotton fabrics, underwear and clothes made of wool, silk and synthetic

materials should not be worn.

3. Staying in the fresh air is useful for chronic dermatoses. At the same time, insolation, exposure to direct sunlight should be extremely limited in case of photodermatosis, lupus erythematosus.

4. Sleep and rest is an important part of the hospital regimen. Severe patients with exacerbations of chronic dermatoses with fever, chills, general weakness are shown strict bed rest.

5. Medical nutrition. It plays a very important role in the complex treatment of most pruritic, allergic and metabolic dermatoses. The main thing is to identify the increased sensitivity of the patient's body to certain foods. In such cases, the first type of specific diet is used - the exclusion of allergen products from the diet. For example, avoiding chocolate, egg white, or fresh cow's milk can lead to rapid resolution of hives, pruritus, and atopic dermatitis in children and adults.

The second type of specific diet is a diabetic diet for chronic furunculosis, lipoid necrobiosis, carbohydrate restriction for pyoderma,

atopic dermatitis, hypochloride diet for rosacea, eczema, hypocholesterol diet for skin xanthomatosis, psoriasis. This type of therapy is aimed at correcting the identified metabolic disorders and diseases of the gastrointestinal tract. in patients with dermatoses, which accelerates the rehabilitation of patients.

Nonspecific diet therapy, widely used in the complex treatment of itchy and inflammatory dermatoses in the acute period, involves a sharp restriction, up to the complete exclusion from the diet of sweet, spicy, salty, fried and extractive foods (for example: honey, jam, chocolate, citrus fruits, mustard, horseradish, peppers, spicy cheeses, smoked sausages, etc.). During the period of exacerbation of chronic skin diseases, alcoholic beverages and beer should be completely excluded. Since ancient times, short-term therapeutic starvation and unloading diet therapy have been used as hyposensitizing therapy. 6. Since mental trauma, functional disorders of the central and autonomic nervous system play a role in the occurrence or exacerbation, as well as in the pathogenesis of a number of dermatoses (eczema, psoriasis, lichen planus, neurodermatitis, etc.), psychotherapy should be an integral part of the overall treatment of such patients. Psychotherapy involves the influence of a doctor on the patient with a word and his attitude towards the patient. The doctor must be able to patiently listen to the patient's complaints, convince him that the disease is curable, explain to the patient in a popular and accessible form why he is prescribed this or that medicine, how to take it and what should be expected from this drug in terms of improving subjective and objective characteristics. diseases.

The main thing is to win the patient's trust, to convince him that his problems are your problems, and you are able to help them solve them. In addition to suggestion, hypnosis is also used, which has a more pronounced psychotherapeutic effect in patients with dermatoses with itching, alopecia areata, psoriasis, pathomymia, etc. To normalize the activity of the central and autonomic nervous system in patients with dermatosis, it is necessary to normalize sleep and lifestyle. For this purpose, you can use an electric probe, acupuncture (acupuncture),

transcutaneous electrical nerve stimulation. Tests for knowledge control

1. Which of the following drugs do not have a hyposensitizing effect? a) staphylococcal vaccine;

b) calcium gluconate; c) zinc oxide; d) diphenhydramine; e) telfast.

2. Glucocorticoid hormones are the basic therapy for: a) psoriasis; b) acantholytic pemphigus; c) epidemic pemphigus of newborns; d) articular dermatitis; e) scabies.

3. The following do not have antifungal activity: a) griseofulvin; b) pimafucin; c) nizoral; d) erythromycin; e) fluconazole.

4. No immunostimulating action: a) pyrogenal; b) polyoxidonium; c) antistaphylococcal immunoglobulin; d) taktivin; e) cyclosporine A (sandimune).

5. Vitamin A deficiency does not lead to: a) the development of scurvy; b) violation of the processes of keratinization of the skin; c) violation of optical adaptation in the dark; d) fragile bones; e) hair loss.

6. Vitamin E deficiency leads to: a) anemia; b) the birth of children with a large weight; c) testicular atrophy and impaired spermatogenesis; d) acromegaly; e) the appearance of foci of vitiligo.

7. Zinc preparations are indicated as etiopathogenetic therapy in the treatment of: a) scabies; b) senile itching; c) enteropathic acrodermatitis;

Tests for knowledge control

1. Acanthosis does not occur in: a) psoriasis; b) lichen planus; c) lupus erythematosus; d)neurodermatitis; e) eczema.

2. Acantholysis happens: a) with dermatitis; b) vegetative pemphigus; c) herpes; d) Kaposi's sarcoma; e) Lyell's syndrome.

3. Follicular hyperkeratosis is not typical for: a) lupus erythematosus; b) Devergie's disease; c) avitaminosis C; d) skin changes in polyglot invasions; e) ichthyosis vulgaris.

4. Diffuse hyperkeratosis is not typical for: a) ichthyosis; b) palmoplantar keratoderma; c) homozoa; d) Dühring's dermatitis herpetiformis; e) verrucous lichen planus.

5. What disease does not cause dyskeratosis? a) lichen planus; b) Darier's disease; c) Paget's disease; d) keratoacanthoma; e) scleroderma.

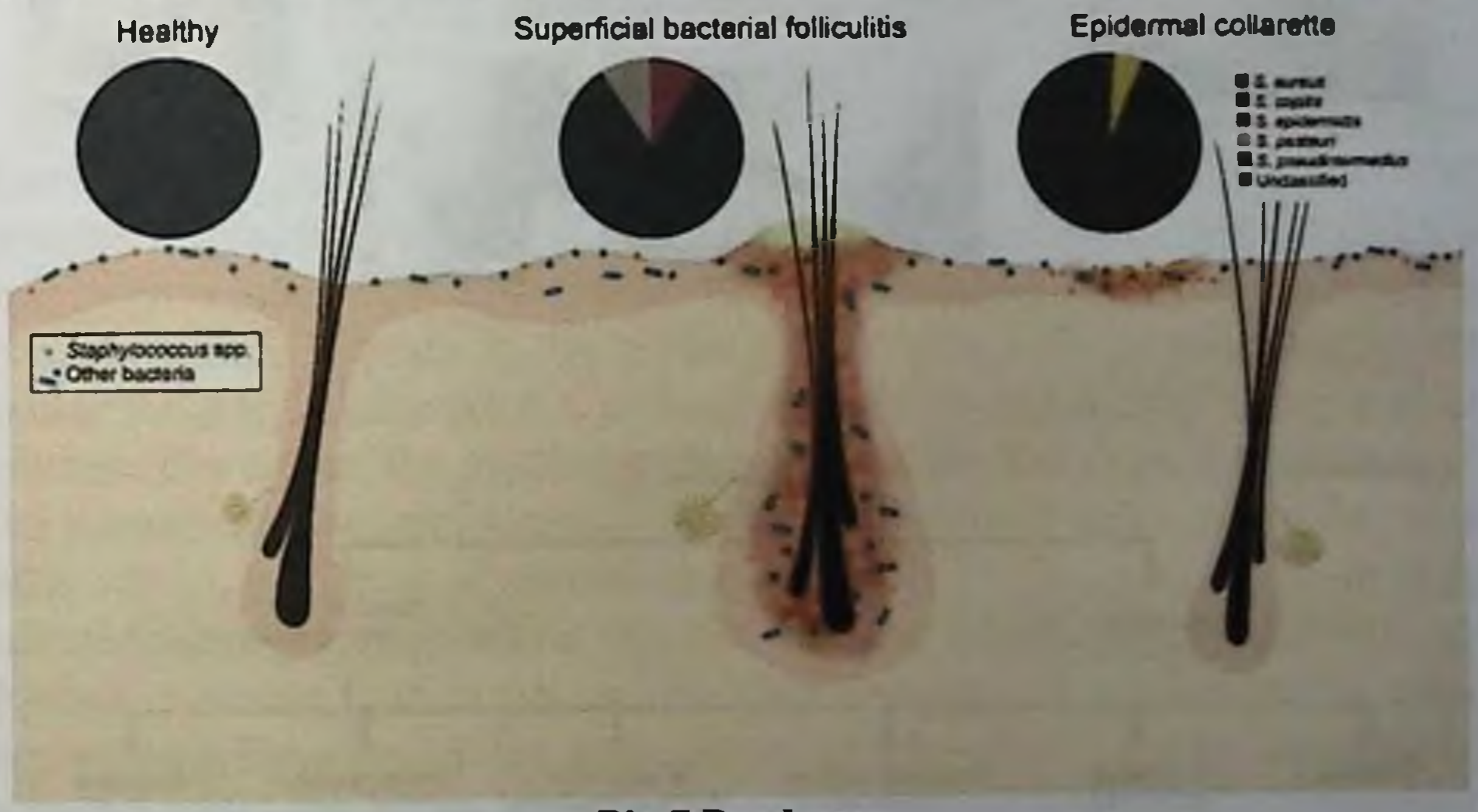
6. Parakeratosis is not observed: a) ichthyosis; b) psoriasis; c) pink lichen; d) lichen planus; e)chronic eczema.

7. Balloon degeneration is typical for: a) genital herpes; b) streptococcal impetigo; c) vulgar impetigo; d) pemphigus vulgaris; e) Duhring's dermatitis herpetiformis.

Answer standards: 1 b; 2 a; 3 d; 4 d; 5 d; 6 a; 7 a.

CHAPTER 2. PYODERMA. UNPLEASANT. DERMATOZOONOSES. CLINICAL CLASSIFICATION OF PURULENT SKIN DISEASES. STREPTO-, STAPHYLODERMA, MIXED PYODERMA. A SIMPLE ANNOYANCE. SCABIES, LICE.

Pustular skin diseases (pyoderma) are the result of skin lesions with pyogenic microbes: staphylococci, streptococci, pneumococci, Proteus, gonococci, Pseudomonas aeruginosa and Escherichia coli. prevalence of pyoderma. They occupy one of the first places in the structure of skin morbidity in the child population of the country. This is due to the exceptionally high prevalence of their pathogens in nature and the high frequency of the transition of non-pathogenic forms to pathogenic ones, as well as the age-related features of the morphology and physiology of the skin in children, and the weaker pronounced barrier function of the skin in the latter. The mechanism of development and clinical course of pyoderma depend both on the type and pathogenicity of the pathogen, and on the state of the protective forces of the microorganism. Intact, clean skin serves as a reliable barrier to the penetration of pyogenic microbes.

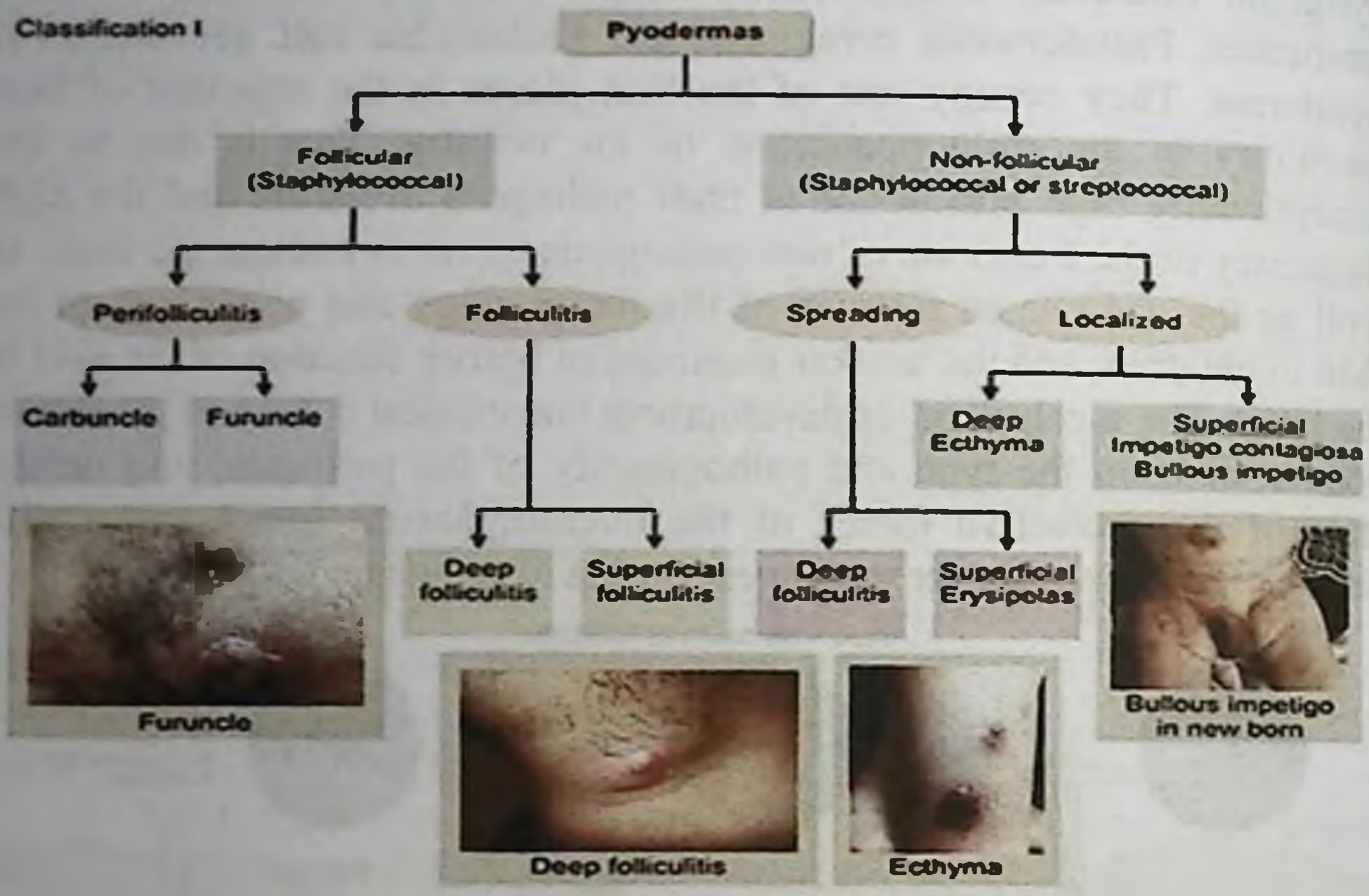


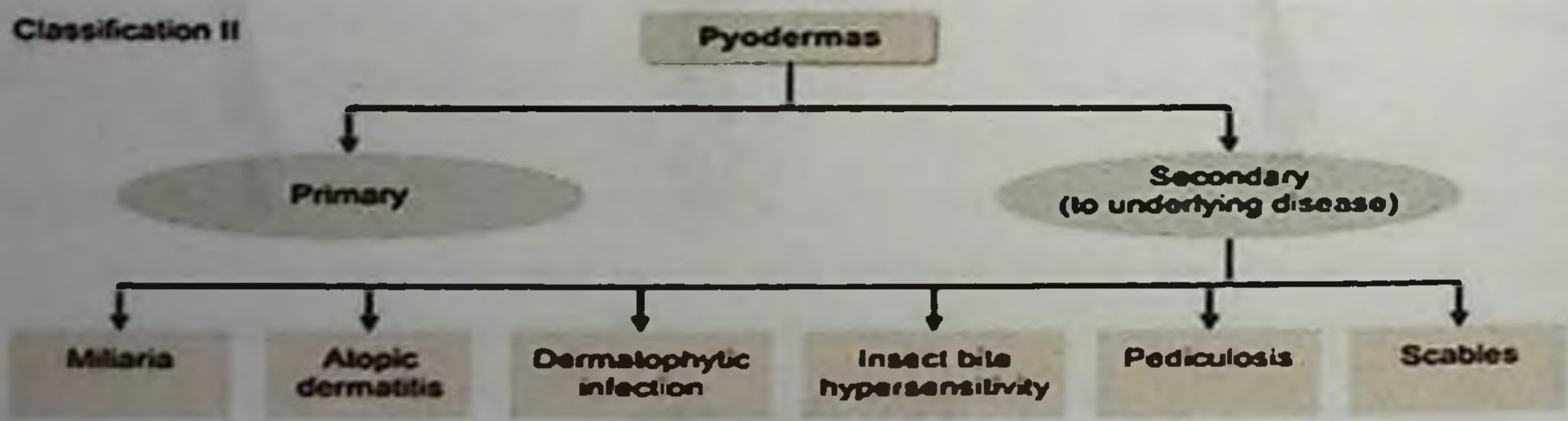
Pic-7 Pyoderma

Dirt, microtraumas, overheating and hypothermia of the body, exposure to drafts, exposure to substances that destroy the water-fatty emulsion film and cause a sharp decrease in the concentration of hydrogen ions on the skin are among the exogenous factors that contribute to the occurrence of pyoderma. skin surface (pH greater than 6.2-6.8 and even

greater than 7). The entrance gate of pyococcal infection can also be insect bites (mosquitoes, mosquitoes, midges, lice, bedbugs, etc.).

Among the endogenous factors contributing to the occurrence of pyoderma, one can name an unbalanced diet, hypovitaminosis, hypothermia, overwork, emotional overstrain, exhaustion of the body during diseases, intoxication, endocrine disorders (diabetes mellitus, etc.).





CLASSIFICATION OF PYODERMA

By the nature of the occurrence and course, primary (occur on the background of healthy skin), secondary (due to any skin pathology (eczema, neurodermatitis) or immunosuppressive therapy) are distinguished; acute (lasting up to 2 months) and chronic (lasting more

than 2 months) pyoderma. According to etiological signs, these diseases are divided into staphyloderma, streptoderma and streptostaphyloderma. According to the depth of skin lesions, superficial (localized within the epidermis) and deep (within the dermis, hypodermis) pyoderma are distinguished. It should be remembered that with pyoderma, the pathogen enters the body, as a

rule, from the outside: in older children, adolescents and adults, through the openings of the sebaceous hair follicles, and in young children, through the openings of the sweat glands.. Therefore, in newborns, older children and adults, staphyloderma predominates (the causative agent is predominantly Staphylococcus aureus), and in young children streptoderma (the causative agent is beta-hemolytic streptococcus).

Along with the above clinical and etiological classification of pyoderma, there is a classification of the latter according to ICD-10, which is based on the topography of rashes (headings: L 00-03; L 08; L 66. 2-6). Currently, all statistical reporting on incidence is carried out according to ICD- 10.

PYODERMA CLINIC

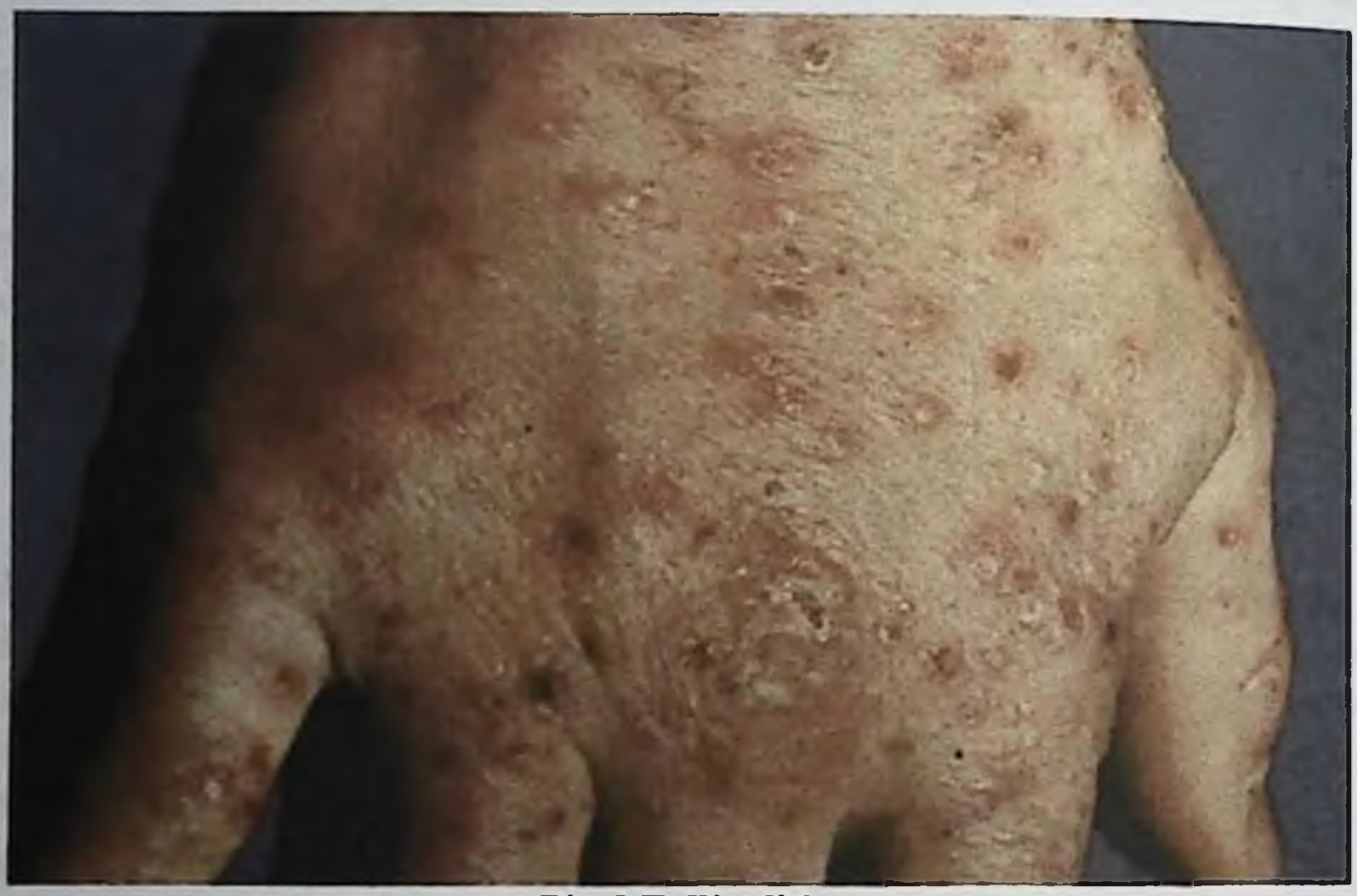
Superficial staphylodermic ostiofolliculitis is characterized by the formation of pustules at the mouths of sebaceous hair follicles. The process begins with the appearance of a pink or red-pink spot around the hair with a diameter of 4-7 mm. A few hours later, a yellowish-white conical pustule appears in the center of the spot with an elevation in the center ranging in size from a pinhead to a lentil and a halo of hyperemia around. The cap of the pustule is dense, tense, by 3-4 days the contents of the pustule dries up and turns into a yellowish-brown crust. After the crust falls off in 1-2 days, a pinkish spot remains in this place, which later completely disappears. When the pustules are opened, small erosions are formed, covered with crusts, which then disappear. Ostiofolliculitis often appears on the skin of the face, beard, scalp, chest, back. The disease usually heals itself. It is necessary to differentiate it from impetigo vulgaris. If the inflammatory process spreads from the mouth of the hair follicle to its deeper sections, then folliculitis is formed.

Folliculitis is a purulent inflammation of both the outer and inner root membrane of the hair follicle. It is customary to talk about superficial and deep folliculitis with an acute or chronic course of the disease.

Superficial folliculitis affects only the upper part (30-50%) of the hair follicle within the dermis, and inflammation increases within 2-3 days.

Clinically, it presents as a rash of hemispherical, rarely conical, often painful, pea-sized red papules and pustules surrounded by a pink-red rim. In the future, yellowish crusts form, after falling off of which there is no scarring or atrophy.

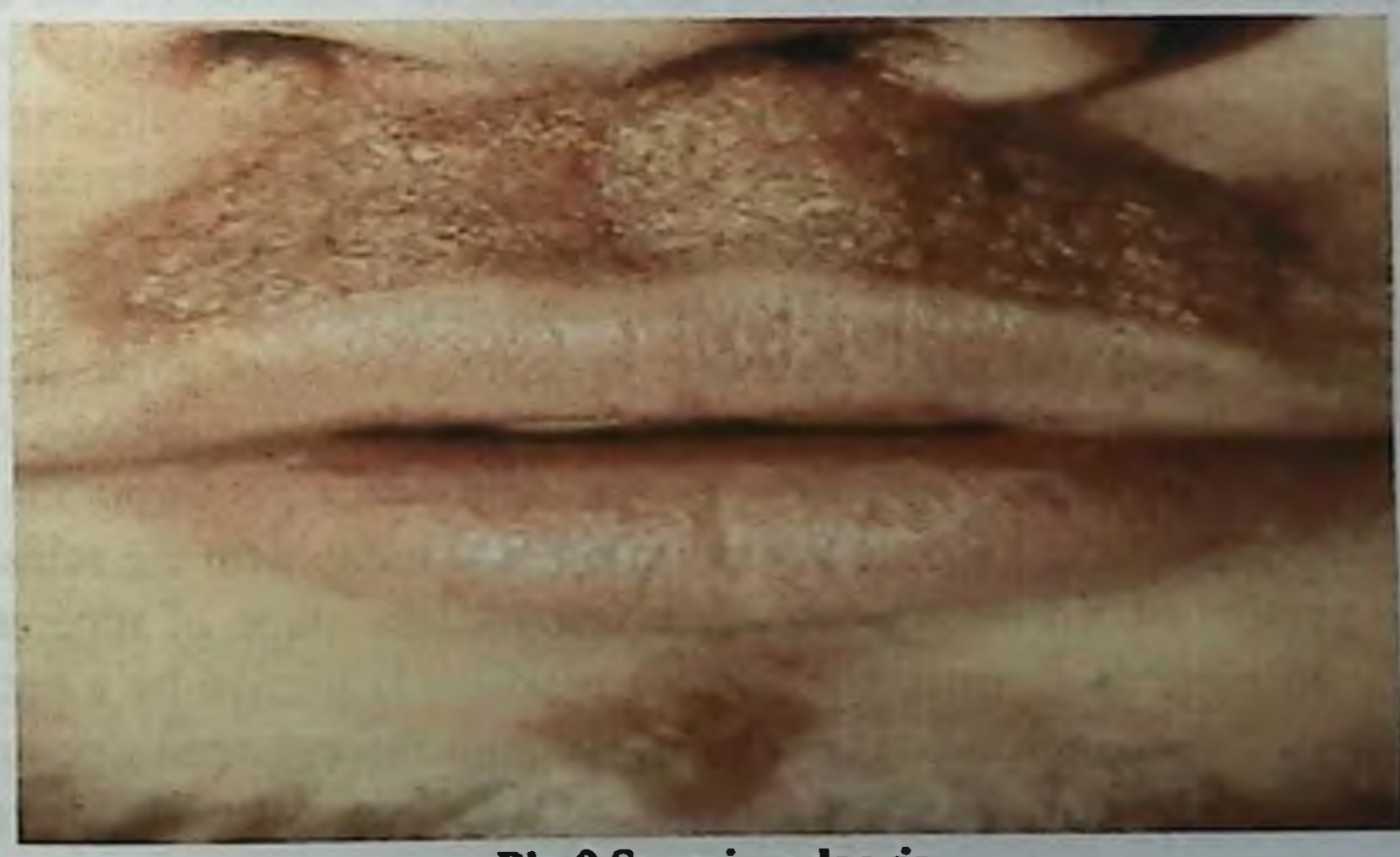
With deep folliculitis, inflammation captures the entire follicle, often surrounding fiber. In evolutionary terms, the infiltrate melts, opens, necrotic masses are rejected, an ulcer is formed, which heals with a scar. Folliculitis can develop on the skin where there is hair. It is necessary to differentiate it with vulgar impetigo, furunculosis.



Pic-8 Folliculitis

Sycosis vulgaris is a chronic inflammation of the hair follicles, manifested by continuously recurrent staphylococcal ostiofolliculitis and folliculitis in the mustache, beard, eyebrows, less often on the skin of the scalp, pubis, chest, and back. The main contingent is men. The causative agent is often Staphylococcus aureus or epidermal. The disease lasts for months and years and is not prone to self-healing. Patients go to the doctor only a few months after the onset of the disease, when elements of the rash are already detected at different stages of development. The face of such patients has an "untidy appearance": the skin in the area of the beard and mustache isinfiltrated, an abundance of pustules, erosions, purulent-bloody crusts, part of the hair in the center of the foci of inflammation has fallen out, the remaining unshaven hair loses its normal appearance, sticking out

in different directions. After healing, the hair grows back in most areas, but not in all, as part of the hair roots die off. Multiple scars remain at the site of resorption of the pustules. In typical cases, the diagnosis of vulgar sycosis is not difficult. First of all, it must be distinguished from parasitic sycosis (infiltrative suppurative trichophytosis). The latter begins acutely, is accompanied by the formation of inflammatory nodes, easy painless hair removal, after which droplets of yellow pus are released from the gaping hair follicles, which flows like honey from honeycombs (kerion celsius, i.e. "honey comb" Celsius "). Microscopic examination reveals zoophilic trichophyton in the hair and skin scales from the foci of inflammation. Parasitic sycoses may resolve spontaneously, without treatment, within 6-9 weeks, going beyond the immune system. Vulgar sycosis should also be differentiated from tuberculosis and lupus erythematosus. Vesiculopustulosis (periporitis) is a staphylococcal inflammation of the sweat pores of the eccrine sweat glands in young children. Its appearance is often preceded by sweating. The disease is manifested by the appearance of a large number of pustules ranging in size from a pinhead to a pea with whitish-yellow contents. A hyperemic border is formed around the pustules. The disease appears from the 3-5th day of life or later, until the end of the neonatal period. Periporitis affects mainly debilitated children who suffer from excessive sweating and do not have proper care. The foci of the disease are usually localized in the axillary and inguinal folds, on the skin of the chest, back, and scalp. The disease lasts from 3 to 10 days with timely treatment and proper care. It is necessary



Pic-9 Sycosis vulgaris

to differentiate periporitis with scabies (paired vesiculopustules, scabies, tick detection) and bullous impetigo.

Piosis Munson (synonyms: tropical pemphigus contagiosum, tropical bullous impetigo) is a representative of tropical pyoderma. It is found mainly in countries with a humid tropical climate. The causative agent is Staphylococcus aureus, sometimes hemolytic streptococcus is also isolated. Children and women are more likely to get sick, as well as Europeans who have come to countries with a hot tropical climate. The disease begins with the appearance of multiple pustules with an inflammatory corolla, often in combination with small conflicts with serous-purulent contents, erosions, and crusts in the area of large folds (inguinal, interstitial, axillary). The general condition of the patient usually does not suffer. After epithelialization of erosions, slight

hyperpigmentation remains. In debilitated and debilitated children, the process can be universal, accompanied by fever, headaches, weakness, and can last for several weeks.

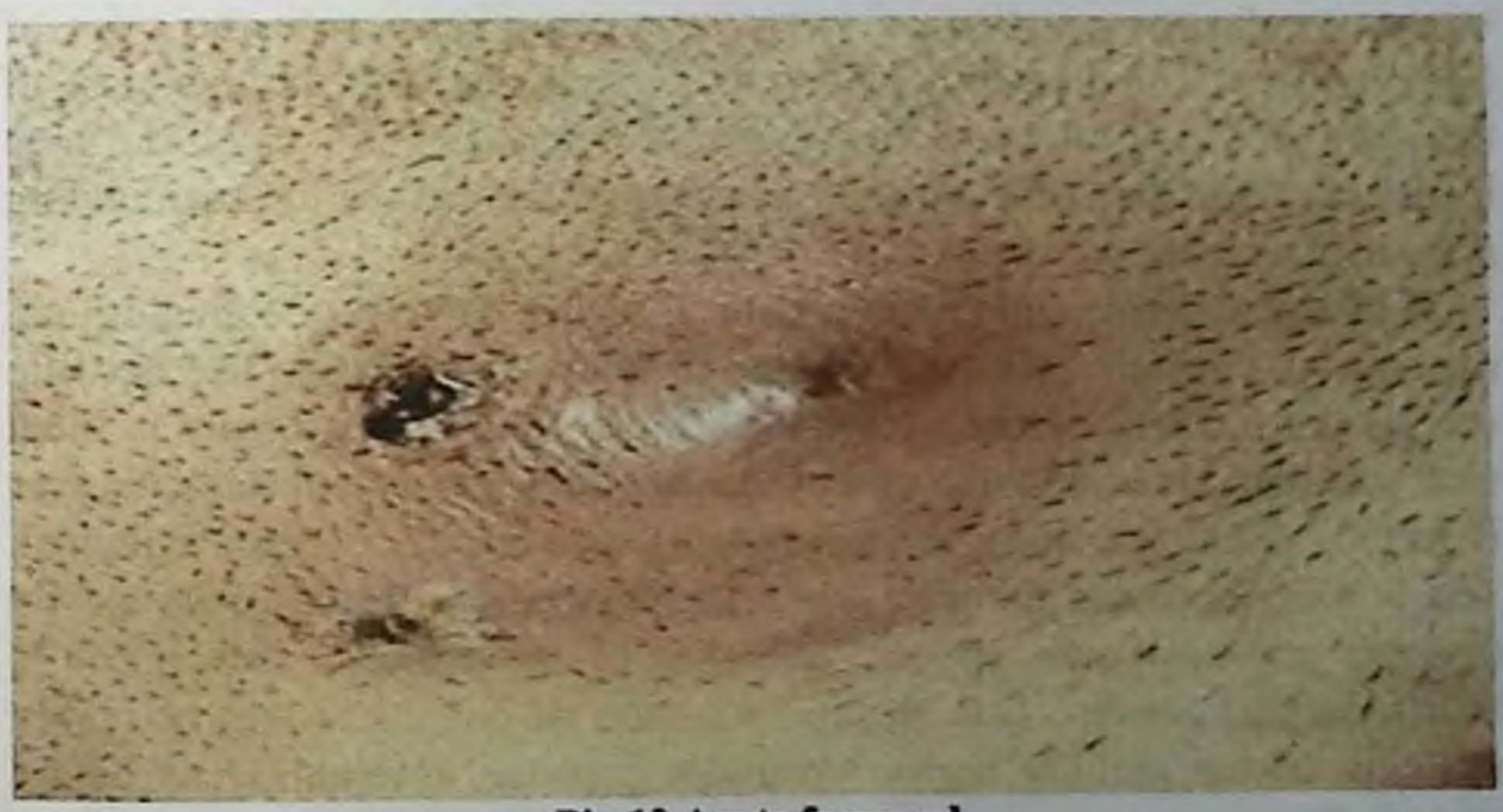
Munson's suppuration should be differentiated from chickenpox (mainly children are ill, skin rash

- papules and blisters with indentation in the center, crusts, scars - appears against the backgroundof general intoxication: fever up to 38.5 ° C and above, general weakness).

Deep staphyloderma Furuncles are an acute purulent-necrotic inflammation of the hair follicle throughout its entire length and adjacent tissues. More often boils are localized on the back of the neck, forearms, thighs, buttocks, face. Furuncles on the nasolabial fold and on the upper part of the face are dangerous, as they can cause thrombosis of the lymphatic and venous vessels, metastasis of infection, the appearance of purulent meningitis and septic conditions. Usually, a furuncle develops from folliculitis, when the inflammatory infiltrate grows along the periphery and in depth within 4-7 days, which leads to the formation of a conical node up to 2-3 cm in diameter or more. The knot is painful, the pain may be throbbing. Usually the first stage of development of a boil - the stage of infiltration - lasts 4-5 days and ends with the formation of a necrotic rod. Then the tissue at the top of the boil melts with the release of a small amount of pus through the hole formed (the stage of ulceration). Further, an ulcer is formed, the bottom ofwhich is formed by a necrotic rod. Within 2-4 days, the rod is rejected through the enlarged opening of the ulcer. Within 4-5 days, the ulcer is filled with granulations and scarred

(scarring stage). Thus, for the maturation and resorption of the boil, an average of 2 weeks is required. At the same time, on time rational therapy, started at the infiltrate stage, allows you to stop the process, and the inflammatory infiltrate resolves within 4-5 days, pain disappears, and usually there is no trace of the boil. The furuncle is differentiated from the nodes of infiltrative- suppurative trichophytosis. Furunculosis can be acute and chronic.

Acute furunculosis is more often the result of general hypothermia or overheating, when several boils (up to 10 or more) form simultaneously in a limited area (acute localized furunculosis) or in different areas of the skin (acute general furunculosis). Chronic furunculosis can also be localized and general, it is a recurrent purulent process in which boils appear at different frequencies over a long period of time. Usually develops in people suffering from diabetes mellitus, chronic gastroenteritis or colitis, HIV infection, hypo- and beriberi; it can also be the result of prolonged physical overwork. A carbuncle is a conglomerate of several boils united by a common infiltrate. In fact, we are talking about purulent-necrotic inflammation of the subcutaneous tissue, affecting several hairline. follicles.



Pic-10 Acute furuncul

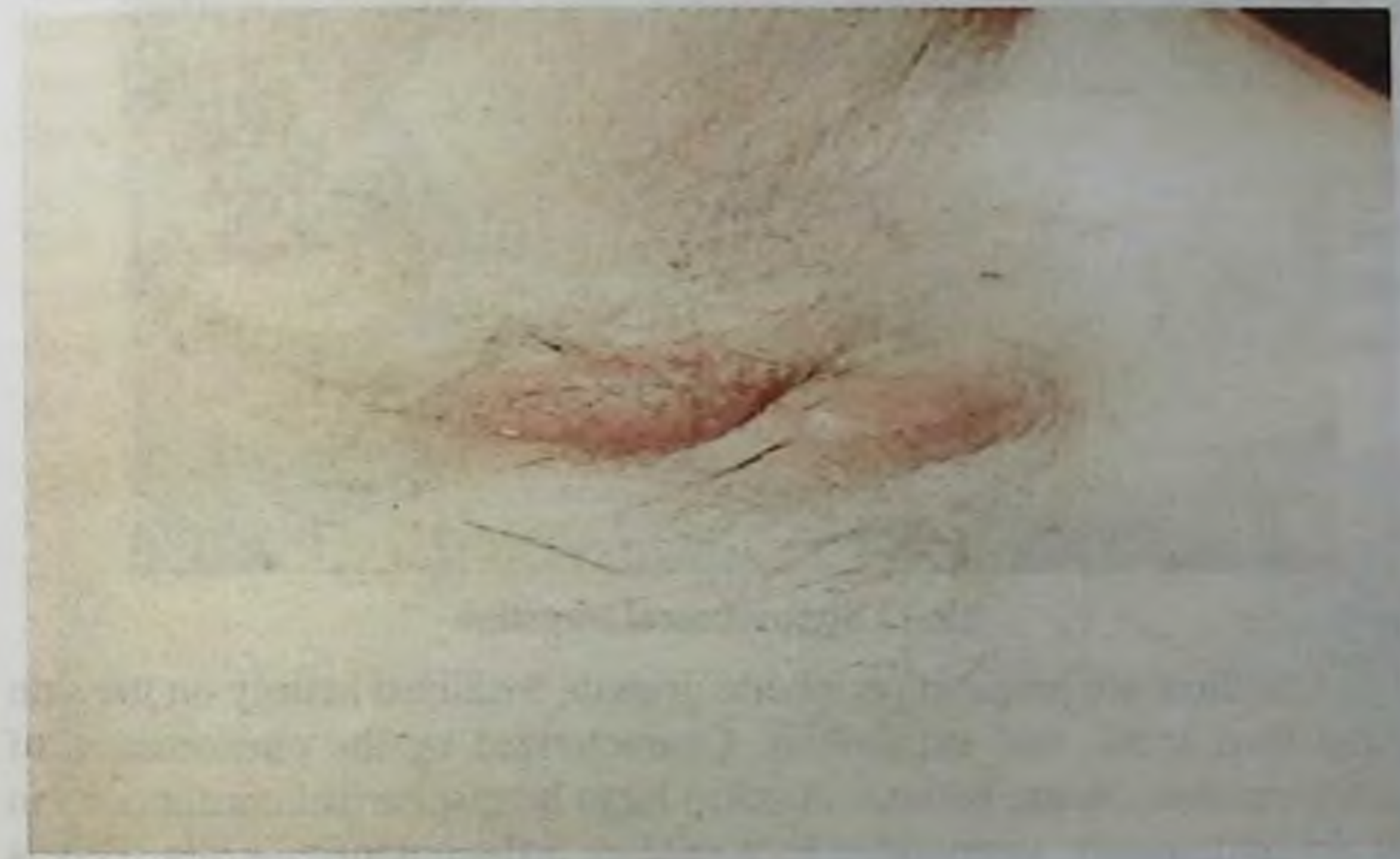
The inflammatory infiltrate grows both due to peripheral growth and involvement of new hair follicles in the process, and due to spread to the underlying tissues - muscles, fascia, periosteum. Already by the 4-5th day of development, the carbuncle turns into a dense inflammatory node (up to 10 cm in diameter), indistinctly delimited from healthy tissue. The central

convex part of it is purplish-blue, while the periphery is bluish-pink. Throbbing, tearing pain is characteristic. Within almost 2 weeks, the painful infiltrate softens, forming several fistulous openings, through some ofwhich a purulent-bloody fluid is released. After the rejection of multiple necrotic rods, a wide, deep ulcer of irregular shape is formed with bluish-covered edges and a loose, uneven bottom. Infiltration around it persists for a long time. Gradually, the ulcer is filled with granulations and heals with a retracted uneven scar. The development of a carbuncle is usually accompanied by fever up to 40-41 ° C, chills, headache, possible confusion, frequent complications, the nature of which depends on the localization of the carbuncle. So, the carbuncle of the chin area can be complicated by osteomyelitis of the lower jaw with perforation into the oral cavity. Differential diagnosis of carbuncle is carried out with furuncle, phlegmon, actinomycosis.

Hidradenitis is an acute purulent inflammation of the apocrine sweat glands caused by Staphylococcus aureus and Staphylococcus aureus. inflammatory the process of hidradenitis is localized in the armpits, less often - on the pubis, around the breast nipples, in the genital area and perineum. The development of the disease is promoted by excessive sweating, alkaline reaction of sweat, non-compliance with the rules of body hygiene. The disease begins with the appearance in the thickness of the skin of a painful node the size of a pea. After 2-3 days, the node increases to the size of a hazelnut or walnut, soldered to the surrounding skin. In the center

of the abscess, which has a conical shape, there is a softening and opening of the node with the release of thick creamy pus. The necrotic core is not formed. Already on the 7-8th day, the abscess cavity is filled with granulations and by the end of the second week a small retracted scar is formed. Near one node, several more glands usually become inflamed. The process becomes chronic: some nodes open and heal, others slowly dissolve and new ones appear. The skin of the affected area becomes bluish-purple, uneven. Due to the formation of conglomerates of nodes, the skin of the axillary region protrudes papillae, resembling a "bitch's udder". The process can last several weeks or even months, there may be a subfebrile reaction. temperature, general weakness, especially with the bilateral location of hidradenitis. The presence of HIV infection, diabetes mellitus, dysfunction of the sex glands, thyroid gland, myxedema aggravate the course of hidradenitis.

Differential diagnosis is carried out with boils, scrofuloderma, phlegmon, multiple abscesses. In weakened, anemic and dystrophic children up to 6 months old, less often in older children (up to 1 year old) who do not receive proper care, multiple abscesses (pseudofurunculosis) may form. With this disease, painful nodules the size of a pea or hazelnut appear on the skin of the back, buttocks, thighs, back of the neck. At first, the knot is dense, but then softens, abscessing of adjacent tissues begins, and fluctuation appears. A yellow-green cream-colored pus is released from the opened node, a necrotic core is not formed. The absence of the rod is explained by the fact that with pseudofurunculosis of the Finger, inflammation of the eccrine sweat glands occurs and the process often recurs. Usually starting from the first month of life, the disease with

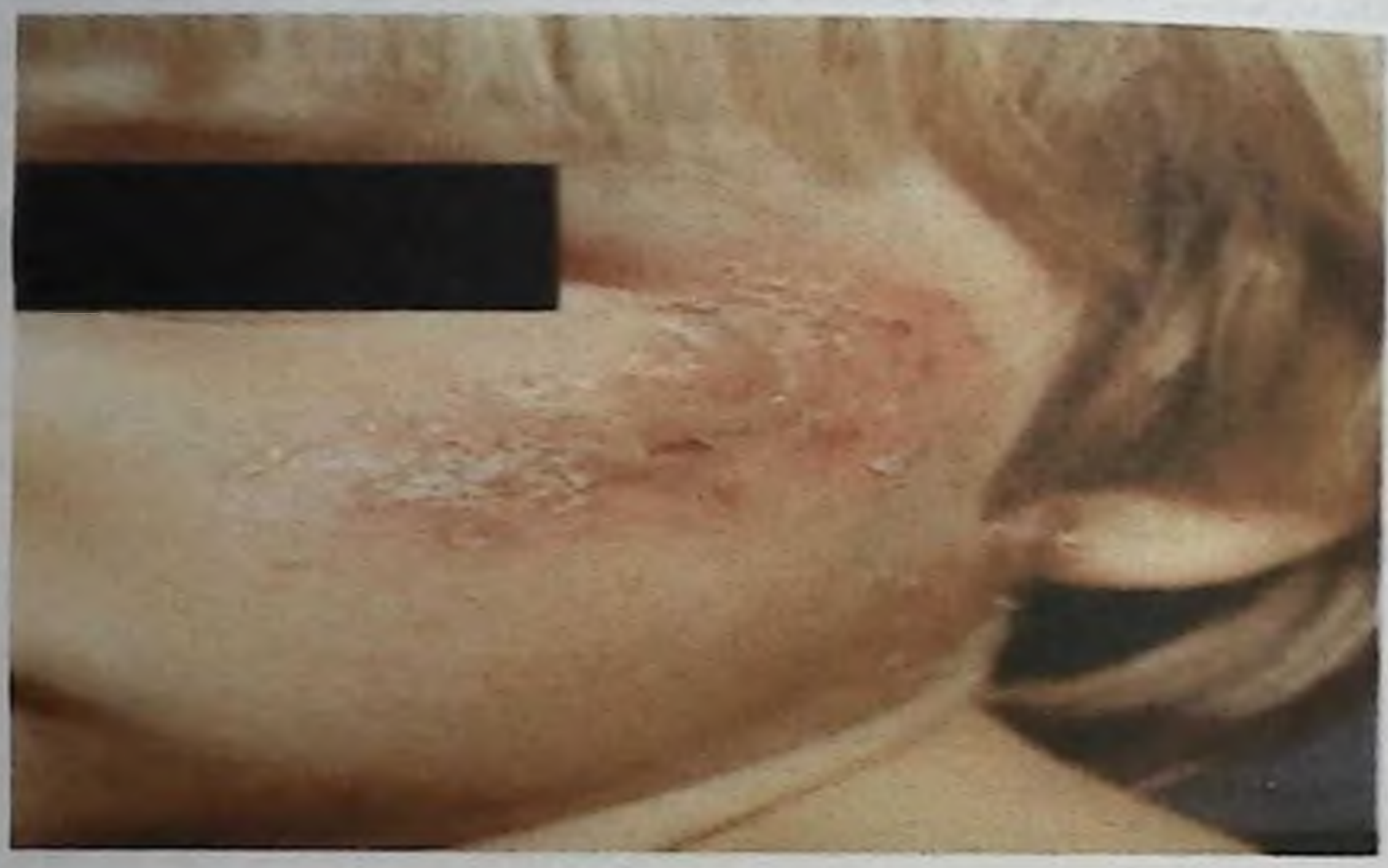


Pic-11 Hidradenitis

irrational treatment can last 2-3 months or longer. Therefore, with pseudofurunculosis on the finger, the general condition of the sick child suffers: the body temperature during subfebrile condition becomes 38-39 ° C, anemia, malnutrition, polyadenitis, hepatosplenomegaly, loss of appetite are observed. General complications: phlegmon, sepsis, pneumonia, purulent meningitis, osteomyelitis. In such cases, the prognosis can be very poor. Most often, proper care of the child and timely treatment exclude serious complications, and the process ends with

recovery. Pseudofurunculosis is differentiated from vesiculopustulosis, furunculosis and scrofuloderma.

Superficial streptoderma The most striking representative of superficial streptoderma is streptococcal impetigo. The primary element of streptococcal impetigo is conflict - a superficial non-follicular bladder with serous-purulent contents, surrounded by a rim of hyperemia. Its tire does not strain and opens quickly. The secretions dry up and turn into yellowish crusts, which falloff on the 4-6th day.



Pic-12 Streptococcal Impetigo

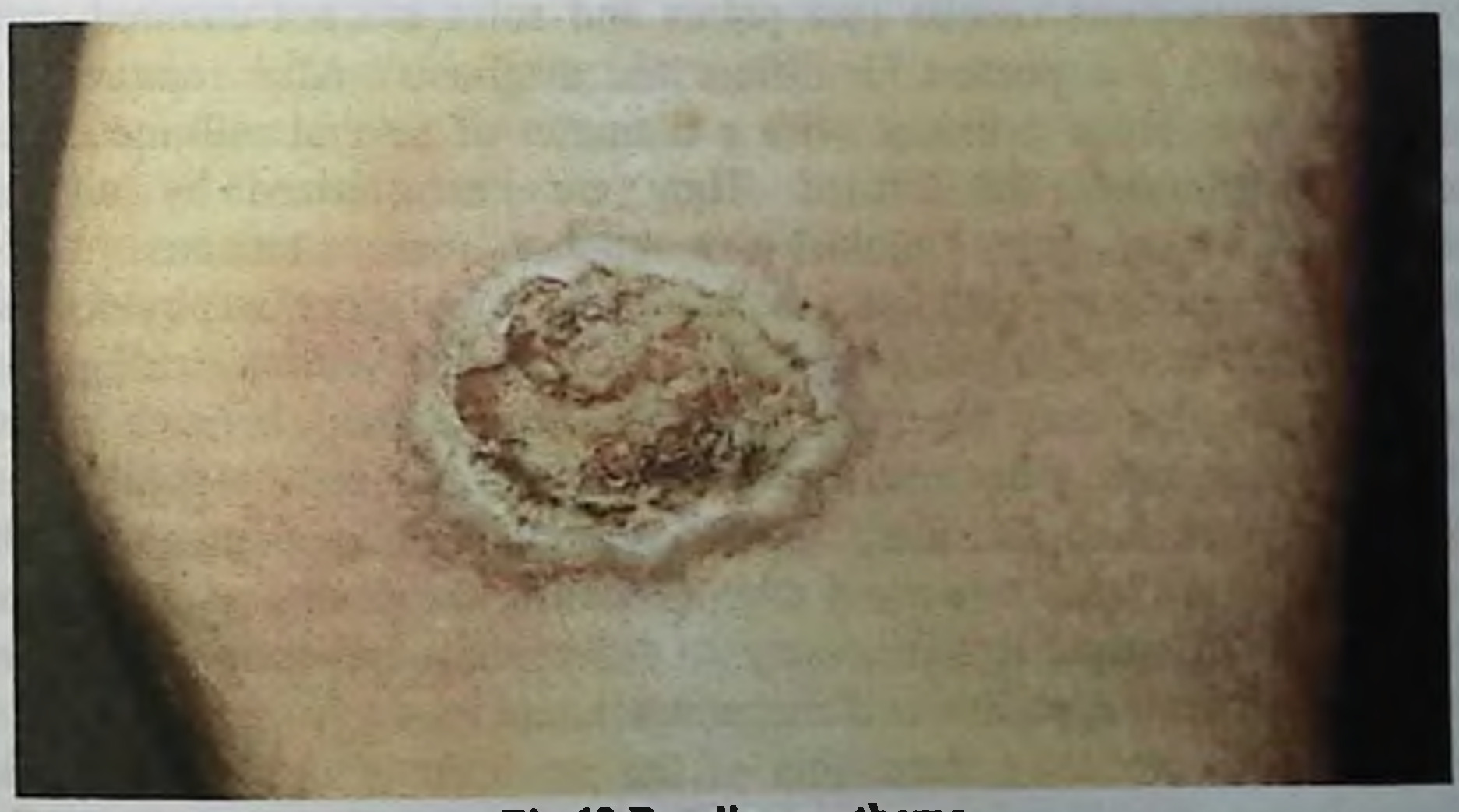
Conflicts are prone to peripheral growth, localized mainly on the skin of the face, trunk, and extremities. Characterized by the phenomenon of autoinoculation. Some patients develop large hemispherical conflicts up to 1-2 cm in diameter, surrounded by a reddish inflammatory corolla. This is the so-called bullous impetigo. It must be differentiated from pemphigus vulgaris, Duhring's dermatitis, bullous toxidermia. In some patients, ring-shaped impetigo is formed, characterized by centrifugal growth, while the skin in the center of the focus begins to epithelize, and small conflicts appear around it.

Children often develop streptococcal stasis, or angular stomatitis, when rapidly opening conflicts appear in the corners of the mouth and shallow painful cracks and erosions are formed, covered with serous-purulent crusts. Diversify with yeast jam. With superficial panaritium, a large conflict appears on the nail phalanges of the fingers, surrounding the nail in a horseshoe shape.

After its opening, erosion is formed with a border of exfoliating epithelium. Over time, the nail plate can become deformed. Differentiate with candidiasis of the nail folds.

Dry, or erythematous-squamous, streptoderma affects children and adolescents and is characterized by the absence of conflicts. Clinically, the disease manifests itself as pinkish-red spots covered with thin whitish scales. The spots are prone to peripheral growth and are localized on the skin of the face, trunk, extensor surfaces of the limbs. Possible itching of the skin in the affected area. Differentiate from vitiligo

Deep Streptoderma Vulgaris ectima is a deep ulcerative streptococcal skin lesion. The disease begins with the appearance of single or multiple conflicts or deep epidermal pustules on the skin of the legs, thighs, torso, less often - arms. Then a brownish-brown crust forms on the surface, surrounded by an infiltrated corolla of a bluish-red color. After removing the crust, a deep ulcer with steep edges and necrotic masses at the bottom is exposed. Healing occurs with the formation of a scar within 2-4 weeks. Ecthyma develops in malnourished people, chronic alcoholics, patients with severe general diseases. Ecthyma vulgaris should be distinguished from syphilitic ecthyma, trophic ulcers, scrofuloderma, and ulcerative induration.



Pic-13 Baseline erythema.

Superficial streptostaphyloderma Impetigo vulgaris is a highly contagious disease and can take on the character of an epidemic in children's groups. On the skin of the face, hands and other open areas, rashes appear at the same time, characteristic of both streptococcal

impetigo and ostiofolliculitis or folliculitis. The elements of the rash are surrounded by a halo of erythema, hyperemia and edema are noted in the affected areas.

Conflicts open early with the formation of rounded or oval erosions bordered by a hyperemic corolla with fragments of the epidermis. Within 1-2 days, erosion is covered with crusts of a grayish or brownish color. After the rejection of the crusts on the 8-9th day, a secondary erythematous spot appears in place of the previous elements. New rashes along the periphery merge with each other and can capture significant skin surfaces. This leads to an increase in lymph nodes, an increase in ESR, and leukocytosis. Patients have an increase in body temperature, malaise, and sometimes pain in the joints.

Impetigo vulgaris is differentiated from pemphigus vulgaris, a vesicular form of erythema multiforme. Epidemic pemphigus of the newborn. This is an epidemic form of pyoderma that occurs in newborns on the 4th-8th day of life. Infection of a child occurs either from the mother or from medical personnel who have a focus of inflammation with pathogenic streptostaphylococci. The disease begins with the appearance on the skin around the navel or abdomen, back, limbs, in the natural folds of multiple blisters with serous-purulent contents (conflicts), surrounded by an erythematous corolla (the palms and soles are not covered with blisters, which is important for differential diagnosis). After rupture of a thin cap of bubbles, erosions with a diameter of several millimeters to several centimeters are formed. They are characterized by uneven scalloped edges, juicy pinkish-red bottom, serous-mucous turbid discharge. Among the pathogens isolated from the bladder, staphylococci, streptococci and other microorganisms are found. With a widespread process, the skin may resemble a second-degree burn. In such cases, children, especially with irrational treatment and reduced immunity, can develop severe complications: sepsis, staphylococcal pneumonia, otitis media, etc. This leads to a sharp deterioration in the general condition of the child, up to death. It is necessary to differentiate epidemic pemphigus of newborns from syphilitic and congenital pemphigus.

Ritter's exfoliative dermatitis of the newborn is one of the most severe forms of epidemic pemphigus of the newborn. The disease usually begins in the first week of a child's life, more often in premature and debilitated children. Microbiological studies have shown that its causative agents are mainly staphylococci of phage group II (phage types 71 or 55/71). It is noted that the earlier Ritter's dermatitis occurs, the more

severe it is. During the course of the disease, 3 stages are distinguished: erythematous, exfoliative and regenerative. Initially, hyperemia appears around the mouth and navel, then either serous edema and epidermolysis occur, or blisters with purulent contents appear on the unchanged skin, which easily burst and form large erosive weeping surfaces. The process spreads rapidly and can cover the entire body within a day. The disease resembles a second-degree burn. With it, the epidermis exfoliates into layers, the Nikolsky symptom is positive (even on seemingly normal skin areas between the blisters). From the first

hours of the illness, the child's condition deteriorates sharply: there is a fever (up to 40-41 ° C), in the blood - hypochromic anemia, left-shifted leukocytosis, increased ESR, early complications - otitis media, pneumonia, abscesses., pyelonephritis, etc. In recent years, abortive forms of exfoliative dermatitis are more common, when lamellar peeling and slight hyperemia of the skin predominate in its clinic; There are no erosions, the general condition of the child is satisfactory.

Ritter's exfoliative dermatitis is differentiated from Lyell's syndrome, SSSS syndrome, Leiner's desquamative erythroderma, and ichthyosiform bullous erythroderma. Staphylococcal scalded skin syndrome, or SSSS (staphylococcal scalded skin syndrome). The causative agent of the disease is staphylococcus phage group II, producing the toxin "exfoliatin A or B", causing detachment of the epidermis directly under the granular layer (F.A. Zverkova, 1994). This syndrome occurs exclusively in children aged 1 month to 5 years. In adults, a more developed immune system allows faster neutralization and elimination of the aforementioned epidermolytic toxin from the body.

The disease is first manifested by the appearance of patchy rashes of a brick-red color on the body (after otitis media, purulent conjunctivitis or catarrh of the upper respiratory tract). At the same time, impetiginous crusts form around the natural openings. Within 1-2 days, the process progresses, as a result of which sluggish large blisters appear (Nikolsky's symptom is positive). Gradually, the entire skin is scalded (burned) and covered with large weeping erosions. The oral mucosa is usually not affected. With a favorable course of the process, by the end of the first week of the disease, epithelialization of erosions begins, resulting in peeling. SSCC syndrome should be differentiated from toxic epidermal necrolysis (Lyell's syndrome). The latter begins acutely with the appearance of blisters and erosions on the skin and mucous membranes. At the same time, the general condition of patients can be very difficult due to

the large loss of fluid, electrolytes, and proteins. The disease can develop at any age and is most often the result of an increased sensitivity of the body to drugs and intolerance to drugs (barbiturates, sedatives, antibiotics, analgesics, sulfonamides, etc.). SSCC is also differentiated from other blistering dermatoses, in particular Leiner's erythroderma desquamative, epidermolysis bullosa congenita, ichthyosis congenita, pemphigus syphilis, and ichthyosiform erythroderma bullosa. Deep streptostaphyloderma Chronic ulcerative pyoderma may be primary or secondary (a complication of impetigo or ecthyma). It develops in patients with malnutrition, diabetes mellitus, chronic venous insufficiency. On the skin of the legs, feet, hands, ulcers of irregular shape are formed, painful on palpation, with covered wounds. the edges; the bottom is covered with granulations and purulent discharge; the skin of the edges of the ulcer and around it is stagnant-bluish. Within a few months, the ulcer tends to peripheral growth, exists for up to a year or more, then, during treatment, necrotic tissue is rejected, the ulcer is filled with granulations and slowly scars. Differentiate with tertiary gummous syphilis, Bazin's indurative erythema, leishmaniasis, sporotrichosis.

Chronic ulcerative vegetative pyoderma differs from chronic ulcerative pyoderma in a more severe and protracted course. The process is localized on the extremities, at first one or more

ulcers are formed at the site of pustules and folliculitis. Separate ulcerated infiltrates can merge, forming an uneven, bumpy surface covered with crusts, after the removal of which ulcers form with papillary-vegetative growths, fistulous tracts and with an abundance of pus in pockets- cones. Next to the primary ulcers, daughter abscesses appear, communicating with the main ulcer through fistulous passages. Subsidiary abscesses can open on their own on the surface of the skin. After the expiration of pus, the fistula may close, but may reopen with the accumulation of pus. The healing of ulcers is accompanied by the formation of scars. The disease is differentiated from collicative tuberculosis, gummous and serpiginating tuberculous syphilis, actinomycosis, blastomycosis.

Tests for knowledge control

1. Which of the following diseases is not caused by staphylococci?
a) superficial folliculitis; b) furuncle; c) vulgar ecthyma; d) hydradenitis; e) vulgar sycosis.

2. Streptococcal impetigo is characterized by everything except: a) conflicts; b) inflammatory corolla; c) high contagiousness; d) thin crusts; e) deep pustules.

3. Which of the following diseases are not typical for young children? a) folliculitis; b) pseudofurunculosis; c) hydradenitis; c) epidemic pemphigus of newborns; d) Ritter's exfoliative dermatitis.

4. With hydradenitis, all of the above are prescribed, except for: a) antibiotics; b) immunomodulators; c) aniline dyes; c) UHF-therapy; e) poultices.

5. Vulgar impetigo is characterized by all of the following, except for: a) conflicts; b) high contagiousness; c) deep folliculitis; d) wet erosive surfaces; e) thick brown-yellow crusts.

6. What pustular skin diseases do not require systemic antibiotic therapy? a) ostiofolliculitis; d) furunculosis; b) carbuncle; e) hydradenitis; c) sycosis vulgaris.

7. In the treatment of pyoderma vulgaris, all except: a) staphylococcal toxoid; d) antibiotics; b) immunofan; e) cytostatics; c) complexes of vitamins and microelements.

Operation standards: 1V; 2G ; _ 3B; 4D ; _ 5V; 6A; 7D.

CHAPTER 3 ALLERGODERMATOSES. DERMATITIS. TOXICODERMA. NORMAL AND ALLERGIC CONTACT DERMATITIS. TOXICODERMA, SULFANILAMIDE ERYTHEMA. MULTIFORM EXUDATIVE ERYTHEMA.

The term "dermatitis" in dermatology refers to acute inflammatory processes in the skin caused by certain exogenous (chemical, physical, mechanical) influences. For dermatitis, a characteristic feature is the subsidence and resolution of the disease process with the cessation of the pathogen.

Dermatitis as a contact inflammatory reaction occurs only when obligate and facultative factors are directly exposed to the skin. The clinical picture of dermatitis depends on the nature of the etiological factor, its strength and individual characteristics of the body and skin, in particular. According to the mechanism of development, dermatitis is divided into simple (artificial) and allergic. Simple, or artificial, dermatitis occurs upon contact with the primary irritant (acids and alkalis, high and low temperatures, radiation exposure, mechanical factors, etc.), which leads to the development of skin inflammation even with a single exposure. For the occurrence of simple dermatitis, the presence or absence of hypersensitivity of the skin to this irritant does not matter. This group also includes occupational dermatitis (from exposure to strong alkalis, acids, etc.). The course of simple dermatitis is affected by the duration of contact and the concentration of the substance. The clinic of simple dermatitis is characterized by intense inflammation, often with the formation of blisters, sometimes necrosis.

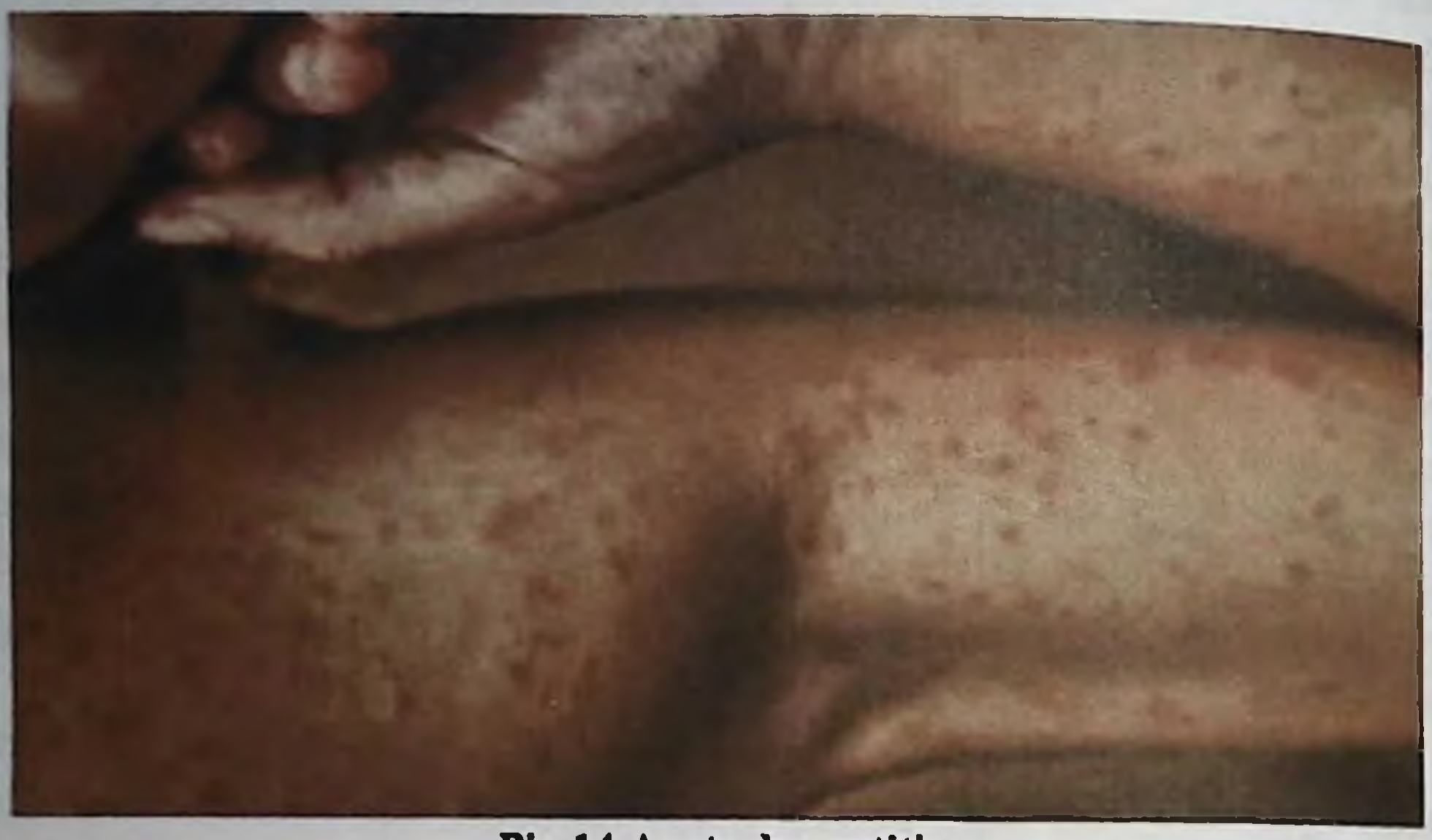
Daundermatitis is clinically manifested by severe hyperemia and swelling of the skin, there may be vesicular and papular rashes, weeping, scales and crusts. Sometimes blisters and large blisters appear as a result of physical and chemical factors. They are characterized by clear boundaries, since the process develops precisely at the point of contact with the irritating factor. As a rule, rashes are accompanied by subjective sensations (heat, burning, itching, sometimes pain). The process is resolved by peeling, sometimes coarsely lamellar, with pigmentation. The incubation period is determined by the active agent, but more often it is absent and is not accompanied by a violation of the general condition. The exception is burns and frostbite of a large area

and depth. Mechanical causes that can cause dermatitis include pressure and friction. One of the most common types of mechanical acute dermatitis is abrasion that usually occurs on the palms of the hands of people engaged in physical labor, and on the feet when walking in uncomfortable shoes. It is clinically characterized by sharply limited edematous hyperemia, against which, with prolonged exposure to an irritating factor, large blisters appear. Scuff marks can also occur in folds due to friction between surfaces, especially in obese people. After opening the blisters, continuing to injure them, erosions are formed that correspond in size to the size of the injured area. Subjectively, patients experience pain and burning. With chronic pressure and friction, the strength of which is relatively small, the affected areas of the skin are compacted, lichenified, which occurs due to skin infiltration, thickening of the epidermis and hyperkeratosis.

Callosity is a chronic form of mechanical dermatitis resulting from prolonged and constant pressure and friction on the hands when performing manual operations, and on the feet when wearing tight shoes. One form of dermatitis in children is called intertrigo, or diaper rash. It occurs when the skin is irritated by the folds of the bedspread, clothing and is manifested by hyperemia, maceration of the epidermis, sometimes wetting (with the appearance of erosion), burning and pain. Chemical causes that cause simple contact dermatitis include strong acids and alkalis, alkali metal and mineral acid salts, skin-acting chemical warfare agents, and many others. With the development of the chemical industry, the number of chemicals that can cause dermatitis is constantly increasing. Strong solutions of these chemicals are obligate irritants and cause dermatitis in anyone.

Acute dermatitis caused by acids and alkalis develops according to the type of chemical burn: erytomatous, vesiculobullous, necrotic. Their weak solutions with prolonged exposure provoke chronic dermatitis in the form of infiltration and lichenification of varying severity. Exposure of the skin to high temperatures leads to the formation of burns (combustio). Burns can be of four degrees. With a first- degree burn, erythema and slight swelling are formed on the affected area of the skin (subjectively - burning and soreness). With a second- degree burn, blisters appear against the background of hyperemia and edema. A third-

degree burn is characterized by necrosis of the superficial layers of the dermis without the formation of a scab.



Pic-14 Acute dermatitis

With a IV degree burn, necrosis of all layers of the dermis occurs with the formation of a scab, the rejection of which reveals an ulcer. The prognosis depends not only on the degree of the burn, the general condition of the patient's body, the addition of a secondary infection, but also especially on the size of the burn area. Exposure to low external temperature leads to tissue damage from cold and is called frostbite (congelatio), which comes in four degrees. With frostbite I degree, the affected area is stagnant-cyanotic, subjectively - a sensation of tingling and itching. With frostbite II degree, the clinical picture is similar to the previous one, but blisters appear on the affected areas of the skin, filled with serous or serous-hemorrhagic contents. With frostbite of the 3rd degree, necrosis of the affected areas occurs with the formation of a scab (subjectively severe pain). With frostbite IV degree, deep tissue necrosis is noted (up to the bones). The occurrence of frostbite is preceded by a latent period, which is characterized by cooling, pallor and insensitivity of the affected area. Most often, frostbite occurs on open and distal parts of the body (fingers and toes, skin of the nose and cheeks, ears).

Frostbite is caused by high humidity, wind, tight shoes, a decrease in the resistance of individual tissues (frostbite suffered in the past, increased sweating, injuries, etc.) and the body as a whole (physical

overwork, stressful conditions, beriberi, weakening of the body after acute or chronic infectious diseases, large blood loss, alcohol consumption Such conditions of the body can lead to frostbite even at a positive temperature (5-8 ° C). Under the influence of prolonged cold in combination with dampness in asthenics, with hypovitaminosis C and A, a peculiar skin lesion occurs - chills (pernions). Increased sensitivity to cold in these individuals leads to frequent relapses of the disease, especially in autumn, and remission occurs in summer.The disease is due to a tendency to acrocyanosis of the limbs, work and living in damp, insufficiently heated rooms. -red in the area of the terminal and middle phalanges of the fingers and joints, or pale red with a bluish tint on the cheeks. When heated, the sensation of itching and burning in the lesions increases, pain appears on palpation. Exposure to the sun on the skin can lead to the formation of acute or chronic solar dermatitis (solar dermatitis). From the entire spectrum of sunlight, the appearance of dermatitis is mainly due to short-wave ultraviolet rays.

Individuals not accustomed to long-term treatment in the sun, those with soft, slightly pigmented skin (more often blondes) may develop acute solar dermatitis, which is manifested by redness, flow, sometimes blisters and vesicles, which occur several hours after exposure. The process is accompanied by burning and pain. General symptoms (headache, vomiting, fever) occur when large areas of the skin are affected. After a few days, the disease ends with peeling and pigmentation. Fishermen, sailors and persons working under the influence of sunlight (in the field, at construction sites, etc.), i.e. having a profession associated with prolonged exposure to sunlight, may develop chronic solar dermatitis, which is manifested by infiltration, pigmentation and dry skin. Various types of ionizing radiation (X-rays, alpha, beta and gamma rays, neutron radiation) can lead to acute or chronic radiation dermatitis. The severity of radiation dermatitis is determined by the dose and penetrating power of radiation, the size of the radiation area, and individual sensitivity.

Depending on the above factors, acute radiation dermatitis may occur. It can be manifested by erythema (with a peculiar purple or blue tint), temporary hair loss, bullous reaction against the background of intense hyperemia and edema (in these cases, the process ends with skin atrophy, persistent alopecia, the appearance of telangiectasias and

pigmentation disorders - “variegated”, “X-ray "skin") and a necrotic reaction, the manifestations of which are the same as in bullous dermatitis, but more pronounced (with tissue necrosis) and is accompanied by severe general phenomena. Repeated exposure of the skin to "soft" X-rays in relatively small doses and exposure to radioactive substances leads to the development of chronic radiation dermatitis. The latter can also be a consequence of acute radiation dermatitis. The process is characterized by poikiloderma (variegation) of the skin in the affected areas, dryness, thinning, loss of elasticity, the presence of telangiectasias, hyperpigmented and depigmented areas, as well as onychodystrophy, itching. Chronic radiation damage to the skin contributes to the formation of papillomas, hyperkeratosis, warty growths in the affected areas, which can malignantly

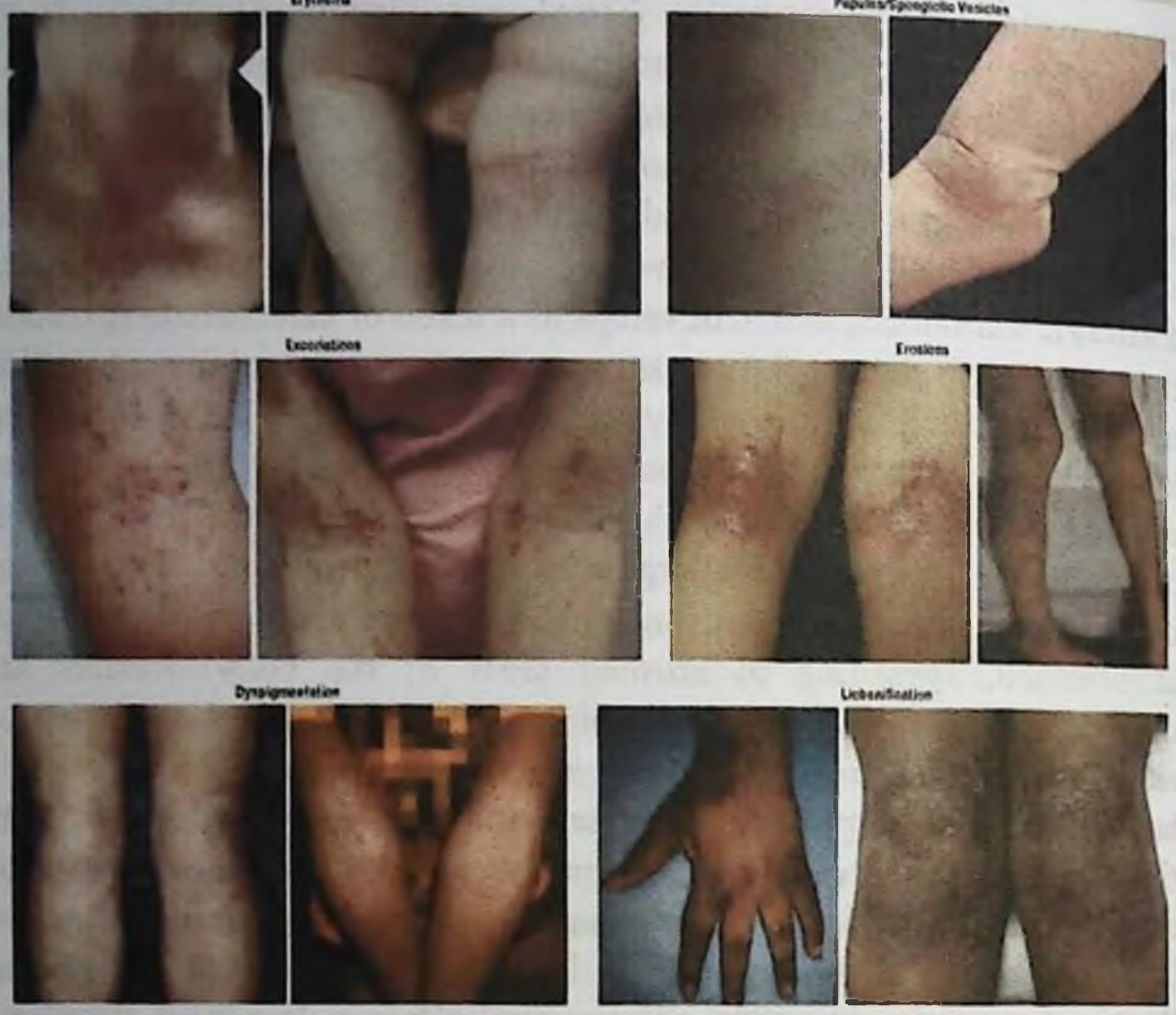
degenerate. In these places, the so-called late radiation dermatitis, including late radiation trophic ulcers and radiation cancer, can develop.

Pathomimia - self-injury of the skin - is also considered artificial dermatitis. Usually they damage the skin of the face with mental disorders or in pursuit of a specific goal. Skin rashes occur as a result of the use of various chemicals or mechanical stress. Common signs are irregular or elongated blisters caused by thermal burns or caused by burning the skin with certain chemicals. A characteristic cause of the disease is the location of the elements of the rash on areas of the skin that are accessible to irritate the patient himself. In the treatment of simple dermatitis, local remedies are most often used. It is important to eliminate the irritant. For chemical burns caused by concentrated acids and alkalis, washing them with plenty of water is an urgent remedy. With erythema with edema, lotions (2% solution of boric acid, lead water, etc.) and corticosteroid ointments (betamethasone ointment, hydrocortisone ointment) are shown, with the vesiculobullous stage, blisters are treated with the preservation of their tires and impregnation of solutions with a disinfectant (methylene blue, gentian violet). etc.) and application of epithelizing and disinfecting ointments (dermatol 2-5%, pantestin, etc.). Treatment of patients with necrotic skin manifestations is carried out in a hospital.

Allergic dermatitis occurs due to repeated contact with chemical allergen agents (turpentine and its components, dinitrochlorobenzene, synthetic adhesives, varnishes, paints, chromium, cobalt, nickel salts,

epoxy resins, etc.), cosmetics (Ursol, paraphenylenediamine), drugs (chlorpromazine, mercury compounds, semi-synthetic antibiotics), insecticides, plants (primrose, sedge, angelica, parsnip, etc.). Allergic dermatitis occurs in response to the contact effect on the skin of an optional irritant to which the body is sensitized and in relation to which the irritant is an allergen (monovalent sensitization). In the pathogenesis of allergic dermatitis, which develops as a result of external allergization (contact allergic dermatitis), cross- sensitization is also observed. In this case, skin sensitization caused by one agent is accompanied by increased skin sensitivity to other substances. Inflammatory changes upon repeated contact with allergens develop some time after the first contact. Clinical manifestations of allergic dermatitis go beyond the zone of action of the pathogen and are not limited to the place of contact with the allergen, spreading to crushed areas of the body. Rashes are polymorphic, vesiculation and weeping are possible. The boundaries of the foci are less sharp than with articial dermatitis. The disease is characterized by a protracted course with relapses with new contacts with the allergen. Recurrent allergic dermatitis often transforms into eczema. Most medical, occupational, and photodermatitis can be allergic. Certain difficulties arise in the diagnosis of occupational allergic dermatitis. At work, occupational allergic dermatitis develops only in individuals with features of the body's reactivity when exposed to the same allergen. In its manifestations, occupational dermatitis resembles true eczema: edema, hyperemia, papular and vesicular rashes, prone to grouping. Contact allergic dermatitis differs from acute eczema in the following ways: the onset of the disease in areas of the skin exposed to the allergen; the boundaries of the lesion are clear and correspond to the site of action of the allergen; resolution occurs after the allergen ceases to act; in contrast to the severe itching characteristic of eczema, dermatitis is accompanied by a feeling of heat, burning, sometimes pain and rarely itching; relapses are possible only in cases of contact

with the allergen. Dermatitis often occurs on the face from paints and cosmetics. 2-3 hours after dyeing the hair on the skin of the face, especially the eyelids, swelling, erythematous rashes, severe itching, vesicular rashes appear. Often there are dermatitis from lipstick, which are manifested by mild hyperemia, dryness, cracks, with subjective



Pic-15 Allergic dermatitis

sensations in the form of a burning sensation, a feeling of tightness, and slight itching. With further use, cheilitis becomes chronic. In spring and summer, cheilitis may worsen due to photosensitizing properties of some lipsticks. Dermatitis from mascara is much less common.

Dermatitis can occur when using theatrical makeup. Currently, allergic contact dermatitis is often observed from any perfume and cosmetic products. The most common cause of such dermatitis are preservatives. Dermatitis from cologne is localized in open areas of the body, including the face, and manifests itself in the form of sharply defined dark brown (mainly located in the form of stripes) erythematous, pigmented spots of various sizes. Dermatitis can develop in people who wear dyed fur. In these cases, skin lesions are caused by ursol derivatives, which are not sufficiently washed out when washing the fur. Most often, dermatitis manifests itself in rainy weather, when the paint comes off wet wool.

Dermatitis begins 5-6 weeks after the start of wearing fur, localized mainly on the neck and lower face. The most common cause of drug-induced allergic dermatitis are antibiotics, sulfonamides, chlorpromazine, novocaine, quinoline derivatives, arsenic preparations, barbiturates, antipyrine, mercury preparations. Contact dermatitis from antibiotics is mainly located on the face, neck, hands; they develop in nurses, doctors, especially dentists, pharmacists; have eczema-like manifestations. The clinical picture of dermatitis from novocaine resembles eczema with localization on the face and neck; it is common in dentists, surgeons, and nurses. In some patients, novocaine dermatitis turns into eczema. Dermatitis that occurs when exposed to mercury can be contact and toxic by the type of toxidermia (develops with any method of introducing mercury into the body). Rashes are diverse: cortical and rubella-like, scarlet-like, urticaria, according to the type of exudative erythema, from papulo-vesicular to severe exfoliativedermatitis and erythroderma.

Contact dermatitis develops when using ointments and creams with mercury in dermatology and cosmetology. Manifestations range from mild to eczema and widespread erythroderma. Allergic dermatitis can be caused by the external use of anesthesin, carbolic acid, formalin, sulfur preparations, aniline dyes, rivanol, undecylenic acid, boric alcohol, lanolin, petroleum jelly, corticosteroids, etc. Among allergic dermatitis caused by the action of plants, dermatitis caused by primrose and bluegrass. Dermatitis, as a rule, occurs in places of skin contact with primrose, mainly on the hands, and when carrying pollen with fingers, on the face, neck and other areas. It proceeds in the form of edematous erythema, eczematous and bullous rashes. There are messages available o contact dermatitis, cheilitis and stomatitis resulting from sensitization to bay leaf in foods.

There are cases of allergic contact dermatitis around the mouth in children who were fed

spinach, carrots, tomato or orange juice. Sometimes contact dermatitis occurs as a result of the action of vanilla, cinnamon and similar spices. The clinical picture of these dermatitis is also eczema-like with itching of varying intensity. The diagnosis of allergic dermatitis is based on the history and clinical picture. Often, to confirm the diagnosis, skin-allergic tests are used if an allergen is suspected (compress, dropper, scarification), which are mandatory for identifying an industrial allergen in the development of occupational allergic dermatitis. The test is carried out after the elimination of clinical changes in the skin. Differential diagnosis

is carried out with eczema, which is characterized by polyvalent (rather than monovalent) sensitization and a chronic relapsing course, as well as with toxidermia, in which the allergen enters the body.

Care. First of all, eliminate the effect of the irritant. Patients are prescribed a diet with the exclusion of irritants, limiting the amount of salt, carbohydrates. Prescribe antihistamines (tavegil, suprastin, diphenhydramine, histafen), hyposensitizing drugs (30% sodium thiosulfate solution, calcium gluconate), in severe cases - systemic corticosteroid hormones (dexamethasone, testosterone, diprospan). To relieve inflammation, powders, lotions from a 2% solution of boric acid, oil suspensions with zinc, sulfur, salicylic acid, xeroform, oil-in-water emulsions, andcorticosteroid creams are used topically.

Urticaria is a toxic-allergic disease, manifested by the sudden appearance of blisters on various parts of the skin, less often on the mucous membranes. The appearance of rashes is accompanied by significant itching. The disease can have an acute and chronic course, manifest itself in the form of acute outbreaks lasting from several hours to several days. There may be cases in a few weeks, months or even years. Etiology and pathogenesis. The development of the disease is based on the hypersensitivity reaction of immediate and delayed action, due to the high content of biologically active substances in the blood serum, which have a toxic effect on the walls of blood vessels, increasing their permeability.

An important role in the pathogenesis of urticaria is played by the mediator histamine, which is usually bound to intracellular tissue proteins in mast cells and basophils. The reaction between the antigen and antibodies fixed on mast cells leads to the activation of proteases (trypsin), under the influence of which histamine is released, as well as serotonin, acetylcholine and bradykinin (slow acting substance) are released, which potentiate the action of histamine. A certain role in the development of urticaria is played by functional disorders of the autonomic nervous system with its regulatory center - the hypothalamus, in which, due to the presence of cholinergic and adrenergic centers, nerve impulses are switched to humoral ones. Urticaria is caused by a variety of endogenous and exogenous factors. It often occurs as an acute exacerbation due to hypersensitivity to food (eggs, milk, mushrooms, strawberries, etc.), drugs (aspirin, antibiotics, therapeutic sera, etc.), cold, heat, sunlight.

Urticarial rashes can appear after insect bites (bugs, fleas, mosquitoes, etc.), touching some plants (nettle), can occur as a manifestation of toxicosis in pregnant women, with diseases of the

hematopoietic organs, malignant neoplasms, etc. Chronic forms of urticaria are usually

associated with dysfunction of the gastrointestinal tract, helminthic invasions, giardiasis of the biliary tract, the presence of focal infection. There are such types of urticaria: acute (artificial, symptomatic), including acute limited angioedema; chronic recurrent, chronic persistent papular, solar. psychogenic.

Clinic. Urticaria is clinically characterized by the sudden appearance on the skin, less often mucous membranes, of a monomorphic, severely itchy urticaria rash, i.e., the primary morphological element in urticaria is a blister. The bubble is a limited, dense, cavityless formation, rising above the level of the skin, round, oval or irregular in shape, pink or white, ranging in size from a lentil to the size of a palm or more, with a flat, smooth surface. The development of a blister is based on a limited swelling of the papillary dermis, which is formed as a result of an acute expansion of the capillaries and an increase in the permeability of their walls. A sharp increase in edema can lead to compression of blood vessels, which explains the appearance of white blisters (porcelain urticaria). Sometimes blisters and blisters develop on the surface of the blisters (papulo-vesicular urticaria). Often, the appearance of rashes is accompanied by headache, fever, joint pain, chills. Acute urticaria (up to 30 days) is characterized by the appearance of severely itchy urticaria, often accompanied by a violation of the general condition, fever, gastrointestinal disorders, and neurotic conditions. Eosinophilia is found in the peripheral blood. With abundant rashes, blisters can merge, forming extensive foci with polycyclic outlines. In some cases, blisters occur on the mucous membranes of the lips, tongue, soft palate, and larynx. In some individuals with hypersensitivity, mechanical irritation of the skin leads to the formation of blisters (for example, when a spatula is passed over the skin, a bright red urticarial edematous strip appears after a few minutes, which is not accompanied by itching, an artificial urticaria occurs). A special form is giant urticaria, acute limited angioedema, in which suddenly there is a sharply protruding limited swelling of the skin and subcutaneous fat, more often on the face or in the genital area of white or pink color, densely elastic consistency. The appearance of edema is accompanied by burning, itching. After 1-2 days, the swelling disappears without a trace. Angioedema may be combined with the usual urticaria. With laryngeal edema, stenosis and asphyxia may occur, requiring urgent intervention (tracheostomy).

Pathogenesis. Immediate allergic reactions: the interaction of the antigen with IgE antibodies fixed on the surface of basophils and mast cells leads to the activation of these cells and the release of histamine, leukotrienes and prostaglandins. These substances increase the permeability of venules and cause the release of biologically active substances from other cells. In sensitized patients, even a small dose of the drug can cause a severe allergic reaction. reaction (anaphylactic shock), especially when administered parenterally. Immune complex allergic reactions: immune complexes activate complement with the formation of C3a and C5a fragments, which cause degranulation of mast cells. Most often, there is a lesion of the face, a significant increase in the eyelids, lips, tongue, genitals. Swelling of the mucous membrane of the larynx can cause airway obstruction. Classification and mechanisms of development Immune: - allergic reactions of immediate type - - immunocomplex allergic reactions. Non-immune (pseudo-allergic

or anaphylactoid reactions): - NSAIDs - - X-ray contrast agents - - ACE inhibitors; - calcium antagonists - preparations containing histamine. Drugs that cause angioedema: - antibiotics and other antimicrobial agents (penicillins, cephalosporins, drugs containing a group of sulfonamides); - antiarrhythmic drugs (amiodarone, procainamide); - immunomodulators, vaccines (antilymphocyte immunoglobulin, levamisole); - cytostatics; - ACE inhibitors (captopril, enalapril); - calcium antagonists (nifedepine, verapamil) - drugs that release histamine. Chronic recurrent urticaria is a chronic disease with intermittent symptoms. relapses and remissions. Characteristic signs are a long course, the appearance of less and less edematous urticarial elements, intense itching, insomnia, irritability, neurotic disorders are possible. Patients develop extensive excoriations, eczema, pustules, and skin pigmentation. Usually develops against the background of focal infection, disorders of the gastrointestinal tract, liver. Chronic persistent papular urticaria is more common in women. A cellular infiltrate joins the edema. On the extensor surfaces of the limbs, the trunk appear dense flat, reddish papulo-urticarial rash, accompanied by itching. Solar urticaria develops with liver diseases and disorders of porphyrin metabolism. In these cases, there is an increased sensitivity of the skin to ultraviolet rays. In open areas, after exposure to the sun, an urticarial rash appears. Cold urticaria occurs when the skin is exposed to cold, located in places of contact with cold.

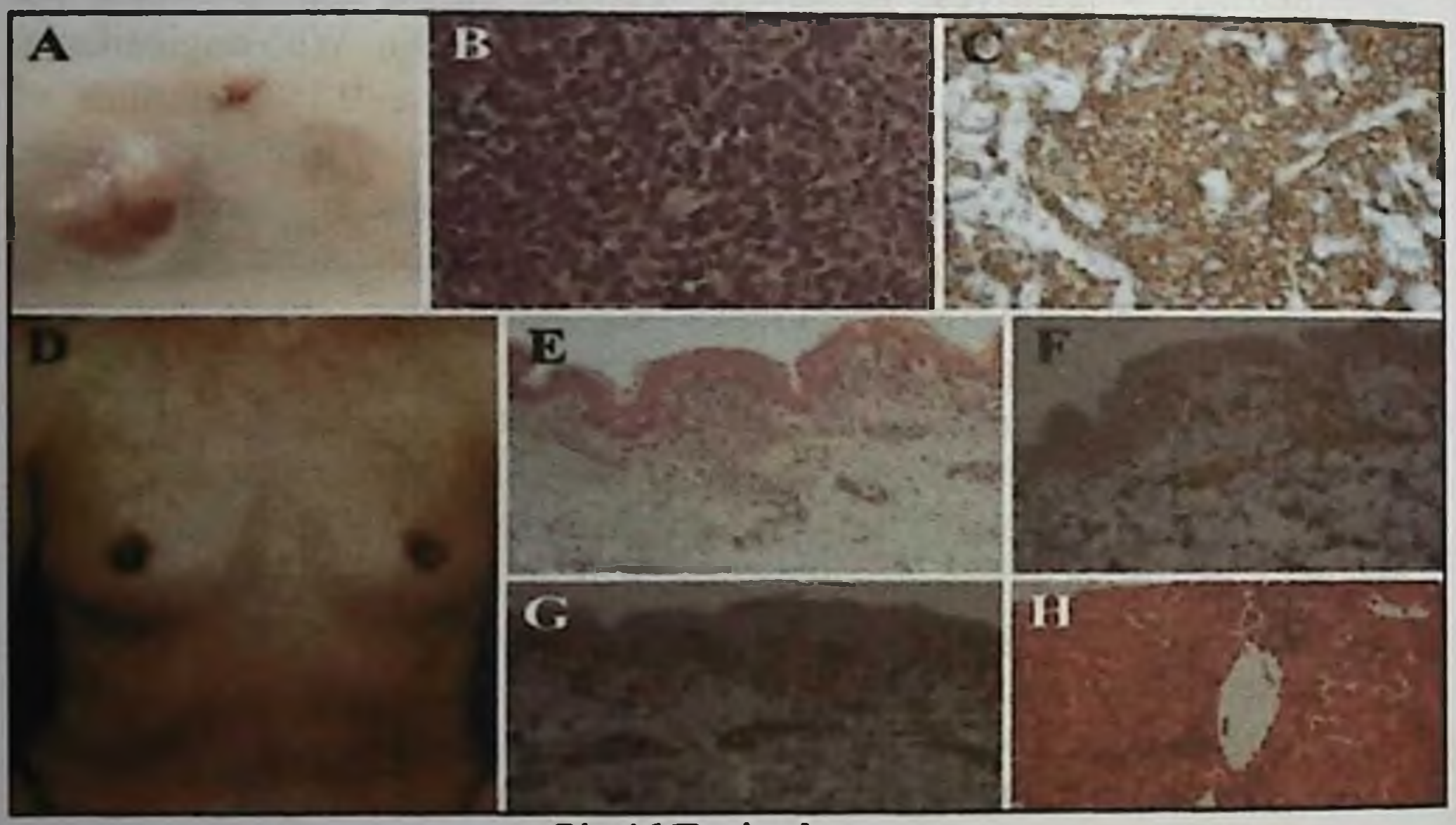
Differential diagnosis is carried out with diseases in which urticarial rashes are one of the symptoms: - grain scabies; - toxicoderma; - Duhring's dermatitis herpetiformis.

Treatment of urticaria depends on the etiology and pathogenesis. Identified pathology of internal organs, nervous and endocrine systems is subject to treatment; existing foci of infection are sanitized. For all types of urticaria, the following is used: - a strict diet with the exception of allergenic and extractive products; - antihistamines (diphenhydramine, suprastin, fenistil, loratadine, tavegil, edem, etc.); - hyposensitizing drugs (10% calcium gluconate solution, 30% sodium thiosulfate solution) - detoxification drugs (polyvidone, sodium chloride, potassium chloride, magnesium chloride, sodium bicarbonate); - enterosorbents (enterosgel, multisorb, atoxil); - sedatives (persen, alora, etc.); - diuretics (lasix, veroshpiron); - laxatives. In severe resistant cases, the following are prescribed: - glucocorticosteroid drugs - - histoglobulin. With angioedema, the following are prescribed: - 0.3-0.5 ml of a 0.1% solution of epinephrine subcutaneously - glucocorticosteroids (dexamethasone, diprospan) - diuretics (furosemide) - antihistamines (suprastin, tavegil). In chronic urticaria, histoglobulin is prescribed subcutaneously. With psychogenic urticaria, antihistamines are ineffective, since the main mediator is acetylcholine, so anticholinergics are prescribed. External treatment of urticaria is symptomatic. Applied means that eliminate itching: - 1% solution of menthol - - shaking suspension with diphenhydramine, diphenhydramine cream, psilobalm - - ointments and creams containing glucocorticosteroids. It is important to constantly take care of the skin, using indifferent creams or emulsions (Lipikar Balm AR, Excipial M Lipolozion, Excipial M Hydrolosion, etc.).

TOXICODERM

Toxidermia is an acute inflammation of the skin that develops as a result of the action of an irritant that enters the body through the respiratory tract, gastrointestinal tract, by subcutaneous, intramuscular and intravenous injections. Mostly drug toxidermia is observed. There are predisposing factors: exogenous and endogenous. Exogenous factors include drugs (antibiotics, sulfonamides, iodine, vitamins B1, B6, B12, etc.), food products, chemicals at work and at home. en- _ common factors - functional disorders of the gastrointestinal tract, liver, kidneys, etc. (lead to autointoxication). Clinic. Characterized by the acute appearance of limited or widespread, usually symmetrical, monomorphic pruritic rashes. Some drugs cause a characteristic clinical picture of the disease: iodine-,

bromine-acne; sulfa drugs - erythematous, red-cyanotic foci of a rounded shape; fixed erythema. Common symptoms may include insomnia, irritability, fever, malaise and arthralgia, a hemorrhagic component. Subjectively noted itching, burning, soreness. Internal organs and the cardiovascular system may be affected. According to the clinical picture and the nature of the rashes, a number of clinical varieties of toxidermia are distinguished. Spotted toxidermia. There are itchy spots (erythematous, hemorrhagic and pigmented), which are more often located in isolation, but often merge, capturing large areas of the skin. Small-lamellar or large-lamellar peeling may occur on the surface of the lesions. Toxic melasma (wartime melanosis). The result of the toxic of effect of oil and coal hydrocarbons that enter the body through the lungs. It also occurs against the background



Pic-16 Toxicoderm

hypovitaminosis of vitamin C. On the skin of the face, neck, upper limbs, less often on the trunk and lower limbs, scaly erythematous rashes first appear. Later, they develop reticular pigmentation, follicular keratosis, telangiectasias, and skin atrophy. Perhaps a violation of the general condition (general weakness, headache, loss of appetite). Papular toxidermia. The appearance on the extensor surfaces of common, islet-inflammatory, hemispherical or polygonal papules on the extremities is characteristic. More often they are observed after taking tetracycline, hingamin, iodine preparations and other medicines. Nodular toxidermia.

There are painful inflammatory nodes that rise above the surface of the skin. Vesicular toxidermia. There are small or large bubbles that are disseminated, surrounded by a rim of hyperemia. Pustular toxidermia. There are pustular rashes or inflammatory papules, in the central part of which pustules are determined. Observed after taking halogen-containing drugs (iodine, bromine, fluorine, etc.), as well as vitamins B6 and B12, glucocorticoid hormones. Bullous toxidermia - common or limited bullous rashes, along the periphery of which there is a border of hyperemia. They are noted after taking iodine preparations. Fixed toxiderms. Predominantly on the mucous membrane of the oral cavity or in the genital area, one or more large erythematous spots with a bluish tinge in the center are formed, on the surface of which blisters may appear, subsequently eroding. With repeated administration of the corresponding drug, a relapse occurs on the same area of \u200b\u200bthe skin. Fixed toxidermia can occur after taking sulfa drugs, antibiotics, salicylates, antihistamines and other drugs. Care. It is necessary to eliminate the causes of the disease. Recommended antihistamines (suprastin, tavegil, diphenhydramine) and hyposensitizing drugs (calcium gluconate, sodium thiosulfate), diuretics, vitamins C and B, rutin. Depending on the clinical picture of the disease, powders, water-borne suspensions, glucocorticosteroid creams or ointments are prescribed.

1. Atopic dermatitis corresponds to the ICD-10 class.1) Ф00-Ф99;
2) G00-G99;
3) L00-L99;
4) Q 00- Q 99.

2. In the pediatric age group with atopic dermatitis, the inflammatory process
1) latent character;
2) acute character;
3) subacute character;
4) chronic.

3. In the infant group, inflammation of the skin is more common.
1) latent character;
2) acute character;
3) subacute character;
4) chronic.

4. In infancy, the form of atopic dermatitis predominates.
1) lichenoid;
2) spring-like;
3) squamous;

4) exudative.

5. Erythematous-squamous form of atopic dermatitis is characterized by age periods

1) children;
2) baby;
3) adolescence;
4) senile.

6. If atopic dermatitis is complicated by a bacterial infection, there is

1) herpetic infection;
2) candidiasis of the scalp;
3) pyoderma;
4) Kaposi's sarcoma.

CHAPTER 4 ECZEMA. NEURODERMATOSIS. MICROBIAL ECZEMA, SEBORRHEIC, PROFESSIONAL AND CHILDREN'S ECZEMA. ATOPIC DERMATITIS. NEURODERMATITIS - LIMITED AND WIDESPREAD CLINICAL FORMS, ITCHING, STROPHULOSIS.

Contact allergic cheilitis is diagnosed mainly in women over the age of 20 years. The etiological factors of allergic contact cheilitis are chemicals that are part of lipstick, plastic dentures, toothpastes, etc. Cheilitis can occur upon contact with metal objects (wind instrument mouthpieces, pens, pencils, and other objects). Occupational contact allergic cheilitis may develop. Pathological changes develop when the body is predisposed to allergic reactions and sensitization to various chemicals. Clinic. Patients complain of severe itching, burning, swelling and redness of the lips. In the anamnesis, contact with various irritating factors and exacerbation of the disease with repeated exposure are noted. On examination, reddening of the red border of the lip is detected, rarely spreading to the skin and mucous membranes. With severe inflammation, small bubbles appear, after opening of which erosion and cracks form. In other cases, the described subjective sensations are accompanied by peeling without a pronounced inflammatory reaction.

Care. In the treatment of contact allergic cheilitis, the establishment and elimination of the irritant is of primary importance. Apply local ointments containing corticosteroids with antiallergic, antipruritic and anti-inflammatory effects (florin, prednisolone, fluorocort and other ointments). Additionally, hyposensitizing therapy is carried out, suprastin, diphenhydramine, fencorol, claritin or other drugs are prescribed orally. actinic cheilitis actinica) is a chronic disease caused by increased sensitivity of the red border of the lips to sunlight. The main causes of this cheilitis are delayed-type allergic reactions to ultraviolet rays, i.e. actinic cheilitis develops in people with sensitization of the red border of the lips to solar radiation. A number of researchers consider the dry form of actinic cheilitis to be an optional precancer. Clinic. There are exudative and xerotic (dry) forms of actinic cheilitis. The exudative form of actinic cheilitis is more common in individuals with hypersensitivity to sunlight, so the clinical picture corresponds to the manifestations of acute allergic contact dermatitis. At the same time, against the background of a slightly swollen red border of the lower lip, areas of bright red erythema appear, small bubbles and erosion appear,

and crusts form on their surface. Patients are concerned about itching, burning, rarely sore lips. With a long course, a persistent increase in the lip develops - macrocheilia. A characteristic diagnostic sign is the exacerbation of the process under the influence of solar radiation, the defeat of the entire red border. In spring, the red border of the lower lip with the xerotic (dry) form of actinic cheilitis becomes bright red, covered with small dry silvery-white scales. The lesion covers the entire surface of the red border. In a number of patients, areas of keratinization are formed on the red border, sometimes there are warty growths. Actinic cheilitis often coexists with actinic dermatitis. With a long course of actinic cheilitis, under the influence of other factors, foci of leukoplakia and malignancy may appear.

Care. It is necessary first of all to recommend to the patient to avoid exposure to the sun as much as possible, to change the profession if it is associated with a long stay in the open air.

Hyposensitizing drugs, nicotinic acid, B vitamins, multivitamins with microelements are prescribed. Topical corticosteroid creams or ointments are used for actinic cheilitis. Symptomatic cheilitis Atopic cheilitis atopica) belongs to a group of symptomatic diseases of the lips, namely: it is believed that this type of cheilitis is a symptom of atopic dermatitis. A typical clinical symptom of diffuse neurodermatitis is pruritus followed by the development of erythema, excoriations and lichenification. Localization of pathological changes is characteristic: on the skin of the face, neck, elbows. In the etiology of atopic cheilitis, genetic factors are of great importance, which can create conditions for the development of an allergic reaction. Allergens can include drugs and cosmetics, foods, and bacterial and physical factors. It is more common in children aged 4 to 17 years. Clinic. Patients with atopic cheilitis complain of itching in the lips, redness, peeling of the red border of the lips. On examination, there is a slight swelling of the red border of the lips with the involvement of adjacent skin areas in the pathological process. It is characterized by lesions of the corners of the mouth that appear to be infiltrated. As the acute phenomena of inflammation subside, peeling and lichenification are observed. Infiltration, dryness of the corners of the mouth lead to the formation of cracks. Lip changes are combined with dry, flaky facial skin.

Care. Substances that cause sensitization are excluded from the diet: caviar, citrus and other fruits, chocolate, strawberries, as well as

spicy and spicy foods. In treatment, preference is given to antihistamines (suprastin, phencarol, diphenhydramine or other drugs), vitamins of group B (riboflavin, pyridoxine, etc.) are prescribed. To reduce itching, tranquilizers are used (seduxen, tazepam, etc.). Hyposensitizing therapy is used - intravenous infusions of 30% sodium thiosulfate solution, histoglobulin. Antiallergic drugs, antipruritic and ointments with corticosteroids are also locally used. With a slight effect of conservative therapy, the persistent course of the disease, Bucca's border rays are used. Eczematous cheilitis (cheilitis eczematosa) is considered as a symptom of eczema, which is based on inflammation of the surface layers of the skin of a neuro-allergic nature. Allergens can be a wide variety of irritants - from microorganisms, drugs to materials for prosthetic structures and components of toothpastes. Clinic. The disease can be acute or chronic. In the acute stage of the disease, patients complain of burning, itching of the lips, swelling and redness. In this case, erythema, swelling of the lips with the spread of pathological changes to the skin is observed. In this case, vesicles, weeping, crusts may appear. Therefore, the polymorphism of the elements of the lesion of both lips and the involvement of the skin in the process are characteristic. The transition to the chronic form of eczematous cheilitis is characterized by a decrease in the severity of acute inflammatory phenomena (edema, hyperemia) and compaction due to inflammatory infiltration, nodular formation, and scales. All the described changes in eczematous cheilitis develop on previously unaltered lips. Clinically, eczema complicated by a secondary infection is manifested by edema, reddening of the lips, vesicular rash, and crusting. With the cessation of blisters, peeling appears.

Care. In the treatment of eczematous cheilitis, hyposensitizing and sedative agents are

used.

General treatment of eczematous lesions is necessary. Outwardly, lubrication of the affected areas with a 5% synthomycin emulsion, ointments containing corticosteroids and antibiotics is used. Macrocheilitis is a disease of the lips characterized by persistent swelling or enlargement of the muscular stroma. The combination of macrochelitis with neuritis of the facial nerve was first described in 1901 by G. I. Rossolimo, and then by Melkersson. Later, Rosenthal described the addition of a folded tongue symptom to these signs. A

disease characterized by a triad of these symptoms is called Melkersson-Rossolimo-Rosenthal syndrome. In the etiology of macrochalitis, importance is attached to the infectious-allergic factor, as well as hereditary predisposition. It is assumed, in addition, that the described syndrome is an angioedema. Clinic. Patients are concerned about itching in the lips, an increase in their size, sometimes the appearance of edema in other parts of the face. Patients note the prolonged existence of edema, sometimes spontaneous improvement occurs, after which a relapse occurs. On examination, the skin in the lip area is shiny, its color is not changed. The skin in the affected area becomes bluish- pink. Localization of edema: one or both lips, cheeks, eyelids, other parts of the face. The defeat of the facial nerve is manifested in the distortion of the face in a healthy direction, smoothing of the nasolabial fold. Folded tongue, apparently, is not a symptom of the disease, but an anomaly of development. Detection of an edematous-folded tongue is considered a pathognomonic sign of the syndrome. Diagnosis of the Melkersson-Rosenthal syndrome can be difficult, since the patient does not always have all three signs at the same time: macrocheulitis and neuritis or macrocheulitis with a folded tongue. Perhaps the presence of one macroheil with the subsequent addition of other signs of the disease.

Differential diagnosis is carried out with angioedema, lymphangioma, hemangioma. It is also necessary to exclude collateral edema of the lips with periostitis and abscess.

Care. In the treatment of macrochalitis, the main role belongs to the combination of immunocorrective, desensitizing and antiviral therapy. Assign corticosteroids (dexamethasone 3- 5 mg / day, 125 mg per course), antibiotic therapy, antihistamines - tavegil, suprastin, fencorol, histoglobulin. Vitamins of groups C, B, PP. Immunocorrectors - T-activin, lycopene, etc. Antiviral drugs - acyclovir, zovirax. A certain effect is given by the use of laser therapy on the area of the lips and the area affected by neuritis of the facial nerve. For persistent cases, usually during the period of remission, stimulating therapy with pyrogenal, prodigiosan and other drugs is carried out. For neuritis, physical factors, Bernard currents, ultrasound, etc. are used. Electrophoresis of heparin ointment or applying it to the lip along with dimexide gave a good result. If hypersensitivity to bacterial allergens is detected, specific hyposensitization with bacterial allergens is used. For cosmetic purposes, surgical excision of part of the lip is used, but this does not

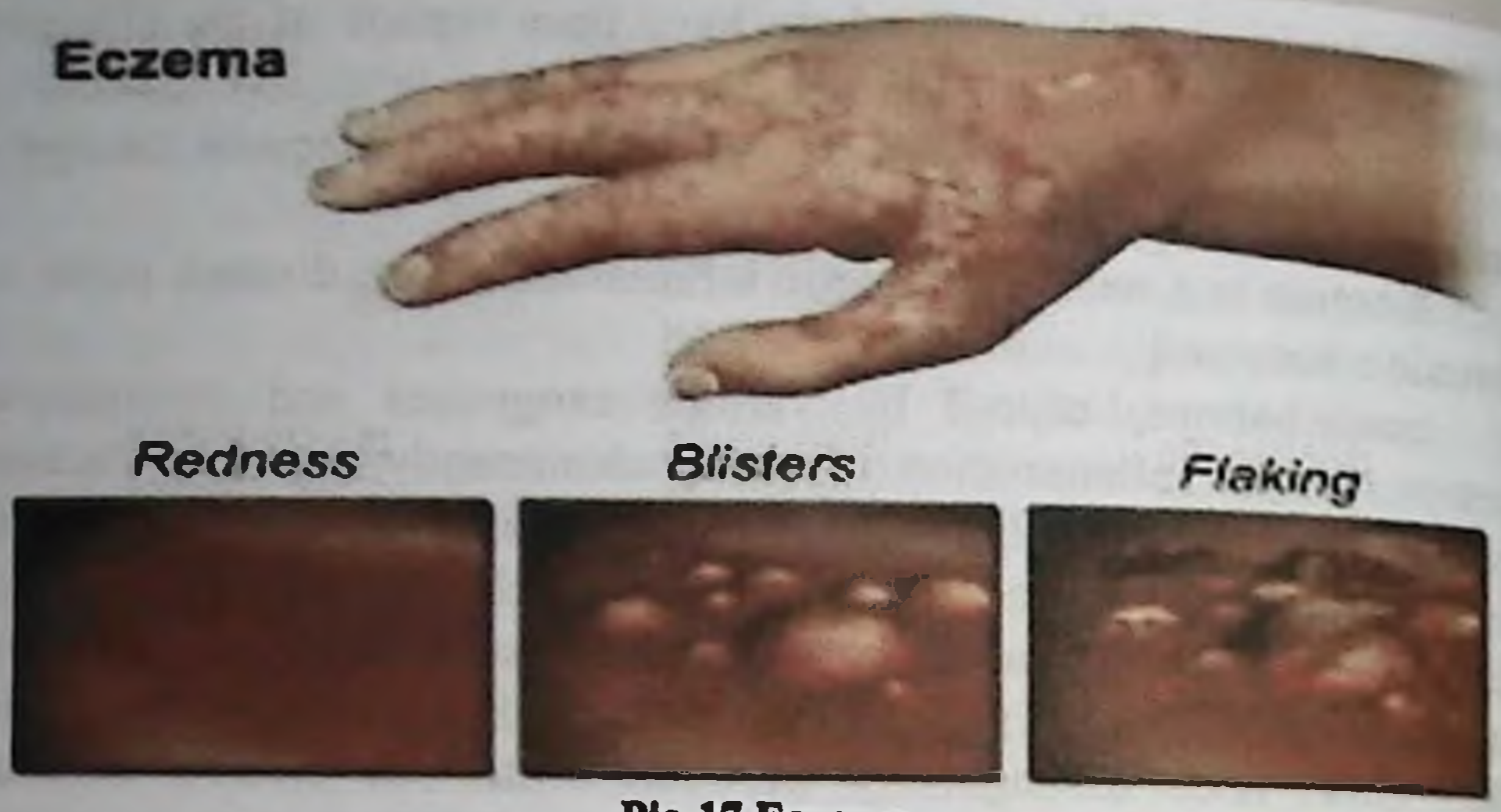
prevent recurrence. Recently, there have been reports of the effective use of hirudotherapy (medical leeches).

Forecast: with timely access to a doctor and adequate therapy - favorable.

Eczema is a recurrent allergic inflammatory skin disease, prone to chronic course and

exacerbations, caused by various exogenous and endogenous factors. Serous inflammation of the predominantly papillary dermis, focal spongiosis of the papillary layer of the epidermis, and polymorphism of pruritic elements, among which vesicles are in the first place, are noted. Eczema can be caused by internal or external factors, and in individuals with a hereditary predisposition, various agents. In 1819 L.-T. Biett distinguished between acute and chronic eczema according to the type of course, and P. Raeder (1823) gave topographic clinical variants of eczema, as well as important differences between true eczema and eczematous rashes of various nature. For example, differences with solar eczema or eczematous rashes from contact with mercury, the juice of some plants, etc. He also described eczema of the extremities, scalp ("seborrheic eczema") and, most importantly, found common signs of these clinically different dermatoses. The classification of eczemas is based on their clinical and pathogenetic features. Thus, eczema is distinguished: - true, including itchy and dyshidrotic forms; - microbial, which includes nummular, varicose, paratraumatic, sycosiform and eczema of the nipples; - seborrheic;

- tylotic (horny, calloused); - children's; - professional. Depending on the duration of the disease, acute (up to 3 months), subacute (from 3 to 6 months) and chronic (more than 6 months) eczema are distinguished. Etiology and pathogenesis. Currently, eczema is considered a polyetiological disease. It develops as a result of a complex effect of neuropsychic, allergic, hereditary, endogenous (endocrine, metabolic), exogenous and other factors. Exogenous influences are extremely diverse. Often these are nickel (coins, keys, jewelry, buckles, spectacle frames), chromium (cement, varnishes, painted leather, furs), paraphenylenediamine (paints, cosmetics), cobalt (cement, ceramics, paints, jewelry, plastic), formaldehydes. (textiles, paints, antiseptics), topical medicines (local anesthetics, antibiotics,



Pic-17 Eczema

adhesive plasters), plants (primroses, tulips), microorganisms and fungi, rubber, mineral oils, crude oil, turpentine, formalin, epoxy resins, bakelite glue, lubricating fluids, etc. The pathogenesis of eczema consists of numerous and diverse links that affect, to one degree or another, almost all body systems. Nervous stress is one of the triggers of eczema, as indicated by the symmetry of the rash and the dependence of the onset or relapse of the disease on mental trauma.

Important information The role of the central nervous system in the pathogenesis of eczema is supported by the successful use of electrosyn, hypnosis and sedatives. The development of the disease largely depends on a combination of genetic predisposition, allergens, and additional non-specific factors: household, occupational, environmental, etc. Patients with true eczema have a positive association of histocompatibility antigens HLAB 22 and Cw 1, which allows us to consider these antigens as genetic markers of eczema in persons of the Caucasoid race, predominantly of the Slavic type (A. A. Kubanova). With an allergic disease in one of the parents (mainly the mother), the probability of developing eczema in the child is 40%, and in both parents - 50-60%. Toxicosis of pregnant women in the mother, nutritional errors, concomitant diseases contribute to the onset of the disease in children. Artificial feeding from the first days of a child's life, early introduction of complementary foods, especially whole cow's milk, semolina porridges with whole or condensed milk, concentrates, meat or fish broths create conditions for the occurrence of exudative diathesis

and childhood eczema. The eczematous inflammatory reaction is considered to be a delayed-type hypersensitivity reaction. A significant role in the pathogenesis of the disease is played by various immune shifts, accompanied by a change in the profile of inflammatory cytokines, prostaglandins, and cyclic nucleotides. In patients with eczema, immunodeficiency is determined by cellular, humoral and phagocytic links. The total number of T-cells and T-helpers is reduced, as well as the functional activity of T- suppressors is reduced.

According to modern concepts, in the development of delayed-type hypersensitivity in eczema, the main role is played by T-lymphocytes (mainly represented by the Th -1 population), which carry specific antigen receptors on their surface. In this type of reaction, immune T-lymphocytes secrete a number of anti-inflammatory cytokines: interleukin-1 (IL-1), IL-2, tumor necrosis factor alpha (TNFa) and 7-interferon. The release of biologically active substances (prostaglandins, leukotrienes, histamine) causes the development of tissue inflammatory reactions, which is clinically manifested by an early allergic reaction in the form of hyperemia, edema, and itching. Antigenic stimulation of CD 4+ lymphocytes of the first order (Th -1) leads to the formation of IL -2, and IL -2-producing ability of CD 4+ in patients with eczema is higher than in healthy people. However, IL-1 and IL-2 are not the only cytokines involved in the pathogenesis of eczema. For example, eczematous reactions caused by cobalt and mercury were accompanied by a significant increase in the synthesis of IL-8 at the site of contact of these metals with the skin.

Another effect of anti-inflammatory cytokines is the induction of the expression of

adhesion molecules on leukocytes and endothelial cells, which stimulates the influx of leukocytes from the vascular layer. channels to the focus of inflammation by their transendothelial migration. Further development and accumulation of immunocompetent cells in the focus of inflammation is controlled by chemokines produced by macrophages and endothelial cells. Cellular infiltrate in the focus of inflammation, consisting of neutrophils, eosinophils and macrophages, contributes to the further development of allergic inflammation in the form of a late allergic reaction. So, type IV allergic reactions ("delayed type", "eczematous type") involve T-lymphocytes (a subpopulation of T-

helpers - TH-1), macrophages and endothelial cells. cells and their secreted cytokines.

The mechanism of such reactions is the penetration of a hapten (low molecular weight antigen) into the skin, where a stable protein complex (hapten is a carrier protein) is formed, which binds to Langerhans cells in the epidermis and is already presented as a full-fledged antigen by T-helper cells and causes a delayed-type hypersensitivity reaction. in the skin. The process ends with the selection of various intermediaries. T cells that enter the regional lymph nodes produce specific memory cells and T effectors that circulate in the blood. In the study of T- lymphocytes from chronic eczematous foci, a profile of either Th -0 or Th -1 with an increased content of interferon 7 was revealed. Eczematous manifestations develop only in sensitized people. They occur after repeated contact with the hapten. no later than 48-72 hours. The polymorphic infiltrate in the skin in eczema is the result of the action of the resulting inflammatory cytokines, primarily TNF- α. In addition, the role of free radical processes ("oxidative stress") in the pathogenesis of eczema cannot be ignored. Free radicals activate phospholipase, which leads to the release of arachidonic acid, from which, after exposure to cyclooxygenase and lipoxygenase, inflammatory mediators arise - leukotrienes, prostaglandins and thromboxanes. Clinic. The true or idiopathic form of eczema is manifested by anti- inflammatory edematous erythema, followed by a rash of a group of tiny seropapules or vesicles (microvesicles), which quickly open without having time to form. In place of rapidly opening seropapules and vesicles, point erosions are exposed - the so-called serous holes, from the depths of which serous exudate is releasedcreating a richly weeping surface. The serous fluid gradually dries up, forming grayish-yellow crusts, under which epithelialization occurs. Thus, during the course of eczema, the following stages are clearly observed: erythematous, papulo-vesicular, wet stage and cortical. Due to the fact that with the wave-like development of the process, all primary elements - erythema, seropapules and vesicles - appear simultaneously, creating one of the most characteristic signs of eczema-evolutionary polymorphism. The transition from an acute condition to a chronic one occurs gradually and is expressed in an increase in infiltration, thickening of the affected area of the skin and an increase in the skin pattern, which is called lichenization. Skin color becomes more

stagnant; on the surface, except for crusts, there is a significant peeling. Along with these moderate symptoms of inflammation, the appearance of fresh seropapules and vesicles continues with the formation of weeping "serous wells" and serous crusts. Such an alternation of islet-inflammatory and infiltrative manifestations is characteristic of true eczema, which is always accompanied by severe itching, which increases as the process aggravates. Foci of eczema do not have clear boundaries. It is relatively rare for a process to be limited to a single site. An eczematous lesion usually begins on the skin of the face, hands and can spread throughout the skin. The rashes are symmetrical with a tendency to spread to the skin of the upper, lower extremities and torso. Patients are concerned about itching of varying intensity, which contributes to the development of neurotic disorders with sleep disturbances up to insomnia. As inflammation decreases, weeping is replaced by the formation of crusts, scales, the appearance of peeling (eczemacrustosum, eczemascuamosis), secondary pigmented vascular or depigmented spots, which gradually resolve. Areas of dryness and peeling are possible. skin with the formation of cracks in the stratum corneum (eczema crackles). With a long chronic course of eczema, calloused hyperkeratotic formations with cracks (eczematuloticum) sometimes appear on the palms and soles of the hands. Often, eczema is complicated by the addition of a pyogenic infection: pustules and purulent crusts appear (eczema impetiginous). The acute process gradually turns into a chronic course and can last for years. Microbial eczema develops at the site of chronic foci of pyoderma: around infected wounds, trophic ulcers, fistulas, abrasions, scratches. Ostro-inflammatory, sharply limited, large and large-scalloped foci with a well-formed torn off stratum corneum along the periphery are formed, which are the remnants of streptococcal conflicts. In addition to seropapules and papulovesicles, weeping erosions, a massive layer of purulent crusts forms on the surface of the foci. The arrangement of the elements is continuous, without areas of healthy skin. The foci are prone to peripheral growth. Around them, on outwardly healthy skin, fallouts are often visible - separate small pustules, dry scaly foci like simple pituriasis. The number and prevalence of dropouts vary greatly. The process is accompanied by itching. The focus of microbial eczema is initially asymmetric and is more often located on the lower extremities. A variety of microbial eczema is the so-called coin-shaped (nummular),

or plaque-like, eczema with the formation of sharply demarcated foci of rounded outlines 1-3 cm in diameter. On their edematous-hyperemic surface, there is abundant weeping, layering of serous-purulent crusts.

Nummular eczema is more often localized on the upper extremities, but in some cases the process can be widespread. Irrational treatment of microbial eczema or dramatization of its foci is accompanied by the appearance of secondary allergic rashes - microbids or allergides. They are polymorphic, their manifestations can be represented by edematous-erytomatous spots, seropapules, vesicles, pustules. With a progressive course, secondary rashes merge and form weeping erosive areas. In such cases, microbial eczema transforms into true eczema. Varieties of microbial eczema are also paratraumatic (near-ear) and varicose eczema. Varicose eczema. Varicose veins of the lower extremities contribute to the onset of the disease.

Elements of eczema are localized in the area of dilated veins, along the circumference of varicose ulcers, areas of skin sclerosis. The development of the disease is promoted by injuries, hypersensitivity to drugs for the treatment of varicose ulcers, skin maceration. Clear boundaries of foci, moderate itching bring together varicose eczema in the clinical aspect with microbial and paratraumatic. Seborrheic eczema develops in the so-called seborrheic areas: the scalp, auricles, in the sternum, between the shoulder blades, in the nasolabial folds. The lesions are plaques formed from erythematous scaly patches with yellowish-brown draining miliary papules.

These rashes merge with each other and acquire an annular, garland-like contour. Seborrheic eczema is directly related to seborrhea and its characteristic neuroendocrine disorders, and is often associated with the presence of Malassezia in the foci. furfur. Occupational eczema occurs as a result of exposure to occupational hazards in workers and employees of certain industries, but its clinical manifestations do not differ from eczema of another etiology. Dyshidrotic eczema is localized on the palms and soles, where, due to the thickness of the stratum corneum of the epidermis, the erythematous stage is poorly expressed, but well-formed "sago grains" predominate. Vesicles either open, turning into erosions, or dry up into layered serous-purulent crusts, and sometimes merge into large multi-chamber blisters. Gradually increasing, the focus of dyshidrotic eczema can spread to the back of the hands, feet and other parts of the limbs. In such cases, the

transformation of eczema into idiopathic occurs with the formation of microvesicles, weeping, "serous wells". In many patients, dyshidrotic eczema is accompanied by trophic changes in the nails. Tilotic (horny, calloused) eczema, like dyshidrotic, is limited to the area of the palms and soles. The erythematous stage is expressed slightly due to the thickening of the stratum corneum in the form of homosols at the site of vesicular elements. Children's eczema is characterized by the severity of the exudative component and often occurs with a genetic predisposition. In children with exudative diathesis, foci of focal chronic infection, bronchial asthma, hay fever, acute respiratory diseases, conjunctivitis, keratitis, and gastrointestinal disorders are often found. Usually occurs at the age of 3-6 months, manifested by clinical signs of true, microbial and seborrheic eczema. Eczema in infants and young children is one of the most common dermatoses. Complications of pregnancy and concomitant diseases in the mother (nephropathy, diabetes mellitus, cardiovascular insufficiency, chronic hepatocholecystitis, foci of chronic infection, etc.) contribute to the development of eczema in newborns. at the same time, increased sensitivity to various foods, frequent acute respiratory infections, tonsillitis, otitis, poor care. Morphological manifestations of eczema in children are identical to rashes in adults. It is only necessary to emphasize the high severity of exudative symptoms with a tendency to merge elements and complicate impetigation. Features of the course of eczema in children are associated with localization, layering of viral infection and the possibility of sudden "eczema" death in young children.

Sycosiform eczema may occur in patients with sycosis complicated by eczema. In these patients, follicular pustules recur, located on the inflamed skin and penetrating the center of the hair (sycosis). With sycosiform eczema, the process goes beyond the hairline, eczematous wells, weeping and severe itching are noted. The skin becomes lichenified. The favorite localization of the process is the upper lip, beard, armpits, pubis. Eczema of the nipples in women is represented by reddish foci, sometimes covered with crusts and scales, accompanied by weeping, cracks, has a sharply defined outline and a very persistent course. Often, eczema of the nipples is a consequence of trauma when feeding a child or a complication of scabies. Histopathology. In the

acute course of eczema in the epidermis, parakeratosis, focal spongiosis and edema of the papillary dermis with vasodilation and a

predominant lymphocytic infiltrate around them are detected. Later, acanthosis, parakeratosis, and spongiosis are detected in the epidermis, combined with pronounced accumulations of lymphocytes in the interspongious spaces. In the dermis, there is a pronounced infiltration of the papillae, mainly by lymphocytes and histiocytes. Diagnostics. With eczema, acute inflammatory edema occurs with a rash of seropapules, microvesicles, the formation of "serous wells" and weeping. True eczema is most similar to atopic dermatitis. However, in atopic dermatitis, persistent white dermographism, a high tone of the pilomotor reflex, and skin infiltration with lichenification predominate. There is no spontaneous prolonged wetting, and if it happens, it is short-term, limited, in places of combing. In atopic dermatitis, improvement is possible in the summer until clinical recovery, and in patients with eczema, a clear dependence of the condition on the season is not observed. In complex diagnostic cases, skin biopsy specimens are examined.

Care. General and external treatment is prescribed individually, taking into account the endogenous and exogenous factors underlying the disease. It is necessary to correct neuroendocrine disorders, sanitize foci of chronic infection, limit contact with water. If possible, identify foods that trigger allergies. If they are not found, then a hypoallergenic diet is prescribed with the exception of chocolate, honey, nuts, citrus fruits, melons, pineapples, seafood, fish, smoked meats, mayonnaise, horseradish, vinegar, mustard, eggs, milk, radishes, radishes, tomatoes, extractives, alcohol. Methods for nonspecific evaluation of pathogenetic therapy are varied. First of all, these are antihistamines for 3-5 weeks (with alternating courses of 10 days). If necessary, after a 2-3-week break, treatment with antihistamines is resumed: prescribe loratadine

0.001 g 1 time per day or fexofenadine 180 mg 1 time per day. With severe skin itching, sleep disturbances, neurotic complaints, antihistamines with a sedative component are recommended: hifenadine 0.025 g 3 times a day, or chlorpyramine 0.025 g 3 times a day, or clemastine 0.001 g 2-3 times a day, or mebhydrolin 2 times a day. 0.1 g 3 times a day. Hyposensitizing agents: inject 30% sodium sulfate solution 5-10 ml intravenously slowly daily or every other day, for a course of 10-15 injections, or 25% magnesium sulfate solution 5-10 ml intramuscularly, or 10% calcium gluconate solution 10 ml intramuscularly, total 10-15 injections daily or every other day. With

severe nocturnal itching, which is not stopped by taking drugs orally, antihistamines are administered intramuscularly: 1% diphenhydramine solution, or 2% chlorpyramine solution, or 0.1% clemastine solution, 1-2 ml at night daily, only 10-15 injections.. In case of sleep disorders, neurotic disorders, tranquilizers are prescribed: oxazepam 0.01 g at night or nitrazepam 0.005 g at night until the psycho-emotional state normalizes. Pancreatic enzyme preparations are prescribed in courses of 3-6 weeks: pancreatin preparations (creon 1 capsule 3 times a day with meals with a small amount of water or mezim-forte 1 tablet 3 times a day before meals); preparations containing pancreatin, cholic acid, pepsin, amino acids (panzinorm forte 1 tablet 3 times a day with meals). With a tendency to constipation, use preparations containing bile components (festal or enzistal 1 tablet 3 times a day with meals). Patients with unstable stools are prescribed drugs that do not contain bile. With severe edematous syndrome, diuretics are used.

Immunomodulatory drugs are used in accordance with the results of an immunological blood test for clinical signs of secondary immunodeficiency: frequent acute respiratory viral infections, viral and fungal infections, recurrent pyoderma, foci of chronic infection that are resistant to adequate therapy. More often, taktivin is used in 1 ml of a 0.01% solution once a day intramuscularly. daily for 5-14 days, or licopid 10 mg 1 time per day for 10 days, or myelopide

3.0 mg 1 time per day for 10 days. Drug testing in vitro using the chemiluminescent method allows you to individually select immunotropic drugs. In the case of persistent, severe eczema with a tendency to turn into erythroderma, prednisolone (or another glucocorticoid) can be prescribed for 2-3 weeks, starting at a dose of 20-30 mg / day and gradually reducing the dose by 1-2 mg (1/4 tablet). In order to detoxify patients with eczematous erythroderma, hemosorption, plasmapheresis, and enterosorption are used. In children, dysbacteriosis, fermentopathy of the gastrointestinal tract are usually observed simultaneously, therefore bifidumbacterin, bifikol, bactisubtil, lactobacterin, colibacterin are indicated. If staphylococcus, Proteus vulgaris or lactose-negative Escherichia coli are detected in the colon after a course of antibiotic therapy, coliprotein and staphylococcal bacteriophage should be used. External treatment is prescribed in accordance with the nature of the inflammatory process. In acute inflammation, fukortsin, a 25% solution of silver nitrate, baths with a

0.01-0.1% solution of potassium permanganate, aerosols with corticosteroids, lotions or wet-drying dressings with anti-inflammatory, antibacterial, astringent solutions in small concentrations are used, because there is no irritating effect, according to the principle "wet - wet" and "irritating does not irritate". For lotions, Burow's liquid is often used (1 tablespoon per glass of water), 0.25% tannin solution, 3% sodium tetraborate solution, 0.25% zinc sulfate solution. Well reduce puffiness and hyperemia, contribute to the epithelization of lotion from tea, decoction of plantain, marshmallow root, chamomile, coltsfoot. In microbial processes, impetiginations, antimicrobial solutions are mainly used - 2-5% resorcinol solution, 0.05-0.5% solution of ethacridine lactate, 0.02% furatsilina solution, 0.01- 0.1% potassium permanganate solution. In the interval between applying lotions, the foci are extinguished with a 0.5-2% solution of silver nitrate, fucorcin, 2% solution of brilliant green and lubricated with vegetable or zinc oil. Lotions are used until the sputum disappears. With microbial eczema, it is advisable to use dressings with staphylococcal bacteriophage for a short time. After removing the phenomena of acute inflammation, removing scales and crusts, pastes and ointments are used. Pastes are not applied to wet areas, scalp and other skin surfaces with hairline. It is also inappropriate to prescribe them for dry skin, severe infiltration and under a compress.

With eczema, pastes are most often used: 5% bornnaphthalene, 3% ichthyol, 5-10% naphthalene, 5-10% tar-naphthalene. lanol, 2-10% ichthyol-naphthalene with the addition of 2- 5% anesthetic. In subacute and especially chronic eczema, ointments are used that soften scales and crusts, facilitating their removal along with the microorganisms present in them. In patients with eczema with hypersensitivity of the skin to many medicinal substances, indifferent ointments are used - zinc, naftalan. In patients without hypersensitivity, ointments containing sulfur, ichthyol (2-10%), tar, ASD (5-10%), 2-10% boronaftalan, 1-3% indomethacin are used.

With severe dryness, a solution of retinol in oil, sunflower, peach oil is added to the ointment. Glucocorticoid preparations used in the form of ointments, creams, suspensions (0.5% prednisolone ointment, sinaflan, sinalar, deperzolon, elokom, etc.) have a pronounced anti-inflammatory, antipruritic effect. In recent years, ascomycin macrolactam derivatives such as tacrolimus and pimecrolimus have been

used to treat eczema. Both drugs, similar in composition and mechanisms of action, are representatives of new immunosuppressants and have a powerful anti-inflammatory effect. Pimecrolimus specifically binds to the cytosolic macrophilin-12 receptor of T-lymphocytes and suppresses their activation, inhibits the synthesis of pro- inflammatory cytokines - interleukin-2 and 7-interferon, as well as interleukin-4 and interleukin- 10 (Th 2- type).) in human T cells. In addition to preventing the release of pro-inflammatory cytokines, pimecrolimus significantly reduces the release of inflammatory mediators from mast cells, which gives grounds to consider the topical pimecrolimus therapy as an alternative to corticosteroids.

For microbial eczema or impetigation phenomena, glucocorticoid ointments with antibiotics or disinfectants are used. Given the possibility of absorption of hormones by the skin, hormonal ointments can be added to conventional ointments and pastes in various proportions (1:1, 1:2, 1:3, etc.), which enhances the effect of the latter. It is convenient to use glucocorticoid agents in the form of aerosols, which also contain antimicrobial substances: oxycyclosols (oxytetracycline, prednisolone), oxycort, polcortolone. In chronic forms of eczema, selective herbal medicine is used during the subsidence of inflammatory phenomena (suerythemic and then erythemal doses daily, for a course of 15-20 sessions). Also recommend phonophoresis of ointments, oxygen therapy. Applications of ozocerite, paraffin, therapeutic mud are prescribed for areas of pronounced lichenization, 10-20 procedures per course. In some cases, other methods are also effective: reflex (indirect) physiotherapy, indirect diathermy, acupuncture, laser therapy. An important role in the treatment of eczema in adults and children is played by a rational hyposensitizing diet.

The prognosis is favorable both for life and for rehabilitation in the process of recovery, especially if the correct preventive measures are followed. Relapses of the disease are possible with all forms of eczema.

The prognosis is much worse if eczema develops in small pasty and asthenic children, in the elderly and people whose body is weakened by infection or intoxication. Prevention.

In the prevention of eczema in children, a rational lifestyle and nutrition of their mothers during pregnancy is important. Children with eczema are at risk of contracting viruses (primarily the herpes virus). In such cases, Kaposi's herpetiform eczema with a severe course and a

possible fatal outcome is not excluded. In the prevention of recurrence of eczema in adults and children, systematic dispensary observation and spa treatment during remission are also of great importance. Good hygiene is important for all forms of eczema. This helps prevent secondary infection.

Small folliculitis should be lubricated with fucorcin or 1-2% aqueous or alcoholic solution of aniline dyes. Baths and showers with extensive skin lesions are temporarily excluded. However, baths, for example, with a decoction of chamomile, and in children's practice with a decoction of bran, oak bark, etc., sometimes have a therapeutic effect. Avoid overheating. With symptoms of varicose veins, wearing rubber stockings or bandaging the legs with elastic bandages is the prevention of varicose eczema. Treat varicose veins (together with the surgeon), ulcers, fissures, fistulas, wounds. Breastfeeding mothers with eczema of the nipples should express milk. With eczema of the skin of the hands, you should not wet your hands with too hot or cold water, wash them manually with synthetic powders. Contact with suspected allergens at home and at work should be avoided whenever possible.

In the prevention of eczema in children, deworming and rehabilitation of foci of chronic or acute focal infection in pregnant women, rational nutrition is important, since allergens circulating in the blood of a woman, penetrating through the placenta, sensitize the fetus. In these cases, after the birth of a child, exogenous allergens with mother's milk enter the already prepared soil. Pregnant women, especially those who had allergic diseases in the family, should not consume large amounts of milk, eggs, sweets. Their food should be rich in vitamins and include a variety of vegetable dishes, boiled meat, lactic acid products, fruits. There are oranges, tangerines, apricots, peaches, strawberries, raspberries, strawberries should be limited. Patients with eczema are not recommended to wear synthetic, flannel or wool underwear. Patients with eczema should be registered in the dispensary.

24. With a common process in atopic dermatitis, the area of skin lesion

1) 10-50%;
2) 5-10%;
3) more than 50%;
4) less than 10%.

25. Prurigo-like form of atopic dermatitis is characterized by

1) bullous rashes;
2) **itchy papules, mainly on the extensor surfaces of the limbs;**
3) cicatricial alopecia;
4) network structure.

26. Prurigo-like form of atopic dermatitis is typical for
1) **children's age group;**
2) infant age group;
3) **adolescent age group;**
4) senile age group.

27. Denny-Morgan symptom
1) additional fold of the upper eyelid;
2) additional knee fold;
3) additional elbow fold;
4) **additional fold of the lower eyelid.**

28. Exudative form of atopic dermatitis is characterized by
1) lichenization;
2) **microvesiculation with the development of weeping;**
3) **formation of crusts;**
4) **erythema.**

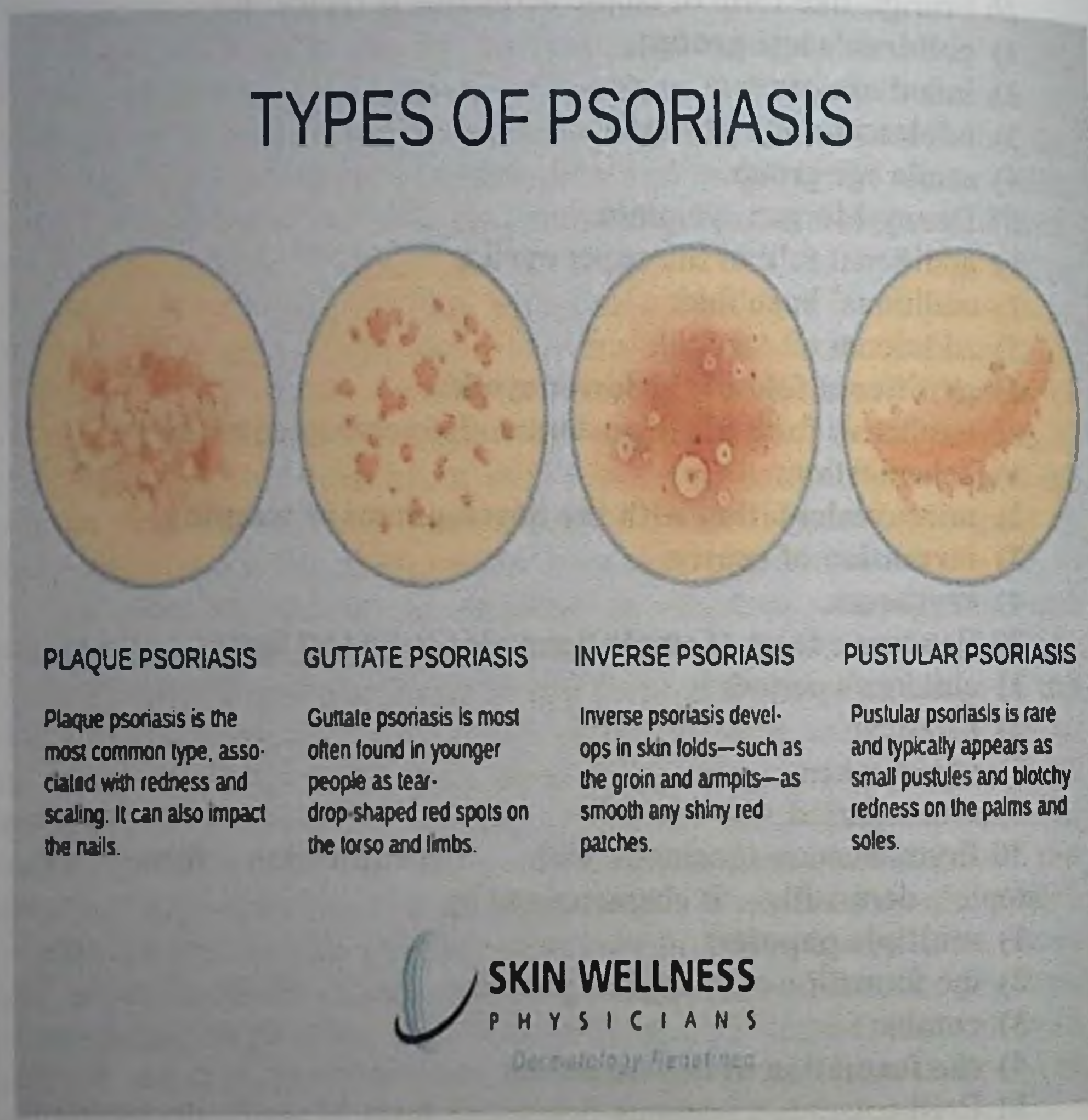
29. Exudative form of atopic dermatitis is typical for
1) children's period;
2) **infancy;**
3) adolescence;
4) senile period.

30. Erythematous-squamous with lichenification form of atopic dermatitis is characterized by
1) **multiple papules;**
2) the formation of pruriginal papules;
3) **combs;**
4) **the formation of lichenization.**

31. Erythematous-squamous-lichenized form of atopic dermatitis is typical for
1) **children's period;**
2) infancy;
3) **adolescence;**
4) senile period.

32. Erythematous-squamous form of atopic dermatitis is characterized by

1) **small papules;**
2) **combs;**
3) **peeling in the form of confluent foci with fuzzy boundaries;**
4) **erythema.**



CHAPTER 5 PAPULAR DERMATOSIS. PSORIASIS, LICHEN PLANUS

Psoriasis (lichen scaly, psoriasis vulgaris) is a chronic relapsing dermatosis with hyperproliferation of epidermal cells, disruption of the keratinization process and an inflammatory reaction in the dermis, changes in various organs and systems. Dermatosis most often occurs at the age of 10-25 years, but it can first appear at any age: cases of psoriasis have been described in children under 1 year of age and in adults aged 82 years. The disease is widespread at all latitudes of the globe, among the population of different nationalities and races, where it occurs with an uneven frequency - from 0.1 to 3% or more. Approximately 15% of patients are diagnosed with psoriatic arthritis, often with multiple joint involvement, often leading to disability. This makes psoriasis not only a medical problem, but also a social one.

Psoriasis is equally pronounced in men and women, but among children it is more common in girls, and among adults - in men (60-65%). The clinical description of a disease similar to psoriasis has been known since ancient times (in the Bible, in Hippocrates, Celsus, etc.). However, it was not until 1841 that Gebra defined psoriasis as an independent disease. Etiology and pathogenesis. None of the many hypotheses of psoriasis is generally accepted. The role of genetic factors is undoubted, as indicated by family cases of the disease, including in twins, and certain correlations with the HLA system. Special genetic studies conducted by V. N.

Mordovtsev et al. have established a multifactorial type of inheritance of psoriasis with incomplete gene penetrance. It was shown that histocompatibility antigens B13 and B17 are significantly more common in patients with psoriasis only with skin manifestations, and B17, B27, B33, B40 - in patients with skin manifestations in combination with arthritis. In addition to the HLA system, another important genetic marker of psoriasis has been identified: the association of dominant forms of psoriasis with the distal region of chromosome 17.

In psoriasis, both autosomal dominant inheritance (probability up to 50%) and hereditary predisposition to the disease may occur under the influence of environmental factors. The formation of psoriatic elements is based on excessive proliferation of keratinocytes, impaired differentiation of epithelial cells, high activity of metabolic processes associated with energy balance, insufficient synthesis of keratohyalin.

Immune disorders play an important role in the development of psoriasis: an increase in the activity of T-helpers with a defect in T-suppressors, an increase in the content of circulating immune complexes, and an increase in titers of autoantibodies to DNA. Cellular infiltrate in psoriasis consists mainly of CD -4 + T-lymphocytes. All these data indicate that psoriasis is an immunogenetic disease in which cytokines and growth factors determine the severity of inflammation and hyperproliferation due to a violation of the cAMP/cGMP second messenger system. Activated macrophages produce pro-inflammatory chemicals. cytokines: tumor necrosis factor alpha (TNF- α) and gamma (TNF-7), interleukins (IL-1a, IL-2, IL-6, IL-8). TNF - α plays a key role. Psoriasis is a systemic process in patients not only with immune disorders, but also with pronounced functional and morphological changes in a number of organs and systems. At the current level of knowledge, psoriasis can be defined as a genotypic dermatosis transmitted by a dominant type with incomplete penetrance and uneven manifestation. This disease is multifactorial in nature: pathogenic factors are changes in enzymatic, lipid, less often protein and carbohydrate metabolism, endocrine dysfunctions and functional disorders of the interstitial brain in the form of an adaptation disease, shifts in amino acid metabolism, often in combination with chronic tonsillitis, influenza and other infectious and allergic diseases. (mainly streptostaphylococcal and viral nature).

The genetic apparatus of cells can be pathogenically affected by filterable viruses, which leads to dysregulation of biochemical processes (viral-genetic hypothesis). In patients with psoriasis, changes in the ratio of histone protein fractions were revealed, which play an important role in the regulation of proliferative activity and DNA synthesis, which make up most of the chromatin. Clinic. Monomorphic papular psoriatic rash is located in most patients symmetrically, mainly on the extensor surfaces of the limbs. Psoriasis can affect any area of the skin, as well as nails and the musculoskeletal system. The primary element is a pink epidermal papule (Pilnov's symptom) of varying intensity: fresh elements are brighter (up to red), long-term ones are more faded. Papules are flat, with a rough surface, covered with silvery-white (silver color is associated with air bubbles) bran-like or small-lamellar scales, which are easily removed. On fresh papules, the scales are located in the center, along their periphery there is a narrow light border. Then the

delamination increases, occupying the entire surface of the element. Firstly, papules in psoriasis

have a regular rounded shape and a diameter of 1-2 mm. As the process progresses, psoriatic papules spread along the periphery, increase and form plaques that reach large sizes and have bizarre outlines. Quite often, papular elements of different sizes and psoriatic plaques are simultaneously found in the same patient.

For the diagnosis of psoriasis, the following are important: symptoms, including the psoriatic triad and an isomorphic reaction, or the Koebner phenomenon. When scraping a psoriatic papule, abundant peeling is constantly noted in the form of silvery-white scales resembling stearin (the "stearin spot" phenomenon), after removing the scales, the shiny surface remains wet (the phenomenon of the terminal or psoriatic film), with further scraping, drip bleeding occurs - the phenomenon of pinpoint bleeding (Auspitz), or "blood dew" (A. G. Polotebnov). These phenomena are based on the pathohistological nature of psoriasis (parakeratosis, acanthosis, papillomatosis). An isomorphic reaction in psoriasis (the Koebner phenomenon) consists in the development of psoriatic rashes on skin areas prone to irritation by mechanical and chemical agents (scratches, pricks, scratches, cuts, friction, radiation, burns, etc.). After skin damage, an average of 7-9 days pass before the development of an isomorphic reaction, although there have been cases of reactions after shorter (3 days) and longer (21 days) periods. Psoriasis can be accompanied by itching (usually in a progressive stage) of varying intensity and a feeling of skin tightness. Depending on the size and nature of the elements of the rash, the following clinical forms of psoriasis are distinguished: - punctate psoriasis (punctate psoriasis) with papules 1-3 mm in diameter; - teardrop-teardrop-shaped or lenticular psoriasis, when the elements reach a size of 4-5 mm; - nummular psoriasis (coin-shaped psoriasis) with rashes up to 2-3 cm in diameter - - lichenoid spiny psoriasis with lichenoid papules - - papillomatous, severrucous psoriasis, in which papillomatous growing foci are formed; - ring-shaped psoriasis, seuorbicular (ring-shaped psoriasis), when the elements form rings - - gurate psoriasis, when the rashes are arranged in the form of arcs and garlands; – psoriasis figurata, seugeographica (curly, or geographical, psoriasis), when the outlines of the foci resemble a geographical map; - seborrheic psoriasis (seborrheic psoriasis) with localization of rashes in

typical seborrheic places in the form of small pinkish scaly spots; - eczematous-eczematous psoriasis, etc.

On the scalp, diffuse peeling or sharply limited layers of scales can occur, often capturing and surrounding smooth skin. From the scalp, psoriasis begins in about 1/4 of cases and is combined with rashes on other areas of the skin in more than 60% of cases. Often, the lesion of the scalp is focal in nature, located on the back of the head, temples, crown in the form of distinctly raised plaques. Psoriasis of the genital area is 1/5 of cases (more often in girls), and in large folds it usually proceeds as an intertriginal process, but with clear boundaries and infiltration in the lesions. The main localization falls on the limbs (up to 85% of cases) and the trunk (more than 70%). With the defeat of the palms and soles (palmar-plantar psoriasis), in 8-12% of cases, extensive rounded scaly patches develop. striped patches. In infancy and in children under 5 years of age, psoriasis is manifested by discharge from lesions, especially often on the scalp, face, skin of the trunk and extremities. Papules form from erythematous patches, sometimes with

weeping and swelling (exudative psoriasis), which is very difficult to diagnose.

With the defeat of the inguinal folds, the process often passes to the mucous membrane of the genital organs. In children of primary school age, the papular-psoriatic rash is bright, juicy and tends to merge, compared with the rash in adults, the exudative effect is more pronounced. a component that forms many scaly crusts and massive serous crusts. Atypical clinical forms of psoriasis, in addition to follicular, verrucous and rupioid, include intertriginal, exudative, pustular psoriasis and psoriatic erythroderma. Intertriginous psoriasis (psoriasis inversa) develops in young children, in patients with severe forms of diabetes mellitus, rheumatism, obesity, and in the elderly. Localization of elements on the flexion surfaces of the limbs, joints, on the skin of the inguinal and femoral folds, on the armpits, palms, soles. The disease is usually manifested by nummular, exudative plaques with clear boundaries, purple-red color, slight peeling, moist and macerated surface, often accompanied by itching and burning.

It is possible that this form is inherently exudative psoriasis. (exudative psoriasis), the clinical picture of which has changed somewhat due to the localization of the process in the folds of the skin. With exudative psoriasis, the elements of psoriasis are covered with

grayish-yellow loose crusts saturated with exudate. Pustular psoriasis (pustular psoriasis) has two clinical variants: the Zumbush type and the Barber type.

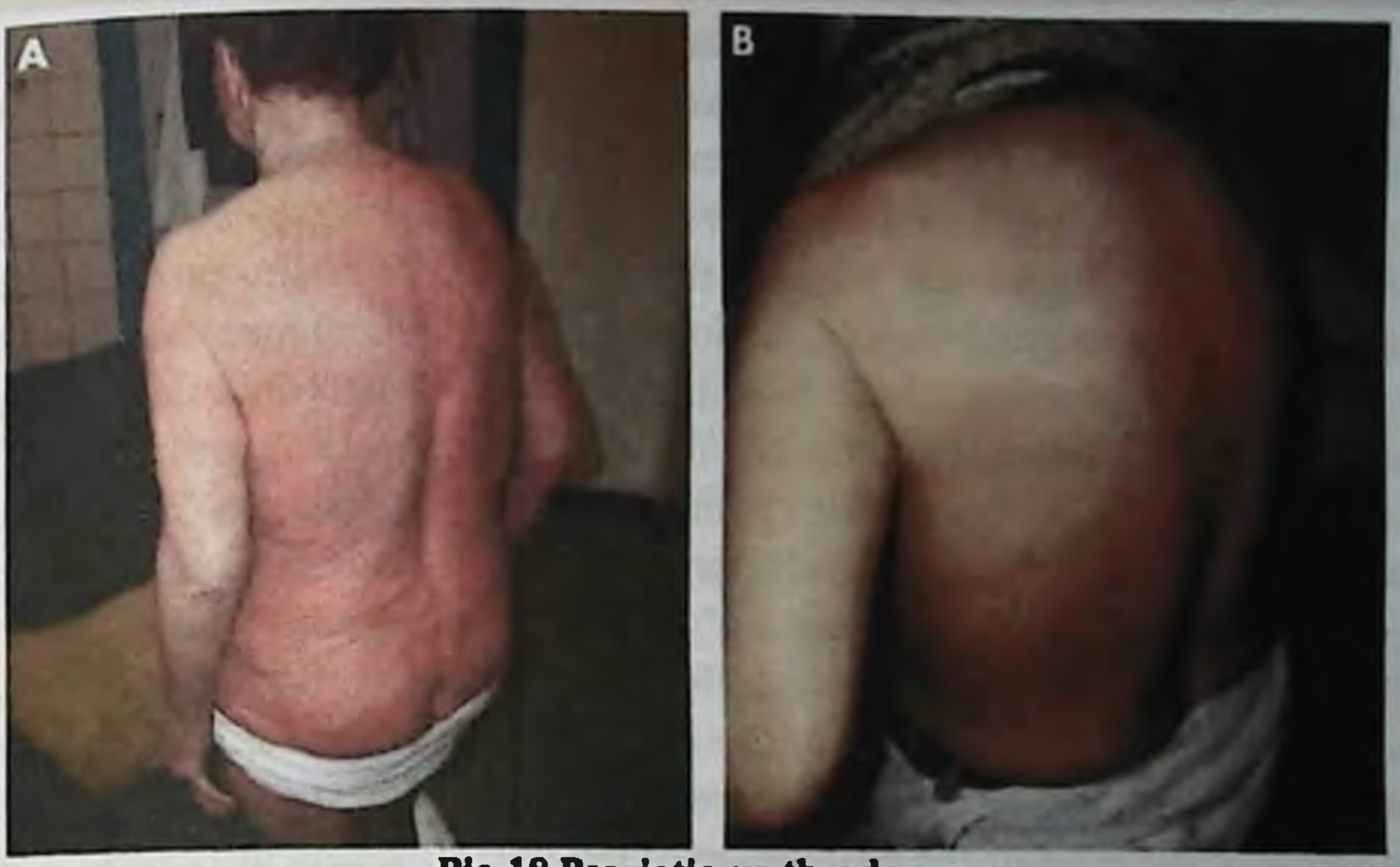
The first type of pustular psoriasis may occur primarily without a previous typical picture of dermatosis, but more often develops secondary as a result of the transformation of psoriasis vulgaris or intertriginous psoriasis.

This is a severe general disease, accompanied by fever, malaise, leukocytosis. First, multiple disseminated erythematous, erythematous-edematous foci appear, which, quickly spreading over the skin, form various figures and scattered bright red foci. Against this background, disseminated pustular elements are located, wrinkling into yellow-brown crusts and crusts. Dermatosis can take a malignant course and end fatally. In the second type, at first, on an erythematous background, small pustular and psoriasis-like foci develop, located on the palms and soles of the hands. symmetric. The contents of the pustules are sterile. Sometimes generalization of the process is possible. Along with the described foci on the palms and soles, other areas of the skin may have typical psoriatic rashes.

Psoriatic erythroderma (erythrodermia psoriatica) develops as a result of exposure to adverse exogenous factors, irritant treatment and is observed in 1.5-3% of patients with psoriasis, often with a decrease in immunological parameters and high allergic reactivity. Stressful situations can also trigger the development of erythroderma. Often the cause of erythroderma does not go away easily. The whole skin is bright red, with a brown tint, tense. In different areas, edema and infiltration are expressed differently. There is a sharp peeling (pityriasis on the scalp, lamellar in other areas), hair falls out, lymph nodes increase, nails are destroyed, purulent paronychia occurs. Without prior psoriasis, this process is difficult to distinguish from other erythroderma. Psoriatic

rashes on the mucous membranes are extremely rare. Damage to the oral mucosa, obviously, is the result of an isomorphic reaction (dental caries, alcohol intake, smoking, etc.). Foci on the mucous membrane of the mouth look like round-oval or irregularly shaped grayish spots with a pinkish-red corolla around the circumference, sometimes slightly rising above the level of the mucous membrane. In about 25% of patients with psoriasis, the nails are affected in the form of punctate onychodystrophy (pinpoint depressions, a symptom of

"thimble"), onychogryphosis (an intensely growing and worn nail plate resembles a bird's beak) and onycholysis (atrophic onychodystrophy). In 10-12% of patients with psoriasis, progressive changes in the joints are associated with skin rashes: arthropathy of the type of deforming polyarthritis, deformities of the joints of the extremities (psoriatic arthritis), stiffness of the spine (arthropathic psoriasis). This most severe clinical form of psoriasis leads to disability and even death. In most patients, joint damage occurs 3-5 years or more after the appearance of the first psoriatic rashes. Sometimes psoriatic arthritis develops simultaneously with skin rashes or precedes them, more often in long-term patients with psoriasis, mainly in men. Inflammatory-dystrophic changes in bones and joints have different intensity and severity. There is a symmetrical multiple lesion of the small peripheral joints of the hands and feet (osteoporosis, mainly in the epiphyses, narrowing of the joint spaces) with a gradual involvement of large joints, sometimes the spine, in the process. In severe forms, resorption of the terminal phalanges of the hands and feet, the appearance of exostoses (osteophytes) are observed. Changes in the joints cause subluxations, dislocations, contractures and ankylosis. Psoriatic arthritis differs from rheumatic arthritis in its negative serological reactions, the absence of acute attacks and heart damage. During psoriasis, there are three stages of the process: progressive, stationary and regressive. In the progressive stage, the appearance of new papules, peripheral growth of old elements and an inflammatory corolla around the papules (corolla psoriatic triad, Koebner phenomenon, the formation of new plaques as a result of the fusion of papules or an increase in old ones) can be noted. plaques. In the stationary stage, fresh elements do not appear, there is no peripheral growth corolla, the Koebner phenomenon is not caused. In the regression stage, the elements of psoriasis flatten, turn pale, peeling decreases or stops, depigmentation of the corolla (pseudo-atrophic Voronov's corolla) appears around the papules, and the elements resolve, that starts from the peripheral or central part and ends with the formation of depigmented cells (less often hyperpigmented) spots. The disappearance of psoriatic rashes indicates remission. After about 20 years, spontaneous remissions are observed in % of cases, usually in the warm season. If the sun exposure causes an exacerbation of the process, remissions occur less frequently. Relapse occurs under the influence of various factors (neuro-



Pic-18 Psoriatic erythroderma

psychic trauma, endocrine disorders, exacerbation of foci of chronic infection, change of seasons, etc.). According to the ratio of climatic and meteorological factors, winter (exacerbation in the cold season), summer and off- season, or mixed, types of dermatosis are distinguished. Winter type of psoriasis is much more common than others. Some scientists distinguish the so-called latent psoriasis, which implies an innate predisposition to the development of dermatoses, metabolic features, and structural and functional changes in the skin. This form of psoriasis can be noted, as a rule, in family members of patients with psoriasis. Histopathology. In the epidermis, parakeratosis, acanthosis in the interstitial areas of the Malpighian layer and thinning of its supraorbital areas, intracellular edema and spongiosis, accumulation in the intercellular spaces of neutrophils penetrating from the dermis (Munro microabscesses) are observed. Above the papule, the stratum corneum is thickened, loosened, and consists mainly of parakeratotic cells with elongated rod-shaped nuclei. Between the plates there are numerous gaps filled with air, which is why the scales have a silvery appearance. In older elements, hyperkeratosis is more pronounced than parakeratosis. papules are found in the stratum corneum regardless of their age. The granular layer disappears or remains as a single intermittent row of cells. In acute cases, there is no shiny layer.

The Malpighian layer above the papillae is thinned, the epidermal outgrowths are elongated. Acanthosis is expressed in the spinous layer between the papillae. The capillaries of the papillary layer are dilated, strongly convoluted, overflowing with blood. In the papillae and the suction layer around the vessels there is an inflammatory infiltrate consisting of lymphocytes and histiocytes. The phenomenon of "stearin spot" is a consequence of the friability of the stratum corneum and the absence of the keratohyalin layer. The phenomenon of "terminal film" develops due to the exposure of a shiny reddish surface, consisting of a number of flattened cells of the spiny layer, as a result of scraping. The phenomenon of "punctate bleeding" is a consequence of a violation of the integrity of the capillary vessels of the papillae, which are easily accessible due to the thinning of the areas of the Malpighian layer above the papillae. An electron microscopic examination revealed intense staining of the cells of the basal layer of the nuclei, which indicates

an increased proliferative activity of epidermal cells. A genetic study reveals an increase in the mitotic index and the content of Burr bodies (a spiralized, genetically inactive form of the X chromosome). Diagnosis of psoriasis is usually not difficult: a characteristic clinical picture, a triad of symptoms ("stearin stain", terminal film, "blood dew"). In differential diagnosis, one should keep in mind psoriasis with pustular syphilis, which is characterized by a pronounced infiltration of elements and positive serological reactions. With lichen planus, the papules are not round, but polygonal in shape, slightly scaly, purple-red.

Psoriasis should also be differentiated from Zhiber's pink lichen, seborrheic eczema, some dermatomycosis and a number of other dermatoses.

Care. For successful treatment, patients must follow the correct regimen. Assign a diet with a decrease in the amount of foods rich in cholesterol (butter, pork, fatty sausages, liver, egg yolk). They recommend fasting days. They provide for a sleep duration of at least 8 hours a day, a daily stay in the air for at least 1.5-2 hours, physical education, smoking cessation and alcohol consumption. Warm baths are shown, especially sea baths.

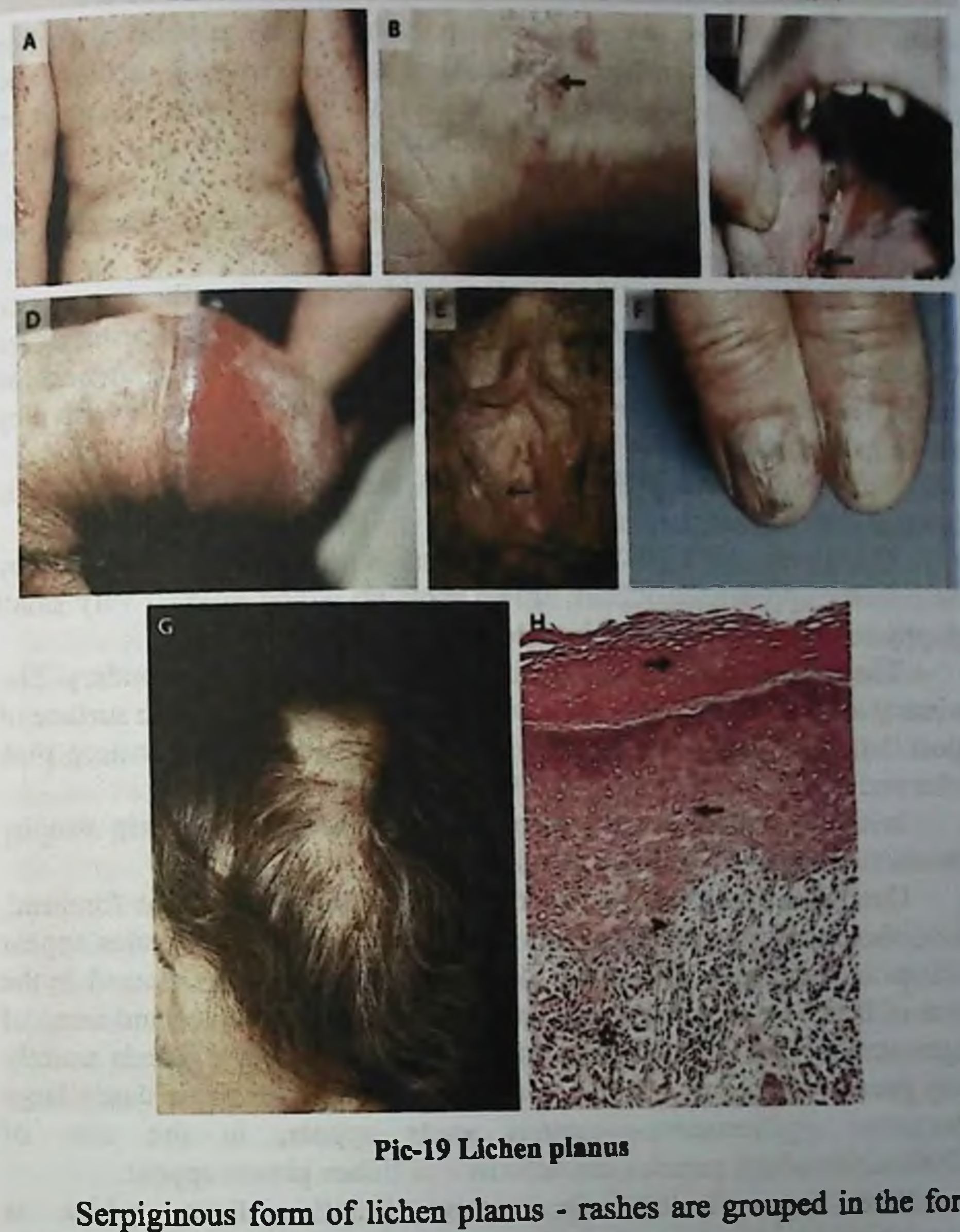
General treatment includes: - detoxification therapy: neogemodez invasions, rheosorbilact; - enterosorbents (enterosgel, activated carbon, carbalong); - drugs affecting the nervous system: sedatives,

tranquilizers, antidepressants - - anti-inflammatory drugs: magnesium sulfate, salicylic acid preparations - - hyposensitizing agents: sodium thiosulfate, calcium preparations - - antihistamines (fenkarol, histafen, tavegil) - - hepatoprotectors, choleretics and agents that normalize the functioning of the gastrointestinal tract (enzymes, probiotics) - immunomodulators, biostimulants, pyrogens - an important place in the treatment of psoriasis belongs to vitamin therapy; Complex vitamins of group B are widely used, especially B1, B2, B5, B6, B12, B15, vitamins A, E, C, PP - biological products. For the treatment of severe forms of psoriasis, glucocorticosteroids (prednisolone, methylprednisolone, betamethasone, dexamethasone), cytostatics (methotrexate), immunosuppressants (cyclosporine), aromatic retinoids (etretinate, acitretin, tigazon) are used. External treatment consists in the appointment of indifferent ointments (boric), keratolytic (2-5% salicylic ointment, karboderm), absorbable (tar and sulfur ointments 2-20%, dibunol liniment 10%, naftalan ointment). The most popular glucocorticosteroid ointments are: non-fluorinated (apulein, advantan, elocom, locoid, hydrocortisone ointment), fluorinated (celestoderm, betamethasone valeriate, fluorocort), difluorinated (sinaflan, flucinar, lorinden, flucar, sinalar), combinations of glucocorticoids and keratolytics (prednicarb, betasalik, diprosalic, lorinden A). In addition, synthetic analogs of vitamin D3 (psorkutan, daivonex), herbal remedies (psoriaten, calendula ointment, fludex) are used externally. Phosphodiesterase inhibitors (5% papaverine ointment). In the winter form of psoriasis, ultraviolet irradiation in suberythemal doses and photochemotherapy are indicated: combined exposure to long-wave ultraviolet rays (PUVA) with photosensitizing agents- psoralens. Resorts with hydrogen sulfide, radon, sulfide, brine baths, mud baths are shown.

Lichen planus (Wilson's lichen) is an inflammatory disease of the skin and mucous membranes, the course of which can be both acute and chronic. Usually the disease occurs in the age range of 30-60 years, more often in women. Clinic. With lichen planus, the skin is more often affected, but there may be an isolated lesion of the oral mucosa and an isolated lesion of the nail plates. The typical form is characterized by a monomorphic rash in the form of small flat papules of a polygonal shape, not prone to peripheral growth. Elements are red-pink in color with a characteristic lilac tint, waxy mother-of-pearl luster and an

umbilical depression in the center 0.2–1.0 cm in size. In the case of merging of individual elements, relatively large plaques are formed. Rashes are localized mainly on the flexion surfaces of the wrist joints and forearms, inner thighs, extensor surfaces of the legs, lateral surfaces of the body, in the genital area. The skin of the face, palms and soles is usually without rashes. Along with the skin oral mucosa may be affected. Mucous membranes are more often affected in the oral cavity (the inner surface of the cheeks, tongue, gums, palate, tonsils), less often in the genitals. First, small papules of a grayish-white color appear, then the papules form plaques covered with a whitish or grayish-white lacy coating. Nail changes are observed in all forms of dermatosis, characterized by the formation of longitudinal ridges, grooves, grooves, the surface of the nail becomes bumpy, there are areas of turbidity. Nails can become thinner, partially or completely destroyed, and therefore there are two types of nail changes - onychorrhexis and onycholysis. With lichen planus in the progressive stage of the disease, a positive Koebner phenomenon is determined (the appearance of rashes in the area of even minor skin lesions) and a positive symptom of the "Wickham lattice" - when the surface of the papules is lubricated with vegetable oil or water, a granular or mesh pattern is determined, due to the uneven growth of the granular layer (granulosis).

Patients are worried about severe itching. When the process regresses, secondary hyperpigmentation usually remains at the site of the papule. In some patients, the process may begin acutely. In acute cases, fever, rapid generalization of rashes, skin edema, erythema are possible, erythroderma may develop with small-plate peeling. Depending on the localization of the elements of the rash, the following forms of lichen planus are distinguished: The ring-shaped form of lichen planus is characterized by the presence of rashes in the form of rings. Ring-shaped elements are usually formed from plaques as a result of the lowering of their central part during regression. Often found in the genital area (glans penis, scrotum). The linear form of lichen planus - the elements of the rash are located linearly along the branches of the nerves of the skin.



Pic-19 Lichen planus

Serpiginous form of lichen planus - rashes are grouped in the form of semicircles or other forms.

Zosteriform lichen planus - elements of the rash are located on one half of the body along the large nerves. The following clinical atypical forms of lichen planus are distinguished: Warty, or hypertrophic form of lichen planus - more often on the lower extremities, in the lumbar region and sacrum, pink-cyanotic flattened warty papules and plaques covered with a small number of scales.. The foci are round or oval in shape, with

clear boundaries. On the periphery, individual small papules are often visible. Distinctive features of this form are excruciating itching and resistance to therapy. Pemphigoid, or bullous form of lichen planus is an exudative form of dermatosis, in which the elements of the rash turn red, blisters and vesicles appear on their

surface, often with hemorrhagic contents. Sometimes blisters form on apparently unaffected skinand mucous membranes.

Follicular, or pointed form - along with flat papules, the appearance of cone-shaped nodules located follicularly is observed. In the center of the nodules is a horny spike, and the affected area feels like a grater to the touch. The elements are never combined. On the scalp, papules resolve by cicatricial atrophy.

The horny form of lichen planus - the surface of the papules is covered with an abundant layer of scales.

The pigmented form of lichen planus is clinically characterized by the primary appearance of dark brown spots on which isolated very small (poppy seed-sized) polygonal papules appear.

The atrophic form of lichen planus can be primary or secondary. The primary atrophic form is characterized by the appearance on the surface of spots that have a rounded shape, with a characteristic purple or deep pink color and thinned epidermis (in the form of papyrus).

In the secondary atrophic form, depigmented areas of skin atrophy remain at the sites of resolved papules.

Coral-like lichen planus - more often on the skin of the forehead, neck, shoulder girdle, abdomen, large, flattened bluish-red papules appear without a central depression, the elements of the rash are arranged in the form of beads or necklaces, alternating with smaller nodules and areas of pigmentation. The erythematous form of lichen planus proceeds acutely with general symptoms (fever, vomiting, diarrhea), in these cases large edematous erythematous-squamous spots appear, in the area of \u200b\u200bwhich papules characteristic of lichen planus appear.

Rare forms include the erosive-ulcerative form, which is characterized by the formation of erosions or small ulcers of irregular or rounded shape with a pink-red velvety bottom on the oral mucosa and on the skin of the legs. Rashes on the mucous membranes are characterized by severe pain. If the oral mucosa is affected, the risk of developing squamous cell carcinoma of the tongue and oral mucosa increases, so such patients are under dispensary observation. According to the prevalence of the pathological process, localized, disseminated and generalized lichen

planus is distinguished, along the course - acute (up to 1 month), subacute (up to 6 months) and with a long course - chronic without remissions or recurring. During the course of the disease, 3 stages are distinguished: progressive, stationary and regressing. The pathohistological picture is characterized by hyperkeratosis, uneven granulosis and vacuolar degeneration of the basal layer. Differential diagnosis should be made with psoriasis, papular syphilis, lupus erythematosus, lichen rosea, limited neurodermatitis. Atypical forms are differentiated from Kaposi's sarcoma, granuloma annulare, warty tuberculosis of the skin, nodular itching, toxicoderma, erythema multiforme.

Care. In acute widespread rashes, antibiotics (macrolides, tetracyclines) and glucocorticoids (prednisolone, diprospan) are used. Sedatives (persen, sedan, bromine preparations), antihistamines (diazolin, tavegil, diphenhydramine), hyposensitizing agents (sodium thiosulfate, calcium gluconate), quinolone preparations (delagil) have a good effect. Systemic immunotropic therapy is used with the use of exogenous interferons (reaferon, interlock) and interferonogens (neovir). With the defeat of the mucous membranes, the use of vitamin A (retinol acetate) is effective for 4-6 weeks. Vitamin E (tocopherol acetate) is used as an antioxidant and allows the complex treatment to reduce the daily dose of glucocorticosteroids. The appointment of a complex of oxidant vitamins, consisting of alpha-tocopherol, beta-carotene, ascorbic acid (tresvit), is effective. It is advisable to use rutin, riboflavin. In severe cases, with generalized rashes, cyclosporine or retinoids are prescribed.

External treatment consists in the appointment of glucocorticosteroid ointments, it is possible under an occlusive dressing, solutions and agitated suspensions with menthol, painkillers, antihistamines, prescribed for intense itching. With erosive and ulcerative foci, epithelial agents are prescribed (solcoseryl, sea buckthorn oil, rosehip oil). In case of damage to the oral mucosa, genital organs, baths or rinses with a solution of sage, chamomile, eucalyptus are prescribed. Among the methods of non-drug therapy, phototherapy, photochemotherapy, PUVA therapy, laser therapy are used. With a warty form, cryodestruction, diathermocoagulation, laser therapy, novocaine blockade are indicated. In the stationary stage of the disease, sanatorium treatment is indicated in sanatoriums with hydrogen sulfide or radon baths.

CHAPTER 6 VIRAL INFECTIONS OF THE SKIN. AIDS. SIMPLE PLANER, PACKAGING IRON. MOLLUSCUM CONTAGIOSUM, GENITAL WARTS, WARTS. SKIN DISEASES IN AIDS.

Human herpes viruses were first identified in 1924. Since then, more than 100 types have been described. All of them are DNA-genomic intracellular parasites. Typical herpesviruses consist of three main components: a nucleotide, a capsid, and a protein-lipid shell. The study of this groupof infections is the most dynamically developing area of modern medicine. This circumstance is explained by several factors. First, herpes infections are ubiquitous. Secondly, they represent a serious economic problem, as they are characterized by chronic diseases. course (lifelong persistence of the virus) with frequent relapses. Thirdly, herpetic infections are the main subject of study in many clinical disciplines . microbiology, dermatology, neurology, ophthalmology, pediatrics, which is explained by the dermatoneurotropic effect of the virus. Fourthly, most representatives of the herpes group have oncogenic properties or are a "background" for oncological diseases. Fifth, herpes infection may be a marker of severe immunoparesis, in particular HIV infection. Herpes simplex (syn.: simple vesicular lichen).

Etiology. The causative agent of herpes simplex is the dermatoneurotropic virus Herpesvirus hominis (HSV) types 1 and 2. This virus belongs to the human herpesvirus genus. Currently, 9 representatives of this genus are known. Common immunobiological features made it possible to distinguish 3 subfamilies - alpha, beta and gamma. Alpha herpesviruses include HSV -1 and HSV

-2 or HCV, varicella-zoster virus, and herpes zoster virus (HSV -3, VHV or VZV). All representatives of the subfamily contain double-stranded DNA, have common structural features and mechanisms of infection and reproduction, have a short reproductive cycle, and are also able to remain in the human body in an inactive (latent) state, mainly in the nerve ganglia. Beta herpesviruses include cytomegalovirus (CMV). The reproductive cycle of this virus is much longer. The reservoir of CMV is the epithelial cells of the excretory ducts of the glands, the epithelial cells of the kidneys and other organs. The infection also proceeds latently. Gamma herpesviruses include Epstein-Barr virus, HSV-6, HSV-7, HSV-8 and HSV-9. The Epstein-Barr virus multiplies only in lymphocytes; the last four viruses have not yet been classified, but are thought to play a role in conditions such as infectious exanthema and chronic fatigue syndrome.

It is believed that HSV-7 is responsible for the development of chronic fatigue syndrome and lymphadenopathy.

HSV-8 was isolated in 1996 from a histological tissue section of an HIV-infected patient with associated Kaposi's sarcoma.

HSV-9 is involved in the formation of secondary immunodeficiencies. HSV types 1 and 2 are closely related - they have almost 50% similarity in the genetic structure. structure. However, each of them is able to produce type-specific proteins that are characteristic only for herpes types 1 or 2. This circumstance allows the use of immunohistological methods to identify a specific

type of herpes in clinically difficult cases. Previously it was thought that HSV-1 affects the skin around the mouth, and HSV-2 is the cause of genital herpes. It has now been proven that any of these types can cause the development of both labial and genital herpes. Pathogenesis. HSV enters the human body through the epithelial surface. After active reproduction within the skin or mucous membranes, the virus infects skin neurons and migrates to sensitive root ganglia, where a latent infection develops.

Primary infection is characterized by severe skin lesions, pain and symptoms of general intoxication. However, an asymptomatic course of the primary infection is also possible, i.e. very often the infection is not diagnosed. HSV is transmitted by direct contact of an infected skin surface of one person with the skin or mucous membranes of an uninfected person. The transmission of herpes infection through public institutions is highly questionable, since the virus cannot survive for a long time outside its usual habitat. It is believed that HSV-1 enters the body in the first three years of a person's life. Most often this happens already when the fetus passes through the birth canal of the mother or when the baby accidentally comes into contact with infected family members. Infection with HSV-2 occurs much later - during the onset of puberty of the individual and the onset of sexual activity. The virus, penetrating through the skin or mucous membranes, enters the regional lymph nodes, blood, and internal organs. In the body, it spreads mainly hematogenously and perineurally, penetrating into the sensory ganglia of the trigeminal nerve and sacral ganglia. In neurons and supporting ganglion cells, the virus genome remains in a subviral form until the end of its life. Even in a latent state, HSV synthesis occurs in small amounts. Immediately after infection, the body produces antibodies to the herpes virus, the titer of which gradually

increases over the next 3-4 weeks and then remains at a relatively constant level throughout life.

Despite the presence of antiherpetic antibodies in human blood, they do not necessarily have a protective effect. In most people, immune mechanisms (mainly cellular) suppress the replication of the virus, thereby preventing the development of the disease clinic. However, in more than 30% of the population, antiviral protection from the immune system does not work, therefore, a clinic of manifest forms of herpes infection develops. Activation of the virus in the latent period occurs as a result of the action of various provoking factors associated with a depressing effect on the immune system. Of these, the most common factors can be distinguished: hypothermia, hyperinsolation, intercurrent viral diseases, injuries, course treatment with steroids and cytostatics, stress. Patohistology. Herpetic lesions of the skin and mucous membranes are characterized by the formation of single-cavity epidermal vesicles due to degenerative changes in keratinocytes in the deep layers of the epidermis. At the same time, areas of ballooning dystrophy and multi-chamber vesicles are found in the upper layers of the epidermis. HSV can cause the formation of intranuclear inclusions in lesions and cells with ballooning degeneration. At the very beginning, the inclusions are diffuse in their development, stained basophilically and fill almost the entire center of the nucleus. Later, they become eosinophilic, more dense and separated from the nuclear membrane with an unstained light halo. Changes in the dermis are characterized by

inflammatory, predominantly vascular reactions: vessel walls are infiltrated with neutrophilic granulocytes, multiple perivascular infiltrates consist of lymphoid cells and neutrophils.

clinical picture. Clinically, four variants of the course of herpetic infection are distinguished.

1. Primary infection. May be accompanied by severe clinical symptoms or be asymptomatic (up to 65% of cases).

2. Non-primary infection. It occurs in people who already have antibodies to one of the types of HSV. And most often there is a variant of non-primary herpes (sexual, non-sexual), when a person who already has antibodies to HSV-1 or HSV-2 becomes infected with HSV-2 or HSV-1 for the first time.

3. Latent infection.

4. Recurrent infection. Recurrent infection may not show up clinically. In 20% of patients, episodes of asymptomatic shedding of the virus are observed.

According to the severity of the course of herpetic infection, there are: degrees: - mild - up to four relapses per year, with isolated foci of rashes, without intoxication and pain syndromes; - medium - more than four relapses per year with limited rashes in various areas, but occurring without signs of intoxication (severe fever) and with locally severe pain; or up to four recurrences per year occurring in different areas. - severe - more than four relapses per year, occurring with severe fever, intoxication, pain syndrome and widespread rashes - rashes. Both the primary manifestation and the recurrence of a herpes infection are characterized by the development of common clinical signs: groups of vesicles with transparent contents ranging in size from 1.5 to

2.0 mm appear, located on slightly hyperemic and edematous skin. Their most typical localization is in places close to natural openings - on the face, around the mouth, nose, sometimes on the mucous membranes of the oral cavity, genital organs, conjunctiva, cornea. It is customary to distinguish between genital and non-sexual herpes. This is due to approximately the same frequency of vesicular elements on the skin surface. on the genitals and elsewhere. Rashes are accompanied by subjective complaints of patients on a burning sensation, itching. Vesicular elements dry up after a few days and turn into serous crusts; with pyogenic flora, crusts can be both purulent and serous-hemorrhagic, depending on the predominance of one or another type of exudate. Bacterial infection of the primary elements is usually accompanied by regional lymphadenitis. After another 2-3 days, the crusts fall off; if the bubbles open, then erosions with scalloped edges are exposed. Erosions are epithelialized; after healing, unstable hypopigmentation remains. The average duration of one episode of herpes simplex is about 10 days. Primary herpes occurs after the first contact with HSV. This often happens in infancy when the immune system is still immature. Primary herpes is characterized by the intensity of clinical symptoms.

In newborns, it proceeds especially hard, according to the type of septic infection, and is characterized by hematogenous dissemination with damage to internal organs (necrotic changes in the parenchyma of the liver, adrenal glands, esophagus, stomach, intestines), oral cavity, skin, substance and meninges (herpetic meningoencephalitis). HSV is usually transmitted to the baby during childbirth through an infected birth canal,

although intrauterine transmission of the virus has become increasingly important in recent years. Transmission of the virus (usually HSV-1) after childbirth is extremely rare. The risk of infecting a child in the presence of genital herpes in the mother is 40%. As a rule, the disease begins on the 5-21st day after infection. It is believed that a primary herpes infection during pregnancy or childbirth is much more likely to cause illness in newborns than to relapse. Individual characteristic skin lesions are observed in approximately 80% of infected newborns.

Acute herpetic stomatitis is one of the most common clinical forms of primary infection. The disease often develops in early childhood. Its occurrence in children is facilitated by teething, in adults - their removal. The incubation period is from 1 to 8 days, after which clinical manifestations develop rapidly - symptoms of intoxication (chills, hyperthermia up to 39-40 ° C, headache, drowsiness, general malaise). In this case, grouped vesicles formed on the oral mucosa can be painful, localized on the buccal surface of the cheeks, on the gum mucosa, tongue, inner surface of the lips, less often on the soft and hard palate, palatine arches., on the tonsils.

Adults can complain not only about the soreness of the rashes, but also about burning, changes in taste sensations. Under the influence of mechanical factors (brushing the teeth, eating), the vesicular elements quickly open with the exposure of an eroded, bright red surface and with remnants of the epithelium on the periphery. When the process is localized on the gum, erosion is more often point-like. As the process spreads, they merge and form more visible areas of erosion with scalloped contours. Vesicular elements are located on an edematous-erythematous background. Regional lymph nodes (submandibular, parotid) react to the process with an increase, often painful, soldered to other tissues.

In weakened children, the process quickly spreads to the internal organs, the central nervous system, and in 25% of cases ends in death. Recurrent herpes is the most common clinical form of herpes in adults. With it, usually the intensity, prevalence, duration of rashes, as well as subjective symptoms are less pronounced compared with acute herpes infection. Extra-sexual localization is more characterized by skin lesions in the area of the nasolabial triangle, conjunctiva and cornea. Genital herpes often affects the genital area, the skin of the buttocks. The course of the disease can be quite long - up to several decades. The frequency of relapses in different patients varies significantly: in some patients, rashes occur 1-3 times a year, in others - up to 5 times a month. Sometimes the

process takes on a long continuous character, when some of the rashes have not yet epithelialized and new elements are already appearing. In women, monthly recurrences of genital herpes may be associated with menstruation. All this leads to mental exhaustion of the patient. The recurrence of herpes is characterized by the development of prodromal phenomena, consisting in the appearance of a burning sensation, itching, tingling at the site of the future rash. Usually, a patient with a history of herpes infection can predict the appearance of a new rash in 12-16 hours. After subjective symptoms, the first signs of an objective clinical picture appear - the skin becomes hyperemic, edematous, later small bubbles with transparent contents appear here. Soon, due to vascular effusion, the contents of the vesicles become cloudy and may become hemorrhagic.

Due to the processes of maceration and traumatization, the vesicles open, forming erosions with scalloped edges. Further, the process can proceed as follows: two options. The first option occurs when the wetting surface of the erosion dries quickly and crusts gradually form. The second option develops in the case of secondary infection, and then not only erosive, but also ulcerative elements with a somewhat compacted bottom and a corolla of inflammatory infiltration along the periphery can form at the site of vesiculation. The average duration of one recurrence of the disease is 10-14 days, but in the presence of secondary pyogenic flora, it can increase. In addition to the classical course of herpetic infection, in 10-30% of cases, atypical forms can also develop: abortive, edematous, zosteriform, disseminated, migratory, hemorrhagic-necrotic, elephantine-like, erosive-ulcerative, rupioid. In these cases, it can be very difficult to make a diagnosis based on the clinical picture alone. The abortive form usually occurs on areas of the skin with severe keratinization and is manifested by papular elements, therefore it is sometimes called pruriginous. Abortive forms also include erythematous and pruriginal-necrotic, in which severe subjective symptoms (itching, burning, soreness) are replaced by either severe swelling and redness of the skin, or the formation of nodules prone to rapid necrotization. The edematous form occurs in areas of the skin with loose subcutaneous fat (paraorbital region, red border of the lips) and is characterized by pronounced tissue edema. Zosteriform recurrent herpes in the clinic is very similar to herpes zoster, as it is located along the nerve trunks, accompanied by neuralgia, headache, and general weakness. In such cases, the diagnosis can only be established based on the results of the virus typing analysis. If several foci of rashes appear simultaneously, then a disseminated form occurs. With a

migratory form, the classic clinical manifestations of herpes simplex often change their localization. In the hemorrhagic-necrotic form, the contents of the bladder become hemorrhagic, and then a focus of necrosis quickly forms at the site of the formed erosive defect. Of particular difficulty is the diagnosis of the ulcerative form of herpes simplex with localization on the genitals. In this case, it is very easy to make a diagnostic mistake and confuse the manifestations of herpes with syphilitic rashes. Ulcerative elements in herpes are formed at the sites of opened vesicles and merged erosions. At the same time, the bottom of the ulcers is soft, juicy, pink-red, sometimes with a grayish-yellow coating.

There is no seal at the base of the ulcer. The lesions are predominantly localized in men on the inner layer of the foreskin, and in women - on the labia majora. Sometimes there is an increase in regional inguinal lymph nodes. The development of these forms of herpes simplex is associated with increased virulence of herpes infection, as well as with a pronounced immunoparetic effect of the virus on the cellular link of immunity. The most common form of herpes is facial herpes. Patients most often associate the manifestation of this infection with hypothermia, climate change, and a stress factor.

Rashes on the face occur sporadically and are localized on the red border of the lips, the skin of the forehead, nose, cheeks. Sometimes the process captures significant surfaces of the skin of the face. Recurrent genital herpes is one of the most urgent medical and social problems of the modern world. Unlike many sexually transmitted infections, genital herpes can affect people who do not belong to the decreed categories, who comply with all norms of moral behavior. Often manifesting genital herpes leads to serious psychosocial problems due to sexual transmission, relapses and lack of effective treatment. HSV belongs to the so-called oncogenic viruses. Many authors point to the "background" nature of herpes infection in a number of tumor processes, such as cervical cancer, prostate cancer, adenocarcinoma and intestinal cancer, nasopharyngeal cancer, neuroblastoma. Some women with cervical cancer have elevated titers of antibodies against HSV-2. In patients with herpetic colpitis and cervicitis in situ carcinoma was detected in 7.0-21.7% of cases. Patients with herpetic diseases of the genital organs should undergo an annual cytological examination (smears from the cervix). as potential precancerous patients. There are a number of publications devoted to the adverse effect of herpes on the course of pregnancy and the important role of this infection in the development of gynecological pathology. So, in

75% of cases of the so-called miscarriage of "unexplained genesis" HSV-2 was detected in the cervical mucus. In 82% of cases, HSV causes long-term inflammatory diseases of the vulva and vagina that are not amenable to traditional therapy; and in 64% of cases, herpes infection in women is atypical. Its most frequent clinical manifestations are persistent itching and burning in the vulva and vagina (65 and 63%, respectively), serous leucorrhoea (58.7%), pseudo- erosion (49.8%) and cervical leukoplakia (16.1%), pelvic ganglioneuritis (29.3%)., acute condylomas of the vulva (27.2%). One of the most dangerous consequences of genital herpes is intrauterine infection of newborns or infection of a child during the passage of the mother's genital tract during childbirth. The risk of developing neonatal herpes varies significantly depending on the form and severity of the herpes infection in the mother and ranges from 0.01 to 75%. The greatest probability of infection with the ensuing consequences for the child is observed during the first infection in the last two months of pregnancy, when a state of viremia occurs. HSV is very often associated with other sexually transmitted pathogens (49%).

At the same time, HSV can potentiate the reproduction, growth and development of one or all pathogens. In the last decade, it has been shown that HSV activates the HIV genome, which is in the provirus stage, and is one of the factors stimulating HIV infection. One of the most severe forms of herpetic infection is Kaposi's eczema herpetiformis (or Juliusberg's acute vaccine-like pustulosis). For more information about this disease, see the "Eczema" section. Diagnosis of herpetic infection in its classical course is not particularly difficult. However, in atypical clinical course, abortive and "erased" forms, laboratory verification methods play an important role in confirming the diagnosis.

organs (in case of death) can be used for laboratory diagnosis of herpes infection. Re-collection of clinical material for repeated studies is recommended, as the diagnostic value of this approach

only increases. In addition, when examining a patient for herpes infection in controversial cases, it is better to use several methods at once (for example, cytomorphological methods and ELISA, RIF and ELISA, etc.). laboratory confirmation of the presence of HSV is the cultivation of the virus, although in everyday practice this is not of great importance. This is due to the availability of faster, relatively cheap and quite informative methods for checking for viruses. It should be remembered that for any method of detecting a virus, the "age" of the element and the place of collection of the material are critical. The best option is to take the

test material from the bubbles. Ulcerative, erosive elements must be fresh, since drying and crusted efflorescences do not provide the substrate necessary for research. All of the above methods allow the doctor to determine whether the clinic of the disease is a manifestation of a herpes infection. But sometimes in the chronic recurrent course of herpes, another problem arises - an adequate immunological response from the patient's body. Then methods come to the rescue, allowing to determine the degree of immunoparesis and possible measures of immunomodulation and control of the effectiveness of the therapy. The main tests of immunological diagnostics are divided, at the suggestion of R. V. Petrov (1984), into tests of the first and second levels. Level one tests are sometimes called orientation tests. They are aimed at determining - the total number of lymphocytes (absolute and relative number) - - the absolute and relative values of T- and B- lymphocytes; – phagocytic activity of neutrophils – – blood levels of the main classes of immunoglobulins (IgA, IgM, IgG) – – complement titer. The second level tests are called analytical tests. These include: - detection of subpopulations of regulatory T-lymphocytes (T- helpers, T-suppressors); - detection of circulating immune complexes (CICS); - detection of spontaneous migration of leukocytes and its inhibition in the presence of PHA, assessment of the functional activity of immunoregulatory cells in RBTL and KOH-A; - detection of B- lymphocytes carrying the following components: surface immunoglobulins of different classes, analysis of the synthesis of immunoglobulins in B-cell culture; - determination of the activity of killer cells, the most important mediators, including interleukins - - determination of complement components, stages of phagocytosis (NCTtest) and the receptor apparatus of phagocytes. The greatest changes in herpetic infection can be found in cellular immunity. Correction of disorders will also be aimed at restoring the quantitative and qualitative composition of lymphocyte populations. Differential diagnosis. Herpetic infection should be distinguished from herpes zoster, streptococcal impetigo. With genital herpes, they differentiate with primary erosive syphiloma, soft chancre, erosive circus balanoposthitis, acute vulvar ulcer, cervical erosion. Herpetic eruptions on the oral mucosa should be distinguished from aphthae, secondary syphilis.

The treatment of herpes simplex is not an easy task, requiring from the doctor not only extensive knowledge about the pharmacokinetics of antiviral drugs, but also the ability to adequately assess the patient's condition, which ultimately affects the choice of drugs. Approaches to the

treatment of herpes infection in Western countries and in the countries of the former USSR differ significantly. So, foreign dermatologists in practice use mainly antiviral chemotherapy drugs. This is explained by the fact that only these drugs have an etiological focus, and therefore there are no significant differences in the treatment of the first clinical episode, relapse of the disease,

and treatment in the interrecurrent period. According to these experts, it is necessary to determine only the frequency of administration and up to the dosage of the chemotherapy drug. As for the duration of the course of chemotherapy, the leading condition is the financial viability of the patient. In contrast to this opinion, our dermatologists are inclined to a different point of view: in addition to etiotropic chemotherapy, great importance is attached to methods of immunological correction, restorative treatment, and specific vaccination. Complex therapy can reduce the frequency of clinical relapses, as well as mitigate their manifestations. The arsenal of etiotropic drugs used to treat patients with herpes has recently expanded significantly. The first synthesized drug with antiviral activity is acyclovir (virolex, zovirax). It is an acyclic analogue of the nucleoside (guanine), a natural component of DNA. It is currently the drug of choice for herpes infections.

The mechanism of action of this drug is associated with its conversion by the enzyme thymidine kinase of virus-infected cells through a series of successive reactions into acyclovir triphosphate, which inhibits DNA polymerase and competitively replaces dioxyguanosine triphosphate in the synthesis of viral DNA. In general, the drug is safe and highly active; in its active form, it selectively inhibits only the synthesis of viral DNA, without affecting the metabolism of the patient's cells. Acyclovir is active not only against HSV-1 and HSV-2, but also against chicken pox and the Epstein-Barr virus. Features of the pharmacokinetics of acyclovir include its weak absorption from the gastrointestinal tract - only 20% of the dose is absorbed. In addition, it is difficult to bind to plasma proteins. These circumstances should be taken into account by the doctor to decide on an adequate dosage and frequency of administration of the drug (at least 4-5 times a day). Acyclovir is contraindicated in pregnancy and in persons with hypersensitivity to it. Acyclovir should be used with extreme caution in patients with severe renal insufficiency. When a recurrence of herpes (oral or genital area) has already occurred, the appointment of antiviral therapy often provides only moderate relief of its symptoms. However, if therapy is started in the prodromal period, its effectiveness can be quite

high. Patients suffering from frequent or severe relapses should be given continuous restraint therapy. They usually start with a dosage of 400 mg 2 times a day or 200 mg 3 times a day, then the dose is increased or decreased depending on the severity of the clinical effect. The course of therapy is 1 year, after which they take a break in taking the drug to assess the need to continue treatment, since it is noted that recurrent infections are characterized by a decrease in the frequency of their recurrence over time. Valacyclovir (Valtrex) is an L -valyl ester of acyclovir, provides a high level of absorption of acyclovir when taken orally, increases its bioavailability by 4-5 times and maintains a high level of its safety and tolerability. It is a specific inhibitor of the herpes virus DNA polymerase. Blocks viral DNA synthesis and virus replication. Valaciclovir is active against Herpes viruses simplex I and II types, Varicella zoster, Epstein - Barr, cytomegalovirus (CMV) and human herpesvirus type 6. For the treatment of herpes simplex, Valtrex is prescribed at a dose of 500 mg 2 times a day. day. The course of therapy depends on the clinical stage of the disease and can range from 3-5, 7-10 days before the prophylactic administration of the drug at 500 mg per day, and then after 1-3 days during the year. Contraindications and side effects of Valtrex are similar to those of acyclovir.

For herpes caused by acyclovir-resistant strains of the virus, other antiviral drugs must be used. new generation drugs. Famciclovir is the active precursor to penciclovir, which is converted to triphosphate in virus-infected cells by the action of virus-induced thymidine kinase. Penciclovir triphosphate exists in the infected cell for more than 12 hours and inhibits viral DNA replication. The action of the drug is similar to that of valaciclovir. Dosage regimen recommended by the manufacturer: 250 mg orally 4 times a day. The duration of therapy is determined by the doctor individually, but on average it is 7-10 days. During pregnancy and lactation, famciclovir can be prescribed only for health reasons. Vidarabine is active against Herpes simplex types I and II, Varicella zoster. Available in the form of a solution and a cream base. It is administered intravenously at a dose of 10-15 mg/kg/day for 5-10 days. Externally applied to the affected areas of the skin 4 times a day with an interval of 4 hours. the duration of treatment is 7 days. Good results were obtained with topical application of the drug in ophthalmic herpes, when vidarabine is applied topically to the conjunctiva 5 times a day. Ganciclovir is a synthetic analogue of guanine, chemically similar to acyclovir. Its greatest activity was observed against CMV, as well as HSV-

1, HSV-2, chickenpox and Epstein-Barr virus. The mechanism of action of the drug is associated with competitive inhibition of viral DNA synthesis, which subsequently leads to the suppression of DNA synthesis due to inhibition of its chain elongation. Ganciclovir suppresses the viral load. DNA polymerase is more active than cellular polymerase. Distributed in all organs, crosses the placenta. The dosage regimen of the drug is individual and, when taken orally, can range from 0.5 g 6 times a day to 1 g 3 times a day. With intravenous administration, the daily dose is 5-10 mg / kg of body weight, the frequency and duration of use depend on the treatment regimen. Contraindications are the same as those of acyclovir. Helepin - in tablets; appoint 0.1 g 3-4 times a day; the course of treatment is 15-20 days. Metisazon - in a tablet of 0.2 g; take 3 tablets 2 times a day for 4-6 hours. day, 1 hour after eating. It is also possible to use other antiviral agents (foscarnet, alpizarin, flacoside, gelpin, ribamidil). In addition to antiviral chemotherapy drugs, interferon preparations and their inducers, which have an indirect antiviral effect, have recently been widely used to treat herpes infection. Interferons in the body of a healthy person are found in trace amounts, mainly in the blood and on the mucous membranes. However, leukocytes of healthy people are capable of synthesizing these glycoproteins upon antigenic stimulation. In patients with chronic viral infections (herpetic infection, CMV, hepatitis, etc.), this symptom is not significantly expressed. In the case of an antigenic attack, all known types of interferons are normally synthesized, but alpha-interferon is of primary importance for local antiviral immunity. human leukocyte alpha-interferon is used as a prophylactic in the form of local applications on the mucous membranes (nasal and oral cavities, genital mucosa) in the acute period of a viral infection. Human immune gamma-interferon modulates the activity of many subpopulations of T- and B-lymphocytes. In this case, the drug can inhibit the synthesis of antibodies and phagocytosis, modify the reaction of lymphocytes. The action of interferon- gamma on human T-lymphocytes lasts about 4 weeks. therefore, maintenance courses of therapy with this drug should be carried out at least once a month. Doses of interferon preparations for parenteral administration are selected individually, since their range is very wide: from several thousand units per 1 kg of body weight to several million units per 1 injection. The course of

treatment is 3-10 injections. Reaferon is a recombinant a2-interferon produced by the bacterial strain Pseudomonas, in the genetic

apparatus of which the human leukocyte gene of a2- interferon is inserted. This drug is completely identical to human a2-interferon. It has antiviral, immunomodulatory and antitumor activity. Assign intramuscularly, subconjunctival and topically. For intramuscular administration, the contents of the ampoule are dissolved in 1 ml of isotonic sodium chloride solution immediately before injection. The drug is administered at 1 million IU 2 times a day for 5-6 days, then for 5 days 1 time per day; if necessary, continue to administer the same dose 2 times a week for 2 weeks. The total dose of reaferon per course of treatment is 15-20 million IU. This drug is contraindicated in allergic diseases, pregnancy. If it is used in the treatment of patients suffering from cardiovascular diseases, it is necessary to carry out hemodynamic control.

Leukinferon is a complex of cytokines of the first phase of the immune response in their natural quantitative and qualitative ratio. In viral infections, interferon should be the leading drug, but since it inhibits hematopoietic processes, it is used in conjunction with leukinferon (one ampoule 1-2 times a week). In mixed infections, most likely caused by viruses (for example, infections of the urogenital tract - STI)), leukinferon is also used in combination with human interferon, but at a lower dose (0.25–1 million IU). However, leukinferon can have side effects: in addition to exacerbating the underlying disease, it can cause severe toxic reactions, as well as leuko- and thrombocytopenia. Neovir stimulates the rapid formation of endogenous interferons in high titers. This drug is indicated for recurrent herpetic infection of various localization, with STIs. Take 6 tablets at a time per os (without chewing), at the same time, half an hour before meals (days of admission: 1st, 3rd, 6th, 9th, 12th, duration of treatment 12 days); or injected intramuscularly at 250 mg 1 time in 2 days; for a course of 7-9 injections. Cycloferon is a synthetic analogue of a natural alkaloid obtained from a Citrus culture grandis. It has an immunostimulating, antiviral and anti-inflammatory effect, normalizes the immune status of the body, stimulating the production of interferon both in immunodeficiency and in other autoimmune diseases. The drug is characterized by low toxicity and the complete absence of mutagenic, teratogenic, embryotoxic and carcinogenic effects. It is administered intramuscularly or intravenously at a dose of 0.25 g once a day for 2 days, then every other day. The average course of treatment is 10-15 injections; if necessary, fix the effect can be repeated after 10-12 days.

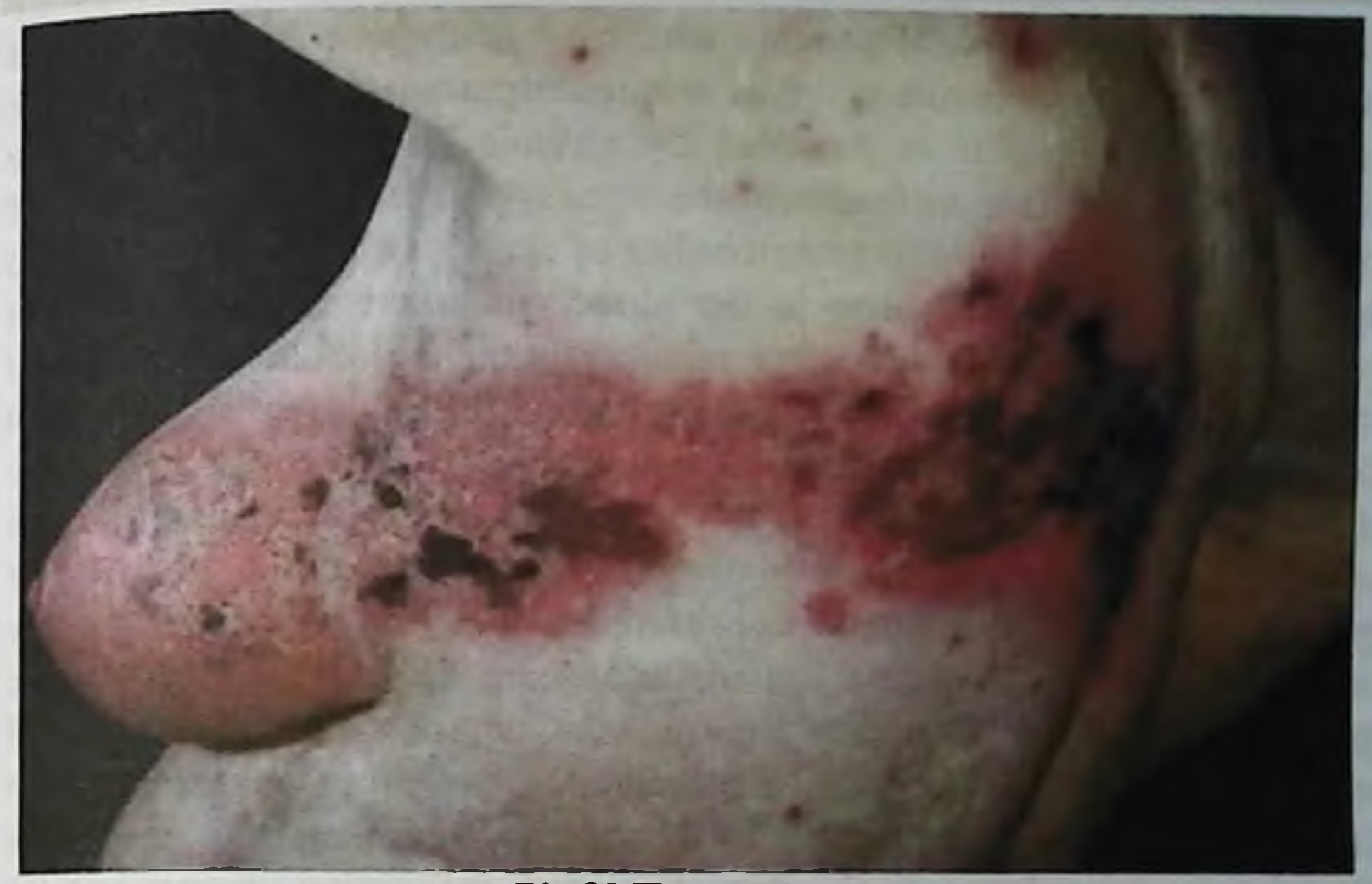
Immunomodulators belonging to the group of nucleic acids also have a pronounced antiviral effect: poludan (polyadenyluridylic acid complex), inosine pranobex or isoprinosine (inosine complex with acetylamidobenzoic acid). Groprinosin (inosine pranobex), along with an immunostimulating effect, also exhibits antiviral activity. The drug enhances the differentiation of pre-T-lymphocytes, stimulates mitogen-induced proliferation of T- and B-lymphocytes, increases the functional activity of T-lymphocytes, including their ability to form lymphokines. Groprinosin significantly enhances the production of interleukin-2 by PHA-activated human lymphocytes and promotes the expression of this interleukin receptor on lymphoid cells. The drug has a thymosin-like effect, stimulating predominantly cellular immunity, and is especially effective in conditions of cellular immunodeficiency. Groprinosin is able to potentiate the action of a number of antiviral drugs,

such as acyclovir, interferon. It is administered orally at 50-100 mg/kg of body weight per day, and this daily dose is divided into equal parts; they are taken at intervals of 6-8 hours. The course of treatment is 5-8 days; repeats during exacerbations. All drugs from the group of nucleic acids are pronounced inducers of endogenous interferon. With prolonged administration, it should be remembered that the RNA and DNA precursors contained in them induce the growth and reproduction of both eukaryotic and prokaryotic cells, i.e. may well stimulate the growth and reproduction of bacterial flora. The arsenal of topical antiviral drugs is also quite wide. When prescribing them, one unshakable rule should be remembered - most of them are effective only in the prodromal period, when there is no vesiculation yet. It is believed that when vesicular rashes appear on the skin and mucous membranes, the processes of viral DNA replication are completed, which means that the main effect of local antiviral drugs does not work, in this case only a "softening" effect can be expected from them. Cream Acyclovir 5% (virolex, zovirax, herpevir, medavir) is recommended to be applied to the affected areas 5 times a day until complete regression of clinical manifestations. Vira-MP (vidarabine phosphate 10%) - gel; it is recommended to apply it on the skin 4 times a day for 7 days. Gossypol-liniment 3% is applied as local applications 4-6 times a day for 5-7 days. Gavisosh (isopropyl deoxyuridine) - ointment; applied to the affected surface 3-5 times a day for 3-12 days. In addition, tromantadine 1%, rhyodoxol ointment 0.25–0.5–1.0%, oxolinic ointment 0.25–3.0%, tebrofen ointment 0.5–5.0%, alpizarin ointment 2.0% are used. -5.0% -

they are also applied to the affected surface 3-5 times a day for 3-12 days. In the presence of erosions and pronounced edema, lotions with DNase, with poludan (a complex of RNA-containing polyadenylic and polyuridylic acids), with a 0.5% solution of zinc sulfate are used. Antiseptic solutions and aniline dyes are also widely used. To stimulate the epithelialization of erosions, laser therapy is applied locally. Good results have been obtained when used as a prophylactic and therapeutic agent. means of topical application of the Spanish drug epigen-intim, which is an extract of licorice root. Produced in aerosol form. For prophylactic purposes, to prevent new episodes of herpes infection, an inactivated herpes culture vaccine is used, containing an HSV virus of antigenic types I and II, inactivated by formalin, grown on a culture of chick embryo fibroblast tissue cells. The vaccine is used only during the period of remission, not earlier than 2 weeks after the complete disappearance of the clinical manifestations of herpes infection. ophthalmic herpes - not earlier than after 1 month. In parallel, as a general strengthening therapy, it is recommended to prescribe vitamin therapy (antioxidant complex), biogenic stimulants. The drug is administered in a single dose of 0.2 ml (control - the formation of a "lemon peel"). The course of treatment of herpetic infection of the skin and mucous membranes consists of 5 injections, which are made at intervals of 3-4 days. The main course includes 1 or 2 such cycles with an interval of 7-10 days between them. After 6 months, a second course of vaccination is carried out (1-2 cycles of 5 injections). During a clinical episode of a herpetic attack of genital herpes, the doctor should advise the patient to refrain from sexual intercourse until the clinical picture of the disease has completely disappeared. During this period, the use of barrier contraception (condom) should be mandatory for all sexual intercourse. Sexual partners of patients with genital herpes should be evaluated and treated if they have herpes.

Herpes zoster (syn.: herpes zoster).

Etiology and pathogenesis. The disease is caused by the Varicella virus. zoster (HSV -3, VOG or ZVZ), which is the causative agent of both chickenpox and shingles. The ZVZ virus, like HSV types I and II, belongs to the alphaherpesvirus family, and therefore has all the characteristic features of this group: a short reproductive cycle, rapid spread through cell culture with apronounced cytopathic effect.



Pic-20 Herpes zoster

For example, the ability to remain inactive in some structures of the central nervous system (mainly in the paravertebral ganglia). However, there is no unequivocal relation to the last statement, since after chickenpox in childhood, stable lifelong immunity remains normal and herpes zoster mainly develops in individuals who cannot indicate a history of chickenpox in childhood. It is believed that chicken pox is a disease of childhood, and shingles is a disease of mature and senile age. This circumstance is explained by many features of the reactivity of the organism at different stages of life. But even in the elderly, a past infection should normally leave a strong immune system. If the disease is prone to relapse, then this should greatly alert the doctor. Most often, recurrent herpes zoster is one of the signs of a paraneoplastic process, therefore, depending on the location of the rash, it is necessary to conduct a diagnostic search aimed at excluding the tumor process. Other reasons for the reactivation of the ZVZ virus can be: chronic diseases (tuberculosis, diabetes mellitus, collagenoses, etc.), acute, severe infections (flu,

SARS, pneumonia, pleurisy, malaria, erysipelas, AIDS, etc.), hypothermia., radiation exposure, etc. About 500,000 new cases of the disease are registered annually in the United States. At the same time, approximately 5% of patients are HIV-infected, and 5% are cancer patients. In the group of HIV-infected patients, 25% of patients suffer from herpes zoster (this is 8 times higher than the average for the age group of

20-50 years) and 7-9% of patients who have undergone kidney or heart transplantation and receive systemic immunosuppressants. The appearance of rashes on the skin is preceded by viremia. A few days after the formation of vesicles, antibodies to the virus appear in the blood serum of patients, represented by immunoglobulins of classes G, M, A. Acute-phase IgM are the first to circulate in the blood, and secretory IgA and IgG appear a little later. The latter are memory immunoglobulins and can be stored in sufficient quantities for a long time (almost for life). Within a few days after the onset of the disease, indicators of cellular immunity are sharply reduced. Patohistology. Morphological skin changes in herpes zoster are similar to those in vesicular lichen simplex, but more pronounced. In the epithelial cells of the basal layer, ballooning dystrophy is noted, due to a sharp intracellular edema, the cell nuclei are pycnotically changed. Inside the nuclei, inclusions in the form of eosinophilic bodies are found. Intracellular edema is combined with intercellular edema, which leads to the formation of blisters in the upper parts of the germ layer. In the dermis, a weak infiltration of neutrophilic granulocytes is detected, which then migrate into the epidermis. In addition, the nerve trunks and the corresponding roots of the sensitive ganglia are affected. In the affected nuclei in ganglion cells, eosinophilic bodies are detected, and with electron microscopy, the herpes virus is detected. Viral particles are also found in the endotheliocytes of skin capillaries and in axons. clinical picture. Shingles is characterized by seasonality. Most often occurs in the transitional periods of the year (spring, autumn). It is characterized by damage not only to the skin and mucous membranes, but also to the structures of the nervous system. The most severe occurs in the elderly and senile age; children under 10 years of age are extremely rare, men get sick more often than women. The prodromal period is characterized by the following phenomena: general infectious disease: hyperthermia (low-grade fever), feeling unwell, headache, often accompanied by nausea and vomiting, general weakness. Neuralgia (often unilateral) of a certain zone of innervation develops somewhat later or simultaneously. The duration of incubation for herpes zoster has not been established.

The clinical picture is characterized by pronounced polymorphism. On hyperemic and somewhat edematous skin along the sensitive branch of the nerve and its branches, there are a few spotty- nodular rashes. After a few hours, they turn into vesicles, 0.2-0.5 cm in diameter, sometimes bullous rashes form. The vesicles are filled with serous contents, which

over time (3-4 days) become cloudy, become hemorrhagic, and with secondary pyogenic flora - purulent. Usually on the 4-5th day after the appearance of rashes, the cavity elements dry out with the formation of yellow-brown crusts. The latter disappear by the end of the 2-3rd week, exposing hypopigmented spots with fresh epithelialization. More often, the process is resolved without a trace. Zoster- herpes does not have a typical localization. Rashes can be found on any part of the skin and mucous membranes (in the mouth, nose, genitals). Most often, the elements of herpes zoster are

located on the trunk along the intercostal nerves, on the limbs, along the branches of the trigeminal nerve, on the scalp, face, etc. The process is characterized by asymmetry of the rashes. There are several clinical forms of herpes zoster. A typical form has been described above. The abortive form of the disease is manifested only by the appearance of a few papular rashes against an edematous-erythematous background. At the same time, the rashes are confined to certain areas, the sensitive innervation of the skin and mucous membranes is preserved. Ulcerative, and as an option - ulcerative-necrotic form, occur when the vesicular elements are prone to rapid opening and the formation of erosive-ulcerative defects. Soon, as a result of the rapid drying of the exudate, the ulcers are covered with massive dense crusts, under which necrosis processes begin to develop rapidly. This form is prone to protracted flow; clinical rashes usually resolve not earlier than after 1-1.5 months. Often, ulcerative elements are resolved due to the formation of cicatricial atrophy. Generalized (common) form of herpes zoster occurs in exceptional cases (2- 4%). In this case, the disease manifests itself as usual, but in the future the process tends to spread rapidly. As the number of vesicular elements on the skin and mucous membranes increases, the severity of the pain syndrome begins to gradually weaken. In most cases, the appearance of elements of the rash is accompanied by an increase in regional lymph nodes, the appearance of polyadenitis. This form is more common in individuals with severe forms of secondary immunodeficiency, developing as a result of systemic use. immunosuppressants (corticosteroids, cytostatics), as well as in cancer patients. The gangrenous form of herpes zoster is the most severe. It is more common in people with severe concomitant pathology of internal organs: gastric ulcer, chronic enterocolitis, diabetes mellitus. Primary blisters are no different from those in the classical course of lichen. But then they quickly change the serous contents to hemorrhagic and, upon opening, immediately expose the ulcerative surface. Ulcerative defects

tend to grow peripherally - deep gangrenous foci are gradually formed. ulcers with offensive discharge. protracted process; after resorption of ulcerative elements, scars form on the skin. A distinctive feature of zoster, unlike, for example, herpes simplex, is the development of pain in the vast majority of cases, which is associated with the involvement of the sympathetic nervous system in the pathological process, since this virus has neurotropism.. One of the most severe complications after the resolution of rashes is the development of postherpetic neuralgic syndrome. At the same time, in places of former rashes, pronounced painful sensations of varying intensity persist for months, and sometimes for several years. In general, postherpetic neuralgia occurs in 10-15% of patients, but its frequency increases sharply with age. So, in people older than 60 years, this complication is observed in more than 50% of cases. Most often, postherpetic neuralgia resolves within the first 12 months. The ophthalmic form of shingles deserves special attention. It is caused by a virus infection of the ophthalmic branch of the trigeminal nerve, and in a third of patients, in addition to it, the nasolabial nerve is also affected. The variety and severity of ophthalmic herpes are associated with the anatomical features of innervation (eyeball, mucous membranes of the upper part of the nasal cavity, frontal and ethmoid sinuses). This situation requires mandatory consultation of an ophthalmologist. Most often, vesicular rashes form on the skin of the wings and the tip of the nose. Often, the process, spreading to the eye, affects the cornea, conjunctiva, sclera, iris, ciliary body. In such cases, an objective examination reveals photophobia, blepharospasm, lacrimation, etc. If the cranial ganglion of the facial nerve is involved in the process, a picture of the Hunt triad appears - facial nerve paresis, ear pain, vesicular rashes on the skin of the corresponding dermatome. With the defeat of the nodes of the vagus and glossopharyngeal nerves, unilateral herpetic eruptions appear on the mucous membranes of the hard and soft palate, pharynx, posterior third of the tongue and pharynx. The lids of the vesicles open quickly, exposing eroded areas with scalloped edges. As a result, a picture of aphthous stomatitis is formed. In patients with normal immunity, vesicular elements disappear 2-3 weeks after their appearance. But complications are possible, which can be local or general. Local complications include bleeding and gangrene; common complications include meningoencephalitis, cerebrovascular accident, Hunt's syndrome, paresis, myelitis, congestive pneumonia, hepatitis, pericarditis, myocarditis,

pancreatitis, esophagitis, enterocolitis, cystitis, arthritis, disseminated skin lesions, bacterial superinfections.

Diagnostics. The diagnosis of shingles is based on the following characteristics. Firstly, this infection is characterized by polymorphism and asymmetry of rashes - papules, vesicles, pustules and erosion can be observed in one area of the skin. Secondly, the correct collection of anamnestic data is of some importance when it is possible to find out that the patient had contact with a patient with chickenpox (or shingles); a correct assessment of the patient's physical characteristics can also help in establishing the diagnosis. Thirdly, the installation of the Tcank probe is also important for diagnosis. Additional laboratory tests are usually not required to diagnose this disease, but in the case of an atypical course, cultivation of ZVZ is recommended, although this method is not recommended. this process is much more labor intensive, time consuming and expensive than HSV cultivation. In clinical conditions, sufficient information can be obtained by setting up immunological methods for recording the humoral response to the introduction of the ZVZ virus and to virus-specific antigens found in infected cells. These are methods such as RIF (direct and indirect), ELISA. They are described above, in the section on herpes simplex, and are not fundamentally different. Differential diagnosis. Shingles should be distinguished from bullous erysipelas, acute eczema and hepatocholecystitis, angina pectoris, food intoxication (before the appearance of rashes), from the zosteriform form of herpes simplex.

Care. The main directions in the treatment of herpes zoster are: elimination of pain, acceleration of healing of rashes, relief of mental and physical suffering, prevention of dissemination of infection and other complications, reduction in the frequency of post-herpetic complications, prevention of the spread of infection. Among the antiviral drugs in the treatment of herpes zoster, acyclovir is the most widely used. Moreover, it should be prescribed as early as possible - in the first 2-3 days from the onset of the disease. The dosage of the drug must be adequate. When taken orally, the drug is recommended to prescribe in a single dose of at least 400-600 mg, and sometimes 800-1200 mg 5 times a day. This approach allows us to hope for a favorable prognosis for the development of postherpetic neuralgia. In the presence of conditions for intravenous infusion, the drug is prescribed at 800-1000 mg 3 times a day. The duration of the course is determined individually. Other antiviral chemotherapy drugs described above, such as famvir and

valocyclovir, are also effective. Widely used in the treatment of patients with herpes zoster immunopreparations with a pronounced antiviral effect - interferons and their inducers, groprinosin, etc. (appointment regimens are described above). From the very beginning of the appearance of rashes, the doctor should make every effort to stop the symptoms of sympathetic pain. Non-steroidal anti-inflammatory drugs (NSAIDs) are the most widely used drugs. In addition to analgesic, they also have an anti-inflammatory effect, which plays an important role in the pathogenesis of herpes zoster neuralgia, when nerve trunks swell and pinch in narrow bone spaces. NSAIDs that have the most pronounced analgesic and anti-inflammatory effect include ortofen. Assign 25 mg 3-4 times a day or intramuscularly 3.0 ml daily. Indomethacin is also one of the most active anti-inflammatory drugs.

Taken during or after meals, 25 mg 3-4 times a day or as rectal suppositories, 0.05 g each. Acetylsalicylic acid (aspirin) is prescribed 0.25 g 3 times a day. Side effects of NSAIDs are also associated with COX inhibition and are expressed mainly in the ulcerogenic effect of these drugs, therefore, people with gastric and duodenal ulcers should be prescribed them with great caution. With severe pain syndrome, it is recommended to use ketorolac either orally at 10 mg 3-4 times a day, or intramuscularly at 1 ml 3-4 times a day. However, this drug is not recommended for long-term use due to severe side effects. Recently, new NSAIDs have appeared with a selective nature of COX- II inhibition, which makes it possible to avoid dangerous ulcerogenic complications. One of them is nimesulide, administered orally at 100 mg 2 times a day. Another selective COX- II inhibitor is meloxicam. It is supposed to be 100 times more potent in inhibiting COX- II than COX- I and superior to other NSAIDs in this respect. Its daily dose can vary from 7.5 to 15 mg. This drug is available in the form of tablets of 7.5 and 15 mg and in the form of rectal suppositories of 15 mg. If anti-inflammatory drugs do not work, you can use the so-called triplets (diphenhydramine, analgin, papaverine), ganglion blockers (gangleron - 1 ml intramuscularly, pahikarpin - 0.05 g 3 times a day). Novocaine blockades, electrophoresis with novocaine during the spread of pain, analgesics have an analgesic effect. Herpes zoster is one of the few conditions in dermatology that allows the use of even narcotic analgesics if traditional pain therapy is ineffective. In order to eliminate the swelling of the nerve trunks, rehydration therapy is prescribed. It boils down to the appointment of furosemide 0.04 g in the morning or in the form of injections of a 1% solution of 2 ml.

It should be remembered that this drug removes potassium from the body, so people with heart failure, as well as long-term use of the drug, it is prescribed against the background of potassium- containing drugs. medicines. You can also prescribe diacarb in tablets of 0.25 g 1 time per day; hypertonic glucose solution (40% solution - 20 ml), magnesia sulfate (25% solution - 5–12 ml). In secondary purulent infection, broad-spectrum antibacterial drugs are used: tetracyclines (tetracycline, metacycline, vibromycin), macrolides (erythromycin, spiramycin, roxithromycin), azalides (azithromycin), cephalosporins (cephalexin, ceftriaxone) and other antibiotics. They are prescribed in an average therapeutic dosage for a course of 7-10 days. In the early stages, vascular drugs (antiplatelet agents, angioprotectors) are used to prevent microcirculation and

intravascular coagulation disorders: curantyl 25 mg 3 times a day for 5-7 days; pentoxifylline 100 mg 3 times a day. Vitamin preparations of group B (B1, B6, B12); vitamins A, E and C; multivitamin complexes; biogenic stimulants (plasmol, FIBS, gumizol, vitreous body, splenin); adaptogens are widely used (drugs of ginseng, eleutherococcus, Manchurian aralia). External therapy for herpes zoster depends on the stage, severity of the process, as well as on the complications that have arisen. Therefore, at different stages of the disease, antiviral, antimicrobial and keratoplastic agents can be used: aqueous solutions of aniline dyes, fucorcin; brilliant green solution; acyclovir, interferon, heliomycin, tetracycline ointments; synthomycin emulsion. With gangrenous and necrotic forms, jelly or ointments of solcoseryl, iruksol, as well as glucocorticosteroid ointments are used externally.

1. Lichenoid parapsoriasis is characterized by all of the following except:

a) Small lichenoid brownish-reddish papules with pityriasis scales; b) brownish pigmentation with telangiectasias after scale involution; c) slight purpura after scraping; d) severe itching; e) long course and resistance to therapy.

2. In the pathogenesis of rosacea, the following factors are important, except:

a) angioedema;
b) exposure to the sun;
c) diseases of the gastrointestinal tract;
d) endocrinopathy;
e) gluten intolerance.

3. Seborrhea contributes to the development of all of the following diseases, except:

a) acne vulgaris;
b) rosacea;
c) seborrheic dermatitis;
d) rhinophyma;
e) xanthelasmas.

4. Trichonodiasis is:

a) nodular formations on the hair shaft;
b) bunches of short hair;
c) twisted hair;
d) spindle-shaped hair;
d) matted hair.

5. In arthropathic psoriasis, it is advisable to prescribe all of the following, except:

a) penicillin and prednisolone;
b) non-steroidal anti-inflammatory drugs;
c) oxyferriscarbon;
d) detoxification agents;
e) antioxidants.

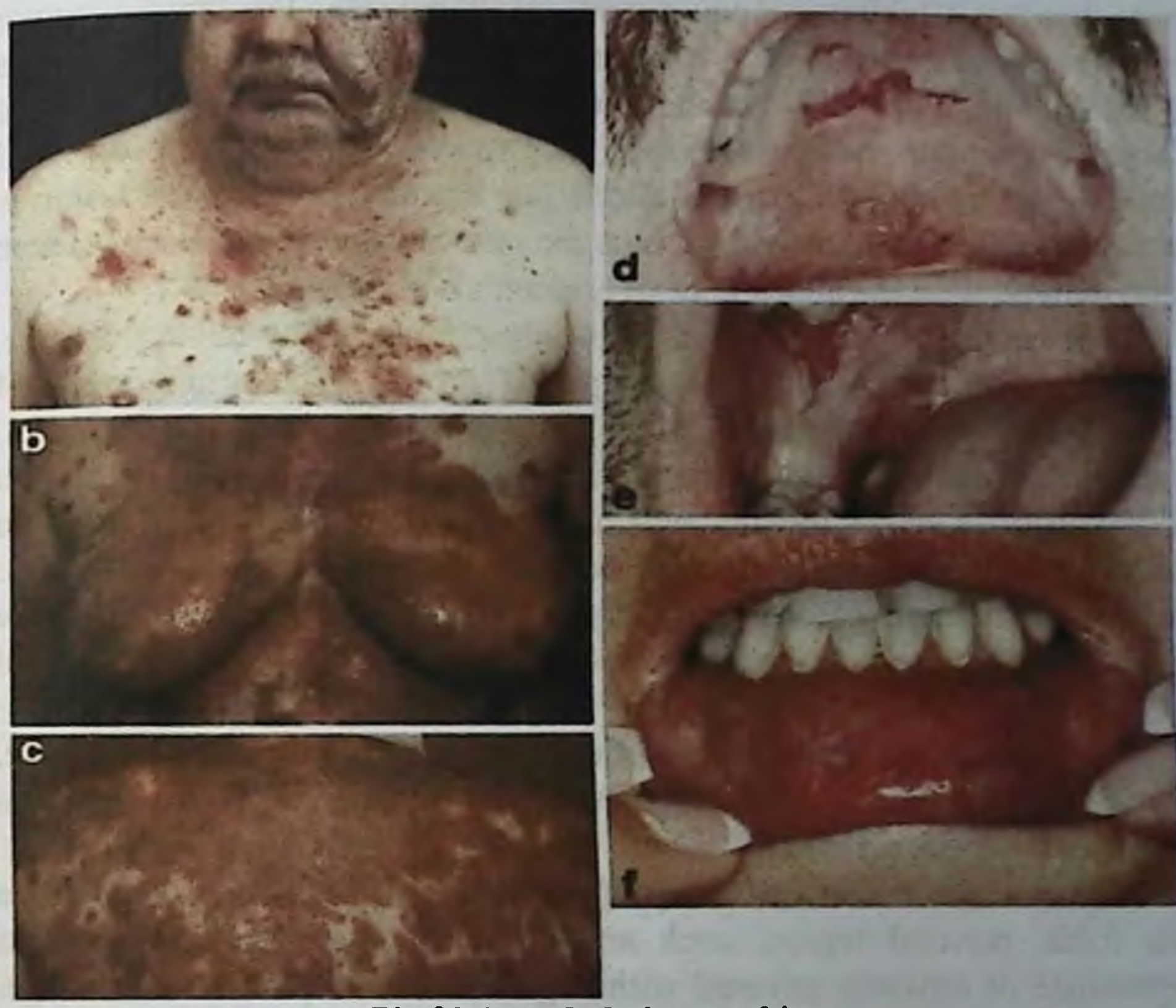
6. Typical elements of the rash in lichen planus have the following symptoms, except:

a) flat, polygonal, reddish-lilac papules;
b) depressions in the center of the papule;
c) waxy shine;
d) Wickham nets on the surface of the papule;
e) predominant localization on the face.

7. Manifestations of plaque parapsoriasis are characterized by the following features,except:

a) yellowish-brown spots with clear boundaries;
b) slight peeling with small scales, sometimes imperceptible, detected on screening;
c) predominant localization on the trunk and lower extremities;
d) no itching;

CHAPTER 7 VESICULAR DERMATOSES. ACANTHOLYTIC ULCER. WITH TUBERCULOUS DERMATITIS. LEVER PEMPHIGOID. COMPARATIVE DIAGNOSIS OF BLISTERING DERMATOSES.



Pic-21 Acantholytic pemphigus

Acantholytic pemphigus is a severe dermatosis with a chronic undulating course, which is based on an autoimmune lesion of the skin and mucous membranes, manifested by acantholysis and blistering. Etiology and pathogenesis are unclear. There are various theories of the onset of the disease: viral, neurogenic, endocrine, metabolic. The leading one is autoimmune. As for the pathogenesis of the disease, it is generally accepted that acantholysis is mandatory.

Breaking the bonds between epidermal cells is mediated by autoantibodies (IgG). They bind to glycoproteins of cell membranes and cause acantholysis. Epidemiology. The frequency of occurrence of true

pemphigus among skin diseases on average does not exceed 1-1.5%, in other words, up to 80% of cases of true pemphigus account for vulgar (common) cases. In recent years,

cases of the disease among young people aged 18 to 25 have become more frequent. Children andadolescents rarely get sick. The familial nature of this pathology has not been confirmed. Pemphigus vulgaris often occurs in the age range of 40-60 years, men and women get sick equally often. Clinic. Pemphigus vulgaris usually begins with a lesion of the oral mucosa, and a skin rash appears after a few months. Initially, the lesion is local in nature, and after 6-12 months the rash becomes generalized. There is no itching, with the appearance of erosion - burning, pain.

On externally unchanged skin, intraepidermal flabby blisters appear, which increase in size due to peripheral growth. The arrangement of elements is chaotic. The lid of the bladder is easily torn and red, painful, weeping erosions are formed that do not tend to heal. A positive symptom of Nikolsky is characteristic, which is determined both directly in the lesion and on the skin of healthy skin. Often rashes are located on the mucous membrane of the mouth, nose, larynx, pharynx, vagina. The bubbles here open so fast that it is very difficult to see them. In place of the bubbles, painful erosions form.

As a rule, the general condition is disturbed: weakness, malaise, weight loss, hoarseness, dysphagia, septic fever, and complications from the internal organs are noted.

Other clinical forms of pemphigus Vegetative pemphigus affects the skin folds, perioral region, neck and scalp. It is characterized by the appearance of erosions covered with vegetation, with purulent discharge. The transition of ordinary pemphigus to vegetative and vice versa is possible. Pemphigus foliaceus is characterized by the appearance of blisters against a slightly hyperemic background, which, when opened, form erosions covered with leaf-like multilayer crusts. In most cases, blisters are indistinct, as they open immediately after formation. The preferred localization is the face, scalp, upper chest, abdomen, but it is possible that the entire skin is affected - exfoliative erythroderma. Erythematous (seborrheic) pemphigus often occurs in people 40-50 years old. It can be transformed into a regular or leaf-shaped form.

First, erythema-squamous rashes appear, resembling seborrhea or discoid lupus erythematosus, localized on the face and scalp. The rash spreads to the skin of the chest and interscapular region. Blisters may appear that are covered with crusts of a grayish-brown color.

Diagnostics. Diagnostic criteria for acantholytic pemphigus include clinical and laboratory signs of acantholysis in the epidermis and epithelium of the mucous membranes.

There are seven main criteria:

1. A characteristic clinical picture of the lesion: blisters on intact skin, long-term erosion on the skin and on unchanged skin of the oral mucosa, conjunctiva, nasal mucosa, and genital organs. On the edge of the erosions, the remains of bubble tires can be observed.

2. Nikolsky's symptom on apparently unchanged skin. With light friction of healthy-

looking skin near the blisters, and sometimes at a distance from them, detachment of the surface layers of the epithelium occurs with the formation of erosions. This symptom is considered the most informative.

3. The marginal symptom of Nikolsky - when pulling with tweezers for fragments of the bladder lid, the epithelium exfoliates beyond the boundaries of visible erosion.

4. Asboe-Hansen symptom - pressing the bubble causes an increase in its squares.

5. The cytological diagnostic method (Tsanku cytodiagnosis) provides for obtaining smears-imprints from the bottom of fresh erosion. To do this, use dry alcohol-free glass, which is tightly applied to the surface of fresh erosion. To obtain smears from erosions on the mucous membrane of the hard, soft palate and pharynx, indirect scraping is used to obtain them. Soft scraping of the erosion surface is carried out with a blunt spatula or Volkmann's spoon, avoiding visible damage to the surface and bleeding, after which the material taken from the bottom of the erosion is carefully removed. put on a glass slide in the form of a smear. The resulting smears are dried and stained according to Romanovsky-Giemsa. Further microscopy of the preparations reveals acantholytic Tcank cells.

These are modified cells of the spinous layer that have undergone acantholysis, which have degenerated and differ from the normal cells of this layer: - they are rounded (oval), scattered, acantholytic cells are smaller in size than normal epidermocytes; - nuclei of acantholytic cells are intensely stained; - two or three large nucleoli can be found in the enlarged nucleus - - the cytoplasm of the cells is sharply basophilic, unevenly colored; a light blue zone is formed around the nucleus, and along the periphery there is a thickening of the color in the form of an intense blue rim - acantholytic cells in pemphigus can form symplastic cells with several nuclei.

6. The histological method of research is one of the main and mandatory in confirming the diagnosis. A fresh vesicle or marginal zone of erosion must be cut with the capture of unscattered skin. The earliest histological changes in the epidermis are intracellular edema and the disappearance of intercellular bridges in the lower part of the spinous layer. Due to acantholysis, cracks form inside the epidermis, and then bubbles are formed, located above the basal layer of cells. A characteristic histological feature is the identification of individual altered spinous cells, which, having lost contact with each other, remain attached to the layer of unchanged basal cells.

7. Immunomorphological studies in some cases play a decisive role in the diagnosis of pemphigus. Already in the early stages of the disease, direct immunofluorescence (DIF) can detect deposits of immunoglobulins on cryostatic sections of the skin or mucous membrane of class G and complement located in the intercellular spaces of the epidermis. Indirect immunofluorescence detects high titers of autoantibodies (IgG) to proteins in the blood and bladder fluid. The height of their titers directly correlates with the severity of pemphigus.

Differential diagnosis is carried out with Dühring's dermatitis, other bullous dermatoses, aphthous stomatitis, Lyell's syndrome and severe forms of toxicoderma. Vegetative pemphigus is differentiated with wide condylomas, vegetative pyoderma, iodo- and bromoderma, seborrheic pemphigus - with seborrheic eczema and discoid lupus erythematosus.

Care. General treatment is the appointment of glucocorticosteroids. An initial dose of prednisolone 80–100 mg/day is usually sufficient to stop the process, sometimes higher doses of

200 mg/day or more are required. The dosage should be at least 1 mg / kg / day. Other corticosteroids (dexamethasone, metipred, etc.) are prescribed in doses equivalent in effectiveness to doses of prednisolone. It is advisable to take 2/3 of the dose after breakfast, 1/3 - after dinner. Pulse therapy is indicated for patients in extremely serious condition - 1000 mg of prednisolone per day in the form of single intravenous infusions. With an adequate dose, a distinct therapeutic effect (cessation of the formation of new blisters, active epithelialization of erosions) occurs after 10-14 days, and it is possible to reduce the initial dose of glucocorticosteroids by 1/4-1/3 from the initial one. dose. Then the dose of prednisolone is gradually reduced to maintenance slowly over several months. Usually patients receive glucocorticosteroids for life.

To increase the effectiveness of glucocorticosteroid therapy and to reduce their dose, cytostatics are prescribed: inside azathioprine 2.5 mg / kg or cyclophosphamide 100-200 mg / day, the duration is determined individually or intramuscularly, methotrexate 10-20 mg 1 time per week (for course 3 -5 injections). With insufficient therapeutic efficacy of glucocorticosteroids and the presence of contraindications to the use of cytostatics, immunosuppressants are prescribed - cyclosporine orally at 5 mg / kg / day in 2 doses, until a clinical effect is obtained, then the dose is reduced to the minimum maintenance dose.. It is necessary to correct and prevent side effects caused by long-term use of glucocorticosteroids, the most severe of which can be fatal.

The main complications of steroid therapy include: 1. Exogenous (drug) Itsenko- Cushing's syndrome with all its manifestations. Its severity decreases with the transition tomaintenance doses of glucocorticosteroids.

2. n immunosuppressive state, manifested by the addition (exacerbation) of a purulent, fungal or viral infection. As a result, it becomes necessary to use general therapy with antibacterial, antifungal or antiviral drugs.

3. Violations of the water-salt balance in the body: - hypokalemia is manifested by a violation of the conduction of the heart muscle, therefore potassium preparations are prescribed (asparkam, panangin, potassium orotate, etc.); - hypocalcemia can cause paresthesia, spasms of striated muscles, osteoporosis and osteomalacia, the development of pathological fractures, in this regard, calcium preparations (calcium gluconate, oxidevit, calcitrin) are prescribed; diuretics.

4. Hypoproteinemia, which develops as a result of protein catabolism, may manifest as protein-free edema due to a decrease in plasma pressure. In this regard, patients are prescribed anabolic hormones (retabolil or nerobol intramuscularly every two to three weeks).

5. Gastritis, esophagitis, gastric and duodenal ulcers, which can lead to severe complications - gastric bleeding or perforation of the ulcer. This circumstance necessitates a sparing diet and prophylactic use of antacids, histamine H2 receptor blockers. The occurrence of peptic ulcer during treatment with glucocorticosteroids dramatically worsens the prognosis of the disease. It is strongly recommended to reduce the daily dose of the hormone while taking cytostatics, switching to injectable use of glucocorticosteroid drugs.

6. Diabetes mellitus develops due to the stimulation of gluconeogenesis and the peripheral anti-insulin action of

glucocorticosteroid hormones, in connection with which the patient is transferred to a hypocarbohydrate diet (Table 9a). With severe hyperglycemia, in addition to the diet, antidiabetic drugs are prescribed, recommended by the endocrinologist.

7. Mental disorders of the patient (insomnia, euphoria, agitation). Sleeping pills and sedatives are prescribed, if necessary, a consultation of a psychoneurologist.

8. Muscle atrophy, vascular fragility, hypercoagulation syndrome, the appearance of atrophic streaks on the skin, steroid acne, etc. Externally, aqueous solutions of aniline dyes, corticosteroid ointments or creams (celestoderm B with garamycin, kremgen, trimistin, polcortolone), agents that improve epithelialization are used. (panthenol, methyluracil ointment), irrigation with anesthetics, baths with potassium permanganate. Recommended diet: exclude irritating and rough foods, salty, smoked meats, animal fats, carbohydrates. They recommend foods rich in protein and vitamins.

1. The primary element of the lesion in lichen planus:
- 1. bubble
- 2. bubble
- 3. knot
- 4. knot
- 5. bump

2. The Latin name of the primary element of the lesion in lichen planus:

1. knot
- 2. ulcer
- 3. papule
- 4. bubble
- 5. tuberculosis

3. Lichen planus refers to:
- 1. to dermatoses
- 2. to allergic conditions
- 3. to bacterial infections
- 4. to viral infections
- 5. obligate precancerous conditions

4. Secondary element of the lesion in lichen planus:
- 1. plate
- 2. ulcer.3) erosion

- 3. peel
- 4. 1), 2) and 3) are correct

5. Atypical form of lichen planus is localized on:
- 1. cheek
- 2. Lip
- 3. gums
- 4. language
- 5. sky

6. The combination of erosive lichen planus with diabetes mellitus and arterial hypertension is asyndrome:
- 1. Grynszpan
- 2. Melkerson-Rosenthal
- 3. Behceta
- 4 Stevens-Johnson
- 5. Shegrena

7. Greenspan's syndrome - severe course:
- 1. leukoplakia
- 2. lichen planus
- 3. chronic recurrent aphthous stomatitis
- 4. erythema multiforme exudative
- 5. recurrent herpes

CHAPTER 8 KERATOMYCOSIS, RINGWORM, CANDIDIASIS. ONYCHOMYCOSIS.

Fungal diseases of the skin (mycoses) is a collective term for lesions of the skin, nails and hair caused by fungi of the genera Trichophyton, Microsporum, Candida, Epidermophyton and, less commonly, mold fungi. Fungi are widespread in the environment and live in the soil, on plants, parasitize animals (zoophilic) and humans (anthropophilic or zooanthropophilic). Routes of infection: - direct route - contact with soil, plants, sick animals or people - - indirect route - through things and objects used by patients, or through animal care items.

Keratomycosis

Diseases of this group are characterized by a predominant lesion of the stratum corneum and hair cuticle with little or no inflammatory reaction and involvement of the skin appendages. versicolor versicolor (versus versicolor). Synonyms: pityriasis versicolor, malassezia skin. The disease was first described by Robin in 1853. Etiopathogenesis and epidemiology. Multicolored versicolor is widespread. It is estimated that up to 10% of the world's population suffers from it. In hot countries, versicolor is more common. In mid-latitudes, most cases of the disease occur in the summer. More often people of young and middle age are ill. The peak incidence occurs at the age of 20 years. The disease is rare in children and the elderly. It is possible to transmit the pathogen from a patient with multi-colored lichen or a carrier, for example, in a common bed, infection on the beaches is also likely.

Lichen versicolor is not considered highly contagious health status. Children can become infected from adults due to the friability of the stratum corneum. The disease is prone to relapse. The causative agent is Malassezia furfur or Pityrosporum orbiculare - lives in the scales of the stratum corneum of the epidermis. Pathogenesis factors: increased sweating, impaired carbohydrate metabolism, vegetodonia; in children - malnutrition, immaturity and friability of the stratum corneum of the epidermis, irrational hygiene regimen. clinical picture. The most common localization of the lesion: chest, back, less often - shoulders, abdomen and scalp in children, but without damage to the hair. The disease is characterized by the appearance of spots of coffee- milk or grayish-pink color of various shapes and sizes, tending to merge. There is no inflammatory response. Occasionally on the surface there is pityriasis versicolor.

Subjectively, only sometimes patients feel a slight itch. After ultraviolet irradiation (more often after sunburn), the colonies of the fungus die off, exfoliate, leaving behind depigmented spots (secondary leucoderma). Diagnosis is based on characteristic clinical symptoms. Confirmation of the diagnosis is carried out by positive iodine test and washing with aniline dyes.

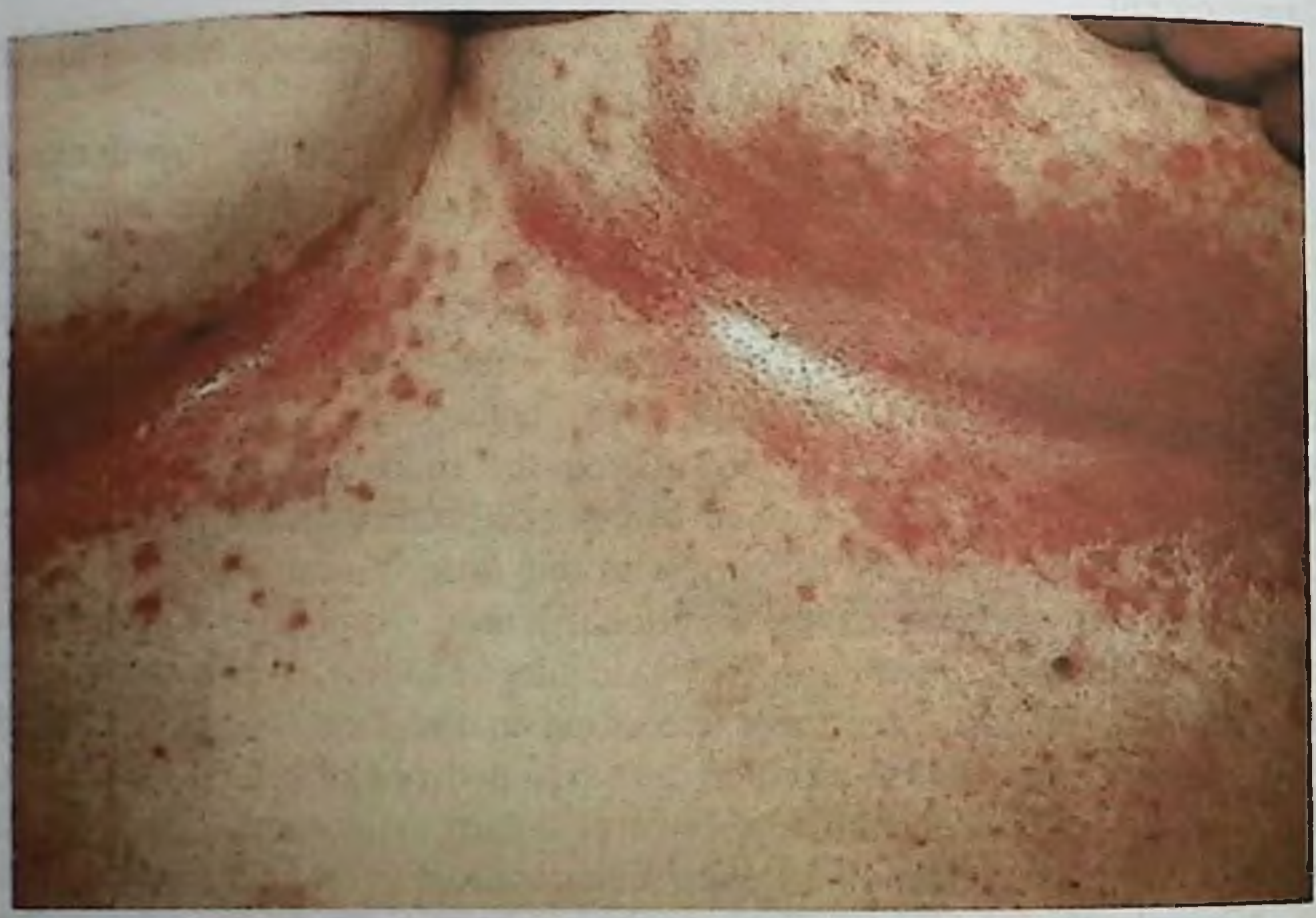
At the same time, due to the more intensive absorption of the reagent by the loosened cornea, lesions are clearly distinguished. The diagnostic technique "strike of the nail" ("chip phenomenon", or Besnier's symptom) can be used, which reveals hidden peeling. Microscopic examination of pathological material (scraping from skin rashes) is desirable. Luminescent diagnostics using a Wood's lamp is possible, revealing lesions in the form of dark brown or reddish-yellow staining.

Treatment for limited forms, as a rule, is carried out by external therapy. With common forms and ineffectiveness of local therapy, treatment with a systemic antimycotic is prescribed. Ketoconazole has long been used to treat Malassezia infections. Recently, modern triazole antimycotics have also been prescribed: itraconazole and, much less frequently, fluconazole. Despite the emergence of new drugs, ketoconazole continues to be considered the most active drug against Malassezia. The low sensitivity of Malassezia to terbinafine explains the ineffectiveness of systemic therapy with this drug. Externally, exfoliating and fungicidal preparations are traditionally used. Apply 3-5% resorcinol or salicylic alcohol, nitrofungin, 3-5% tincture of iodine, 5-10% mineral or tar ointment. Possible treatment according to the Demyanovich method (60% sodium hyposulfite solution and 6% hydrochloric acid solution), 20% benzyl benzoate emulsion, Nizoral cream, Miconazole, antifungal aerosols. Treatment lasts 10-14 days. Ketoconazole is effective in the form of Nizoral 2% shampoo. For systemic therapy, you can prescribe ketoconazole (Nizoral) 200 mg / day and itraconazole (Orungal) also 200 mg / day for 1 week. There is evidence of the effectiveness of fluconazole ("Diflucan"), administered once at 400 mg / day. Treatment should end with a course of UV therapy until the skin color is normalized. cure criteria. Treatment is carried out until the disappearance of clinical manifestations. Microscopic examination of pathological material should give a negative result. Prevention. To prevent relapse, it is important to eliminate sweating and treat visceral pathology. It is advisable to examine all family members of the patient or those in close household contact.

Disinfection of underwear, bed linen and clothing should be mandatory. Shampoo containing ketoconazole can be used to wash the body. In the spring, general UVI is recommended.

Candidiasis of the skin and mucous membranes

Candidiasis (syn. - candidiasis, soormycosis) is a fungal disease of the skin, nails, mucous membranes caused by yeast-like saprophytic fungi of the genus Candida (or Monilia). First described by B. Langenbeck in 1839, fungi in tissues were first discovered in 1843 by Berg, and in 1923 Berhaut isolated the genus Candida from yeast-like organisms. Etiology and epidemiology. About 150 species of fungi of the genus Candida are known.



Pic-22 Candidiasis Of The Skin And Mucous Membranes

About 20 types of pathogens can cause candidiasis. Of these, patients most often distinguish eighttypes. Among these eight, in turn, four types-C are the main ones. albicans, C. _ tropicalis, C. _ parapsilosia, C. _ glabrata. C. _ albicans - unicellular microorganisms of relatively large size, oval, round shape, form pseudomycelium (threads of elongated cells), blastospores (kidney cells sitting on pseudomycelium constrictions). The main features that distinguish fungi of the genus Candida from true yeast are the following: the presence of pseudomycelium, the absence of ascospores (spores in sacs inside cells) and their characteristic biochemical

properties. The disease is widespread throughout the world. Candidiasis differs from other opportunistic mycoses in that it is mainly endogenous infection. The most common causative agent of candidiasis is C. albicans and other pathogenic species of Candida - permanently or temporarily live on the mucous membranes or skin of a person, most often in the intestines.

Exogenous infection is less common. Some types of Candida are found in the soil, contaminate food: vegetables, fruits, meat and dairy products, bakery products. Perhaps intrauterine infection and infection of the newborn during passage through the birth canal. Sexual transmission is not uncommon. In addition to carriers and people, infection with candidiasis can occur from pets.

Currently, the prevalence of opportunistic yeast-like fungi of the genus Candida is increasing

among healthy people and patients with mycoses. There is an increase in the incidence of candidiasis among people of different age groups. However, children and the elderly are more commonly affected.

Pathogenesis and risk factors. In the pathogenesis of candidal infection, the decisive role belongs not so much to the pathogen as to the state of the macroorganism. At the same time, the pathogenic properties of fungi (secretion of proteolytic enzymes and hemolysins, dermatonecrotic activity and degree of adhesiveness) play a role in the development of the disease. Like most deep forms of candidiasis, superficial forms are classified as endogenous infections, with the source of the pathogen in the patient's own body.

Nosocomial and HIV-associated infection usually presents only with oral candidiasis. A number of factors increase the risk of this disease: contact with patients with candidiasis, high humidity and ambient temperature, human contact with rotting vegetables, fruits, berries, chronic infections (pneumonia, tuberculosis, etc.), cancer, metabolic disorders, metabolic disorders substances, hypovitaminosis, vegetovascular dystonia. The development of candidiasis is facilitated by the use of antibiotics, corticosteroids, cytostatics, and radiation therapy. Favorable background - endocrinopathies (diabetes mellitus, etc.), immunodeficiency states, AIDS. The development of candidiasis in children of the first year of life is facilitated by frequent regurgitation with the reflux of acidic stomach contents into the oral cavity, which creates a favorable environment for the growth of fungi of the genus Candida. In older people, wearing dentures has a great influence on the development of the disease. The main clinical forms of candidiasis:

1. Superficial candidiasis (candidiasis of smooth skin and mucous membranes, candidal onychiaand paronychia).
2. Chronic generalized (granulomatous) candidiasis.
3. Systemic (visceral) candidiasis.
4. Candidamycins.

Candidiasis of the skin and nails Candidal intertrigo. A disease of smooth skin, often begins with the defeat of large folds (intertriginous candidiasis, candidal intertrigo). In adults, the folds of the perineum, buttocks, groin and armpits are affected, the folds on the abdomen and neck in obese people, in women the vulva and skin under the mammary glands are often affected.

Subjective sensations include itching and burning, and in cases of advanced or complicated bacterial infection, pain. With secondary pyococcal infection, regional infections, lymphadenitis occurs. At the beginning of the disease, a whitish strip of macerated stratum corneum appears in the depth of the folds. Here surface cracks and erosion are formed. Then erythematous-edematous foci with vesicles, papules, pustules, erosions with weeping are formed. The affected areas have clear polycyclic edges bordered by a hidden halo of exfoliating epidermis. The surface of the erosions is smooth, shiny, bluish-red, sometimes macerated with a whitish bloom. New foci appear along the periphery as a result of autoinoculation. Candidal diaper rash is differentiated from inguinal epidermophytosis, erythrasma, seborrheic eczema, psoriasis of the folds.

With the spread of inflammation from the folds to neighboring areas, candidiasis of smooth skin occurs. There are erythematous and vesicular varieties. Smooth skin lesions can occur spontaneously anywhere under compresses, wet dressings, or prolonged bathing. In women breastfeeding newborns with oral thrush, lesions appear in the periarticular region. The course of the disease is acute, the foci tend to resolve after the elimination of factors provoking maceration. Differential diagnosis of such lesions is carried out with microbial eczema. Candidiasis of the interdigital folds. It is more often observed in school-age children and people working in personal plots, as well as in confectionery and fruit and vegetable enterprises. In the folds of the hands or feet between the 3rd and 4th or 4th and 5th fingers, maceration foci appear with stagnant- hyperemic skin, surrounded by a covered exfoliated rim of the epidermis. On the periphery there are rashes in the form of seropapules and vesicles.

Differential diagnosis is carried out with dyshidrotic eczema and lesions caused by dermatophytes. Candidal onychia and paronychia. They

occur more often in association with smooth skin or mucosal lesions, but may be isolated in defeat. Paronychia is characterized by edema, infiltration, hyperemia of the nail folds, and the absence of supraungual skin (epinichia). The supranail ridge moves along the surface of the nail. Often paronychia and onychia co-exist. The introduction of fungi occurs from the lateral areas, the periarticular roller. Therefore, the free edge of the nail remains intact. The lateral edges of the nail are deformed - as if cut off. The surface of the nail is also deformed and has an uneven thickness. Candidiasis of the oral mucosa and pharynx Candidiasis of the oropharynx is divided into acute and chronic. The latter is subdivided into recurrent and persistent. By localization, candidal stomatitis, glossitis, tonsillitis, pharyngitis, gingivitis, as well as cheilitis and angular stomatitis (congestion) are distinguished.

The most common form of oropharyngeal candidiasis is known as "thrush". Any part of the oral cavity and pharynx can be affected, most often the cheeks, less often the palate, tongue, then the rest of the departments. First, pinpricks appear, white grains, then films resembling yogurt and having a curdled appearance. The plaque is easily removed by scraping with a spatula, after which you can see its bright red base, sometimes with a bleeding surface. Lesions are painless, although erosion and ulceration may occur with injury and the addition of bacterial flora. In the presence of erosion, the plaque may acquire a brownish-brown hue. With the defeat of the tongue(glossitis), plaque is observed not only on the back of the tongue, but also on the lateral surfaces, in the folds; the tongue is enlarged due to edema, the filiform papillae are flattened. With candidal tonsillitis on the tonsils, in addition to plaque, whitish plugs form, but swallowing is painless, the temperature is low. body temperature does not rise, regional lymph nodes are not enlarged.

Cheilitis is characterized by redness of the red border of the lips, its dryness, burning, tightening, gray scaly scales. Yeast glossitis and stomatitis should be differentiated from desquamative glossitis, leukoplakia, lichen planus. Candidiasis of the corners of the mouth (angular stomatitis, hyperemia) can form independently without affecting the oral mucosa. The skin in the corners of the mouth becomes inflamed, macerated, moist, covered with a white, easily removable plaque, after the removal of which a red smooth eroded surface is exposed. The process is often bilateral. Sometimes it can spread to the red border of the lips. Differential diagnosis should be made with bacterial, usually streptococcal, congestion, syphilitic papules, and chancre.

The chronic form of oropharyngeal candidiasis occurs in patients with HIV infection, AIDS and other forms of immunodeficiency. It is characterized by a long persistent course, resistance to therapy. Clinical signs are frequent lesions of all parts of the oral cavity, sometimes the presence of more difficult to detach films with an erosive, bleeding base. 8.3.3. Urogenital candidiasis According to the clinical course of candidal infection, a primary episode of urogenital candidiasis and recurrent urogenital candidiasis (with 4 or more relapses per year) are distinguished. A severe variant of the most recurrent form is persistent vulvovaginal candidiasis. With it, the symptoms of the disease persist constantly with varying degrees of severity, usually subsiding after treatment. By localization in women - vaginitis, candidal cervicitis, colpitis, urethritis, bartholinitis; in men - balanitis and balanoposthitis. In women, subjective symptoms of vulvovaginal candidiasis are itching and burning, pain. These symptoms may be constant or worsen during the day, evening or night, or after walking for a long time. In the area of the vulva and labia itching is intense, accompanied by scratching. Sometimes it prevents you from urinating. Pain and burning worse during intercourse. With the development of candidal vulvitis or vulvovaginitis, spilled edematous-hyperemic foci appear with the presence of a white point of the smallest plaque. Plaque is easily removed, under it an erosive surface with serous or serous- hemorrhagic discharge is exposed. When making a differential diagnosis, the most common vaginal infection, bacterial vaginosis, should be considered first. At the same time, subjective symptoms (itching, burning, dysuria) are characteristic of candidiasis, bacterial vaginosis and other vaginal infections. In men, candidiasis most often occurs after sexual intercourse. Burning and itching in the area of the glans penis may appear within a few hours. In mild cases, they last no more than 1-2 days and disappear, resuming after the next sexual intercourse. There is moderate hyperemia, slight superficial peeling. Separations are rare. Papules may appear on the skin of the head and foreskin, turning into superficial pustules and vesicles, and then into characteristic rounded erosions, bordered by a white stripe of macerated epidermis, with polycyclic outlines when confluent. There may be a whitish coating on the surface of these elements. erosion formation occurs most often on adjacent areas of the scalp and the inner leaf of the preputial sac. Candidiasis of the urethra is observed much less frequently. Unlike candidal balanoposthitis, banal balanoposthitis is accompanied by severe hyperemia, weeping, the absence of a characteristic lesion border, and often an unpleasant odor.

Differential diagnosis should also be carried out with primary and secondary periods of syphilis,

erosive rashes observed in acute bacterial infections, sexually transmitted infections. Diagnosis ofcandidiasis is based on the presence of a clinical picture characteristic of the patient and laboratory examination data. When making a diagnosis of superficial forms of candidiasis, it is sufficient to detect fungal elements during microscopic examination of pathological material (pseudomycelia and budding yeast cells). Skin flakes, scrapings from nails and mucous membranes are examined. Sowing on the Sabouraud nutrient medium and isolating the culture of the fungus are necessary to determine the type of pathogen. At the same time, the detection of fungi in the absence of symptoms of the disease is not an indication for treatment. However, at present, polymerase chain reaction, immunofluorescence reactions, passive hemagglutination, etc. are also used to diagnose candidiasis.

Treatment of candidiasis Before starting treatment of a patient with superficial forms of candidiasis, especially those resistant to previously used methods of therapy, it is necessary to organize a study of the patient's blood sugar and consult an endocrinologist. With all forms of candidiasis, it is necessary to strive first of all to eliminate the factors contributing to the onset of the disease. Treatment should be comprehensive and based on the appointment of pathogenetic and strengthening treatment, sanitary and hygienic measures, and then the use of general and external antifungal drugs. Systemic therapy. The leading antifungal drugs of general action for a long time were polyene antibiotics: amphotericin B, nystatin, levorin, amphoglucamine, mycoheptin, pimafucin. Amphotericin B is administered intravenously at 250 IU per 1 kg of body weight, per course - 2 million IU, vials of 50,000 IU + 5% glucose ampoule as a solvent. Currently, it is used exclusively for health reasons for the treatment of patients with chronic, generalized (granulomatous) candidiasis, sepsis, with severe damage to internal organs. Nystatin

- a daily dose (for adults 6-10 million IU) is taken in 4 doses, tablets of 250,000 IU or 500,000 IU, the course of treatment is up to 4 weeks. Levorin - ampoules of 200,000 units of a solution in 5 ml of H2O, inhalations 1-2 times a day for 15 minutes (7-10 days). Amphoglucamine tablets

0.1 g (100,000 IU) 2 tablets. 2 times a day (3-4 weeks). Mycoheptin - tablets of 0.05 g. 250 mg 2 times a day after meals for 10-14 days. Pimafucin (natamycin) - tablets of 100 mg. Assign 1 tablet 4 times a day

after meals (from 10 days to several months). Currently, imidazole derivatives are widely used clotrimazole, ketoconazole ("Nizoral"), miconazole, econazole. Capsules Nizoral take 0.2 g 2 times a day for 2-4 weeks to 1 year on the background of pathogenetic therapy. Among modern fungicidal preparations for the treatment of candidiasis, triazole derivatives are increasingly used: fluconazole (diflucan, mycosist), as well as itraconazole (orungal, orunite). Adults with persistent thrush with candidal glossitis are prescribed fluconazole according to the following scheme. The first day - 400 mg 1 time per day, then 200 mg per day, the duration of the course is 7-14 days. Intaconazole is prescribed 100-200 mg per day for 7-20 days. During pregnancy and lactation, systemic drugs are not used. Outdoor therapy. Preparations for local etiotropic therapy of candidiasis are divided into antiseptics and antimycotics. Antimycotics (polyene antibiotics, imidazoles, triazoles) are prescribed in the form of solutions, aerosols, gels, creams, ointments, drops, chewable tablets. Antiseptics (diamond green, methylene blue, Castellani liquid, etc.) with antifungal activity are usually prescribed in

the form of lubrication or rinses. Lubrication is carried out with 1-2% alcohol or aqueous solutions, applying them to previously dried surfaces. Antiseptics are less effective than antimycotics, resistance develops rapidly, and chronic use can lead to irritation. On the mucous membranes, the best effect is provided by Lugol's solution, diluted 2-3 times, 10-15% solution of borax in glycerin. Local antiseptics are recommended to be alternated every week. In patients with weeping erosive rashes, treatment consists in prescribing lotions with a 1% solution of tannin, 0.25% solution of silver nitrate, 2-3% - borax and bicarbonate, 1-2% aqueous solution of aniline dyes. In the future, lubrication of lesions with a 1-2% alcohol solution of aniline dyes, fucorcin, 5-20% solution of borax in glycerin is practiced. The following ointments have an anti- candidiasis effect: dekamin 0.5-2.0%, nystatin 0.5-1.0%, ketoconazole, Kanesten cream, Clotrimazole, Nizoral, Pimafucin, Mycozolon ointment and others

Treatment of oropharyngeal candidiasis should primarily be based on etiotropic therapy. The most important therapeutic measure is the eradication of the pathogen in the oral cavity. In the future, relapse prevention and correction of local and general predisposing factors are carried out. In most cases of oropharyngeal candidiasis, topical therapy is prescribed. Topical polyene antimycotics used in the treatment of oropharyngeal candidiasis include nystatin, levorin, natamycin, and amphotericin. Imidazole derivatives include miconazole, econazole,

clotrimazole and others. Antiseptics with antifungal activity are usually prescribed in the form of lubricants or rinses. Any topical medication should remain in the mouth for as long as possible. Antiseptics are widely available and available, but they are less effective than antimycotics, resistance develops rapidly, and chronic use leads to mucosal irritation. The use of modern antiseptics (chlorhexidine bigluconate, etc.) is more effective. Rinsing is carried out after meals twice a day. In most cases, urogenital candidiasis can be treated with topical antifungals and antiseptics. Local antifungal agents are available in the form of vaginal suppositories and tablets, creams, solutions for douching. For example, suppositories of econazole 150 mg, 1 suppository in the posterior vaginal fornix at night for 3 days or suppositories with natamycin 100 mg, 1 suppository for 6 days. Vaginal creams are used to treat vulvitis, pills and suppositories-vaginitis. Currently, systemic drugs are very often used. One of their important advantages is the convenience of prescribing compared to vaginal forms for topical use. The duration of systemic therapy for uncomplicated urogenital candidiasis is minimal. Fluconazole is prescribed 150 mg once, itraconazole 400 mg once (in 2 doses) or 200 mg for 3 days, ketoconazole (nizoral) 200 mg for 5 days. Fluconazole is considered the most effective drug against Candida albicans. In the treatment of skin candidiasis, it is necessary to solve three problems: eradication of the pathogen in the focus (appointment of antiseptics and antimycotics), sanitation of a possible endogenous source of the pathogen in the intestine or genitourinary tract, correction of conditions predisposing to candida intertrigo. In the first case, locally prescribed: aniline dyes (alcohol or aqueous solutions, the latter are better suited for large folds) or 2% aqueous solution of resorcinol, 0.25% solution of silver nitrate, 0.1% solution of rivanol or ointments and pastes containing 10% boric acid. Of the local antimycotics, you can use drugs of any class in the form of 1-2% creams, ointments, solutions, for example, antimycotics-imidazoles. In the presence of inflammatory phenomena in the focus, as a rule, due

to the addition of a bacterial infection. In addition, the use of combined preparations containing an antimycotic along with an antiseptic or antibacterial antibiotic, as well as a steroid hormone, should be prescribed. External agents are used until the skin lesions are completely resolved, and then for another 1 week. To eliminate the intestinal or urogenital reservoir of candidal infection, antimycotics are usually prescribed orally for candidal intestinal dysbiosis (nystatin in tablets of 4-8

million units / day or natamycin 0.4 g / day), as well as in the form of a suppository or douching for vaginal candidiasis.

The duration of treatment is 1-2 weeks. You can combine the first two tasks by using a systemic antimycotic: fluconazole, itraconazole, or ketoconazole. The use of these agents leads to the widespread destruction of the pathogen both in skin foci and in all its reservoirs outside the skin.

8.3.5. Prevention of candidiasis In patients with diabetes mellitus, malignant neoplasms, blood diseases, immunodeficiency, as well as in those receiving massive antibiotic therapy, it is necessary to conduct control studies for yeast-like fungi. If fungi of the genus Candida are detected again (at least 2-3 times) in an amount exceeding 104 colonies per 1 ml (or 1 g) of pathological material when sown on a nutrient medium, anti-candidiasis treatment should be recommended even in the absence of clinical manifestations of candidiasis. With urogenital candidiasis, attention should be paid to the correction of intestinal dysbacteriosis, the treatment of sexual partners. In the maternity hospital for mothers and newborns with symptoms of candidiasis, separate household items and medical instruments are isolated, which are disinfected by boiling for 30 minutes or by autoclaving. Medical personnel with candidiasis of the interdigital folds or nails should not be allowed to work in maternity hospitals, children's departments of hospitals and clinics.

Tests for knowledge control 1. According to the current classification, what group of mycoses does versicolor versicolor belong to? a) deep mycoses; b) keratomycosis; c) pseudomycosis; d) candidiasis; e) dermatomycosis.

2. hich of the above tests and symptoms are used for the differential diagnosis of lichen versicolor? a) test with tincture of iodine (Balzer test); b) symptom of stearin staining; c) a symptom of "ladies' heels"; d) sample with 50% potassium iodide solution; e) diascopy.

3. Which of the drugs is most effective for the treatment of tinea versicolor? a) nystatin; b) amphotericin B; c) macrofoams; d) ketoconazole (Nizoral); e) azithromycin.

4. The detection of what confirms the diagnosis of versicolor versicolor on microscopy of the affected skin scales? a) disputes; b) mycelium; c) ovoid bodies; d) pseudomycelium; e) coccoid- rod flora.

5. What pathogen causes versicolor versicolor? a) red trichophyton; b) microsporum sanis; c) candida; d) malassezia; e) streptobacilli.

e) esicular rash.

6. What antimycotic is considered the most effective in the treatment of candidiasis? a) nystatin;

b) levorin; c) fluconazole; d) pimafucin; e) clotrimazole.

Answer standards: 1 b; 2 a; 3 d; 4 b; 5 d; 6 d; 7 a; 8 B.

Dermatomycosis (syn. dermatophytosis) is a large group of fungal diseases caused by dermatophytes-filamentous fungi from the genera Epidermophyton, Trichophyton, Microsporum, capable of affecting all layers of the epidermis, hair and nails. These fungi are widely distributed in nature and have a pronounced contagiousness.

There are different types of epidermomycosis and trichomycosis. Epidermomycosis includes three nosological forms: athlete's groin, athlete's foot and rubrophytosis. Inguinal epidermophytosis (syn. marginal eczema - eczema marginatum, tinea cruris). Fungal infection of mainly large skin folds, interdigital folds of the feet, and rarely of the nails of the feet.

Dermatophytosis

- Dermatophyte
  - — Sexual
    - Arthroderma
  - — Asexual
    - Macroconidia
      - —Trichophyton
      - —Microsporum
      - —Epidermophyton

Pathogen - Epidermophyton floccosum (syn. Epidermophyton inguinale). Epidermophyton floccosum accounts for less than 2% of all pathogens of dermatophytosis. Mostly men are ill. Epidemiology and pathogenesis. Increased sweating, impaired carbohydrate metabolism, elevated ambient temperature contribute to the occurrence of inguinal epidermophytosis. The disease is most common in warm, humid climates. Infection can occur by contact, but most often through personal hygiene items: washcloths, underwear, thermometers and other items that the patient has come into contact with.

clinical picture. The most typical localization is inguinal, intergluteal, underwing folds and folds under the mammary glands. Foci can also be located on the skin of the trunk, limbs, genitals, scalp. Anti-inflammatory effects are usually observed. The foci are represented by rounded spots with clear boundaries. When merged, they give polycyclic forms with a well-defined edematous peripheral roller with the presence of vesicles, micropustules, crusts, scales on it, sometimes with weeping discharge, so the disease can be similar to eczema. In the center of the foci, there is a resolution of the pathological process, peeling.

Subjectively, patients feel itching. In a chronic course, the disease usually worsens in the summer. With damage to the skin and nails on the feet, this disease is clinically similar to rubro-

and epidermophytosis. Histological examination reveals acanthosis, hyperkeratosis, and moderate perivascular infiltration of the dermis. Fungi are found in all layers of the epidermis. Diagnosis of inguinal epidermophytosis is based on a characteristic clinic and detection of a pathogen in the lesions. It is necessary to differentiate the disease from rubrophytia, candidiasis and eczema of the folds, erythrasma, diaper rash, epidermophytosis of the feet.

Care. External antifungal agents are used: - azole compounds (clotrimazole, miconazole, ketoconazole, econazole) - - allylamine compounds (terbinafine, naftifine) - - 3% salicylic ointment 2 times a day as monotherapy or in combination with 2% alcohol solution of iodine - - salicylic acid (2%) - sulfuric acid (10%) - tar (5-8%) ointment. In inflammatory phenomena, combinations of antifungal drugs with corticosteroids are prescribed: natamycin + neomycin + hydrocortisone, mycosolone for 7-10 days, as well as desensitizing and antihistamine drugs. The total duration of treatment of inguinal epidermophytosis is from 2 to 3-5 weeks.

Athlete's foot. A chronic fungal disease localized on the skin of the arches and interdigital folds of the feet, with frequent lesions of the nail plates.

Pathogen - Trichophyton mentagrophytes var. interdigitale. Trichophyton interdigitale accounts for 5-10% of all foot mycosis pathogens in urban areas and 40-50% in rural areas. In children of preschool and early age, epidermophytosis of the feet does not happen. Epidemiology and pathogenesis. Infection can occur through direct contact with a sick person, but more often indirectly through infected objects. Baths, saunas, showers, swimming pools, gyms, someone else's shoes,

socks and stockings represent the greatest epidemiological danger. From patients with epidermophytosis, exfoliated epidermis, particles of nails destroyed by the fungus, fall on the feet of a healthy person. The disease is promoted by hyperhidrosis of the feet, flat feet, insufficient hygienic nail care, and wearing tight shoes. shoes, metabolic and endocrine disorders, impaired vascular tone of the lower extremities, dysfunction of the nervous and immune systems, high ambient temperature. The penetration of the fungus into the epidermis contributes to the violation of the integrity of the epidermis and nail plates (microtraumatization, diaper rash, abrasions). The incubation period for epidermophytosis is not defined, since after infection there is a long period of adaptation of the pathogen from the saprophytic form to the virulent one. clinical picture. There are erased (squamous), intertriginous, dyshidrotic forms and epidermophytosis of the nails. The scaly form is manifested by peeling of the skin of the arch of the feet. The process can extend to the lateral and flexion surfaces fingers. Since erythema and peeling are not expressed, the disease often goes unnoticed by the patient. The intertriginous form can develop independently. But more often it is a consequence of untreated long-term squamous epidermophytosis. It begins with a thin peeling in the area of the 3rd or 4th interdigital folds. It resembles diaper rash with a crack in the depth of the fold, surrounded by a scaly whitish keratinized layer of the epidermis. Accompanied by itching, burning, sometimes soreness. The course is chronic, with exacerbations in the summer. The dyshidrotic form occurs either primary or is a consequence of the progression of the squamous form. Accompanied by severe inflammation, the appearance of blisters on the arches, lower lateral surface and on the contact

surfaces of the toes with a thick horny cap, transparent or opalescent contents (the so-called "sago grains"). Bubbles are located in groups, tend to merge and form large multi-chamber bubbles when the tire is stressed. After opening the blisters, a weeping erosive surface is formed, often with purulent discharge. In some patients, lymphadenitis occurs, the temperature rises, and the general condition worsens. This form is often accompanied by allergic rashes-epidermophytosis on the skin of the trunk, limbs, face. One of the complications of epidermophytosis of the feet is erysipelas of the lower leg. These forms are quite conditional, since a combination of clinical forms is possible, the transition of one form to another, which depends on the reactivity of the organism, motor activity and the nature of the patient's treatment. Epidermophytosis of nails (onychomycosis), mainly of the 1st and 5th toes, is observed in 20-30% of

patients. Fingernails are not affected. The nail fades, becomes yellowish-gray in color, gradually deforms, subungual hyperkeratosis develops with the destruction of the nail plate, "corroded" of its free edge. Primary nail changes are observed at the free edge in the form of yellow spots and stripes. Patohistology. In the squamous form, acanthosis and hyperkeratosis are noted. The stratum corneum is 2-3 times thicker than the rest of the epidermis; the lustrous layer is usually absent. In the dyshidrotic form, significant acanthosis, hyperkeratosis, and focal parakeratosis are observed. In the Malpighian layer - intercellular edema with a large number of vesicles, exocytosis. In the upper layers of the dermis, edema, perivascular inflammatory infiltration of lymphocytes, histiocytes, fibroblasts and neutrophilic granulocytes. Threads and chains of fungal spores are found in the horny and prickly layers of the epidermis. With onychomycosis of the nail bed - parakeratosis, smoothness of the papillae of the dermis, swelling in the nail bed. reticular layer. Fungal elements are found in the keratinized and parakeratotic masses of the nail bed. Diagnosis of all forms of epidermophytosis is based on clinical symptoms, characteristic localization and a mandatory double microscopic examination of skin scrapings or nail sections, or on a cultural study on Saburo's medium. Differential diagnosis is carried out with microbial and dyshidrotic eczema, dry lamellar dyshidrosis, candidiasis and rubromycosis. Epidermophytosis of the nails is differentiated from trophic onychodystrophy and rubromycosis of the nails.

Care. In the presence of acute inflammatory phenomena, lotions are prescribed from a 1-2% solution of resorcinol, a 0.1% solution of ethacridine, a 0.25% solution of silver nitrate, a solution of potassium permanganate 1:6000-1:8000. The vesicle membrane is cut off with scissors or pierced with a needle in compliance with the rules of asepsis. Then apply solutions of aniline dyes (brilliant green, methylene blue, etc.), pastes and ointments with antimycotics, most often from the group of imidazoles (ketoconazole, miconazole, clotrimazole, bifonazole, econazole, etc.) or allylamine compounds (1% terbinafine cream, 1% cream or solution of naftifine). Combinations of antifungal drugs with glucocorticoids are used for islet-inflammatory manifestations of trunk mycosis. They also have antifungal activity and are widely used in practice: tar 2-3%, sulfur 3-10%, salicylic acid 2-3%, tincture of iodine 2%, Teymurov's paste, nitrofungin, zincundan, undecin, mycoseptin. The total duration of treatment is from 1-2 to 3-5 weeks. In the presence of allergic rashes, it is necessary to prescribe hyposensitizing therapy and

antihistamines, B vitamins, rutin, ascorbic acid. When pyoderma is attached, short courses of

antibiotics are carried out that do not activate the fungal infection: erythromycin, lincomycin, tetracyclines, fusidine sodium, etc.

Treatment of squamous epidermophytosis can be carried out with ketoconazole (Nizoral) 1 tablet (200 mg) 2 times a day for the first two days, then 1 tablet 1 time per day. The course lasts from 2 to 6 weeks. Terbinafine (lamizil, terbizil) is prescribed 1 tablet (250 mg) per day for 2 weeks. Itraconazole (orungal) is prescribed 200 mg 2 times a day after a heavy meal for 7 days. Outwardly, with hyperkeratosis on the feet, the epidermis affected by fungi is exfoliated with Arievich's ointment (Ac. Salicylici 12.0, Ac. Benzoici 6.0, Vaselini 82.0). After detachment, the above ointments with antimycotics are applied to the young keratinizing epidermis.

Treatment of onychomycosis with epidermophytosis is carried out similarly to the corresponding form of rubromycosis (see the section "Rubromycosis"). Rubromycosis (syn. rubrofitia) is a fungal disease prone to exacerbations and chronic course with predominant localization of foci on the skin of the feet, frequent lesions of the nail plates of the feet and hands, large folds, skin of the trunk and extremities, less often - face, neck and scalp. Pathogen - anthropophilic Trichophyton rubrum.

There are three varieties of this mushroom: gypsum, fluffy and velvety. The most virulent and aggressive is hypoid. The disease was first described by the Japanese dermatologist Ota in 1922 and has long been endemic to the countries of the Far East and Southeast Asia. After the Second World War, mycoses began to spread rapidly in Europe and America. At present, the hypoid variant of trichophyton red has become the leading causative agent of mycosis of the feet.

Epidemiology and pathogenesis. Infection occurs through prolonged contact with patients with rubrophytia and the use of various things of the patient (washcloths, pumice stone, scissors, shoes; when visiting baths, pools, where skin flakes affected by the fungus and particles of broken nails can remain on the floors and flooring for a long time). Often the disease is familial.

The pathogenesis of this disease is similar to epidermophytosis. The skin of patients with rubromycosis is characterized by increased dryness, a tendency to pronounced hyperkeratosis, reduced resistance of keratinocytes and downy hair cuticles.

clinical picture. The incubation period has not been established. It is possible to carry the infection for a long time. There are several clinical forms of rubromycosis: rubrophytosis of the feet, hands and feet, rubromycosis of the nails and a generalized form. Rubromycosis of the hands and feet can manifest itself in the following clinical forms: intertriginal, dyshidrotic, squamous, hyperkeratotic with single or multiple lesions of the nails. Usually the process on the feet is preceded by damage to the skin of the palms and rear of the hands. Most often, the disease begins with the 4th interdigital space and quickly spreads to all interdigital folds (intertriginal form), where cracks with peeling around them are found. Then the process spreads to the skin of the soles, which becomes infiltrated, dry (squamous-hyperkeratotic form). On the skin of the

soles, increased keratinization is observed, folds are clearly visible, in which there is a pronounced cystic and small-focal peeling. The process can spread to the lateral and dorsal surfaces of the feet, as well as the hands, where a pronounced peripheral ridge consisting of nodules is observed along the periphery of the foci.

bubbles and crusts. Subjectively, patients feel itching. This form is more common than others with localization both on the palms and soles, and on other areas of the skin. With a dyshidrotic, exudative form, which is more often observed in children, adolescents and young people, vesicles, erosion, and weeping predominate in the clinical picture.

Rubromycosis of the nail plates is possible as an isolated process. But it is usually combined with other forms of rubrophytia. More often (87%) the nails of the feet are affected. There are three types of their lesions: normotrophic, hypertrophic and atrophic. With a normotrophic lesion, only the color of the nails is changed (from grayish yellow to brownish brown). Leukonychia appear in the thickness of the nail - spots and stripes of white color. The free edge of the nail is either unchanged or slightly jagged, and the nail plate is shiny. In the hypertrophic type, the nail thickens due to subungual hyperkeratosis, fades, crumbles easily, and becomes beak-shaped. Significant thickening and curvature of the nails leads to the fact that they resemble the claws of birds (onychogryphosis). In the atrophic type (rarely seen in adults), the nail plate is thinned to the point of lysis and rejection from the nail bed. Sometimes the nail only partially remains on thenail fold.

The generalized form of rubromycosis exists at first as a limited lesion, and then against the background of favorable factors (immunodeficiency, visceral, neuroendocrine pathology, the use of

cytostatics, corticosteroids, antibiotics, etc.), the process becomes generalized. The skin of the trunk, large folds, buttocks, limbs, neck, face is affected.

At the same time, erythematous-squamous (superficial), follicular-nodular (deep) and exudative- erythrodermic types are distinguished. Generalized rubromycosis is characterized by polymorphism of rashes and the similarity of clinical manifestations with eczema, psoriasis, parapsoriasis, granuloma annulare, recurrent syphilis, Dühring's herpetiform dermatosis, chronic trichophytosis, Darier's disease and other dermatoses. Histopathological changes in rubromycosis are similar to those in epidermophytosis. The diagnosis of all forms of rubromycosis is based on clinical symptoms, characteristic localization and a mandatory 2-fold microscopic examination of skin scrapings or nail sections, as well as on a cultural study on Sabouraud's medium with the isolation of a red trichophyton culture.

Treatment of rubrophytia should be complex-symptomatic, etiotropic and pathogenetic. It is in many ways similar to the treatment of similar forms of epidermophytosis. In acute cases, antihistamines and hyposensitizing drugs, sedatives, and lotions are symptomatically prescribed. After the anti-inflammatory effects subside, aniline dyes, nitrofungin, fucorcin, 2% tincture of iodine, antifungal pastes and ointments are prescribed (see Treatment of epidermophytosis).

Treatment of onychomycosis is carried out with the appointment of a systemic antimycotic. The systemic antimycotic drugs terbinafine (lamizil, terbizil, lamiter, binafine) and itraconazole (orungal, orunit) are currently considered safe for the treatment of rubromycosis, their regimens do not differ from those for epidermophytosis. Terbinafine is prescribed 1 tablet (250 mg) per day for 3-4 months, depending on the degree of nail damage. Intraconazole is prescribed in the form of pulse therapy - 400 mg / day (in 2 divided doses of 200 mg) for 7 days. With onychomycosis of the hands, two courses of pulse therapy are carried out with an interval of 21 days, with onychomycosis of the feet - 3 courses or more. Griseofulvin is prescribed at the rate of 18 mg per 1 kg of body weight. The daily dose is divided into 3 doses per day (for adults more often at the rate of 1 tablet (125 mg) per 10 kg of body weight), washed down with water. season with a teaspoon of vegetable oil. The first month the drug is prescribed daily, the second month - after 1 day, then (2 weeks) 1 time in 3 days at the same daily dose.

Treatment is carried out for 8-10 months. Ketoconazole is used 1 tablet (200 mg) per day during or after meals for 6-7 months. Fluconazole is prescribed 150 mg once a week on the set day, after meals, until healthy nails grow back. In parallel, it is advisable to prescribe immunomodulators (histagglobulin, pyrogenal, cycloferon, methyluracil, etc.), aloe, splenin, multivitamins. If there is no effect from the treatment, a second course is carried out using another antimycotic, as well as using angioprotectors (xanthinolnicotinate, pentoxifylline) and hepatoprotectors (essential phospholipids). The best effect is achieved with a combination of systemic and external therapy with hardware cleaning of nails or cleaning of nail plates after applying ureaplast to the affected nail plate under adhesive plaster for 5 days. Prevention of epidermophytosis and rubromycosis. Dispensary observation of patients is carried out for 1 year with control visits 1 time per quarter. Personal prevention consists in using only your own shoes, observing the rules of hygiene of the skin of the feet, disinfecting shoes, bed and underwear. At home, disinfection of shoes is carried out according to the method proposed by V. M. Leshchenko: with a cotton swab, abundantly moistened with a 25% formalin solution, they wipe the insole and lining of the shoe. Then the shoes are placed in a plastic bag. After 24 hours, the shoes are aired until the smell of formalin disappears. Socks, stockings and underwear are disinfected by boiling for 10 minutes. In order to prevent relapses, the skin of the feet after the disappearance of the manifestations of the disease is lubricated with antimycotic agents for 2-3 weeks. Prevention of inguinal epidermophytosis includes the fight against sweating, disinfection of common items. After the end of treatment and the resolution of mycosis, the foci are prophylactically lubricated for 2-3 weeks with 2% iodine tincture or 1% terbinafine cream.

trichomycosis

Among dermatomycosis, some mycologists separate trichomycosis separately, i.e., mycoses that affect not only smooth skin, but also appendages, primarily hair, less often nails. These include microsporia, trichophytosis and favus. In the last 10 years, microsporia has been most common in the Republic of Belarus (40-50 cases / 100 thousand people are registered annually). Trichophytosis is much less common (no more than 1-2 cases / 100 thousand population). Over

the past 10 years, favus has not been registered in our country. Microsporia is a highly contagious fungal disease of the skin and hair,

rarely caused by various types of fungi of the genus microsporum. More than 20 species of Microsporum fungus are known. There are anthropophilic, zoophilic and geophilic microsporums. The representative of anthropophilic microsporums is a rusty microsporum (M. ferrugeneum). Infection occurs from a sick person through direct contact, as well as through contaminated objects (hats, clothes, bedding, combs, hairdressing tools, etc.). The disease is highly contagious. Fluffy microsporum (M. lanosum or m

. canis) is a zoophilic fungus that parasitizes on the skin of animals (cats, dogs). It is microsporia fluffy that is currently almost the only causative agent of microsporia registered in the Republic of Belarus. Human infection with fluffy microsporia occurs in 80-85% of cases from cats (kittens), less often from dogs through direct contact with a sick animal or with objects contaminated with the hair of sick animals. Cases of infection of children after the game were recorded in the sandbox (the sand is contaminated with the broken hair of sick animals). It is known that the causative agent of microsporia remains viable in hair outside the animal's body for up to ten years, and in skin scales for up to seven years. It should be noted that 2.4% of clinically healthy animals can be carriers of fluffy microsporum and be a source of infection in humans. In 2% of cases, person-to-person transmission occurs.

Pic-23 Trichomycosis

The seasonality of zoophilic microsporia is characteristic, mainly associated with epizootics of the disease among cats and dogs, with offspring in cats and more frequent contacts of children with these animals in the summer. The first litter of cats occurs in March-May. The second offspring occurs in late August-November. An increase in the incidence of zoophilic microsporia occurs in June-July and September-November. The seasonality of the incidence of microsporia can be associated with seasonal migration of the population (pioneer camps, vacations) and natural and climatic conditions.

The transmission of the pathogen occurs both through direct contact with a sick animal, and through objects and things contaminated with scales and wool, containing mycelium and fungal spores. Rusty and fluffy microsporums affect mainly children aged 4 to 11 years, but sometimes young women with thin and delicate skin also get sick.

Gypsum microsporum (M. gypseum) is a soil fungus that parasitizes in the soil of vegetable gardens and greenhouses, but can infect cats, dogs, mice, rats, horses, and even poultry. Gardeners, gardeners and flower growers are more likely to get sick, mostly adults (with damage to smooth skin, scalp, less often nails), and children often get sick.

Clinic. After the incubation period, which with zoophilic microsporia is 5-7 days, with anthropophilic microsporia it can be extended up to 4-6 weeks, foci appear on the smooth skin or scalp. With geophilic microsporia, the incubation period is not clear. With microsporia caused by a rusty microsporum on the scalp, the foci are small, numerous, irregularly shaped with slight reddening of the skin and fine-lamellar peeling, with a tendency to merge into large foci, located more often along the edge of the scalp with a transition to nearby areas of smooth skin. The boundaries of the foci are indistinct. Not all hair breaks off, usually at a height of 6-8 mm and above.

On smooth skin there are foci of a rounded or oval shape. shapes, often like rings that fit into each other. In the focus, hyperemia and peeling of the skin, more pronounced along the edge, are noted. Downy hair is often affected. Microsporia caused by fluffy microsporum manifests itself when the scalp is affected by the formation of 1-2 large foci from 3 to 5 cm in diameter, regular round or oval outlines and several small ones from 0.3 to 1 cm. All hair in the foci is usually broken off at a height of 4 - 5 mm above skin level, look like cut. Therefore, this form of microsporia and trichophytosis of the scalp is popularly called ringworm. The surface of the foci is covered with grayish scales. Affected hair is easily removed,

their basal part is covered with a whitish loose coating. Inflammatory phenomena in the focus can be of varying severity: from acute to minor. With the development of acute inflammatory phenomena in large foci, swelling, hyperemia, the appearance of pus and the presence of yellow crusts are noted, cervical, behind- the-ear and occipital lymph nodes often increase. Along with the described typical form of microsporia of the scalp, there are also atypical variants: infiltrative, suppurative (deep), exudative, seborrheic and trichophytoid forms. With the trichophytoid form, for example, all the foci are small, without inflammation, with slight fine-lamellar peeling. The favorite localization

of foci of zoophilic microsporia is the parietal and temporal regions, although the occipital region is more often affected. On the smooth skin of open and closed areas, small foci of hyperemia (1-2 cm in diameter) are formed, with a roller-like edge, covered with vesicles and crusts. There are many foci, they can merge. Fluffy hair (80-85%) in the focus is often affected, lesions of the eyebrows, eyelashes, eyelids are described. In young women with hypertrichosis, it is possible to develop follicular-nodular elements up to 3 cm in diameter in the lower leg, the so-called deep form of microsporia of smooth skin. The clinic of microsporia caused by gypsum microsporia resembles the clinic of loose microsporia. Diagnosis of microsporia is based on clinical and epidemiological data. To clarify the diagnosis, a luminescent method, microscopic examination of pathological material (hair, skin scales), cultural diagnostics are used to identify the type of pathogen in order to determine the scope of anti-epidemic measures. Under the light of Wood's fluorescent lamp, hairs affected by rusty microsporum glow with a bright green glow, in contrast to microsporia caused by fluffy microsporum, when the hair glow is pale green. Microscopically long and fluffy hair affected by microsporiosis is usually wrapped on the outside with a sheath consisting of small, round, randomly arranged spores resembling a mosaic (ectothrix type). Small spores and filaments of mycelium can also be seen inside the hair. The type of pathogen can only be determined by culture. Fluffy microsporum is sown in more than 95% of patients.

Differential diagnosis is carried out with trichophytosis, alopecia areata, discoid lupus erythematosus. Trichophytosis is a contagious fungal disease from the group of dermatophytosis that affects smooth skin, scalp, long and fluffy hair, nails. By origin, the following groups of trichophyton are distinguished: - Anthropophilic group: Tr. violaceum, tr. tonsurans, etc. - zoophilic group: Tr. plaster, tr. verrucosum, etc. By the nature of hair

damage: - Tr. endotrix spores are located inside the hair (causative agents of anthropophilic trichophytosis) - - Tr. ectothrix -spores are located outside the hairline (causative agents of zoophilic trichophytosis). Epidemiology. With anthropophilic trichophytosis, infection occurs through contact with a sick person or his things (hats, bedding, combs, etc.). Intrafamilial transmission of trichophytosis predominates, although infection is possible in hairdressing salons, boarding schools, and schools. Mostly children suffer.

Zoophilic trichophytosis affects mainly rural residents. Tr carriers. gypseum, usually rodents (rats, mice). A person becomes infected mainly through contact with straw, hay, and mouse hair infected with fungus. Carrier Tr. verrucosum of cattle, especially calves, and the pathogen, together with fragments of wool and skin scales, is scattered on pastures and cowsheds. The incidence of zoophilic trichophytosis is higher in winter. Clinic. There are superficial trichophytosis, chronic trichophytosis and infiltrative-suppurative trichophytosis. Superficial trichophytosis is caused by anthropophilic fungi. The incubation period is about 1 week. Smooth skin and/or scalp may be affected. Superficial trichophytosis of the scalp affects almost exclusively children under 14 years of age. There are one or more isolated foci 1-2 cm in size, irregular in shape, not prone to fusion. In the area of the focus, the skin is hyperemic, edematous, covered with bran-like grayish scales. The hair in the focus lost its luster, elasticity, part of it

breaks off at a height of 2-3 mm from the skin surface or at the level of the stratum corneum (black dot form). Trichophytosis of smooth skin affects mainly exposed areas of the skin and can occur at any age, regardless of gender. One or more edematous foci with clear boundaries appear, pink-red in color, their surface is covered with scales and small bubbles. Over time, the focus takes the form of a ring. Itching is moderate. Nails with superficial trichophytosis are practically not affected. Chronic trichophytosis is most often the result of untreated trichophytosis acquired in childhood and occurs mainly in women. In this case, the scalp, smooth skin and nails, more often fingers, can be affected in isolation or in various combinations. When the scalp is affected, mainly in the occipital region, only a slight bran-like whitish peeling and hardly detectable "black dots" at the site of broken hair can be found. It should be remembered that "black dots" may be the only clinical symptom of the disease (black dot trichophytosis in adults). Chronic trichophytosis of smooth skin affects areas subject to friction (buttocks, shins, extensor surfaces of the knee and

elbow joints, less often the trunk). Sometimes the process takes on a general distribution. The foci are represented by pinkish-blue spots without clear boundaries, with a scaly surface. Downy hair is often affected, which in the future will be a constant source of relapses. The nail plates on the fingers are often affected. Most often, the nail lesion begins with the distal free edge or from the side of the nail plate. A whitish-gray spot is formed, the nail gradually becomes uneven, dull, dirty gray with a broken edge of the nail plate and subungual hyperkeratosis. Nail folds usually do not change. Usually, chronic trichophytosis of adults has a poor clinic and lasts for many years. Patients are difficult to identify and can be a source of infection among children. Diagnosis of superficial and chronic trichophytosis is based on the clinical picture, epidermological data in the focus, the results of microscopic examination (detection of filaments of septic mycelium in smooth skin scales, detection of spores inside the hair) and a standard cultural study.

Differential diagnosis is carried out with microsporia, seborrheic eczema, alopecia areata, Broca's pseudopelade. Infiltrative-suppurative trichophytosis is caused by zoophilic fungi. The source of infection are sick animals, less often - a sick person. The incubation period for infection with gypsum trichophyton is 1-2 weeks, and for infection with warty trichophyton it can be extended up to 1-2 months. The scalp may be affected, in men the beard and mustache area (parasitic sycosis), as well as smooth skin.

With the localization of foci on smooth skin, the disease manifests itself in the formation of clearly defined infiltrated plaques (1-2 or more), which consist of many fused folliculitis at different stages of development. The foci can merge, forming bizarre shapes. On the surface of these plaques there is an abundance of vesicles, pustules, papules, crusts, fluffy hair can also be affected. The process ends with the formation of a scar. With infiltrative-suppurative trichophytosis, it is localized on the scalp, sharply defined hemispherical nodes of a bluish-red color with an abundance of folliculitis, erosion and crusts on their surface are formed in the beardand mustache. The hair in the foci is loose, some of them fall out, the rest are easily epilated. The crusts are removed along with the hair, after which the inflamed surface is opened with an abundance of enlarged mouths of hair follicles, from which, when pressed on the inflamed area, pus is released in the form of copious drops or even streams. The Roman physician Celsus described this phenomenon as "honeycombs" - the kerion of Celsus (translated from Greek kerion -

honeycombs). Gradually, the consistency of the knots from dense becomes pasty. At the peak of the disease, the general condition worsens: there is malaise, moderate fever, and regional lymphadenitis. Even without treatment, the process is resolved after 2-3 months, leaving cicatricial alopecia and persistent specific immunity. Diagnosis of infiltrative-suppurative trichophytosis is carried out on the basis of clinical and epidemiological data, the diagnosis is confirmed by mycological examination (microscopy - hair damage by the type of ectothrix and cultural diagnosis). Differential diagnosis. Infiltrative-suppurative trichophytosis of the scalp, beard and mustache is differentiated from vulgar sycosis (less pronounced inflammatory process in the focus, no tendency to self-heal, long-term course of many months), with impetiginous syphilis (history, the presence of other manifestations of syphilis, including serological reactions)

Treatment of microsporia and trichophytosis. With microsporia, general and local treatment is used. The most effective treatment for microsporia, trichophytosis and favus is the fungistatic antibiotic griseofulvin. It is used in tablets of 125 mg and in the form of a suspension specially designed for young children. Fatty foods promote better absorption of the antibiotic from the gastrointestinal tract into the blood. It is not recommended for patients with microsporia, suffering from both acute and subacute diseases of the liver, kidneys, blood, and with its intolerance. Staff and parents should ensure that children take antibiotics regularly so that they do not throw away the pills. Griseofulvin is prescribed at the rate of 22 mg / kg of body weight, with trichophytosis and favuses - 18 mg / kg (2.5 tablets are equivalent to 4 teaspoons of suspension). It is used immediately after the diagnosis is established, the results of clinical blood and urine tests are obtained, and the presence of contraindications to its use is excluded. The daily dose is administered in 3 divided doses. Scheme of application: in a full dose daily for 3-4 weeks, until the first negative test for fungi is obtained (this study is carried out once a week, starting from the end of the second week from the start of treatment), then the antibiotic is taken at the same dose every other day 2 weeks, and in the future - only 2 days a week (2 more weeks). It is better to take with a meal with a teaspoon of vegetable oil. In the absence of griseofulvin, microsporia and trichophytosis can be treated with terbinafine (Lamisil) or itraconazole (Orungal). Terbinafine is prescribed for children weighing 10 to 20 kg at a dose of 94 mg (3/4 tablets of 125 mg) 1 time per day, with a body weight of 20 to 40 kg - 187 mg (1.5 tablets

of 125 mg). mg), with a weight of more than 40 kg and adults 250 mg per day. day. The duration of treatment for microsporia is 8- 12 weeks, for trichophytosis and favus - 4-6 weeks. It is advisable to prescribe itraconazole to children in the form of a solution (10 mg / ml solution) at the rate of 5 mg / kg of body weight, the dose is given 1 time per day at lunch.

Treatment of microsporia of the scalp lasts an average of 4 to 8 weeks, trichophytosis and favus - 4-6 weeks. If the doctor has only 100 mg itraconazole (orungal) capsules at his disposal, then the

treatment is best done by pulse therapy: each cycle of taking the drug lasts 1 week, the cycles are repeated after 2 weeks. At the same time, children with a body weight of 20-30 kg receive 100 mg of the drug daily, with a body weight of 30-40 kg - alternate doses of 200 and 100 mg 1 time per day, with a body weight of 40-50 kg - 100 mg 2 times a day. day, with a body weight of morethan 50 kg and adults - 200 mg 2 times a day. There are 2 or 3 courses. In chronic trichophytosis of adults, along with antifungal treatment, pathogenetic therapy is prescribed to eliminate disorders that have developed mycosis (vegetoneurosis, endocrinopathy, hypovitaminosis, etc.); vitamin therapy, sometimes immunotherapy. Patients with infiltrative-suppurative trichophytosis are prescribed general antimycotic and hyposensitizing therapy (antihistamines, calcium preparations).

External treatment. It is carried out simultaneously with the intake of griseofulvin or other systemic antimycotics. It consists in the following: - weekly shaving of the scalp and washing with hot water and soap 2-3 times a week - - in the morning any antifungal ointment is rubbedinto the lesions (salicylic (3%) - sulfuric (10%) ointment on petroleum jelly; sulfuric (10%) - tar (3%) ointment on vaseline; ointments Batrafen, Mycospor, Miconazole, Travogen, Tolmitsen, Exoderil, Lamisil). The head should be covered with a gauze bandage during the day, thebandages are burned in the evening; - in the evening, the scalp is smeared with 2-5% tincture of iodine. When treating isolated foci of microsporia on smooth skin without damage to downy hair, one can limit oneself only to external antifungal agents (2-5% tincture of iodine in the morning, ointments listed above in the evening). The duration of treatment is about 2 weeks. With multiple lesions of smooth skin or in the presence of single foci with damage to downy hair, griseofulvin and external therapy are prescribed. In recent years, an effective shortened method for the treatment of microsporia and trichophytosis of smooth skin has come into practice

with a liquidof the following composition: salicylic acid - 10.0; chinosol - 10.0; dimexide-72.0; distilled water-8.0. The foci are lubricated with this lotion 2 times a day until the clinical manifestations resolve and the fungi disappear. The duration of treatment is 7-14 days (epilation and the introduction of systemic antimycotics are not required). In the treatment of microsporia ortrichophytosis of smooth skin, the first control test for fungi is carried out after the resolution of the clinical manifestations of the disease, and in case of damage to the scalp, 14 days after the start of treatment, after the disappearance of clinical manifestations and the glow of the hair, then after 3-4 days to 1- th negative test, and then every 5-7 days. After discharge from the hospital or the end of outpatient treatment, the patient should be under dispensary observation for 3 months with damage to the scalp and smooth skin with the involvement of downy hair and 1 month with microsporia or trichophytosis of smooth skin without damage to downy hair. hair. The first control examination is carried out 10 days after discharge from the hospital, then once a month. With infiltrative-suppurative trichophytosis, the crusts are first removed and the pustules are opened, then wet-drying dressings are prescribed with some kind of disinfectant solution (2% boric acid, 0.05% chlorhexidine bigluconate, Alibur liquid, etc.). After stopping wetness and reducing the severity of inflammation, gels, creams or ointments containing ichthyol (3-7%), tar (5-10%), sulfur (5-10%), or ointments and creams with antimycotics are recommended. Favus is a rare, relatively low-contagious fungal infection of the skin and its appendages. It has not been registered on the territory of the Republic of Belarus for more than 10 years. The causative agent is the anthropophilic fungus Trichophyton. schonleinii. The disease is often focal or familial. Infection occurs through close and prolonged contact with a sick person or through infected objects. The disease usually begins in childhood, although adults often get sick. Clinic. The scalp is predominantly affected, sometimes nails, and smooth skin is rarely affected. The favus of the scalp can occur in the form of a typical scutular form or in the form of atypical forms - squamous or impetiginous. The scutular form of the favus is characterized by the fact that at the end of the second week after infection and the introduction of the fungus into the hair, a red itchy spot appears on the follicle around the hair, and then a scutula (scutula) forms in the area. his place. Skutula is a pure mushroom culture. Outwardly, it is a rounded formation of an ocher-yellow color, shaped like a saucer, pierced in the center with hairs up to 1.0–1.5 cm in size. It has a dense texture, dry to the touch. After removal of the

cheekbone, a pink-red moist depression is found under it. In place of the resolved focus, cicatricial atrophy remains. Hair in the lesions lose their luster, have an ash-gray color, dull, reminiscent of an old wig. They do not break off, as with microsporia, but are easily epilated. A characteristic barn or mouse smell emanates from the head of a patient with a favus. Subjectively - there may be slight itching in the affected area. In the squamous (pithyriasiform) form, scutulae are not detected, the clinic is represented by limited erythematous-squamous foci with an abundance of whitish-yellow scales.

The impetiginous form is manifested by the formation of pustules at the mouths of the hair follicles and massive yellow crusts. The process ends with the development of cicatricial alopecia. Favus of smooth skin, as a rule, accompanies the defeat of the scalp. A scutular form may develop, with the scutula usually being small and not coalescing into plaques. The scaly form of the favus of smooth skin is manifested by foci of skin peeling, which resemble foci of trichophytosis. Over time, typical scooters may appear on their surface, which tend to merge with each other. In adults, the nail plates on the hands may be affected. The pathological process begins from the free edge of the nail plate with the appearance of grayish-yellowish spots, then subungual hyperkeratosis slowly forms, the nail plate fades, its edge breaks off. Favus persists for many years without treatment. In malnourished patients suffering from other serious diseases (tuberculosis) as a result of hematogenous metastasis of the pathogen, favus lesions of the gastrointestinal tract, lungs, and favous meningoencephalitis are described. Diagnostics. The diagnosis of favus is made on the basis of the clinical picture. Inspection with a Wood's lamp helps, while the affected hair glows a dull green color. Microscopy of the affected hair reveals a rare septic mycelium, groups of spores of various sizes, with large polygonal spores resembling the heel of the foot; as well as gas bubbles and droplets of fat. It is desirable to obtain a culture of the pathogen, which on a Petri dish looks grayish-white, wrinkled, brain-shaped (reminiscent of a morel). Differential diagnosis. Favus of the scalp is differentiated with discoid and disseminated lupus erythematosus, seborrheic eczema (squamous form), with impetigo vulgaris (impetiginous form), favus of nails with onychomycosis caused by other fungi.

Care. Treatment of patients with favus is based on the same principles as the treatment ofmicrosporia and trichophytosis.

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. Tests for knowledge control

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. Make 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 with antibiogram.
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 in the forehead. Restless chills and feeling unwell.

Sick for 5 days. The disease is associated with the fact that she tried to squeeze out an abscess in 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

began to suffer from headaches and general malaise.

C. The patient

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 C, headache, general malaise appeared.

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 8 1.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 10 1.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 and microscopic 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

Main literature:

1. Pakirdinov A.B. Dermatovenerologiya. Darslik. 2020 y.
2. Eshboyev E.X., Tashkenbayeva U.A., Teri-tanosil kasalliklari va ularning laboratoriya tashxisoti. Darslik. Toshkent. – "Navroz". 2019 y.
3. Eshboyev E.X. Dermatovenerologiya. Darslik. 2019 y.
4. Rodionov A.N., Zaslavskiy D.V., Sыdikov A.A. Dermatologiya. Illyustrirovannoye rukovodstvo poklinicheskoy diagnostike. – Moskva. "Granisa". 2018 g. (po rekomendasii minzdrava).
5. VaisovA.Sh., Teri-tanosilkasalliklari. Darslik. - Toshkent, "Yangi asr avlodi".

Additional literature:

1. Arifov S.S., Eshbayev E.X. Teri-tanosil kasalliklar. Darslik. - Toshkent. "Entsiklopediya". 1997 y.
2. Atif Hasnain Kazmi. Color atlas of clinical dermatology. New Delhi, India. "Jaypee Brothers Medical Pub" 2015 g.
3. Olisova O.Yu., Kojnыye I venericheskiye bolezni. Uchebnik. – Moskva. "Prakticheskaya medisina". 2015g.
4. Chebotarev V.V., Tamrazova O.B., Chebotareva N.V., Odines A.V.,Dermatovenerologiya. Uchebnik. – Moskva. "GEOTAR Media". 2013g.
5. Pasricha J.S., Ramji Gupta. Illustrated Textbook of Dermatology. New Delhi, India. "JP Medical Ltd". 2013y.
6. Korotkiy N.G. Kojniye I venericheskiye bolezni. Uchebnik. – Moskva. "Medisinskoye informatsionnoye agentstvo". 2011 g.
7. Skripkin Yu.K., Butov Yu.S., Dermatovenerologiya. Natsionalnoye rukovodstvo. – Moskva. "GEOTAR Media". 2009 g.
8. Skripkin Yu.K., Kubanova A.V., Akimov V.G. Kojnыye I venericheskiye bolezni. Uchebnik. -Moskva, "GEOTAR Media" 2009g. 19
9. Ananyev O.L., Anisimova Ye.V. Kojno-venericheskiye zabolevaniya. Uchebnik. "EKSMO", 2006 g.
10. Pol K. Bakston Dermatovenerologiya. Uchebnik. Rossiya. "Binom", 2005 g.
11. Elinor Ye. San. Dermatologiya. 100 sluchayev iz praktiki. Uchebnik. Rossiya. "Binom", 2001 g.

Internet saytlari:

1. www.mediashhera.aha.ru.//dermatol//derm-mn.htm;
2. www.matrix.ucdavis.cdu//DOJ.html;
3. www.cc.emory.edu //WHSCIV/medweb. dermatology.html;
4. www.Mosbycom//Mosby//Periodicals// medical//CPDM//dm/html;
5. www.skindex.com.; 6. www.ama-assn/org//journals//standing//derm//dennhome.htm.;
7. www.Crawford.com//epo//mm wz//wor ld. html.
8. www.ziyonet.uz;
9. www. info@ minzdrav.uz;
10. www.info@tma.u

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Chapter-1

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