MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN

CHIRCHIK BRANCH OF TASHKENT STATE MEDICAL UNIVERSITY

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CHRONIC SKIN DISEASES: CLINICAL FEATURES, EPIDEMIOLOGY AND PREVENTION

The Electronic Study Guide

From the discipline of "Dermatovenerology"

For the students of medical institutes

Chirchik 2026

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The electronic study guide was prepared based on the standard program, working program, current curriculum of the discipline "Dermatovenerology" and the qualification requirements for students of the specialties "Dermatovenerology" for the directions of 60910100 – Dentistry, 60910200 – General medicine, 60910300 – Pediatrics, 60910400 – Preventive medicine

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This electronic study guide was reviewed at the TSMU meeting and recommended for approval.

This electronic study guide was reviewed at a meeting of the TSMU Scientific Council and recommended for approval.

LIST OF ABBREVIATIONS

Ig - immunoglobulin
RW - Wassermann reaction
HIV - human immunodeficiency virus
SCF - scarlet fever
HSV - herpes simplex virus
WHO - World Health Organization
IHD - ischemic heart disease
IFA - immunoenzyme analysis
STI - sexually transmitted infections
SGD - skin and genital dispensary
CSC - clinical serological control
CSR - classical serological reactions
SMR - false positive reactions
Exercise therapy - physiotherapy exercises
ICD - International Classification of Diseases
NSAIDs - non-steroidal anti-inflammatory drugs
PCR - polymerase chain reaction
RIBT - white treponema immobilization reaction
IFR - immunofluorescence reaction
MR - microprecipitation reaction
PGR - passive hemagglutination reaction
KBR - complement fixation reaction
ECT - erythrocyte sedimentation rate
UHF - ultrahigh frequency flows
UGH - urogenital chlamydia
UT - ultrasound therapy
UV - ultraviolet light

ANNOTATION

Chronic skin diseases can be caused by rashes or other pathologies on the skin or pathological processes in other organs. Nowadays, scientific and practical research aimed at studying the features of the development of chronic skin diseases, increasing the effectiveness of patient treatment, improving diagnostic methods, and preventing disease recurrence is of great importance.

Despite numerous studies of chronic skin diseases, their combined forms are still a complex problem, creating additional difficulties for diagnosis and treatment. The pathogenesis and etiological factors of this group of diseases are not fully understood. In the diagnosis of chronic skin diseases, examination and identification of internal organs are of decisive importance. This manual provides detailed information on the causes, treatment methods and complications of chronic skin diseases, as well as recommendations and instructions for the treatment of chronic skin diseases. Predominant contributors to the development of persistent skin conditions encompass a range of elements. These include a patient's limited understanding of health-related matters, disregard for a wellness-oriented lifestyle, adverse residential circumstances, neglect of fundamental hygiene practices, and contact with detrimental occupational substances. Research within the spheres of social and hygienic studies has revealed a link between the emergence of long-term skin ailments and factors such as dwelling and living arrangements, the presence of inadequate sanitary conditions within the household, and a lack of regular physical exertion. The individual traits of a patient suffering from a sustained skin disorder, alongside their ability to adjust to the illness and their emotional and psychological well-being, play a substantial role.

Cultivating a culture of healthy living among the populace, commencing in early life, is achieved through the implementation of initiatives that educate individuals about health risk factors, encourage motivation, and establish environments conducive to a healthy lifestyle, incorporating exercise and athletic pursuits.

This educational resource, titled “Chronic Skin Diseases”, is crafted for medical students in undergraduate programs at institutions of higher learning and serves as a crucial tool for all primary care physicians in the accurate diagnosis and therapeutic strategies for mycotic infections, complies with educational standards and is recommended for use in the educational process.

INTRODUCTION

The number of chronic dermatoses remains high and is an urgent problem of modern dermatology. The most common nosologies in the structure of chronic dermatoses include atopic dermatitis (AD), eczema and psoriasis. According to WHO, more than 20% of the Earth's population suffers from skin diseases.

Long-term skin disorders frequently manifest in conjunction with underlying systemic illnesses, which can intensify the symptoms of the dermatological condition. For instance, an analysis of patients within a dermatological hospital revealed that nearly 30% exhibited comorbid conditions. The persistent state of stress, often experienced by these individuals, along with the nature of their occupations—involving strenuous physical work and exposure to detrimental environmental influences—significantly contributes to the progression of these ailments. Key risk factors include a patient's limited understanding of medical information, negligence regarding a healthy lifestyle, inadequate living environments, disregard for personal hygiene standards, and exposure to harmful workplace elements.

Research in social hygiene has established a correlation between the development of chronic skin conditions and variables such as residential circumstances, the presence of substandard sanitary conditions within the household, and insufficient engagement in physical activity. The psychological profile of a patient with a persistent dermatological issue, including their ability to adapt to the illness and their emotional state, is of considerable importance. Fostering a healthy lifestyle within the population, beginning in childhood, is achieved through initiatives that educate individuals about health risk factors, cultivate motivation, and provide an environment conducive to healthy living, encompassing physical exercise.

Consequently, in individuals with chronic skin and subcutaneous tissue diseases, particularly psoriasis, eczema, and atopic dermatitis, endocrine influences, metabolic irregularities, concurrent illnesses, and occupational exposures play a pivotal role in the disease's pathogenesis. These factors, recognized by both national and international medical experts, are instrumental in the development of these dermatological conditions.

Acute inflammation of the epidermis and dermis under the influence of dermatitis.

Classification: a) simple contact dermatitis, b) allergic dermatitis, c) toxicoderma.

Simple contact dermatitis. The causes of simple contact dermatitis can be mechanical irritation due to mechanical pressure, friction (tight shoes, bandages,

plaster bandages, etc.); physical irritants - high and low temperatures (burns, frostbite, solar dermatitis); chemical agents - strong acids and alkalis, salts of alkali metals and mineral acids, which affect the body both in domestic and industrial conditions; biological factors - primrose, butterbur, meadowsweet, etc.

Clinically, the condition presents with redness and swelling, exhibiting distinct margins, alongside the emergence of small blisters and larger bullae, and immediate erosions at the point of contact with the irritant, accompanied by sensations of burning and pruritus. The progression is rapid, demonstrating no propensity for dissemination, and resolves upon removal of the offending agent. Systemic therapy is typically unnecessary; cessation of exposure to the irritant and topical application of pastes, lotions, or corticosteroid creams are usually sufficient. Conversely, allergic dermatitis arises from the body's heightened sensitivity. A diverse array of chemical substances can act as allergens, including pharmaceuticals such as novocaine, antibiotics, mercury-based compounds, resorcinol, iodine, analgesics, and antihistamines. Furthermore, synthetic textiles, various fragrances and cosmetics, and phenol-formaldehyde resins can trigger reactions. Plant-derived allergens include those found in primroses, chrysanthemums, tulips, saffron, snowdrops, dandelions, jasmine, poplar, citrus fruits, garlic, radishes, and carrots. Individuals with a familial predisposition, those experiencing psychological stress, and those with chronic infections are more susceptible to allergic reactions. The onset of the disease typically occurs within three months of initial exposure. The clinical presentation is characterized by a wide range of manifestations, developing after a latent period rather than immediately upon contact, and affecting areas beyond the point of exposure, indicative of systemic allergen distribution. Upon an erythematous and edematous base, small vesicles, papules, weeping lesions, and erosions develop. Resolution occurs following allergen removal, though recurrence is possible with re-exposure. Skin eruptions are associated with pruritus and burning. Treatment involves systemic desensitization with calcium supplements, antihistamines, and adsorbents, alongside the elimination of allergen exposure. Topical treatments include soothing suspensions, pastes, and glucocorticoid ointments

PRIMARY MORPHOLOGICAL ELEMENTS

Primary morphological elements are rashes that appear on intact skin in the early stages of the disease. Primary elements are conditionally divided into two groups, depending on the mechanism of their appearance and the stage of inflammation (alteration, exudation, proliferation). Primary proliferative morphological elements are distinguished: macula, papula, tuberculum, nodule, and primary exudative morphological elements: vesicle, bulla, pustule, and urtica.

A macula is a limited discoloration of the skin that does not rise above the skin surface and is not palpable. Depending on the cause of the spot, it is divided into vascular, pigmented and artificial types, and into inflamed and non-inflammatory spots (Figure 10).

MACULA

A change in the color of the border of the skin. Spots are divided into inflammatory (roseola, erythema) and non-inflammatory (petechiae, ecchymosis, hemorrhages). Depending on the cause of their appearance, they are divided into vascular, pigmented and artificial types

Vascular spots - are observed due to temporary reflex dilation of blood vessels, inflammation or injury.

Inflammatory vascular spots include roseola and erythema. Roseola is a pink-red spot up to 1 centimeter in size (erythema is larger than 1 cm), resulting from temporary dilation of blood vessels. When pressed with a magnifying glass (diascopy), it disappears, when the pressure is stopped, it reappears and may be accompanied by subjective irritation, itching, and in rare cases, pain. Later, the surface becomes crusted or disappears suddenly. These rashes occur in dermatitis, the secondary stage of scabies, and other diseases.

Non-inflammatory spots include spots that appear as a result of pathological dilation, injury, or changes in permeability of blood vessels, as well as pigmented and artificial spots.

Spots that appear as a result of increased vascular permeability or vascular injury are called hemorrhagic spots, and they do not disappear during diascopy and change color over time. A straight-line hemorrhagic spot is called a vibrissae, and hemorrhagic spots, depending on their size, are called petechiae, purpura, ecchymoses, and hematomas.

Pigmented spots - appear as a result of the loss (depigmentation or vitiligo), decrease (hypopigmentation) or increase (hyperpigmentation) of the pigment melanin in skin cells.

Artificial spots - appear as a result of the penetration of any dye or chemical substances into the skin. Artificial spots often occur in people of various professions and are considered a "stigma", that is, a professional sign.

A nodule (papule) is a primary, proliferative, non-cavity, superficial element rising above the skin surface, up to 1 cm in size, which appears as a result of pathological changes occurring in the epidermis and dermis. Over time, it changes, is absorbed, and a spot remains in its place. A nodule occurs in the secondary stage of psoriasis, lichen planus, and lichen.

A tubercle (tuberculum) is a primary, proliferative, cavity-free, elevated above the skin surface, up to 1 cm in size, deep element that appears as a result of pathological changes in the dermis. The tubercle later undergoes necrosis, becomes ulcerated, and finally forms a scar. In some cases, the tubercle is absorbed, forming an atrophic scar in its place. The tubercle occurs in the tertiary stage of cutaneous tuberculosis (tuberculosis), leprosy (leprosy), malignant lesions (leishmaniasis), and ulcers.

A nodule (nodus) is a primary, proliferative, cavityless, elevated above the skin surface, from 1 cm to an egg-shaped, deep element, which is formed and grows in the dermis and subcutaneous fat layer. Later, the nodule is absorbed and ends with an atrophic scar or wound, forming an ulcer, scar. A nodule occurs in the tertiary stage of skin tuberculosis, leprosy, severe wounds and ulcers.

A vesicle (vesicula) is a primary, exudative, cavityless, elevated above the skin surface, containing serous fluid or blood in its cavity, up to 1 cm in size. The roof, bottom and fluid-filled space between them are distinguished. Later, the vesicle dries up and forms a crust or ruptures, forming erosion and then a crust, and disappears, forming a temporary pigmented spot. A blister is found in various herpes diseases.

A blister (bulla) is a primary exudative, hollow, raised above the skin surface, with dimensions greater than 1 cm. A blister differs from a vesicle only in size. The blister stores serous fluid, forming a crust, erosion, and pigmented spot after itself. This rash occurs in dermatitis and purulent wound diseases (Fig. 10).

A pustule is a primary, exudative, hollow, raised above the skin surface, containing pus in its cavity, and can be a superficial or deep element. Pustules can be located around the hair follicle or in the superficial and deep dermis layers of the epidermis, and then rupture, leaving erosion or an ulcer, crust, pigmented spot, or scar in their place. This rash occurs in purulent skin diseases and secondary abscesses.

Hives or hives (urtica) are primary exudative, raised above the skin surface, without cavities, arising quickly and quickly passing due to swelling within the cells. They do not leave any complications and occur in donkey feed disease and insect bites.

SECONDARY MORPHOLOGICAL ELEMENTS

Secondary spot – Pigmented spot (pigmentation, depigmentation) can appear after all primary rashes, in their place. Pigmented spot appears due to temporary accumulation, loss or decrease in the skin pigment melanin in some area. Pigmented spots are darker (hyperpigmentation) or whiter (hypopigmentation) than the original skin color, and in some cases, pigment is completely lost in some part of the skin – this is called vitiligo or depigmentation.

Squama. The shedding and separation of the cells of the stratum corneum of the skin as a result of their development is a physiological phenomenon. In some diseases, excessive scaliness or flaking occurs. Dandruff comes in different colors and sizes, and based on these signs: floury, scaly, and plate-like dandruff are distinguished, and large plate-like migration of dandruff is called exfoliative dandruff. Dandruff can also develop primarily: in scabies, soft leukoplakia, parakeratotic dandruff occurs in exfoliative dermatitis. In ichthyosis, hyperkeratotic dandruff is formed.

Erosion, erosion (erosio) - is a superficial defect of the skin, mucous membranes or red border of the lips on the surface, epidermis, therefore they disappear without a trace. Erosion is formed as a result of rupture of the roof of the primary hollow elements: bubble, vesicle, abscess.

Ulcer (ulcus) - is a deep defect of the skin, the bottom of which is located in the dermis or hypodermis. Therefore, the wound always ends with a scar. Ulcers mainly occur after a lump, nodule or deep abscess. The edges and bottom of the wound are different. The size, shape, depth, appearance of the edges and bottom of the wound are different and have a specific appearance in different dermatoses. If a single, non-fused lump is injured, a wound with a diameter of up to 0.5 cm is formed. If a knot is injured, a somewhat larger and deeper wound is formed. The edges of the wound may be carved, hanging down, or have a plate-like appearance. The bottom and edges of the wound may be soft or dense in consistency, and the bottom may be covered with purulent, necrotic, purulent-bloody mass, covered with pus-like granulations, and may bleed easily. Deep wounds can reach the bone and injure it. When examining a wound, attention should be paid not only to its shape, size, or bottom. At the edges of the wound, there are often remnants of the main pathological process, and the presence of characteristic discharges in its depth can provide information about the process that led to the wound.

Crack (fissura, ragades) - a violation of the integrity of the skin due to drying out and loss of elasticity of the skin. Cracks are straight and often occur in areas of skin stretching: folds, corners of the mouth. If the crack is deep enough and injures the dermis and even the hypodermis, it causes severe pain, the surface is covered with a bloody crust, and when it heals, a scar is formed. Superficial cracks are observed only in the epidermis, do not heal and heal without a scar.

Fissure (FISSURA) A fissure occurs when the skin dries out and loses its elasticity. Fissures can be superficial or deep

ULCUS (Ulcer) A deep and fat-filled break in the skin

SQUAMA (Squama) A scaling or peeling phenomenon resulting from parakeratosis and hyperkeratosis

KERATOSIS: A tendency for keratin cells to proliferate

Crust (crusta) - is observed as a result of drying of exudates inside vesicles, pustules, or as a result of drying of the exudate on the surface of erosions and wounds. Therefore, the crusts are serous, bloody, purulent, and rise above the skin surface. Newly formed crusts are soft and easily removable, erosion or ulcers are observed under them. Long-standing crusts are thick, hard, and tightly adhere to the underlying tissue.

The color of the crust depends on the nature of the exudate, if it is formed from serous exudate, it is a brownish-yellow crust, from serous-purulent exudate - a yellowish-blue crust, from purulent exudate - a dirty-brown or blue crust, and from hemorrhagic exudate - a black bloody crust. The thickness of the calluses varies, and in many cases depends on the process in the skin and when the callus was formed. Calluses that stick together, thicken as a result and rise above the skin are called rupia. Depending on the appearance, color, and size of the callus, it is possible to determine what primary elements it is formed from. In addition to calluses, there are also scaly calluses, which are calluses that have absorbed the serous exudate of the epidermis and stuck together. Unlike calluses, scaly calluses do not break or crumble.

Scar (cicartix) is a new tissue formed as a result of deep injury to the dermis and hypodermis, as a result of wound healing. The scar is mainly composed of collagen fibers. The skin on the surface of the scar is usually smooth, shiny, and does not have a skin pattern or hair follicles. New scars are pale pink, later turning white, and in some cases, the scars are hyperpigmented. After some diseases, the scar surface is layered, bumpy, bumpy, and sucker-like, while some have a pocket-like appearance. The size and shape of the scars vary.

Atrophic scars are usually scaly, have a soft consistency, and are thin, sunken below the skin surface. In some cases, they are rough, dense, thick, and raised above the skin surface and are called hypertrophic or keloid scars. If the scar appears without a wound, it is called cicatricial atrophy (scarred thin skin), and the vessels underneath are visible, easily folded, and resemble crumpled cigarette paper. Scars of various shapes and sizes are observed in various diseases: star-shaped, stamp-shaped, mosaic-shaped, bridge-shaped, pocket-shaped, keloid-hypertrophic, and atrophic types are distinguished.

CRUST (CRUSTA) Appears as a result of the drying of serous, blood or purulent fluid on the skin.

SCAR (CICATRIX) A wound on the skin that remains after deep cracks in the skin. The scar can be normotrophic, atrophic, hypertrophic (keloid)

SPREADING (VEGETATIO) Or spreading. This phenomenon is often observed in the form of nodules, blisters, and ulcers located in skin folds, where the epidermis and the superficial layer of the dermis grow.

LICHENIFICATION - roughening of the skin (LICHENIFICATIO)

Lichenification is a specific change in the skin. In it, the skin becomes dry, the lines on the surface, that is, the skin patterns, become clearly visible, and this area of the skin becomes inflamed, rough and hyperpigmented

SECONDARY MORPHOLOGICAL ELEMENTS.

Vegetation (vegetatio) - means spreading. It is accompanied by thickening of the spinous layer of the epidermis and growth of the sebaceous layer of the dermis. The appearance of the vegetation resembles a rooster's crown or cauliflower, has a soft consistency, the surface is dry, brownish, in some cases it is eroded, reddish, secreting serous, serous-purulent exudate. The vegetation often forms on the surface of ulcers, wounds or nodules. In some cases, there may also be a primary rash (condylomas with sharp tips). Lichenification (lichenificatio) is a drying, thickening and coarsening of the skin, which is observed due to strong infiltration and acanthosis in the epidermis and sebaceous layer of the skin. The skin texture is enhanced. Such skin is persistently hyperemic, dense, and the surface is scaly. Lichenization is usually accompanied by the addition of small nodules, chronic inflammatory infiltrate and severe itching and occurs in neurodermatitis and other chronic skin diseases.

A lesion is a linear skin defect (often a superficial skin defect) that occurs mainly as a result of scratching, scratching, or flaking. If the defect is only in the upper part of the epidermis, a linear scaly appearance occurs. As a result of severe itching, scratching occurs, extending to the deeper dermis, bloody crusts appear on the surface, and hypo-depigmented spots and scars are also observed in the place of excoriation. Excoriation occurs in skin and systemic diseases accompanied by itching.

Atrophy (atrophia) - occurs due to a violation of skin trophic, the skin becomes thinner, slightly sunken compared to the surrounding skin, loses its natural color, is hypo-pigmented, and is observed in the form of foci. Atrophic rash occurs in diseases such as scleroderma and erysipelas.

Thus, there are 8 types of primary and 10 types of secondary rashes that occur on the skin. If the primary rashes on the patient's skin are the same, we call this a monomorphic rash, that is, the same rash. For example, with scabies - the skin has the same rashes, that is, nodules. Now, if two or more, several types of primary rashes are formed, we call this a truly polymorphic rash, that is, a variety of rashes. For example, in the secondary stage of scabies, the patient may have nodules, roseola, and purulent rashes on the skin at the same time. So, there are 3 types of primary rashes here, which are called true, true polymorphic rashes. However, there can also be false, false polymorphic rashes on the skin. In this case, you can see several different secondary rashes that appear on the skin as a result of the same

primary rashes occurring at different times and their changes. For example, there are several blisters on the skin and at the same time secondary rashes with oozing, crusting, and purulent rashes. That is, there are secondary rashes that appear as a result of some of the blisters bursting, crusting, and the blisters drying out, and an infection settling in the crust and blisters. It is essential for students to learn how to look for, see, and correctly identify a rash on a patient's skin in practice. Because tomorrow, after hearing a patient's complaint during work, the doctor's first clinical action will be to look for any changes on the patient's skin, give it a correct assessment, and determine a skin or systemic disease through the rash, that is, to think about the diagnosis.

The skin is the outer covering of the body and performs a number of complex physiological functions. The skin actively participates in the metabolism of substances, mainly in the exchange of water, minerals, fats, carbohydrates, vitamins and energy. The skin participates in the life processes of the organism and performs a number of necessary functions: immune, protective, secretory, respiratory-excretory, resorption, thermoregulatory, receptor, metabolic, and others.

Protective function - the barrier properties of the skin protect the skin and the whole organism from various mechanical, light, and microbial influences. The skin is protected from drying out by its dense stratum corneum. The stratum corneum is resistant to weak physical and chemical influences. Protection from various microbes is carried out due to the stratification and migration of the epithelium, the secretion of sweat and sebaceous glands. The skin's surface is covered by a slightly acidic, aqueous-lipid film, which functions as a natural barrier, inhibiting both microbial proliferation and the absorption of external compounds. This protective layer, known as the acid mantl d3 effectively impedes the invasion of pathogenic microorganisms, while its constituent low molecular weight fatty acids actively suppress the growth of harmful bacteria and fungi. Furthermore, the presence of melanin within the skin provides a crucial defense mechanism against the detrimental effects of solar radiation. Melanin's capacity to absorb ultraviolet light shields the body from potential damage caused by excessive sun exposureRespiration-excretion and resorption function. The resorption properties of the skin depend on the functional activity of the sebaceous follicles, the state of the water-fat mantle, and the maturity of the stratum corneum. The resorption properties of the skin are weak due to the presence of physiological hyperkeratosis and the absence of sebaceous glands in the skin of the palms and soles.

In areas with a large number of sweat glands and a poorly developed stratum corneum, the resorption properties of the skin are good: (drugs are applied) fat-soluble drugs - iodine, dimedrol, resorcinol, salicylic acid, etc. The skin facilitates

a minimal level of gas exchange, involving the uptake of oxygen and the expulsion of carbon dioxide, though these processes occur on a very limited scale.

Regarding thermoregulation, the body employs a variety of adaptive mechanisms to maintain a stable core temperature. Beyond the insulating properties of the stratum corneum, the outermost layer of the epidermis, the fibrous substances of the dermis and hypodermis are also of significant importance. Also of significant importance are the blood-lymphatic circulation and the secretory properties of the sweat-sebaceous glands.

Secretory function of the skin. This function is carried out due to the secretory activity of keratinocytes, immunoregulatory cells and the activity of the sweat-sebaceous glands. The output of sebaceous glands, known as sebum, is a complex mixture comprising various components. This secretion includes fatty acids, cholesterol esters, aliphatic alcohols, trace amounts of hydrocarbons, free cholesterol, glycerol, and minimal quantities of nitrogen-containing and phosphate-containing compounds. The secretion of the sebaceous gland is mainly in the form of a liquid, semi-liquid substance.

Mixing with sweat, sebum forms a thin film of water-lipid mantle on the skin surface, which protects the skin by having a bactericidal and fungistatic effect. Beyond their role in producing secretions, sebaceous glands also function in the elimination of substances from the body. Alongside sebum, these glands facilitate the excretion of various toxins, medium-sized peptides, and a range of pharmaceutical compounds, including iodine, bromine, antipyrine, and salicylic acid, etc. are secreted. In different people, in different areas of the skin, sebum secreted in different amounts is observed. The areas where the most sebum is secreted are: the hairy part of the head, forehead, cheeks, nose (1000 sebaceous glands per 1 sq. cm), the central part of the chest, the interscapular area, the shoulder girdle, the intercostal area.

Sweat glands produce sweat, cool the skin, and ensure a relative constancy of body temperature. The secretion of eccrine sweat glands is a liquid, slightly saline reaction, containing various dissolved inorganic and organic substances in addition to water. Through sweat, The body eliminates a range of pharmacological agents through the skin, including iodine, bromine, mercury, quinine, and antibiotics. On average, the body discharges between 750 and 1000 milliliters of perspiration each day, and when high temperatures are observed, up to several liters of sweat can be excreted.

The excretory function of the skin is observed along with the secretory function. Along with sweat-fat secretions, along with organic, inorganic substances, products of mineral metabolism, carbohydrates, vitamins, hormones, enzymes,

microelements and a large amount of water are excreted from the body. Sweating is a constant, continuous process, and there are invisible sweat and profuse sweat.

He skins plays a significant role in metabolic processes through its capacity for storage. Owing to the water-attracting characteristics of connective tissue cells, elastic, collagen, and reticular fibers, as well as the subcutaneous adipose layer, the skin retains fluids, minerals, vitamins, and trace elements both within and between cells. Additionally, it serves as a repository for carbohydrates, cholesterol, iodine, bromine, amino acids, and various metabolic byproducts. Consequently, cutaneous manifestations can reflect systemic metabolic disturbances, ranging from persistent pruritus associated with hepatic dysfunction to pyogenic eruptions indicative of latent diabetes. Vitamins exert a profound influence on skin health. The B-complex vitamins are crucial for maintaining normal oxidation-reduction reactions. Vitamin PP facilitates the elimination of metabolic waste and detoxification. Vitamins A, E, and D act as anti-infective agents, stimulate protein synthesis, regulate keratinization, and promote epithelial regeneration during inflammatory conditions.

Furthermore, the skin functions as a complex sensory organ. It not only shields the body from external stressors but also acts as a multifaceted analyzer, possessing an extensive network of receptors. This sensory function is mediated by a diverse array of sensory nerve endings and specialized corpuscles distributed throughout the skin's layers. The skin has the ability to perceive pain, temperature and pressure. The sensation of tactile effects is strongly developed in the fingertips, in the area of large folds, and in the mucous membrane of the tongue. The functional state of the receptor area of the skin is inextricably linked with the central and autonomic nervous systems. The skin is exposed to various influences from the external environment, the central nervous system and internal organs and returns the necessary response.

The development of the skin commences during the initial weeks of fetal growth, originating from the ectodermal and mesodermal germ layers of the embryo. The ectoderm gives rise to the epidermis, while the mesoderm differentiates into the dermis and the subcutaneous adipose tissue. In the early gestational period, specifically at 3 to 4 weeks, the ultrastructure of the skin reveals a single layer of columnar cells in certain regions, whereas the palmoplantar areas exhibit two layers of these cells.

By 6 to 7 weeks of embryonic development, the epithelial covering of the fetus is composed of two distinct layers: the basal layer and the developing dermis. At the seven-month mark of gestation, all fully developed layers of the epidermis are discernible, with the emergence of keratinized cells in the palmoplantar regions. This stage also witnesses the formation of elastic and collagen fibers, hair, nails, and hair follicles. Initially, the basal membrane, characterized by a smooth contour,

transitions to a wavy configuration as the dermis develops, with cytoplasmic extensions mirroring the dermal contour along its longitudinal axis. In the final stages of gestation, the skin's structure undergoes refinement, culminating in a fully functional organ capable of executing a comprehensive and diverse array of physiological roles.

The epidermis is the upper, multilayered part of the skin, consisting of 5 layers of cells. The cells differ from each other in shape, number and functional state. The basis of the epidermis is the basal or main layer (Stratum basalis), then the spinous (Stratum spinosum), granular (Stratum granulosum), shiny-transparent (Stratum lucidum) and horny (Stratum corneum) layers are distinguished. The cells of the outer horny layer are constantly migrating and renewing. Therefore, the horny layer is conditionally divided into two layers: a layer of somewhat dense keratinocytes - located on the surface of the transparent layer and is also called the connective, and the surface layer - easily migrating, consisting of completely keratinized keratinocytes.

Under the basal layer, consisting of a number of prismatic cylindrical cells and on the border of the dermis, the basement membrane is formed from stellate growths of cells. The basement membrane provides a strong connection between the epidermis and the dermis.

The basal layer's keratinocytes exhibit continuous proliferation, undergoing active mitosis. Consequently, the cytoplasm of these cells is rich in structures essential for DNA and RNA storage, namely ribosomes and mitochondria. This activity is vital for the generation and replenishment of the epidermis's superficial layers. Interspersed among the basal layer's cells are melanocytes, responsible for melanin pigment production, as well as specialized cells: Langerhans cells, which are dendritic epidermocytes, and Merkel cells, which function as sensory receptors.

On the surface of the basal layer is a spinous layer consisting of 3-8 rows of cells. These cells have numerous cytoplasmic tumors (spines or bridges, acanthus). The cells have a dense cell membrane - tonofibrils and tonofilaments. Cytoplasmic growths (acanthus) connect cells with each other and form a network of channels between them, through which intercellular fluids circulate.

Desmosomes and tonofibrils provide the internal supporting skeleton of cells, protecting cells from mechanical injuries. The spinous layer also contains white tumor-like epidermocytes, which, together with keratinocytes, perform an immunoprotective function.

After the spinous layer, a granular layer consisting of 1–3 layers of cells is located, the cells of this layer consist of 3–4 rows of cells in the palmar-soleus area. These cells have a rhomboid, flattened shape in areas close to the skin surface, and cylindrical and cubic in the lower part. The number of structures storing DNA and

RNA in the nuclei of keratinocytes is sharply reduced, and granular inclusions - keratohyalin are observed in their cytoplasm. Due to the formation of tonofibrillar-keratohyalin structures in the cells of the granular layer, this layer is often called the keratohyalin layer.

In the protoplasm of the cells of the granular layer, the product of keratohyalin reduces the secretion of a growth factor for epidermal cells, leads to the accumulation of substances that stop mitotic division, polypeptide kelons. Since mitotic division occurs in the cells of the basal, spinous and granular layers, these layers are often generalized and are also called the growing layer of the epidermis (Malpighian layer).

Within the cells of the stratum granulosum, the process of keratinization involves the synthesis of keratohyalin, and subsequently, eleidin. This eleidin is then transformed into the stratum lucidum, a translucent layer that retains the eleidin. This layer is particularly prominent in the skin of the palms and soles. Ultimately, keratinocytes are derived from the eleidin substance, which then constitute the stratum corneum, the outermost layer of the epidermis.

The stratum corneum is somewhat thick and consists of tile-like, nuclear-free, plate-like cells that are tightly packed together. The surface cells of the stratum corneum are constantly migrating and renewing. Epidermis - performs protein synthesis, pigment formation, protective and immunological functions.

The epidermis is bounded by the basement membrane. The basement membrane is 40–50 nm thick and has an uneven contour, repeating the direction of attachment of the epidermis to the dermis. The main function of the basement membrane is a barrier function, limiting the penetration and diffusion of circulating immune complexes, antigens, autoantigens and other biologically active mediators. In addition, the basement membrane is actively involved in the exchange of substances between the epidermis and the dermis.

The dermis or the main skin (cutis propria) is composed of cellular elements, fibrous substances and intermediate substances. The thickness of the dermis is 0.49–4.75 mm. The connective tissue part of the skin is conditionally divided into two layers: the surface, the subepidermal layer - papillary (Stratum papillare) and the deep, reticular (Stratum reticulare).

The superficial portion of the dermis extends beneath the epidermal ridges, establishing a foundation for the sebaceous glands situated beneath the stratum spinosum and stratum basale. This layer is composed of an unstructured matrix and delicate fibrous connective tissue, interspersed with a multitude of cellular components, vascular structures, and neural terminations. The cellular constituents of the dermis include fibroblasts, fibrocytes, histiocytes, mast cells, and specialized pigment-containing cells, known as melanophages. A network of small blood

vessels resides within the sebaceous glands, providing essential nutrients to the epidermis, dermis, and nerve endings.

The deeper reticular layer of the dermis exhibits a denser, coarse fibrous structure, constituting the majority of the dermal thickness. The dermal stroma is primarily composed of bundles of collagen fibers, enveloped by a network of elastic fibers, with cellular elements distributed throughout the matrix. The structural integrity of the reticular layer is crucial for the skin's tensile strength.

The hypodermis, or subcutaneous adipose layer, comprises interconnected bundles of connective tissue, housing varying quantities of spherical adipocytes. Within the hypodermis, blood vessels, nerve fibers, nerve endings, sweat glands, and hair follicles are located. The hypodermis terminates at the fascia, and in certain anatomical regions, it may merge with the periosteum or muscle aponeuroses. The vascular and lymphatic systems permeate these dermal layers. The arteries that nourish the skin form a wide network of bundled vessels in the lower parts of the hypodermis. From these networks, small arteries spread, which divide and connect with each other, forming a network of anastomoses and forming the subdermal arterial network. The subdermal arterial network branches and forms anastomoses, rising straight or obliquely upwards, forming the surface vascular network of the dermis at the border of the reticular and sebaceous layers. From these vascular bundles, arterioles begin, forming the dermal sebaceous arteriole network. The capillary density in this area is 16–66 capillaries per 1 sq. mm of skin.

Hair follicles, sweat and sebaceous glands are supplied with vessels. The venous system of the skin begins with postcapillary venules, which are formed from the sebaceous layer and form four venous bundles in the hypodermis (Fig. 6). One of the distinctive features of the vessels in the skin is that they form anastomoses with the same and different vessels to a very high degree.

Arteriovenous anastomoses, glomuses, are common in the skin, which are short connections of arterioles and venules without capillaries. They participate in maintaining body temperature and maintaining the level of interstitial tension, which in turn ensures the functioning of capillaries, muscles, and nerve endings.

The lymphatic system of the skin is observed in the form of capillaries, which are located on the surface and deep vascular networks. Lymphatic networks form anastomoses with each other and have valves. After passing through the hypodermis, they form a wide bundle of bundles at the borders of the aponeurosis and fascia. The receptor function of the skin is of great importance. Since the skin is considered a barrier organ between the external and internal environment, it is also subject to various influences. The skin is innervated by the central and autonomic nervous systems and is considered a sensory receptor area. There are nerve endings of various types in the skin: branched, nodular. They innervate sweat, sebaceous glands, hair

follicles and vessels, in addition to which they have their own capsules, corpuscles and nerve endings. The main nerve bundle of the skin is located in the subcutaneous fat layer. From this bundle, large nerve fibers go to the skin formations and the sebaceous layer, spreading in the form of cylindrical axons to the superficial branches, sebaceous layer and epidermis. They reach the granular layer in the epidermis, where the nerve fibers lose their myelin sheaths and become thinner. In addition to free nerve endings, there are special nerve formations in the skin that receive various influences. Capsular-sensory corpuscles (Meissner corpuscles) provide sensation. Cold is felt by Krause's tubules, and Ruffini corpuscles, which sense heat, and plate-shaped corpuscles, which sense pressure (Fater-Pacini corpuscles), are distinguished. Free nerve endings located in the epidermis provide sensations of pain, itching and burning. Sensory corpuscles are located in the sebaceous layer, consist of a thin connective tissue capsule, and are composed of special receptor cells. In addition to these, the skin contains many vegetative nerve fibers, which are located on the surface of all blood vessels, as well as in short capillaries. They regulate the functional activity of the vascular network.

Skin formations (hair, nails, sweat and sebaceous glands) (Fig. 4). The formation of hair begins at 2–3 months of gestation. Basal cell growths appear in the epidermis, which later develop into hair follicles. By 4–5 months, the first hairs, in the form of fine hairs, spread over the surface of the body. The palms, heels, red border of the lips, mammary glands, labia minora, glans penis and the inner layer of the glans penis are excluded from hair.

The part of the hair that rises from the skin is called the hair shaft, and the part inside the skin is called the hair root. The part of the hair that protrudes from the skin has a depression called the funnel. The hair shaft and root consist of three layers: the central one - the medulla, the cortex and the cuticle. The part that forms the medulla is located mainly in the root part inside the skin and reaches the funnel part of the hair follicle. The bulk of the hair shaft is made up of keratinized cells, which are located tightly to each other. The distal part of the hair root is called the hair bulb. The hair bulb provides hair growth. Blood vessels and nerves enter the hair follicle from the hypodermis. The funnel of the hair follicle is covered with 1–3 rows of epidermal cells. The excretory duct of the sebaceous gland opens into the funnel part. Hair color depends on the amount of pigment produced by melanocytes in the medulla of the hair. Hair is divided into feather, hair (beard, eyelashes, eyebrows, genitals) and long hair (hair on the head). Hair growth continues slowly, at a rate of 0.3–0.5 mm per day. Nails begin to develop from the 3rd month of the embryo. First, the nail plate is formed, the epithelium on its surface thickens slightly and is slightly immersed in connective tissue. Then, from the epithelial part of the nail plate - the matrix, a dense nail root is formed. The further development of the nail is

inextricably linked with the keratinization process. Therefore, the nail is made of dense, tightly attached horny plates and has a shiny surface, located in the nail plate. The base of the nail plate and the skin folds on both sides are limited by the nail pads. The posterior nail pad surrounds the proximal part of the nail in an arcuate manner, and the horny plate of the thin epidermis forms the skin over the nail, a small part of the nail root is visible from under the posterior pad, which is called the nail bed. Nail growth is due to matrix cells.

Beyond the formation of the epidermis, hair, and nails, the ectodermal layer of the developing fetus also gives rise to the sweat and sebaceous glands. The genesis of sweat glands initiates during the second month of gestation. While these glands are structurally developed at birth, their functional capacity remains limited. By the age of two, a significant increase in the functional activity of these glands is observed. Normal sweat glands exhibit differentiation, varying in their mode of secretion. Structurally, these glands are tubular, releasing secretions through both the activity of secretory cells and passive diffusion processes. The distal portion of the sweat glands is situated at the junction of the dermis and hypodermis. The extended excretory duct ascends perpendicularly towards the skin surface, culminating in a spiral or serpentine-shaped opening. There are a lot of sweat glands in the palms, soles, and face. Sweat glands are absent on the glans penis, the outer surface of the labia minora, and the inner sheet of the glans penis. In other areas of the skin, they are scattered. 200–800 sweat glands can be observed on 1 sq. cm of skin. Nails begin to develop from the 3rd month of the embryo. First, the nail plate is formed, the epithelium on its surface thickens slightly and is slightly immersed in connective tissue. Then, from the epithelial part of the nail plate - the matrix, a dense nail root is formed. The further development of the nail is inextricably linked with the keratinization process. Therefore, the nail is made of dense, tightly attached horny plates and has a shiny surface, located in the nail plate. The base of the nail plate and the skin folds on both sides are limited by the nail pads. The posterior nail pad surrounds the proximal part of the nail in an arcuate manner, and the horny plate of the thin epidermis forms the skin over the nail, a small part of the nail root is visible from under the posterior pad, which is called the nail bed. Nail growth is due to matrix cells.

In addition to the epidermis, hair and nails, sweat and sebaceous glands arise from the ectodermal layer of the fetus. Sweat glands begin to develop in the 2nd month of pregnancy. By the time of childbirth, the sweat glands are sufficiently developed, but their functional activity is observed to be low. By the age of 2, the functional activity of the sweat glands increases. Normal sweat glands are differentiated, and they differ from each other in the type of secretion. Normal sweat glands are tubular in structure, they secrete secretion not only due to the activity of

secretory cells, but also with the participation of diffusion processes. The distal part of the sweat glands is located on the border of the dermis and hypodermis. The long excretory duct is directed vertically to the surface of the skin and ends with a corkscrew-shaped, snake-like fissure. In the palms, soles, and face areas, apocrine skin glands, unlike eccrine skin glands, produce secretion with the participation of glandular cell substances, therefore, some cells are in the stage of migration. Apocrine glands also have a tubular structure, but differ from eccrine sweat glands in size, are somewhat deeper and have a specific location. They are located near the areolas of the anal, genital, and mammary glands. The excretory ducts of the glands open into the sebaceous and hair follicles. The full development of apocrine sweat glands is observed in children by the age of one year, and their functional activity begins only with puberty. The activity of apocrine sweat glands depends on the secretion of the gonads. Therefore, apocrine glands are among the secondary sexual characteristics, there are many sweat glands. Sweat glands are not found on the glans penis, the outer surface of the labia minora, or the inner layer of the glans penis. In other areas of the skin, they are scattered. 200–800 sweat glands can be observed on 1 sq. cm of skin.

Sebaceous glands, classified as having a compound alveolar structure, utilize a holocrine mechanism for secretion. These glands are notable for the sebaceous transformation of their secretory cells. The structure of the primary excretory ducts of these glands mirrors that of the epidermis. Typically, sebaceous glands are associated with hair follicles, with their excretory ducts emptying into the upper segment of the follicle. Each follicle is usually surrounded by six to eight sebaceous glands, resulting in the natural oil coating of hairy skin. Occasionally, sebaceous glands release their secretions directly onto the skin's surface via their excretory ducts. Regions where secretions are discharged independently of hair follicles include facial skin, the glans penis, the prepuce, and the labia minora. Notably, the skin of the palms and soles lacks sebaceous glands. The secretions produced by these glands play a critical role in the physiological, immunological, and biochemical processes of the skin.

The oral mucosa, like the skin, is divided into 3 layers. Epithelium, mucosa, and submucosa.

The epithelial layer differs from the skin in that the oral mucosa does not have a horny, shiny, and granular layer on its surface, but consists of only two layers - the basal and spinous layers.

Epithelial cells can become horny on the hard palate, tongue, and gums. However, it is worth noting that in some chronic diseases, horny and granular layers appear due to inflammation. As a result of keratinization, the appearance and color

of the mucous membrane can change. Such changes can often be observed in lichen planus, leukoplakia, and erythema multiforme.

The submucosa is located deeper and consists of blood vessels, collagen, and elastic fibers, and in addition, salivary glands are also located in it.

The structure of the tongue consists of 3 types of suckers, which are: fibrous, crown-shaped and round. The upper surface of the tongue is covered with fibrous suckers, and crown-shaped suckers are round in shape, 1–2 mm above the level of the tongue and are located mainly behind the tongue. The root of the tongue does not have suckers, but consists of a lot of lymphatic tissue.

The mucous membrane of the tongue does not have a horny, shiny and granular layer, but only spinous layer cells, the nuclei of which are very light in color. The sucker-shaped layer is very developed in the tongue.

The lip is made up of circular muscle layers, covered on the outside with skin, and on the inside with a mucous membrane, which is supplied here with many different glands. The lip consists of 3 parts: skin, red border and mucous membrane. The glands open towards the oral mucosa (Fig. 7).

The red border of the lip is divided into 2 zones, outer and inner. In the outer zone there is a modified horny layer in the epithelium, and in the inner zone there is a transition to the mucous membrane - the so-called Klein zone. In general, the oral mucosa is supplied with blood and lymphatic vessels. Lymphatic networks are located in large numbers on the root of the tongue and in the tonsils. The oral mucosa has a nerve receptor apparatus, which forms reticular nodes and enters the cilia, and at the ends of the nerve fibers form ganglia, which in turn innervate the salivary glands.

Salivary glands are located in the mucous membrane of the oral cavity and are divided into 2: major and minor. The major ones are located around the ears, under the jaw and tongue, are long and open into the oral cavity. The minor or minor salivary glands are located in the lips, soft palate and pharynx.

The main physiological functions of the mucous membrane of the oral cavity are the following - protective, temperature, secretory, resorptive (absorption), receptor, etc.:

1. It performs the function of mechanical and enzymatic processing of food.

2. It moistens food that has entered the mouth with the secretions of its salivary and serous glands.

3. It examines and analyzes food that has entered the oral mucosa and senses and removes foreign objects.

4. The oral mucosa has sensory nerve fibers, which, in turn, perceive pressure and pain.

5. In addition, it is resistant to temperature and chemical factors.

6. The oral microflora is very rich, but the mucous membrane protects them.

7. Another protective function of the oral mucosa is to prevent plaque formation, which is carried out under the influence of ribonucleic acid and glycogen produced by the basal layer cells.

Hair (pili) is a product of the skin, which is found on almost 96% of the body surface. Usually, the most densely haired part of the body is the hairy surface of the head, where their total number reaches 100,000. There is no hair on the palms, heels, pink part of the lips, on the glans penis, on the labia majora and minora. Long (scalp hair, mustache, beard and pubic hair, underarm and pubic hair), hard or hairy (eyebrows, eyelashes, nostrils and earlobes) and pubic hair cover almost the entire body (Fig. 8).

Hair consists of two parts: the hair shaft, which protrudes from the skin, and the root, which is located in the skin. The hair shaft extends into the hair follicle and lies on the skin. The sebaceous glands secrete their products into the hair follicle. The hair root continues in the deep layer of the dermis to the border of the subcutaneous fat cell, where it ends with a hair bulb. The hair cuticle does not have the same structure in the lower and upper parts of the hair root. In the area of the hair bulb, the cuticle consists of cells (cylindrical) with a cuticle. As they move to the upper side of the root, these cells become curved, flattened and horny. The horny epithelial cells thin and lie on top of each other.

The cortex of the hair consists of several rows of flat, horny cells elongated in the direction of the hair. Only in the area of the hair bulb are there tonofibrils in the cytoplasm of these cells. The cells of the cortex contain granules of the pigment melanin, which determines the color of the hair. The horny cortex cells contain nuclear residues, pigment and air bubbles, and hard keratin granules. Hard keratin is poorly soluble in water, acids and alkalis, and it contains a lot of the sulfur-containing amino acid cystine. In the cells of the cortex, the process of keratinization occurs without intermediate stages, that is, keratohyalin and elleidin accumulate in the cells. The better the keratin is developed, the thicker and more elastic the hair is.

The hair shaft is not present in fine hair, but in long and hard hair, several rows of large, polygonal cells contain acidophilic trichohyalin, small air bubbles, and a small amount of pigment spots in the cytoplasm.

In the lower 2/3 of the hair root, the nucleus of the shaft cell becomes denser and the cell becomes slightly keratinized. In the upper part of the root, the hair shaft cells are completely keratinized.

The hair root is oriented obliquely relative to the skin surface and forms a hair bulb. The hair follicle, which is embedded in the hair bulb from the bottom, consists of sparse fibrous unformed connective tissue. This tissue is rich in blood vessels and nerve endings. The hair is nourished by the follicle. The epithelium of the hair bulb

covering the follicle is cambial cells, due to which hair grows. The cells located above the hair follicle, and the cambial cells covering the lowest parts of the hair shaft and cortex, form the inner epithelial part of the hair cuticle. As the hair bulb cells move away from the hair follicle, that is, from the source of nutrition, they undergo a process of keratinization. As a result, the cells turn into elongated keratinized bodies. During the keratinization process, the hair cortex is the most intensive. Hair color is also preserved in the cells of the cortex. Hair graying occurs as a result of a decrease in pigment formation and, at the same time, an increase in air bubbles in the hair follicle cones.

The hair root or follicle is located in the hair follicle, which is surrounded from the outside by a fibrous dermal sheath - the hair follicle. The hair follicle, in turn, is divided into an inner and outer epithelial sheath. The inner epithelial sheath of the hair root is a product of the hair bulb and disappears above in the area of the excretory ducts of the sebaceous glands.

The outer epithelial sheath of the hair root is a continuation of the Malpighian layer of the epidermis and continues to the bulb.

As you approach the hair bulb, the inner and outer sheaths become thinner and consist only of the basal layer. The hair follicle consists of connective tissue, from which 2-3 layers of collagen fibers directed to the outer longitudinal direction can be distinguished. As mentioned above, the hair root is oriented obliquely relative to the skin surface. Hair has its own muscle - the hair lifting muscle. It is absent or poorly developed in the beard, in coarse and fine hair, in the armpit hair. This muscle consists of obliquely located smooth muscle cells, one end of which is connected to the sebaceous layer of the skin, and the other end is connected to the hair follicle. The contraction of this muscle causes the hair to move. The hair root becomes perpendicular to the skin surface. As a result, the hair stands up, the hair shaft rises slightly above the skin surface and takes on the appearance of skin. This condition, which often gives the appearance of goosebumps as a result of the external temperature cooling, is a protective function of the body, and muscle contraction also leads to narrowing of the blood vessels, as a result of which heat is retained in the body. As a result of this muscle activity, the sebaceous glands are also compressed and their secretion lubricates the hair.

Hair replacement. Hair grows in its place for several months to several years and, after going through 3 stages of development, eventually falls out, and therefore, hair is periodically replaced throughout a person’s life. The first stage is a period of active growth lasting 3–10 years, the second stage is a relatively calm period lasting 3–4 months, and the last stage is the hair shedding period lasting 1–6 months. Thus, a person can normally lose up to several hundred hairs every day. This process begins with atrophy of the hair follicle and impaired blood supply to the hair follicle

cells. As a result, the hair follicle cells lose their ability to reproduce and most of them die. The hair follicle turns into a follicle, and hair growth stops. The hair follicle separates from its follicle and rises along the sheath that forms the outer epithelial sheath, up to the point of attachment of the hair's lifting muscle. The lower, loose part of the epithelial sheath collapses and becomes a strip of cells. At the end of this strip, the follicle is restored again and covered with preserved cambial cells, resulting in the formation of a new hair bulb. A new hair begins to grow in this bulb. The new hair grows along the epithelial strip, and the strip becomes its outer epithelial sheath. As a result of further growth, the new hair pushes out from under the old hair. This process ends with the fall of the old hair and the appearance of a new hair on the surface of the skin. If the blood supply to the hair follicle stops, new hair does not grow in its place. The nail is a product of the epidermis and consists of hard, horny plates. The development of the nail begins in the 3rd month of pregnancy. First, the nail bed is formed. The epithelium covering the outer surface of the tips of the fingers and toes thickens, sinks into the underlying connective tissue, and the nail begins to form. The nail grows very slowly and is fully formed only at the end of the vibrio's life. The nail is divided into a body, root, two lateral and free parts. When the body of the nail is located in the nail bed, the lateral edges are embedded in the skin folds. The free edge of the nail protrudes from the nail bed. The root of the nail is the base that is embedded in the nail fold. Only part of the root is visible from the nail fold in the form of a pale and whitish crescent (especially on the nails of the big toes). The undifferentiated cells of the nail root that provide nail growth form the nail matrix. The matrix cells divide regularly and become keratinized. The keratinized epithelial cells move into the nail plate, and as a result, the nail grows. The nail grows by an average of 0.12 mm per day.

The nail bed consists of the epithelium and the dermis. The epithelium is formed in the growing layer of the epidermis. The flat nail plate located on the epithelium is made up of flat polygonal keratinized cells densely arranged in a tile-like pattern. If the nail plate thickens due to the nail bed epithelium, the nail grows in length due to the matrix.

The dermis of the nail bed is attached to the bones of the fingers. There are no suckers in the dermis. The nail bed area of the dermis is rich in blood vessels and nerve endings. Here, perpendicular fibers of the dermis are directly connected to the bone by joining the fibers of the periosteum. This structure plays an important role in practical medicine (an example is the inflammatory process that begins in the nail and causes bone injury).

CAUSES OF SKIN DISEASES

Skin tissues are constantly growing, differentiating, and regenerating organs. Changes in these processes lead to changes in various functions of the skin, and as

a result, various dermatoses. Such conditions occur when adaptation-compensation mechanisms fail to ensure that the skin can perform its functions normally. This excludes skin changes caused by unconditional influences. The effects of such influences always cause a certain reaction in the skin of all people. Examples of such influences are high-concentration solutions of acids and alkalis, large amounts of light energy, including X-rays, high and low temperatures.

The skin is continually subjected to pathological influences originating from both environmental (exogenous) and systemic (endogenous) sources. Endogenous factors arise from systemic diseases or alterations within individual organs and physiological systems. It is also worth noting that diseases often arise from the combined effects of external and internal factors, and in some cases, they are formed from the effects of several exogenous factors, one of which can serve as a precursor to the onset of the disease, and the other as a direct cause of the disease, for example, the development of pyoderma.

Exogenous etiological factors include mechanical, thermal, light, chemical substances and various infectious agents. Endogenous etiological factors are distinguished by their diversity. These include diseases of internal organs, especially liver, gastrointestinal diseases, metabolic disorders, endocrine, nervous system, hematopoiesis, vascular system, genetic factors, etc.

Metabolic disorders, in particular, disorders of carbohydrate, fat, mineral-water metabolism, lead to impaired metabolism of these substances in the skin, and these changes can cause various pathological conditions in the skin. For example, hyperglycemia causes furunculosis, itching, and disorders of fat metabolism cause the formation of xanthomas. The pathogenesis of many xanthomas is hypo- and avitaminosis, mainly with a deficiency of vitamins A, C, RR, P and B, many dermatoses are observed.

For example, phrynoderma occurs as a result of vitamin A deficiency, and pellagra and other diseases occur as a result of nicotinic acid deficiency.

The occurrence of dermatoses can be caused by psychogenic and changes in the central nervous system (CNS) and the autonomic nervous system. It is known from the beginning that eczema, urticaria, and skin itching are caused by primary changes in the CNS, which are caused by psychological factors. It is also known to science that various dermatoses occur as a result of changes in the autonomic nervous system.

The occurrence of dermatoses can also be caused by changes in the endocrine glands. For example, skin myxedema occurs in patients with thyroid disease, in Addison's disease - bronze skin color, and simple acne is associated with the activity of the gonads.

The etiology and pathogenesis of a number of diseases are associated with immune changes. First of all, such changes are associated with allergic and autoimmune processes.

The role of focal infection in the pathogenesis of some diseases is significant: most often these are chronic tonsillitis, sinusitis, otitis, carious teeth, etc. Focal infection can cause sensitization of the body, and on the other hand, reduce humoral immunity and cause immunodeficiency. The importance of focal infections in the pathogenesis of urticaria, erythema multiforme, psoriasis, lichen planus and other dermatoses has been proven.

Hereditary factors play a significant role in the development of a number of diseases. In particular, ichthyosis, xeroderma pigmentosum, epidermolysis bullosa, keratoderma, atopic dermatitis, psoriasis and other diseases are among them.

Some diseases can occur as a result of metastasis of tumors and infectious diseases or as a result of the transition of subcutaneous tissue diseases to the skin, for example, a type of skin tuberculosis - scrofuloderma.

It should also be noted that the etiology and pathogenesis of some dermatoses have not been fully studied.

Consequently, dermatological conditions frequently arise from a confluence of factors, rather than a singular cause. Multiple adverse influences, when creating a conducive environment for the development of a skin disorder, can render the skin susceptible to disease initiation by otherwise negligible triggers.

The etiological factors of the above-mentioned skin diseases can be divided into 3 groups. The first group includes the main causative factors, i.e., internal organs (changes), changes in the nervous system, leading to the occurrence of dermatoses. For example: calcinosis or xanthomatosis due to metabolic disorders, leukemic rashes with pathology of the blood-forming organs, skin lymphoma, etc., hereditary changes - causing congenital diseases, for example, ichthyosis, thyroid gland changes - leading to skin myxedema.

The second group includes risk factors, i.e., factors that change the internal organs, nervous system, metabolism, as well as hereditary factors, which serve as preparatory factors for the occurrence of the disease.

The third group consists of a set of determining factors, among which stress is in the first place.

Stress and other etiological factors can be a risk factor or a determining factor. This depends on the state of the body at the time of exposure to this or that factor. Dermatological patients often associate the onset or exacerbation of their diseases with stress, in particular, they consider psychogenic factors to be the main cause of the disease. Based on this, it can be said that the emotional factor is primary, and skin changes can occur as a result of it.

In the context of familial susceptibility to specific diseases, particularly dermatological conditions, certain heritable traits are transmitted from parents to offspring. These encompass characteristics related to metabolic pathways, homeostatic mechanisms, organ functionality, and systemic physiological processes, biochemical reactions occurring in cells and other signs, all of which make them show the same external and internal manifestations, even the response to the impact is similar. These similarities lead to the same predisposition to this or that disease. However, this predisposition may not always lead to the same disease, for this additional influences are needed that further increase the predisposition. From this it is clear that parents and children do not always suffer from the same disease.

The pathophysiological mechanisms underlying skin diseases are multifaceted and intricate. In addition to neurological influences and inherited predispositions, the development of numerous dermatological conditions is significantly influenced by the inherent resilience of the body and alterations within the immune system. If the allergic factor plays a major role in the pathogenesis of some diseases, then in erysipelas and purulent wounds - an autoimmune process, and in other dermatoses - secondary cellular immunodeficiency plays a role.

In some dermatoses of allergic genesis, the disease is observed as a result of allergens directly contacting the skin or entering the body in various ways, while in another group, allergies are caused by infectious agents (infectious, bacterial allergies). In this case, foci of secondary infection cause infectious diseases. In still other cases, the factors that cause allergization in the body are autoallergic processes, which occur due to pathologies of internal organs, metabolic disorders, etc. These changes can occur together and are accompanied by immunodeficiency states and autoimmune reactions.

Thus, a certain cause, a certain etiological factor, can cause different skin diseases, depending on the mechanism of their action on the skin.

DIAGNOSTIC BASIS OF SKIN DISEASES

The diagnosis of diseases of the skin, oral mucosa and red border of the lips is based primarily on the results of a careful examination and examination. The doctor's communication with the patient begins with listening to the patient's complaint and considering it in depth. Depending on the type of dermatosis, patients may complain of various complaints: itching, burning, pain, stinging, pulling, etc. In different people, these sensations may be developed to different degrees in the same disease.

Some diseases may occur without subjective sensations. After the patient's complaint is identified, the patient's history of the disease and the patient's vital signs are collected. After that, the patient is examined. After observing the skin and visible

mucous membranes, this information is supplemented with clinical and laboratory examination data.

In the process of determining the history of a skin disease, it is necessary to determine the duration of the disease, its causes, the causes to which the patient attributes the onset and exacerbation of the disease. Then the nature of the course of the disease is studied: the tendency to relapse, seasonal dependence, the presence of remissions, their duration are determined. If the patient has been treated before, its nature and effectiveness are determined. The role of water, soap, food, medicines, and harmful effects in the profession on the skin process is studied.

When collecting a patient's life history, in order to determine the role of external factors in the pathogenesis of the disease, it is necessary to pay attention to information about home conditions, working conditions, past illnesses, family or hereditary diseases, and harmful habits. When talking with the patient, it is necessary to try to determine the state of the patient's nervous system, his reaction to various stresses, and his attitude to his illness. It is advisable to examine the patient in a warm, naturally lit room. To view the oral mucosa, a wooden spatula is used, directing a slit electric light, which cleans the observed area well from saliva. It is necessary to examine the entire surface of the skin, otherwise the patient may not notice areas that are not subjectively bothersome, but are covered with rashes, such rashes: scars, pigmentations remaining in place of the displaced rashes are of diagnostic importance in some cases.

TOXICODERMIA.

Toxicoderma represents a systemic toxic-allergic response, triggered by the introduction of an allergenic substance via various routes, including the gastrointestinal tract, respiratory system, and other pathways. Exogenous triggers predominantly involve pharmaceuticals, dietary components, and chemical substances that gain entry into the body through ingestion or inhalation. Furthermore, drug-induced toxicoderma can occur following intravenous, intramuscular, subcutaneous, or intradermal administration, as well as topical application. Endogenous factors encompass autointoxication resulting from metabolic byproducts due to compromised hepatic, renal, or gastrointestinal function. Allergic toxicoderma is mediated by the presence of circulating antibodies. Predisposing factors include a history of allergic conditions, familial predisposition, and heightened immune sensitization. Common causative agents include sulfonamides, antibiotics, barbiturates, amidopyrine, B-complex vitamins, vitamin PP, folic acid, antihistamines (e.g., diphenhydramine), and even corticosteroids.

Clinically, patients exhibit erythematous, papular, vesicular, or bullous eruptions across the skin's surface. Systemic manifestations include fever, headache, malaise, and gastrointestinal disturbances. A distinct reaction, characterized by

erythematous macules on the hands, forearms, and mucosal surfaces of the genitalia and oral cavity, followed by the development of bullae, occurs after sulfonamide administration. This is known as sulfonamide or fixed erythema. Subsequent exposure to sulfonamide drugs is contraindicated in these individuals. Prolonged use of bromine-containing medications may lead to the development of bromine acne in susceptible individuals. When taking iodide preparations, iodide acne or tuberous iododerma appears on the skin in the form of dark red hemispherical tumors, which, when pressed, cause purulent discharge. The course of toxicoderma due to exogenous causes is acute. After the allergen is removed from the body, each rash resolves. Toxicoderma of endogenous origin occurs in a chronic form.

Therapeutic intervention necessitates the prompt elimination of the offending allergen, coupled with detoxification protocols, administration of laxatives and diuretics, adsorbents, and agents for hyposensitization and antihistamine therapy. Topical applications include shake lotions, pastes, and corticosteroid ointments.

EPIDERMAL NECROLYSIS (LYELL'S SYNDROME)

Etiological factors for this condition encompass a variety of pharmaceuticals (e.g., sulfonamides, antihistamines, analgesics, sera), chemical agents, and spoiled food products (e.g., processed meats, seafood). The primary pathogenic mechanism involves a severe allergic reaction, which, while intense, is distinct from anaphylactic shock. Underlying chronic infections, such as tonsillitis and cholecystitis, can contribute. A combination of allergic, toxic, and infectious factors may be implicated.

Clinical Presentation: The disease onset is marked by a sudden elevation in body temperature (38°C-41°C), accompanied by the appearance of bullous or erythematous-bullous lesions on the skin. A positive Nikolsky sign is observed, indicating epidermal detachment. Extensive epidermal sloughing leads to painful erosions and bleeding. The skin appears scalded. Severe systemic manifestations include high-grade fever, headache, somnolence, prostration, signs of dehydration, circulatory compromise, renal dysfunction, cardiac irregularities, and other organ system disturbances. The prognosis is often unfavorable.

Treatment: Management is conducted in an intensive care setting. High-dose glucocorticoid therapy (e.g., prednisolone 80-150 mg/day or more) is administered, alongside hemosorption, hemodilution, unithiol, sodium thiosulfate, antihistamines, and supportive measures to maintain fluid, electrolyte, and protein balance. Meticulous patient care is crucial to prevent secondary infections. Topical therapy, analogous to burn management, includes aerosolized steroids and antibiotics (e.g., panthenol, oxycort) applied multiple times daily.

Prevention: Avoidance of medications with a known history of patient intolerance is essential. Immediate hospitalization is indicated for patients with toxicoderma accompanied by systemic deterioration or fever.

ECZEMA

Eczema is a chronic relapsing inflammation of the surface layers of the skin. Functional, neurotrophic or organic changes in the central and peripheral nervous system, endocrine diseases, liver and stomach diseases, metabolic disorders, hypo- and avitaminosis play an important role in the development of the disease. With eczema, polyvalent sensitization develops as a result of contact with various irritants, which leads to allergic restructuring of the body. The microbial factor (pyococci , pathogenic fungi) plays a certain role, which contribute to the sensitivity of the skin to both pathogens and the products of their vital activity.

Eczema Classification: Eczema is categorized into true, microbial, occupational, seborrheic, and infantile forms, each presenting with acute, subacute, or chronic clinical manifestations. True eczema is characterized by the development of microvesicular and micropapular lesions on a background of hyperemic and edematous skin. Subsequently, these vesicles rupture, forming crusts, beneath which epithelialization occurs. Key features of true eczema include polyvalent sensitization, symmetrical lesion distribution, indistinct lesion borders, gradual transition to unaffected skin, true and evolutionary polymorphism, and frequent localization on the upper extremities and face. Lesions may also appear at sites distant from the primary eruption, termed eczematids. Chronic eczema exhibits a less pronounced inflammatory response and lichenification within the lesions. Microbial eczema arises in individuals with heightened sensitivity to microbial factors or their metabolic products. Distinct subtypes include nummular eczema, characterized by round, sharply demarcated lesions; varicose eczema, associated with varicose symptoms; and paratraumatic eczema, occurring at sites of trauma, wounds, or surgical incisions. Microbial eczema commonly localizes to the lower and upper extremities, particularly in areas prone to friction.

Clinical Presentation: Microbial eczema lesions are round, sharply defined, hyperemic, bluish, edematous, and infiltrated. Microvesicles, lamellar desquamation, yellowish crusts, peripheral extension, and asymmetrical distribution are observed. Eczematous foci may develop around isolated pustules on otherwise healthy skin. Seborrheic eczema primarily affects the scalp, face, chest, and interscapular region. Clinical features include erythema and desquamation with yellow scales, distinct lesion borders, potential weeping, and frequent involvement of the retroauricular region. Subjective pruritus is common, especially in children with gastrointestinal disorders. Infantile eczema typically localizes to the face, scalp,

and retroauricular areas. Erythematous macules, vesicles, erosions, weeping, and yellow crusts are observed. Kaposi's varicelliform eruption (eczema herpeticum) results from contact with herpes simplex virus in individuals with pre-existing eczema, neurodermatitis, or other skin conditions. The incubation period averages one week. The disease presents with high fever (up to 40°C), severe systemic toxicity, grouped vesicles or pustules with central umbilication on inflamed skin, and potential involvement of the genitalia and oral mucosa. Regional lymphadenitis, stomatitis, keratoconjunctivitis, meningeal signs, encephalitis, pneumonia, gastrointestinal disorders, and secondary infections may occur. In debilitated children, involvement of internal organs and the nervous system can lead to fatal shock.

Treatment: Kaposi's varicelliform eruption is treated with antibiotics, antiviral agents, anti-varicella-zoster immunoglobulin, B vitamins, vitamin C, and antihistamines. Severe cases may require glucocorticoids. During the initial three days, topical therapy is avoided to prevent shock. Subsequently, aniline dyes and antibiotic ointments are used. Prevention involves avoiding contact with individuals with herpes simplex virus. Occupational eczema results from exposure to workplace hazards, typically affecting exposed skin areas. Pruritus and clinical manifestations of true eczema are observed. Diagnosis relies on detailed occupational history, including sanitary and technical workplace conditions, duration of exposure, and symptom improvement during time away from work. Skin testing (e.g., patch, scratch, prick, intradermal) is performed to identify specific allergens.

The treatment consists in the appointment of hyposensitizing agents (calcium preparations, in severe cases, glucocorticosteroids), antihistamines, vitamins of groups B, C, PP. Physiotherapy methods include inductothermy and acupuncture. Local treatment depends on the stage of each process - lotions, pastes, ointments, creams. It is necessary to exclude spices, salty, smoked, fatty, concentrated broths, coffee, cocoa and other spicy dishes from the diet. Sanatorium-resort treatment using hydrogen sulfide and radon baths is recommended.

PROFESSIONAL SKIN DISEASES

"Occupational dermatoses encompass skin disorders arising from exposure to specific workplace hazards. These hazards can be categorized as chemical, physical (mechanical), or infectious (parasitic).

In the pathogenesis of occupational skin diseases, allergic processes, mediated by antigen-antibody interactions, play a pivotal role. Antigens (allergens) are classified as either proteinaceous or chemical (haptens). Contact allergens are the primary drivers of occupational skin diseases. Upon entering the body, haptens bind to skin, blood, and tissue proteins, forming complete antigens that induce antibody

production. Exposure to an allergen can lead to increased sensitivity to a single irritant (monovalent sensitization) or multiple irritants (polyvalent sensitization). Sensitization can occur after varying periods, ranging from one to two weeks to several months or years. Allergic reactions are classified as immediate (occurring within 10-20 minutes, up to 4-6 hours) or delayed (manifesting after 6-8 hours or later).

Exogenous and endogenous factors significantly influence the development of occupational dermatoses. Exogenous factors include dust, gases, and pollutants in the work environment, thermal extremes, and skin trauma. Endogenous factors encompass nervous and endocrine system disorders, gastrointestinal diseases, individual skin characteristics (e.g., sweat and sebum secretion, pH), pre-existing allergic conditions, and age-related variations. Focal infections and microbial susceptibility also contribute. The development of occupational skin diseases is thus a complex interplay of exogenous, endogenous, social, and immunological factors. Given the clinical similarity between occupational and non-occupational dermatoses, accurate diagnosis requires specialized knowledge.

Occupational diseases are characterized by rapid symptom improvement upon cessation of work (weekends, holidays) and prompt recurrence upon resuming work. Diagnosis relies on detailed occupational history, including duration of exposure, allergic history, and identification of potential workplace irritants. The presence of similar skin conditions in coworkers exposed to the same hazards is also relevant.

The causal relationship between the disease and the patient's occupation is established through sanitary-hygienic workplace assessments, clinical data, skin testing, and other investigations. Occupational hygiene specialists, dermatologists, safety engineers, workshop managers, and technologists collaborate in workplace evaluations. Identifying specific contact allergens and exposure durations is crucial.

Skin testing, particularly patch and prick tests, is essential for diagnosing occupational allergic dermatoses. Patch tests involve applying allergen solutions to the skin, typically on the upper abdomen or back, with readings taken after 24 hours. Prick tests involve applying allergen solutions to gauze patches affixed to the skin, usually on the flexor surface of the wrist or inner arm, with readings taken at 24, 48, and 72 hours. Positive reactions manifest as erythema, edema, and papulovesicular eruptions. Transient exacerbations of existing lesions or systemic reactions may occur. Standardized concentrations of test substances are used.

The diagnosis of occupational dermatoses requires an integrated assessment that includes a detailed medical and occupational history, thorough clinical examination, analysis of workplace conditions, relevant laboratory investigations, and review of industrial documentation concerning harmful factors and chemical exposures. Diagnostic conclusions should be based on recognized classifications of

occupational skin diseases and specify the severity and distribution of lesions, the causative occupational agent, associated complications, and concurrent diseases. In addition, well-defined recommendations regarding the patient's work activities should be formally conveyed to the attending physician, occupational health authorities, and the employer to ensure appropriate management and prevention.

PSORIASIS

Psoriasis (lichen planus) is a chronic, recurrent dermatosis marked by papular lesions affecting the skin and mucous membranes, with possible involvement of the nails and joints. The disease affects approximately 2% of the world's population.

Despite extensive research, the exact etiology and pathogenesis of psoriasis have not been fully elucidated. Multiple hypotheses have been proposed, including viral, infectious-allergic, neurogenic, endocrine, and metabolic mechanisms; however, none sufficiently explains all aspects of the disease. Contemporary concepts highlight the dominant role of genetic predisposition, with heredity being a key determinant. In certain cases, disease-associated genes may manifest in descendants before clinical signs appear in parents, a process known as genetic anticipation. Various external factors—such as infections, psychological stress, and physical trauma—may trigger disease onset in genetically susceptible individuals by promoting accelerated proliferation and abnormal differentiation of epidermal cells. Overall, psoriasis is considered a multifactorial disorder arising from complex interactions between endogenous and environmental influences in individuals with an inherited susceptibility.

Clinical presentations of psoriasis include vulgar, exudative, arthropathic, erythrodermic (generalized and localized), and pustular forms. Vulgar psoriasis is characterized by the appearance of pink, rounded epidermal papules covered with silvery-white, easily detachable scales. Scraping these scales reveals a stearin-like appearance (stearin spot phenomenon), followed by a moist, shiny red surface (terminal film phenomenon). Further scraping elicits punctate bleeding (Auspitz's sign or "blood dew"). These phenomena reflect underlying parakeratosis, acanthosis, and papillomatosis.

The progression of psoriasis is classically divided into three stages: progressive, stationary, and regressive. The progressive stage features small, bright pink papules with central scaling and a positive Koebner phenomenon (development of new lesions at sites of skin trauma). The stationary stage exhibits a negative Koebner phenomenon, paler lesions, scaling covering the entire papule, and a Voronov's ring (pale halo) surrounding the papule. The regressive stage involves lesion resolution, either centrally or peripherally, leaving behind hypo- or hyperpigmented macules. Staging is crucial for guiding therapeutic decisions. Disease course can be seasonal, with summer, winter, and non-seasonal patterns.

Nail involvement manifests as nail plate deformities, pitting (Heller's symptom), yellow-brown discoloration, thinning or thickening with claw-like deformities (psoriatic onychogryphosis), and transverse grooves (Beau's lines).

Arthropathic psoriasis begins as vulgar psoriasis, but progresses to involve joint pain and swelling, particularly in the small joints of the hands and feet, ankles, and wrists. Severe deforming arthritis and ankylosis can develop, leading to disability. Psoriatic erythroderma can arise spontaneously or following infections, vaccinations, or sun exposure. Lesions coalesce into generalized erythema, with brick-red skin and edema. Secondary infections, fever, increased pruritus, and skin hardening can occur. Erythroderma can be complete or partial. Exudative psoriasis is characterized by abundant exudative scales or crusts due to pronounced exudation. Pustular psoriasis, less common, presents with sterile pustules on non-inflamed skin (von Zumbusch type) or on the hyperemic palms and soles (Barber type). Infantile psoriasis often manifests as erythematous patches with maceration, peripheral desquamation, and predominant involvement of the anogenital folds (inverse psoriasis), mimicking diaper dermatitis, candidiasis, or eczema. Vesicular and pustular elements, such as osteofolliculitis or streptococcal impetigo, may accompany papular lesions. Exudative forms of psoriasis are relatively common in children, accounting for approximately 40% of cases.

Management and Differential Diagnosis of Psoriasis

The treatment of psoriasis represents a significant clinical challenge and should be individualized according to the clinical form and stage of the disease. Therapeutic decisions must consider the morphology and extent of skin involvement, seasonal variability, patient age, previous drug responsiveness, and the functional status of internal organs. Systemic therapy should be comprehensive and may include sedative agents, neuroleptics, desensitizing medications, nonspecific immunomodulatory agents (such as aloe extracts, adenosine triphosphate, pyrogenal), and vitamin supplementation.

In cases characterized by pronounced infiltrative psoriatic plaques, enzymatic therapy with hyaluronidase (Lidase) is recommended. The drug is administered subcutaneously or intramuscularly at a dose of 64 units daily, with a treatment course consisting of up to 15 injections. Intravenous infusions of hemosorption solutions (200–400 ml) every three days, as well as intramuscular administration of pyrogenal, are also commonly employed. Immunomodulatory therapy is selected based on the patient’s immune status and may include agents such as T-activin, leukidin, thymalin, methyluracil, splenin, placental extracts, and related preparations.

Correction of metabolic disturbances forms an integral part of treatment. To normalize lipid metabolism, lipoic acid and dipromonium are prescribed. In patients with hepatic dysfunction, hepatoprotective and detoxifying agents—including

Sirepar, methionine, unithiol, Essentiale, LIV-52, and Karsil—are indicated. Aromatic retinoids (e.g., tigason, etretinate) are used in selected cases, along with antioxidants aimed at reducing lipid peroxidation. Cytostatic agents and systemic corticosteroids are reserved for exceptional circumstances when conventional therapies prove ineffective.

During the progressive stage of psoriasis, treatment should be gentle and non-irritating. To arrest disease progression, autologous blood therapy combined with calcium chloride has been employed. This procedure involves intravenous administration of 10 ml of a 10% calcium chloride solution, followed by intramuscular injection of the patient's own blood drawn through the same venous access. Clinical observations indicate that a series of five daily injections with gradually increasing volumes (2, 4, 5, 6, and 8 ml) may be sufficient to suppress the appearance of new lesions.

Topical therapy must be applied cautiously, as inappropriate treatment may precipitate psoriatic erythroderma. Keratoplastic agents, such as 2–3% salicylic acid ointments or creams, are commonly prescribed. In the stationary stage, topical oil-based therapies are widely used in combination with hydrotherapy and physiotherapeutic modalities, including ultraviolet A (UVA) and PUVA therapy. Spa and resort-based rehabilitation is recommended when indicated.

Patients diagnosed with psoriasis should be enrolled in long-term dispensary follow-up. During remission, supportive and preventive therapy should be administered, taking into account the seasonal pattern of exacerbations.

Differential Diagnosis

In most cases, the clinical presentation of psoriasis vulgaris is sufficiently characteristic to establish a diagnosis based on differential features distinguishing it from other dermatoses, including papular syphilis, lichen planus, seborrhea, seborrheic eczema, neurodermatitis, pityriasis rosea, parapsoriasis, discoid lupus erythematosus, and Reiter's disease. Histopathological findings play a crucial role in challenging cases and typically include parakeratosis, acanthosis with elongation of rete ridges, thinning of the suprapapillary epidermis, absence of the granular layer, Munro microabscesses, vascular dilation, and perivascular lymphocytic infiltration.

Psoriasis differs from papular syphilis by its brighter coloration, more superficial localization of papules, profuse scaling, peripheral growth with plaque formation, and the presence of characteristic psoriatic phenomena. Syphilis is additionally distinguished by lymphadenopathy, systemic manifestations, and positive serological tests. Differentiation from lichen planus is usually straightforward due to the polygonal, violaceous papules with central depression, minimal scaling, frequent mucosal involvement, predilection for flexural surfaces, and absence of psoriatic pathognomonic signs.

The clinical features of psoriasis vary depending on lesion localization (intertriginous areas, scalp, nails), disease severity (exudative, rupioid, pustular forms), extent (psoriatic erythroderma), joint involvement (psoriatic arthritis), and age of onset, including pediatric cases. Scalp psoriasis may persist in isolation for prolonged periods and can mimic seborrhea, particularly in early stages when inflammatory changes and scaling predominate without infiltration. Unlike seborrhea, psoriasis does not cause hair thinning or alopecia and often demonstrates plaque formation extending beyond the hairline (the "psoriatic crown").

Key features distinguishing psoriasis from seborrheic eczema include drier scales, clearer lesion borders, minimal exudation, absence of vesiculation, and lack of intense pruritus. Family history and systemic signs should also be considered. Limited neurodermatitis may resemble isolated psoriatic plaques in the occipital region, particularly in climacteric women; however, silvery scaling, psoriatic phenomena, and absence of lichenification favor psoriasis.

Rupioid psoriasis must be differentiated from pyococcal and syphilitic lesions. In psoriasis, crusts form on an infiltrated base, removal does not reveal purulent discharge, and pinpoint bleeding may be observed. Syphilitic rupia is typically asymmetric, ulcerative, painful, and accompanied by positive serological findings.

Intertriginous psoriasis, more common in children and the elderly, presents as smooth, shiny, sharply demarcated plaques with minimal scaling and occasional moisture. Lesions in interdigital spaces appear as macerated, well-defined whitish areas without marked erythema. These forms require differentiation from candidiasis, dermatophytosis, and erythrasma. In contrast to psoriasis, candidiasis is characterized by brighter erythema, indistinct borders, peripheral maceration, satellite lesions, and acute inflammation.

Pustular psoriasis of the palms and soles should be distinguished from dyshidrotic eczema, which presents with vesicles rather than pustules and typically evolves into weeping eczematous lesions. Psoriatic erythroderma requires differentiation from erythrodermic presentations of cutaneous T-cell lymphoma, eczema, neurodermatitis, and congenital ichthyosiform erythroderma. In prolonged or atypical cases, histological examination is essential. Unlike psoriasis, mycosis fungoides predominantly affects older individuals, is associated with severe pruritus, lymphadenopathy, hair loss, palmar hyperkeratosis, and progressive skin atrophy.

Congenital non-bullous ichthyosiform erythroderma (lamellar ichthyosis) typically manifests at birth and persists throughout life, clearly distinguishing it from acquired psoriatic erythroderma.

LICHEN PLANUS

The precise etiology of lichen planus remains elusive, with proposed theories including viral, neurological, and allergic origins. The classic clinical presentation

involves the appearance of polygonal, pink-red papules on the trunk, limbs, oral mucosa, and genitalia, characterized by a central umbilication. Upon illumination, a Wickham's striae pattern becomes visible, resulting from uneven thickening of the stratum granulosum. An isomorphic Koebner phenomenon is typically observed. Pruritus is a common complaint. In addition to the typical form, hypertrophic, atrophic, bullous, and pigmented variants are recognized. The hypertrophic or verrucous form manifests as elevated papules with papillomatous-keratotic proliferations, predominantly located on the shins, arms, scrotum, and sacral region. The atrophic or sclerotic form is characterized by atrophic scarring at the sites of previous papules, commonly found on the head, trunk, axillae, and genitalia. It presents with papules and atrophic macules of a yellowish-brown hue, characteristic of lichen planus. Annular lesions with a slightly brownish-blue raised border and a brown center may also occur. The bullous or pemphigoid form, a rare variant, is characterized by the development of bullae on erythematous plaques and papules, accompanied by intense pruritus, and most frequently located on the lower extremities.

Pigmented lichen planus presents as multiple brownish macules on the face, trunk, and limbs. Diagnosis requires careful examination to identify typical papular elements. Linear lichen planus, where papules are arranged in linear streaks, is more common in children and often follows nerve pathways. Zosteriform lichen planus exhibits typical papules along nerve dermatomes, resembling herpes zoster. Lichen planus can be localized, disseminated, or generalized. The disease course can be acute (up to 1 month), subacute (up to 6 months), or chronic with prolonged remissions or relapses. Infantile lichen planus is characterized by a tendency for papules to coalesce, with edema and erythema. Due to the increased vascularity, hydrophilicity, and abundance of interstitial fluid in children's skin, exudative features are prominent, leading to vesicular and bullous forms. Diffuse erythema of a dark red color with edema and scaling (erythematous lichen planus) may also occur. Identification of characteristic papules at the periphery of erythematous areas aids in diagnosis. In severe cases, erythroderma with scalp and nail involvement can develop.

Clinical Features and Variants of Lichen Planus

In clinical practice, alongside the classical presentation of lichen planus, a number of rare and atypical forms are observed, including verrucous, follicular, atrophic, pemphigoid, coral-shaped, ulcerative, vegetative, pigmented, and flattened variants. The classic form of cutaneous lichen planus is characterized by symmetrical eruptions of small, shiny, polygonal papules with a reddish-violet coloration and a distinct central umbilication. Lesions are predominantly localized

on the flexor surfaces of the limbs, lateral aspects of the trunk, genital region, as well as the palms and soles.

Papular elements often arrange themselves in annular, garland-like, arcuate, linear, or zosteriform patterns. Scaling is usually minimal and inconspicuous, although psoriasiform desquamation may occasionally occur. A fine reticular pattern on the surface of papules, known as Wickham's striae, becomes particularly evident after application of oil and reflects underlying hypergranulosis. Nail involvement may be present, manifesting as longitudinal ridging and fissuring of the nail plates. During the active progressive phase of the disease, the Koebner phenomenon is frequently positive.

The disease course is typically chronic and accompanied by pruritus of varying intensity. Acute onset is uncommon but may occur, occasionally leading to confluent polymorphic eruptions progressing to erythroderma. Oral lichen planus demonstrates considerable clinical variability and may present as white linear thickenings resembling leukoplakia, opalescent porcelain-like plaques, or delicate reticular white lines and dots consistent with Wickham's network. The buccal mucosa, lateral and dorsal surfaces of the tongue, and the floor of the oral cavity are most frequently involved.

In addition to asymptomatic lesions, exudative-hyperemic, erosive-ulcerative, and bullous variants may develop on mucous membranes. Subjective symptoms are generally absent in typical cases, while pain and burning sensations are primarily associated with erosive lesions.

The verrucous (hypertrophic) form is relatively rare and is characterized by marked hyperkeratosis and the formation of sharply demarcated, warty plaques, most commonly on the anterolateral surfaces of the lower extremities. These lesions are significantly elevated above the skin surface and are often associated with intense pruritus. Typical lichen planus papules may appear at the periphery of such plaques or on the oral mucosa. The vegetative form is distinguished by papillomatous growths on the lesion surface.

The follicular variant presents with keratotic follicular papules, which may subsequently lead to scarring alopecia when the scalp is affected. The atrophic form is marked by regression of lesions with the development of cutaneous atrophy, often surrounded by a persistent infiltrated rim of brownish-cyanotic discoloration.

The pemphigoid variant represents the rarest form and is characterized by the development of vesiculobullous lesions containing clear fluid, usually accompanied by pruritus. Blisters may arise within papular or plaque lesions, as well as on erythematous or clinically unaffected skin. The coral-shaped form is defined by large, flattened papules arranged in a bead-like or reticular pattern, predominantly

affecting the neck, shoulder girdle, chest, and abdomen, often accompanied by surrounding hyperpigmentation.

The ulcerative variant primarily affects the lower extremities and is characterized by painful, small ulcers with infiltrated red-cyanotic margins, while typical lichen planus lesions may be present elsewhere on the skin. The pigmented form may manifest either as brown-ochre nodules typical of lichen planus or as diffuse areas of pigmentation resembling poikiloderma, in which papular elements are difficult to identify.

Pathomorphological Characteristics

The histopathological features of lichen planus are diverse and closely correlate with its clinical variability, while maintaining distinct diagnostic criteria. Hallmark histological findings include hyperkeratosis, irregular thickening of the granular layer, pronounced hypergranulosis, acanthosis with pointed elongation of rete ridges, vacuolar degeneration of the basal cell layer, and a dense, band-like lymphocytic infiltrate in the upper dermis closely apposed to the epidermis.

In hypertrophic forms, additional findings include marked acanthosis, severe hyperkeratosis, papillomatosis, and disruption of the dermoepidermal junction due to lymphocytic exocytosis. Atrophic variants demonstrate thinning of the epidermis with flattened rete ridges, reduced hyperkeratosis and granulosis, and perifollicular lymphocytic infiltrates often admixed with active melanocytes, particularly in pigmented forms. Focal damage to the basement membrane is commonly observed as a result of lymphocyte migration.

The follicular form is characterized by dense lymphoid infiltration at the lower portion of hair follicles, with destruction of the external root sheath and loss of its clear boundary with the dermis. In the pemphigoid variant, subepidermal blister formation occurs in addition to classic lichenoid histological features. The coral-shaped form is distinguished by prominent neovascularization of venous-type vessels and frequent dermal extravasations. In mucosal lesions, hyperkeratosis and hypergranulosis are typically absent, while parakeratosis is often present.

Differential Diagnosis

Lichen planus should be differentiated from psoriasis, papular syphilis, toxidermia, lichenoid and verrucous cutaneous tuberculosis, prurigo nodularis, diffuse and localized neurodermatitis, pemphigus, and flat juvenile warts. Differentiation from psoriasis is usually straightforward due to differences in lesion distribution, morphology, and scaling. Psoriasis predominantly affects the scalp, extensor surfaces, and trunk, whereas lichen planus favors flexural areas, the abdomen, and mucous membranes. Psoriatic papules exhibit prominent scaling and positive psoriatic phenomena, which are absent in lichen planus.

Differentiation from papular syphilides is particularly important in annular lichen planus involving the genital region or oral cavity. Syphilitic papules are typically brownish-red, oval or round, lack central depression, demonstrate peripheral clearing with a Biett's collar, and are seldom pruritic. Associated systemic features and positive serological tests are essential diagnostic clues.

Drug-induced toxidermia, especially lichenoid reactions caused by medications such as antibiotics, sulfonamides, antimalarials, psychotropic agents, and bismuth compounds, may mimic lichen planus. Careful drug history, clinical course, laboratory investigations, and histological examination are often required for accurate diagnosis. Compared with lichen planus, toxidermia is usually more acute, accompanied by systemic symptoms, and follows a shorter disease course.

Treatment Principles

Management of lichen planus should be individualized and comprehensive. During the acute phase, especially in the presence of severe pruritus, H1 antihistamines, vitamin A, aromatic retinoids (e.g., tigason), vitamin E, and combined vitamin preparations are recommended. In chronic cases, Actovegin may be beneficial. Antimalarial agents such as chloroquine or delagyl, often combined with systemic corticosteroids, may be effective in selected patients.

In chronic, generalized, or mucosal forms, systemic corticosteroid therapy is often indicated in combination with conventional treatment. Immunomodulatory therapy may include exogenous interferons or interferon inducers. Adjunctive methods such as hypnotherapy and electrosleep have demonstrated favorable effects. Local therapy consists of topical glucocorticoids, chloroethyl cryotherapy, intralesional hydrocortisone injections, diathermocoagulation, laser therapy, and PUVA treatment.Photodermatoses: The history is of great importance in the diagnosis of the disease, since the rash may have disappeared by the time the doctor examines the patient. When there is a chronological relationship between the rash and the patient's exposure to sunlight, the diagnosis is not so difficult, but such a relationship may not always be observed. This is because some patients think that they are "allergic" to photoprotective ointments, but in fact they are affected by sunlight, from which photoprotective agents cannot fully protect. Other patients may complain of a constant rash on the face and exposed areas of the skin due to taking one of the drugs with a photosensitizing effect (thiazide diuretics, doxycycline, minocycline, sulfonamides, antidepressants, etc.), without even suspecting the role of sunlight in the development of the disease. In cases of universal erythroderma, such as psoriasis or persistent solar erythroderma, the skin is more sensitive to sunlight.

The age of the patient is of great importance, since some photodermatoses are observed only at a certain age (for example, solar pruritus is always observed in

childhood), while other types can occur at different ages (for example, polymorphic photodermatosis). It is important to determine how long after exposure to sunlight the rash appeared (solar urticaria - after a few minutes, polymorphic photodermatosis - after a few days), the duration of the rash (solar urticaria - hours, polymorphic photodermatosis - weeks), and what sensations it was accompanied by.

Itching is characteristic of polymorphic photodermatosis, solar urticaria, and pruritus; pain and burning are characteristic of erythropoietic protoporphyria. If the rash appears not only after exposure to sunlight, but also from being near heat sources (fire, tandoor, stove, torch, oven), then the causative agent of the disease is not ultraviolet light, but infrared light. If the rash is observed only in spring and summer, then there is an increased sensitivity to UV-B (wavelength 290–320 nm), and if it is observed throughout the year - to UVA (wavelength 320–400 nm) or the visible range (wavelength 400–800 nm). There is another way to determine the radiation of the pathogenic range, for this it is necessary to determine whether the patient is protected from sunlight by window panes: UV-A has the property of passing through glass, while UVB does not.

It is also important to know what medications the patient is taking and what photoprotective agents he uses. Information about these is also important - both during the period of the rash and before the rash. It should also be remembered that the photosensitizing effect can persist for several months after the drug is discontinued. When collecting information about the patient's profession, hobbies, recent trips, it is necessary to pay attention to whether he was exposed to sunlight or in contact with plants. Often, a preliminary diagnosis can be made during the patient's examination: for example, photophytodermatitis in amateur gardeners, red rash in electric welders, or it can also be found in secretaries-typists, computer operators, because they work in rooms lit by fluorescent lamps during the day. At the end of the anamnesis, the patient's general condition is asked, with special attention paid to the symptoms of collagenosis and porphyria.

Location of rashes. The location of rashes is the main diagnostic sign of all photodermatoses. Rashes appear on open areas of the body: primarily on the forehead, cheeks, nose, supraauricular areas, back and side areas of the neck, front and upper chest, and on the palms of the hands and feet. Areas where rashes are not observed are also of diagnostic importance, since sunlight does not fall on these areas: eyelids, nasolabial folds, between the fingers, the skin behind the supraauricular areas, and the skin under the chin and mouth. Photodermatoses are characterized by a clear border between the lesion and healthy skin, areas that correspond to the hourglass or the edge of clothing. However, there are exceptions to this rule. Not all exposed areas of the skin are always affected (this depends on local immunity and the protective properties of the darkened area). In addition,

rashes can also be observed on “closed” areas of the skin (due to insufficient protection by clothing). In addition, photosensitization can be so strong that the rash spreads to areas not exposed to sunlight, even leading to erythroderma. However, even in such cases, the rash initially appears on areas of the skin that have been exposed to sunlight.

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Treatment and prevention. General principles of treatment and prevention of photodermatosis:

1. Avoidance of all drugs and other types of agents that have the property of showing photosensitizing effects.

2. Avoid exposure to sunlight between 10:00 and 16:00.

3. Use of photoprotective agents with a high level of protection.

4. Wear hats and clothing that protect from sunlight.

5. People who are extremely sensitive to UVA rays should be warned that many photoprotective agents are weak and that they are allowed to be exposed to the sun only for certain periods of time, wearing protective clothing on sunny days.

6. Local treatment - according to skin changes. In mild cases, corticosteroid ointments. In severe cases, courses of prophylactic phototherapy (UV-V) or PUVA therapy (the dose of irradiation should be low), oral corticosteroids and immunosuppressants are prescribed.

ITCHY DER

NEURODERMATITIS (ATOPIC DERMATITIS)

Hereditary factors play a decisive role in the pathogenesis of atopic dermatitis. In early childhood, the condition frequently develops on the background of exudative diathesis and increased allergic sensitivity. Environmental influences and psychoemotional stressors often contribute to disease exacerbation. The course is typically characterized by seasonal relapses, most commonly occurring in the autumn and spring periods. Persistent and intense pruritus represents the leading clinical symptom and is present throughout the day.

The primary morphological element is a papule that initially resembles normal skin in color but gradually assumes a brownish-pink shade. As individual papules merge, areas of infiltration and lichenification are formed. The skin of patients with neurodermatitis is usually dry, exhibits a grayish discoloration, demonstrates an exaggerated pilomotor response, and shows white dermographism, reflecting predominance of sympathetic nervous system activity. Patients frequently present with increased irritability, sleep disturbances, and heightened reactivity to external stimuli.

Limited neurodermatitis most commonly affects the posterior and lateral surfaces of the neck, the occipital region, flexural areas of the elbows and knees, the medial thighs, and the genital region. Lesions typically display zonal structure: the central area shows marked infiltration and lichenification, the intermediate zone consists of shiny papules, and the peripheral zone is characterized by pigmentation. Diffuse neurodermatitis involves extensive areas of the skin, including the extremities, face, and trunk, and is manifested by widespread polygonal papules, infiltration, lichenification, and multiple excoriations.

Atopic dermatitis usually originates in childhood in the form of exudative diathesis or infantile eczema and may later progress into neurodermatitis. The disease is based on a genetically determined abnormal immune response to allergens, reflecting an inherent predisposition to allergic reactions. Associated conditions such as bronchial asthma, helminthic infestations, and eosinophilia are frequently observed.

Management of neurodermatitis must be comprehensive and individualized. Dietary regulation plays a central role, as numerous food products and additives can provoke or aggravate symptoms. Patients are advised to eliminate chocolate, coffee, cocoa, mushrooms, honey, berries, orange- and red-colored vegetables, marmalade, jam, caramel, cow's milk, and eggs from the diet. Reduction of carbohydrate and sodium intake is also recommended. Potatoes and cereals should be soaked for 12–18 hours prior to cooking. Meat products, including beef, lean pork, rabbit, turkey, and chicken, should be prepared using a double-cooking method, involving preliminary boiling for approximately 30 minutes followed by cooking in fresh water.

A diet enriched with vegetable oils, pureed pumpkin, white cabbage, cauliflower, turnips, green apples, pears, and bananas is considered beneficial. Non-specific hyposensitization therapy includes the use of antihistamines and immunomodulatory agents. Medications influencing the sympathetic nervous system, ganglion blockers, and nicotinic acid derivatives may also be employed. Vitamin B complex supplementation is indicated, along with physiotherapeutic procedures such as ultraviolet irradiation, diathermy, electrosleep, reflexotherapy, and inductotherapy.

Topical therapy consists of antipruritic ointments and creams. Sanatorium and resort treatment in dry, warm climates is recommended. Patients should avoid wearing synthetic and woolen clothing. Regular dispensary follow-up, conducted two to four times per year, with assessments by a general physician, neurologist, otolaryngologist, and dentist, is essential for long-term disease control.

RTICARIA

Urticaria is a dermatosis based on toxic–allergic reactions and may arise under the influence of both endogenous and exogenous factors. Internal triggers include disorders of the gastrointestinal tract, parasitic infestations (such as helminthiasis and giardiasis), and dysfunctions of the nervous system. External provoking agents comprise food allergens, medications, contact with synthetic fabrics, certain plants, cosmetic products, floral odors, insect stings, as well as physical factors including exposure to cold, heat, and sunlight. In pediatric patients, urticaria is most frequently associated with food hypersensitivity, particularly to eggs, cow's milk, chocolate, citrus fruits, strawberries, mushrooms, and processed meat products.

The pathogenesis of urticaria is primarily linked to mast cell degranulation, resulting in the release of histamine and other biologically active mediators. These substances increase vascular permeability, leading to plasma leakage into the dermis and the development of localized edema within the papillary layer. Clinically, this process manifests as transient wheals, which are the hallmark lesions of urticaria.

Urticaria is conventionally divided into acute and chronic recurrent forms. Acute urticaria is characterized by the sudden appearance of wheals on any area of the skin, which usually resolve spontaneously within minutes to several hours. Individual lesions may reach considerable size, as observed in giant urticaria or acute localized angioedema (Quincke's edema). Angioedema presents as sharply circumscribed swelling of the skin and subcutaneous tissue, most commonly involving the face or genital region. The affected area typically has a dense, elastic consistency and a pale, porcelain-like coloration. Edema usually subsides within several hours or, less commonly, within one day. Acute urticaria is often accompanied by intense pruritus and a burning sensation, and lesions may regress rapidly even in the absence of treatment.

Red dermographism is frequently observed in acute urticaria. In some cases, the disease persists for more than one month and evolves into a chronic form. Chronic recurrent urticaria is marked by prolonged or repeated exacerbations, severe pruritus, sleep disturbances, irritability, secondary lichenification, and excoriations. Bacterial superinfection may complicate the disease course. Involvement of mucous membranes, including the nasal cavity, oral mucosa, and larynx, poses a serious risk, as pronounced edema in these areas may result in life-threatening airway obstruction. Systemic manifestations such as chills, fever, general weakness, joint pain, and gastrointestinal symptoms may accompany urticarial attacks. A distinct variant, dermographic urticaria, is characterized by wheal formation in response to mechanical irritation of the skin.

Management of urticaria is based primarily on identification and elimination of the causative factor. In cases related to ingestion of allergens or toxins, gastrointestinal decontamination using laxatives and diuretics may be indicated, depending on the timing of exposure. Pharmacological treatment includes the administration of antihistamines (with the exception of diphenhydramine, which may provoke urticarial reactions), intravenous calcium chloride, intramuscular calcium gluconate, and intravenous sodium thiosulfate. In the presence of laryngeal edema, urgent subcutaneous injection of epinephrine is mandatory. Severe or refractory cases may require systemic glucocorticoid therapy. In chronic urticaria, dietary regulation is essential and involves exclusion of spicy foods, canned products, sweets, and alcohol, along with measures aimed at maintaining normal bowel function.

PRURITUS

A distinction is made between idiopathic pruritus and secondary pruritus, which manifests in conjunction with systemic conditions such as jaundice, diabetes mellitus, hematological disorders, malignancies, hepatic, renal, gastrointestinal, and

pancreatic dysfunctions, as well as central nervous system disorders. Pruritus also accompanies a wide spectrum of dermatological diseases. Senile pruritus may be associated with atherosclerotic vascular changes, metabolic disturbances involving cholesterol and nitrogen, and other age-related factors.

While primary skin lesions are not consistently observed in cases of pruritus, excoriations and hemorrhagic crusts are common findings, typically occurring on otherwise normal skin. Pruritus can be categorized as generalized or localized. Localized pruritus frequently affects the external genitalia and anal region. Common etiologies of localized pruritus include inflammatory genital conditions, trichomoniasis, candidiasis, gastrointestinal diseases, hemorrhoids, urinary tract infections, helminthiasis (particularly pinworm infection), prostatitis, and the use of contraceptive agents. Prolonged pruritus can lead to infiltration and thickening of the affected skin, with a predisposition to secondary bacterial infections at the sites of excoriation.

Management: Identifying and addressing the underlying cause of pruritus is essential. Symptomatic relief is achieved through the administration of antihistamines, hyposensitizing agents, and tranquilizers. Topical therapies include corticosteroid ointments, antipruritic suspensions, creams, and pastes.

INFANTILE AND ADULT PRURIGO

Infantile prurigo, also known as children's urticaria, papular urticaria, or strophulus, is a dermatological condition exclusively affecting children, typically commencing in infancy or early childhood (3-4 years). Predisposing factors include hypersensitivity to cow's milk, citrus fruits, egg whites, strawberries, and nutritional deficiencies in both the child and mother. Functional gastrointestinal disorders, helminthiasis, and genetic predispositions also contribute. The condition manifests as wheal-like lesions on the trunk, extensor surfaces of the upper extremities, and buttocks. Central papules are observed within the wheals, and characteristic papulovesicles or small vesicles with serous fluid are present. Severe pruritus leads to excoriations, erosions, and hemorrhagic crusts. Post-inflammatory pigmentary changes and white scars may persist. Systemic symptoms include regional lymphadenopathy and secondary bacterial infections. White dermographism is a common finding. With increasing age, infantile prurigo may evolve into adult prurigo or localized/diffuse neurodermatitis.

Management of infantile prurigo focuses on eliminating causative factors, including dietary adjustments and correction of gastrointestinal dysfunctions. Antihistamines, calcium supplements, vitamins A, B2, B3, B5, B6, B15, and histaglobulin are indicated. Topical therapies include antipruritic pastes, creams, and

corticosteroid ointments. Starch baths and generalized ultraviolet radiation are beneficial.

Adult prurigo, or transient prurigo, is more prevalent in women aged 20-40 years. Contributing factors include gastrointestinal, neurological, and psychiatric disorders, dietary indiscretions, autointoxication, and endocrine imbalances. Predominantly affecting the extensor surfaces of the limbs, buttocks, and trunk, adult prurigo presents with severe pruritus and papulovesicular or wheal-like lesions. Excoriations, hemorrhagic or serous crusts, and post-inflammatory hypopigmentation or hyperpigmentation are observed. Recurrences are common in spring and autumn. Management involves dietary modifications and addressing underlying etiologies. Antihistamines and hyposensitizing agents are recommended. Topical antipruritic creams and ointments are utilized.

Nodular prurigo is characterized by nodular lesions, associated with endocrine disorders, endogenous intoxication, and neuropsychiatric conditions, with a higher incidence in women. Severe pruritus of the extremities is followed by the development of papules and nodules, occasionally with vesicles. Nodules can reach several centimeters in diameter and may exhibit verrucous surface changes. Lesions are discrete and resolve over time, leaving depigmented scars.

Nodular prurigo is challenging to treat. In addition to hyposensitization and antihistamine therapy, diathermocoagulation, laser therapy, intralesional hydrocortisone injections, cryotherapy with liquid nitrogen, and chlorethyl irrigation are employed.

PRURIGO: INFANTILE AND ADULT FORMS

Infantile prurigo, characterized by terms such as children's urticaria, papular urticaria, or strophulus, is a dermatological condition exclusive to pediatric patients, typically manifesting during infancy or early childhood, often by the age of 3-4 years. Key etiological factors include heightened sensitivity to bovine milk, citrus fruits, egg albumen, and berries, alongside maternal and child nutritional deficiencies, gastrointestinal dysfunctions, helminthic infections, and inherited susceptibilities. The clinical presentation is marked by the appearance of wheal-like lesions on the trunk, extensor aspects of the limbs, and gluteal region. Central papules are observed within these wheals, and characteristic papulovesicular eruptions or small vesicles containing serous fluid are noted. Intense pruritus leads to excoriations, erosions, and hemorrhagic crusts. Post-inflammatory pigmentary changes and hypopigmented scars may persist. Systemic manifestations may include regional lymphadenopathy and secondary bacterial infections. White dermographism is a frequent finding. With increasing age, infantile prurigo can evolve into adult prurigo or localized/diffuse neurodermatitis.

Therapeutic strategies for infantile prurigo emphasize the elimination of precipitating factors, including dietary adjustments and correction of gastrointestinal disturbances. Antihistamines, calcium supplements, vitamins A, B2, B3, B5, B6, B15, and histaglobulin are indicated. Topical therapies include antipruritic pastes, creams, and corticosteroid ointments. Starch baths and generalized ultraviolet irradiation are considered beneficial.

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Nodular prurigo is characterized by nodular eruptions, associated with endocrine dysfunctions, endogenous intoxication, and neuropsychiatric conditions, with a higher prevalence in women. Severe pruritus of the extremities precedes the development of papules and nodules, occasionally with vesicles. Nodules can attain several centimeters in diameter and may exhibit verrucous surface changes. Lesions are discrete and resolve over time, leaving depigmented scars.

Nodular prurigo presents a therapeutic challenge. In addition to hyposensitization and antihistamine therapy, diathermocoagulation, laser therapy, intralesional hydrocortisone injections, cryotherapy with liquid nitrogen, and chlorethyl irrigation are employed.

PRURIGO: INFANTILE AND ADULT MANIFESTATIONS

Infantile prurigo, also designated as children's urticaria, papular urticaria, or strophulus, is a dermatological condition exclusively observed in the pediatric population, typically initiating during infancy or early childhood, frequently by the age of 3-4 years. Predisposing factors include heightened sensitivity to bovine milk, citrus fruits, egg albumen, and berries, alongside maternal and child nutritional deficiencies, gastrointestinal dysfunctions, helminthic infections, and inherited susceptibilities. The clinical presentation is characterized by the appearance of wheal-like lesions on the torso, extensor aspects of the limbs, and gluteal region. Central papules are observed within these wheals, and characteristic papulovesicular eruptions or small vesicles containing serous fluid are noted. Intense pruritus leads

to excoriations, erosions, and hemorrhagic crusts. Post-inflammatory pigmentary changes and hypopigmented scars may persist. Systemic manifestations may include regional lymphadenopathy and secondary bacterial infections. White dermographism is a frequent finding. With increasing age, infantile prurigo can transition into adult prurigo or localized/diffuse neurodermatitis.

Therapeutic interventions for infantile prurigo focus on the elimination of precipitating factors, including dietary adjustments and correction of gastrointestinal disturbances. Antihistamines, calcium supplements, vitamins A, B2, B3, B5, B6, B15, and histaglobulin are indicated. Topical therapies include antipruritic pastes, creams, and corticosteroid ointments. Starch baths and generalized ultraviolet irradiation are considered beneficial.

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SEBORREA

Seborrhea predominantly affects seborrheic regions of the skin, including the scalp, face, chest, and interscapular area, and develops as a result of excessive sebum production associated with hyperactivity of the sebaceous glands. Clinically, seborrhea is classified into three main forms: oily (liquid), dry (thick), and mixed.

The pathogenesis of oily seborrhea is largely linked to functional disturbances of the autonomic nervous system. This form is characterized by an increased concentration of free fatty acids in the sebum. In contrast, dry (thick) seborrhea is more closely associated with gonadal dysfunction and is marked by a reduced level of free fatty acids along with an increased proportion of bound fatty acids and cholesterol. A distinctive feature of this variant is follicular hyperkeratosis, which leads to obstruction of follicular openings and subsequent comedone formation. The involvement of microbial flora, particularly streptococci and staphylococci, may further aggravate the disease process.

The severity and persistence of seborrhea are influenced by multiple contributing factors, including disorders of the gastrointestinal tract (such as gastritis, peptic ulcer disease, and colitis), hyperandrogenism, chronic focal infections, vitamin deficiencies, and insufficient skin hygiene.

Oily seborrhea most commonly affects the nose, cheeks, and forehead, where dilatation of sebaceous gland ducts results in excessively greasy and shiny skin. The hair becomes oily, adheres together, and exhibits increased shedding, which may eventually lead to thinning or alopecia. Frequent complications of oily seborrhea include seborrheic eczema, comedones, atheromas, and hair loss.

Dry or thick seborrhea typically develops during adolescence, most often between 16 and 20 years of age, and generally presents with less pronounced exacerbations compared with the oily form. The facial skin appears thickened, with visibly enlarged follicular openings and dilated sebaceous ducts. Sebum in this variant has a dense, paste-like consistency and is discharged as thick oily material. After cleansing with warm water and soap, the degreased skin surface often appears dry and slightly glossy for several hours.

In some individuals, seborrheic areas remain persistently dry and thickened, with evident signs of follicular hyperkeratosis. Both true and false white acne lesions may be observed. False white acne represents superficial sebaceous gland cysts containing a curd-like mixture of fat and keratinized cells, commonly localized on the face, chest, and back. These cysts may empty spontaneously under mechanical pressure during washing.

Atheromas are a common complication of dry seborrhea and represent inflammatory cysts of the deep sebaceous glands. In cases of acute inflammation, abscess formation may occur, and following drainage of purulent contents, healing often results in scar formation. Comedones are another frequent manifestation of seborrhea and represent the initial stage of acne development. A comedone consists of accumulated keratinized epidermal cells forming a follicular plug, typically capped by a darkened surface.

Comedones obstruct dilated follicular openings and, when compressed, release a whitish, pasty material. They may appear not only on the face but also on the chest, back, posterior neck, auricular regions, retroauricular folds, shoulders, lateral trunk surfaces, and the occipital scalp. Small comedones can progress to papular or pustular acne, while inflammatory changes around larger lesions often result in the formation of atrophic scars.

Mixed seborrhea represents an intermediate form between oily and dry variants. The condition usually begins between the ages of 12 and 14 as oily seborrhea confined to the facial skin. Over time, acne lesions increase in number, accompanied by the development of comedones and atheromas, with subsequent spread to all seborrheic zones. The disease course typically extends until approximately 26–28 years of age. Clinical improvement is often noted during the summer months under the influence of ultraviolet radiation. Mixed seborrhea is frequently complicated by pustular dermatoses, alopecia, and rosacea.

Treatment involves the administration of high doses of vitamins A, E, and C, along with iron supplements and general tonic therapy. Patients are advised to reduce intake of carbohydrates, salt, fats, spicy foods, and seasonings. Local therapy includes the application of sulfur-containing preparations, salicylic acid, resorcinol, and other keratolytic and antiseborrheic agents.

CHRONIC CONNECTIVE TISSUE DISEASES: LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus (SLE), also known as erythema multiforme, is a chronic disorder characterized by connective tissue damage. The precise etiopathogenesis remains unclear. While various theories (autoimmune, genetic, endocrine, viral, etc.) have been proposed, none fully elucidate the disease's complexity. Environmental factors, including mechanical and chemical trauma, ultraviolet radiation exposure, certain medications, ionizing radiation, and focal infections, are recognized as significant contributors. A central role in the pathogenesis is attributed to heightened skin sensitivity to ultraviolet light, dysregulation of connective tissue fibrous component metabolism, and autoantibody production.

The increased cutaneous sensitivity to ultraviolet radiation is linked to the presence of coproporphyrin in sebaceous gland secretions. The photodynamically modified sebaceous secretions act as a source of polyautoantigens, which enter the bloodstream and induce the immunologic alterations characteristic of SLE. A specific aggressive protein, the antinuclear factor (ANF), has been identified. This IgG class protein penetrates leukocytes, localizing to their nuclei, ultimately leading to the formation of lupus erythematosus cells (LE cells). LE cells are predominantly

observed in systemic SLE and less frequently in chronic forms. Antinuclear autoantibodies are detectable in the serum of patients with systemic SLE.

Classification of lupus erythematosus: 1. Chronic lupus erythematosus, encompassing four clinical subtypes: discoid, diffuse, centrifugal erythema of Biett, and Irgang-Kaposi deep lupus erythematosus. 2. Systemic lupus erythematosus, categorized as acute, subacute, or chronic. Discoid lupus erythematosus commonly localizes to sun-exposed areas: the nasal dorsum, cheeks, forehead, chin, scalp, and vermilion border of the lips. The primary lesion is an erythematous plaque with edema, progressing to infiltration. Lesions coalesce, forming erythematous-infiltrative plaques covered with adherent keratin scales. Removal of these scales elicits pain (Benier-Meshchersky sign). The undersurface of the removed scale reveals a horny spike (lady's slipper sign). The pain and lady's slipper sign are attributed to follicular hyperkeratosis in discoid lupus erythematosus. The cutaneous process culminates in atrophic scarring. Thus, this form is characterized by erythema, follicular hyperkeratosis, and atrophy. Within the lesion, three distinct zones are discernible: central atrophic scarring, a middle zone of infiltration and hyperkeratosis, and a peripheral erythematous border.

Chronic Cutaneous and Systemic Lupus Erythematosus

Diffuse cicatricial erythema is characterized by the presence of multiple erythematous and scaly lesions accompanied by follicular hyperkeratosis and areas of pink atrophy. The disease course may be associated with low-grade fever, elevated erythrocyte sedimentation rate, anemia, leukopenia, and arthralgia. Clinical observations indicate the possibility of progression to systemic lupus erythematosus.

Biett’s centrifugal erythema presents as edematous erythematous patches with well-demarcated borders, lacking hyperkeratosis and atrophy. Lesions are most frequently localized on the cheeks and forehead and are not accompanied by subjective symptoms. Transition to systemic lupus erythematosus is also possible in this form.

Irgang–Kaposi deep lupus is manifested by dense, deep-seated subcutaneous nodules of bluish-pink coloration that are not adherent to underlying tissues. Lesions typically occur on the cheeks, nose, scalp, trunk, and extremities.

The diagnosis of chronic cutaneous lupus erythematosus is primarily based on characteristic clinical features, while histological examination is utilized in diagnostically uncertain cases. All patients must undergo thorough evaluation to exclude or confirm systemic lupus erythematosus.

Diagnostic Criteria for Systemic Lupus Erythematosus

According to established criteria, systemic lupus erythematosus is diagnosed based on a combination of clinical and laboratory findings. Cutaneous manifestations include facial erythema, alopecia, ulcerations of the oral or

nasopharyngeal mucosa, marked photosensitivity, and capillaritis of the fingertips. Renal involvement is indicated by proteinuria, erythrocyturia or leukocyturia, and the presence of urinary casts.

Laboratory abnormalities of diagnostic significance include anemia, leukopenia, detection of LE cells, high titers of anti-DNA antibodies, antinuclear antibodies at titers exceeding 1:100, thrombocytopenia (below 100×10^9/L), and reduced complement activity (CH50 below 35 units). Differential diagnosis should include papulonecrotic cutaneous tuberculosis, rosacea, cutaneous leishmaniasis, tuberculous syphilis, dermatomycoses, and alopecia areata.

Clinical Features of Systemic Lupus Erythematosus

Systemic lupus erythematosus predominantly affects young women and may arise de novo or evolve from chronic cutaneous forms. Cutaneous involvement is characterized by edematous erythema on the face, often extending to the neck and chest. Polymorphic eruptions, including papules, vesicles, bullae, crusted lesions, and erythematous-hemorrhagic spots, may appear on the trunk and extremities. Healing typically results in pigmentation or mild cicatricial atrophy. Mucosal involvement is relatively uncommon.

The general condition is often impaired, with manifestations such as fever, fatigue, weakness, and weight loss. Renal involvement occurs in approximately half of patients and is manifested by proteinuria, hematuria, and urinary casts; progression to uremia indicates an unfavorable prognosis. Cardiac involvement may present as endocarditis, pericarditis, or myocarditis, while pulmonary complications include pneumonia and pleuritis. Generalized lymphadenopathy, hepatosplenomegaly, and joint involvement are also frequently observed.

The acute form of systemic lupus erythematosus resembles a severe septic condition and may occur without skin manifestations in up to 20% of cases. Subacute disease is characterized by predominance of musculoskeletal symptoms, whereas the chronic form follows a prolonged course with recurrent exacerbations. Laboratory findings commonly include elevated ESR, anemia, leukopenia with lymphopenia, thrombocytopenia, hypoproteinemia, hypergammaglobulinemia, proteinuria, and the presence of LE cells, which are detected in 60–84% of patients.

Treatment Principles

Therapy of lupus erythematosus includes the use of antimalarial agents with photosensitizing properties. Quinoline derivatives such as hydroxychloroquine, chloroquine, delagil, and presocil are administered at a dose of 0.25 g twice daily in 10-day courses with weekly intervals. Vitamin therapy includes B-group vitamins (B_2, B_5, B_{12}) with hypophotosensitizing effects, as well as vitamins A, C, E, and P to support connective tissue metabolism.

Immunomodulatory agents with anti-inflammatory and immunostimulatory properties, such as taktivin or thymalin (administered as a 10-injection course), apilak (0.01 g three times daily for 10 days), and systemic corticosteroids are used according to disease severity. Topical therapy consists of corticosteroid and photoprotective ointments or creams.

In systemic lupus erythematosus, systemic corticosteroid therapy is indicated at doses of 60–80 mg per day. Combined regimens involving corticosteroids and antimalarial drugs may be employed. In refractory cases, cytostatic agents, anabolic steroids, potassium and calcium supplements, and additional immunomodulators may be prescribed. Streptomycin and sulfonamides are contraindicated.

Prevention

Prevention of disease relapses requires long-term dispensary follow-up. Prior to periods of intense solar exposure, photosensitizing agents and photoprotective measures are recommended. Protective creams and powders containing salol (5–10%), tannin, or para-aminobenzoic acid may be used. Patients should avoid direct sun exposure, engage in indoor work, and utilize protective accessories such as umbrellas and scarves that reflect ultraviolet radiation. Sanitation of chronic infectious foci is essential. It should also be noted that pregnancy may provoke disease exacerbation.

SCLERODERMA

Scleroderma is a chronic disorder characterized by structural and functional disorganization of connective tissue, with involvement of the musculoskeletal and neuroendocrine systems. Among systemic connective tissue diseases, systemic scleroderma ranks third in prevalence after rheumatism and rheumatoid arthritis, whereas localized (cutaneous) forms account for approximately 0.3% of all dermatological conditions. The disease has been known since ancient times. A classic literary depiction of advanced scleroderma was provided in 1874 by I.S. Turgenev in his work “The Living Relics,” where the severe clinical manifestations of the disease were vividly described.

Despite extensive investigation, the exact etiology of scleroderma has not been fully clarified. Nevertheless, a number of triggering and predisposing factors have been identified, including acute and chronic infections, mechanical trauma, prolonged vibration exposure, ionizing radiation, physical and emotional stress, as well as the use of certain medications, vaccines, and sera.

The pathogenesis of scleroderma is largely associated with pronounced vascular disturbances, particularly persistent vasospasm, which is mediated by increased accumulation of biologically active substances such as serotonin and hyaluronic acid. These processes result in fibrinoid degeneration of connective

tissue, followed by edema and progressive indurative sclerosis of the skin. Genetic predisposition is also considered to play a contributory role. Scleroderma may develop at any age, although it is more frequently observed in women. In recent years, an increasing incidence has been reported among children, including cases occurring in the neonatal period.

Clinically, scleroderma is divided into localized and systemic forms. Localized scleroderma encompasses several clinical variants, the most common of which is the plaque form. This variant evolves through a sequence of characteristic stages: inflammatory edema, induration, and subsequent atrophy. The disease typically begins with the appearance of a dense, edematous, pale pink lesion with indistinct margins, most often located on the trunk and extremities, and less commonly on the face, neck, or oral mucosa. As the process progresses, the central portion of the lesion gradually becomes paler, acquiring an ivory-white coloration, while a bluish-pink or violaceous peripheral rim persists. The presence of this peripheral ring represents a key clinical sign of active disease.

With further progression, marked induration develops: the affected skin becomes tightly bound to underlying tissues, smooth and shiny, and cannot be folded. Normal skin markings disappear, and appendageal structures such as hair follicles, sweat glands, and sebaceous glands undergo atrophy, resulting in reduced hair growth, sweating, and sebum production. Sensory perception in the involved area is also diminished. In the later stages, the peripheral violaceous ring gradually fades, the induration softens, and pronounced cutaneous atrophy becomes evident.

Linear and Other Forms of Scleroderma

The linear variant of scleroderma is most frequently observed in childhood. The disease process follows the typical sequence of stages, beginning with erythema and edema, followed by induration and subsequent atrophy. Lesions commonly arise on the scalp and extend to the forehead and nasal bridge, producing a characteristic appearance resembling a scar caused by a sword strike. In some cases, linear lesions are distributed along nerve pathways or within the Zakharyin–Ged zones on the trunk and extremities. When ulceration and mutilation occur, deeper structures, including underlying tissues, may become involved.

White spot disease, also known as scleroatrophic lichen, represents a superficial form of scleroderma that predominantly affects women. Lesions are most often localized on the upper back, interscapular region, chest, and genital area. Clinically, small white or pearly macules appear, often with shallow sclerotic depressions and signs of cutaneous atrophy. In some cases, lesions are surrounded by a narrow violaceous or pink rim. The disease course typically culminates in superficial atrophy.

Differential diagnosis of localized scleroderma includes lupus erythematosus, parapsoriasis, adult-onset scleroderma, neonatal scleroma, and certain myopathies.

Systemic (Diffuse) Scleroderma

Systemic or diffuse scleroderma usually begins acutely or subacutely, presenting with general malaise, sleep disturbances, pruritus, arthralgia, and fever. This is followed by the development of dense cutaneous edema, during which the skin becomes tense, non-foldable, and exhibits a marbled appearance. In the infiltrative stage, the skin acquires a wood-like density, becomes firmly adherent to underlying tissues, and displays a waxy, smooth surface. As sclerosis progresses, movements and respiratory excursions become restricted, and facial involvement produces a mask-like appearance. Advanced stages are characterized by atrophy of the skin, subcutaneous tissue, and muscles, giving the patient a cachectic, skeletal appearance. Ultimately, most internal organs and organ systems are affected.

Diagnostics and Differential Diagnosis

There are no pathognomonic laboratory tests specific for scleroderma. However, laboratory findings often include elevated erythrocyte sedimentation rate, hyperproteinemia (exceeding 85 g/L), hypergammaglobulinemia, and detection of antibodies to DNA or antinuclear antibodies. Differential diagnosis should be conducted with scleroderma-like conditions, particularly dermatomyositis.

Treatment of Scleroderma

During the progressive phase of the disease, therapy may include high-dose penicillin, administered in courses of up to 15 million units daily, with two to three treatment cycles and intervals of 1.5–2 months between courses. The therapeutic effect of penicillin is attributed to its active component, penicillamine, which inhibits the formation of insoluble collagen.

For both localized and generalized forms, hyaluronidase (Lidase) is prescribed at a dose of 64 IU administered subcutaneously or intramuscularly once daily, typically for a course of 15 injections, with repeat courses after 2–3 months. In severe cases involving deep fascial structures or in the absence of response to penicillin and enzymatic therapy, systemic corticosteroids are recommended.

To stimulate connective tissue metabolism, biogenic stimulants such as splenin, vitreous body extract, and aloe preparations are used. Immunomodulatory agents aimed at normalizing immune responses and collagen synthesis are also indicated; for example, taktivin may be administered subcutaneously at 1 ml daily for two weeks. Vasodilators (including theonikol, complamine, and undecalin), along with vitamins B, A, E, and C, are commonly prescribed. Physiotherapeutic modalities—such as ultrasound therapy, electrophoresis with zinc sulfate, paraffin or ozokerite applications, and other thermal procedures—are widely utilized.

Preventive measures focus on identifying and eliminating harmful environmental and occupational factors, as well as timely treatment of chronic infectious foci to prevent disease progression and systemic involvement. All patients with scleroderma require regular medical follow-up.

Dermatomyositis

Dermatomyositis is a diffuse connective tissue disease with incompletely understood etiopathogenesis. Viral or virus-mediated genetic mechanisms are postulated. The disease is associated with immune dysregulation, predominantly affecting cellular immunity, as well as disturbances of the nervous and endocrine systems, metabolic abnormalities, increased sensitivity to ultraviolet radiation, hypothermia, trauma, and certain medications. Dermatomyositis predominantly affects women of all age groups and is classified into idiopathic and paraneoplastic forms.

Clinically, dermatomyositis presents with cutaneous, muscular, and systemic manifestations. Early symptoms include erythema and edema of the eyelids, face, and hands, muscle weakness, and fever. Acute forms are characterized by pronounced weakness, myalgia, hyperhidrosis, chills, vomiting, and high fever. Facial involvement often begins with periorbital erythema and edema, subsequently extending to the cheeks, nose, neck, chest, back, joints, and palms.

The disease typically follows a prolonged course, with desquamative erythema, telangiectasia, and congestive lichenoid papules. Hyperpigmentation or depigmentation and cutaneous atrophy resembling poikiloderma are common. Less frequently, scarlatiniform rashes, vesicles, and bullae may appear. Generalized lymphadenopathy and dystrophic calcinosis of subcutaneous tissues may develop. Mucosal involvement is characterized by edema, hyperemia, erosions, and ulcerations.

Muscle involvement manifests as pain, weakness, and reduced motor activity. Severe cases may be complicated by aphonia, dysphagia, arthritis, and arthralgia. Visceral involvement results from muscle pathology and may include myocarditis, myocardial dystrophy, and gastrointestinal dysmotility, with widespread multiorgan involvement.

Acute dermatomyositis occurs predominantly in children and is often marked by early skin manifestations preceding internal organ involvement. In pediatric cases, arthritis, polyserositis, pronounced exudative reactions, and vesiculobullous lesions are more frequent. Skeletal muscle involvement initially affects the shoulder girdle and masticatory muscles, progressing to include the pharyngeal, laryngeal, cervical, intercostal, diaphragmatic, and hand muscles, leading to contractures and

functional impairment. Visceral complications, autonomic dysfunction, and peripheral neuropathies are consistently observed.

Paraneoplastic dermatomyositis occurs in association with malignant neoplasms in individuals over 40 years of age, more commonly in men. It is frequently linked to adenocarcinomas of the female reproductive organs, prostate cancer, seminoma, thymoma, plasmacytoma, melanoma, thyroid carcinoma, and hematological malignancies. In many cases, dermatomyositis precedes the clinical detection of malignancy and serves as an unfavorable prognostic indicator. Successful treatment of the underlying tumor often leads to remission of dermatomyositis, while tumor recurrence is accompanied by disease relapse.

Diagnostic evaluation should be particularly thorough in patients over 40 years of age presenting with persistent fever, elevated ESR, resistance to corticosteroid therapy, and edema. Diagnosis is based on characteristic cutaneous and musculocutaneous findings and is confirmed by laboratory and instrumental studies, including creatinuria, elevated aminotransferase levels (especially AST), muscle biopsy, and electromyography. Differential diagnosis includes systemic lupus erythematosus, rheumatoid arthritis, rheumatic fever, scleroderma, thrombophlebitis, endarteritis, and infectious diseases such as trichinosis, infectious mononucleosis, brucellosis, and typhoid fever.

Treatment and Prevention of Dermatomyositis

Systemic therapy primarily involves corticosteroids, most commonly prednisolone, along with nonsteroidal anti-inflammatory drugs and cytostatic agents such as methotrexate, azathioprine, or prospidin. Comprehensive treatment regimens also include vitamins B, C, and E, ATP, anabolic hormones, and appropriate topical therapies.

Preventive measures emphasize early detection of potentially associated malignant and systemic diseases, as well as avoidance of skin trauma and hypothermia.Skin porphyria late skin porphyria is mainly observed in those in adulthood, the name of the disease also refers to this. Patients complain to the doctor that as early as they first met, not that their skin had increased sensitivity to sunlight, but that the skin of the hands became thinner and, as a result of minor effects, bubbles, scratches, wounds remained formed.

The diagnosis is easy to confirm: patients return yellowish-reddish light when their urine is illuminated by a wood lamp. Unlike other types of porphyria, late cutaneous porphyria does not experience acute abdominal pain, polyneuropathy, and respiratory failure.

The disease is observed in 30-50 years old, it is rare in children. Older than 60 years, at the expense of men taking estrogens (in the case of prostate cancer),

women can get the disease at the age of 18-30 at the expense of taking peroral contraceptives. The disease occurs almost equally in men and women.

Provoking factors: ethanol, estrogens, hexachlorobenzene (fungicide), chlorinated fe-zeros, iron preparations, tetrachlordibenzo-p-dioxin. A disease attack can be caused by a high amount of Chlo-rixin.

Risk factors: diabetes mellitus, hepatitis C, VICh-infection.

Clinic. The disease begins slowly. Puffs on the surface of the limbs arise from the influence of sun nu-ri, in most cases, as early as the time of redness of the skin. Patients complain of extremely thin and easy scarring of their skin and the appearance of painful erosions after less severe injuries.

In erythropoietic protoporphyria and late cutaneous porphyria, the liver is hastened. And in the case of VA-riegat porphyria, vegetative neuropathy and acute pain in the abdomen are superstitious.

Skin rashes are located on the surface of the face, neck, collar, scribal surfaces of the hands, paws of the limbs. On the skin, blisters, erosion, thickets, scars are observed. Blisters with a tense roof are observed on the surface of the seemingly healthy te-Ris. The bubble, bubbles burst and slow-ending erosions form in its place. Erosion ends by forming pinkish atrophic scars. Hypertrichosis is observed in the facial area and is one of the main complaints of patients. The injured skin area is sclerodermywhich is thickened, waxy,yellowish-whitish in the oven. The facial skin is mostly bruised around the eyes. Open areas of the skin, on the other hand, are hyper-pigmented with diffuse melanin (not always).

Diagnosis. The diagnosis is made based on the clinical picture of the disease, the observation of a characteristic yellowish-reddish light return when irradiated with Wood's lamp (yori-tilgan), and an increase in the amount of porphyrins in urine.

Differential diagnosis: Pseudoporphyria. Contributing factors are: drugs (naproxen, cyclospo-rin, ibuprofen, dapsone, furosemide, tetracyclines, pyridoxine); hemodialysis; solarium administration; – liver cell cancer;systemic red runner; sarcoidosis; Shagren's syndrome; hepatitis C.

In dyshydrotic eczema, the skin of the palms and soles is injured, and the surfaces of patients are injured.

The clinical picture of acquired bullez epidermolysis is very similar, the skin is easily damaged, bruising spots appear easily, blisters are formed from exposure to sunlight, blisters are sub-epidermal, inflammation of the derma becomes less developed.

It is necessary to stop drinking alcohol and eliminate other provoking factors (extrogens, chlorinated phenols, tetrachlordibenzo-p-dioxin).

Take 500 ml of blood, between 1-2 weeks, until the amount of hemoglobin is reduced to 10 g%.

Chlorhine is prescribed if blood intake is prohibited due to anemia. It is necessary that the treatment is carried out by an experienced doctor, otherwise an outbreak of the disease and even liver failure can be observed. Chlorhine can cause long-term remission, while in some patients it can eliminate metabolite outcrosses, even leading to complete recovery. The most effective method is to take 3-4 times of blood and then a small amount of chlorhine tay-in.

BULLOUS DERMATOSES: PEMPHIGUS VULGARIS

The etiopathogenesis of pemphigus vulgaris remains incompletely understood. Various theories have been proposed, including chloride retention, toxic, neurogenic, enzymatic, bacterial, viral, and autoimmune origins. Clinical presentations are categorized into vulgar, foliaceous, vegetans, and seborrheic (erythematous) forms. Pemphigus vulgaris is the most prevalent variant, predominantly affecting women over 40 years of age. A unifying characteristic across all pemphigus subtypes is acantholysis, leading to the formation of intraepidermal vesicles in the oral mucosa and skin. Acantholysis is clinically assessed using the Nikolsky sign, wherein lateral pressure on seemingly unaffected skin adjacent to a blister induces epidermal detachment, or by observing epidermal detachment beyond the blister margin upon traction with forceps. The Asboe-Hansen sign, indicative of blister extension upon application of downward pressure, is also observed.

Pemphigus vulgaris typically initiates with the appearance of bullae on the oral and pharyngeal mucosa, which rapidly rupture, resulting in painful, bright red erosions with epithelial remnants. Labial erosions are often covered with thick, friable, hemorrhagic crusts. In severe cases, the entire oral mucosa may be transformed into a persistent erosive surface. Isolated mucosal involvement may precede cutaneous manifestations by several days to months. These patients often seek dental consultation and may be misdiagnosed and treated for ulcerative or aphthous stomatitis, erythema multiforme, or fungal infections.

Cutaneous lesions, primarily located on the chest and back, manifest as tense bullae with clear or slightly turbid contents, arising on apparently normal skin. Large bullae may exhibit a pear-shaped configuration due to gravitational forces on the exudate (pear sign). Both Nikolsky and Asboe-Hansen signs are positive. Following bullae rupture, erosions develop, which eventually re-epithelialize, leaving behind pigmented macules. Progressive deterioration of the patient's general condition, including weakness, fever, and secondary infections, is observed. Without appropriate intervention, the condition can be fatal. Diagnosis of pemphigus vulgaris

is based on clinical presentation, positive Nikolsky and Asboe-Hansen signs, and the identification of Tzanck cells (acantholytic cells with modified spines, large nuclei, and a narrow rim of basophilic cytoplasm). Immunofluorescence (IF) studies are employed for confirmation. Alterations in water and electrolyte balance, specifically sodium chloride retention, are frequently noted. Histopathological examination, revealing intraepidermal bullae, provides the definitive diagnosis.

Vegetating pemphigus is characterized by the formation of bullous lesions not only on the oral mucosa but also in periorificial areas and intertriginous zones, including the axillae, inguinofemoral folds, inframammary regions, and periumbilical area. Following rupture of the blisters, papillomatous vegetations develop on the eroded surfaces and may spread extensively. Over time, these lesions gradually regress, erosions undergo epithelialization, and residual hyperpigmented macules remain.

The general condition of affected patients is significantly compromised, and mortality may occur as a result of severe complications. Pemphigus foliaceus is clinically manifested by superficial, flaccid, flat bullae that rapidly rupture, leaving erosions covered by thin, lamellar scales or crusts. Nikolsky’s sign is markedly positive, while mucous membranes are typically spared. Alopecia and nail dystrophy may develop. Fatal outcomes are often associated with progressive cachexia or secondary infections.

Seborrheic (erythematous) pemphigus usually begins with the appearance of yellowish scales or crusts on the skin of the face and scalp. Removal of these crusts reveals an eroded surface. Subsequently, bullous lesions may appear on the chest and back, rapidly drying to form crusts. Nikolsky’s sign is positive. Clinically, the lesions may resemble seborrheic eczema or impetigo. The disease course is prolonged, and progression to pemphigus foliaceus is possible.

Pemphigus is relatively rare in childhood and predominantly affects girls between the ages of 2 and 15 years. Among the various forms of pemphigus vulgaris in pediatric patients, the foliaceus variant is encountered most frequently.

BULLOUS DERMATOSES: PEMPHIGOID GROUP

Within the pemphigoid group, bullous pemphigoid is the primary condition resembling pemphigus vulgaris clinically. Lever's disease, or nonacantholytic pemphigus, and cicatricial pemphigoid, are distinct entities characterized by a benign clinical course, subepidermal vesicle formation, absence of acantholysis, and a negative Nikolsky sign. Bullous pemphigoid typically affects individuals over 50 years of age and may initially manifest on the oral mucosa. The precise etiopathogenesis remains unclear, though autoimmune mechanisms linked to potential paraneoplastic syndromes, toxic exposures, or metabolic disturbances are

implicated. Clinically, tense bullae with serous or hemorrhagic contents, up to 1 cm in diameter, develop on erythematous and edematous oral mucosa. These lesions subsequently rupture, forming erosions that tend to re-epithelialize. Similar lesions may appear on the skin. In children, oral mucosal involvement is more frequent than in adults. Papular and erythematous-urticarial lesions may also be present on the skin, mimicking erythema multiforme or toxic epidermal necrolysis.

Diagnosis relies on clinical presentation, cytological and histological findings, and immunofluorescence studies.

Treatment involves excluding underlying malignancies, leukemia, or lymphogranulomatosis, followed by the administration of glucocorticosteroids, cytotoxic agents, and diaminodiphenylsulfone (DDS). Topical therapy parallels that of pemphigus vulgaris. Cicatricial pemphigoid is characterized by conjunctival, oral mucosal, and cutaneous bullae that heal with scarring and adhesions. It predominantly affects women over 50 years of age. Initial manifestations typically involve the oral mucosa or eyes, followed by potential involvement of the pharynx, larynx, nose, esophagus, urinary tract, and rectum. Ocular involvement begins as catarrhal conjunctivitis, progressing to bullae formation and subsequent adhesions, leading to symblepharon, narrowed palpebral fissures, and blindness. Mucosal involvement can result in pharyngeal, oral commissure, tongue, and tonsillar destruction. Laryngeal, esophageal, and urinary tract strictures, phimosis, and vaginal atrophy may also occur. Cicatricial pemphigoid must be differentiated from pemphigus vulgaris.

Combined sulfone therapy is often the most effective treatment. Benign mucous membrane pemphigoid, a purely oral condition, is characterized by nonacantholytic bullae. The etiology and pathogenesis are unknown, and it predominantly affects women over 20 years of age.

Clinic. On the mucous membrane of the oral cavity, tense bubbles with serous content and a dense coating appear, which disappear after a few hours without a trace or open, quickly forming slight painful erosions that epithelialize. The process is chronic, the course is good. Treatment is symptomatic.

DURING HERPETIVORM DERMATITIS

The precise origins and mechanisms of dermatitis herpetiformis remain incompletely understood. However, a notable association with heightened sensitivity to gluten, a protein found in grains such as wheat, rice, corn, oats, rye, barley, and millet, has been observed in affected individuals. The onset of this dermatosis can be precipitated by inflammatory processes within the gastrointestinal tract, hepatic dysfunction, ascariasis, malignancies, lymphocytic leukemia, exposure

to iodine and bromine-containing medications, prior infections, and autoimmune phenomena.

Clinically, the condition is characterized by a pleomorphic eruption, encompassing erythematous macules, vesicles, bullae, and papules, arranged in grouped configurations. A cardinal symptom is intense pruritus, often preceding the cutaneous lesions. Tense bullae develop on slightly erythematous and edematous skin, subsequently rupturing to form erosions that heal with residual hyperpigmentation. The Nikolsky sign is consistently negative. Acantholytic cells are absent, but vesicle contents exhibit a significant eosinophilic infiltrate. Patients may demonstrate increased sensitivity to iodine preparations, as evidenced by a positive Jadassohn patch test. Histological examination reveals subepidermal vesicle formation and eosinophil accumulation.

In pediatric patients, prodromal symptoms such as fever, malaise, arthralgia, and dyspeptic complaints may precede the cutaneous eruption. Preschool-aged children typically present with large, tense bullae on erythematous, edematous skin, predominantly on the trunk and thighs. Small papular and papulovesicular lesions are also characteristic. Grouped bullae may localize to the inguinal and axillary folds, face, and extremities. Mucosal involvement is common in children. The scalp is typically spared. Treatment involves sulfone derivatives, including DDS, dimosiphone, dapsone, avlosulfone, promacetin, and sulfetron, potentially in combination with glucocorticoids. Bromine, iodine, barbiturates, and amidopyrine are contraindicated. Topical therapy mirrors that of pemphigus.

Preventive measures include strict adherence to a gluten-free and iodine-restricted diet. Patients require long-term follow-up and registration in a specialized dermatological clinic.

Eczema

Eczema is a recurrent chronic disease in which the skin is neuroallergic, accompanied by intense itching, with chin polymorphism and aso-siy rash on acute inflamed skin, with small blisters (reminiscent of boiling water).

Etiology and pathogenesis. At the origin of eczema, changes in the nervous system, weakening of protective forces, hypovitaminosis, the role of autointoxication is high, which is mainly the development of polyvalent sensitization against exogenous and endogenous Al-lergenes.

Clinic. According to clinical course eczema is acute, moderately acute and chronic eczema; according to its pathogenetic nature, clinical appearance and etiological factors – divided into chin, microbial, seborrheic, professional and pediatric eczema types (appendix VIII).

ChIN eczema (EXEMA ACUTUM

Chin eczema, acute eczema begins at once, accompanied by severe itching of the skin and the appearance of redness, swelling and numerous blisters of the skin, which are signs of acute inflammation. Pu-phacchas burst quickly, small droplet hydration begins on the skin (serous wells or eczematous wells), and there are also small nodules, pus around the inflamed furnace, and, mainly, sharp inflamed foci with indistinct borders, symmetrically located on the skin of the hands, feet and other areas of the body, are visible. In addition to severe itching, the patient sometimes complains that it seems to ache and overheat. In the clinical course, primary and secondary rashes can be varied and vary, and the patient is diagnosed with a condition of chin and false polymorphism (Kreibix triangle). In some patients, depending on which one of the morphological elements is most common in the clinical picture of the disease, conditionally wet, papules, vesicles, pustules and squamous varieties are distinguished.

As a result of the treatment of the clinical picture characteristic of acute eczema, which we have cited above, less spontaneously changes, the inflammatory process decreases, and a clinical manifestation characteristic of mid-acute eczema occurs. It is observed that there is no hydration in the patient's skin, there is a reduced cessation of new blisters and nodular rash, and there are secondary rashes on reddened and swollen skin in small quantities, and the patient notices a decrease in itching.

Subsequently, often without the influence of additional etiological factors and functional injury of the patient's internal organs, inflammatory infiltration characteristic of chronic eczema on the skin becomes rivo-jlib, the limited furnace becomes dense and thickened, and in the form of lichenification.

Eczema does not always last the same, sometimes it is suddenly sharpened and a new large number of different-ma-type rashes, often blisters overflow, giving rise to the clinical picture of acute eczema. When detected, this relapse will be more associated with a violation of the patient's own diet, medication intake or problems of the nervous system and other internal organs.

Microbial eczema (PARATRAVMATIC, varicose, nummular)

Microbial eczema may also have rashes characteristic of the above-mentioned chin eczema, but more pus is prominent, and the border of inflamed foci on the skin becomes clear and settles asymmetrically.

Microbial eczema appears as a secondary disease in a patient who has been hasty for a long time with a more purulent (fungal) disease, as a result of the development of sensitization, and is divided into nummular, para-traumatic and varicose types.

OCCUPATIONAL-RELATED (PROFESSIONAL) ECZEMA

Occupational eczema localizes primarily in exposed parts of the body's skin, and the onset, outbreak, and recurrence of the disease is caused by the development of sensitization, as a result of the long effects of allergens in the patient's workplace production.

The patient may first be diagnosed with monomorph, followed by polymorphic sensitization.

The clinical course of Professional eczema and dermatitis is practically no different from the above-mentioned dermatitis and eczema. Only in the periods of onset, professoinal eczema can be treated more mildly-Di and more so, injuring open parts of the body and being limited, so that hydration with eczematous wells can be very rare.

Since Professional morbidity is associated with the responsibility of the enterprise, it is necessary for the Attending Physician to provide medical care to the patient and refer him to a muta-chassised organization in order to identify and make a final diagnosis of the disease. In Uzbekistan, it is a clinic of the Institute of scientific investigation of sanitary, hygienic and Professio-nal diseases (Nii SGiPZ).

Diagnosis of the allergen by developing and returning the disease at launch, close-up commander and recovering when on leave, the patient's show, thinking that professional factors play a role, and then, through a test-taker test performed on the patient's skin is the basis for diagnosis.

Seborrhea eczema (EXEMA SEBORRHOICUM).

Seborrhea eczema-more commonly develops on the hairy part of the head, the area around the ears, the face, armpits, chest and other areas of the skin where fat-secreting glands are abundant. Attention of those around-attracts, the patient's skin glistens, is visible. In its clinical form, there will be blotches, nodules and flakes, yellowish, oily, with clearly distinguishable borders and overlapping. The disease begins with a period of sexual arousal, and the patient is disturbed by severe itching. In the clinical course of seborrheic eczema, there are no blisters and no hydration.

Histopathology of eczematous skin. Lymphocytic and histiocytic infiltrate is observed in the epidermis in spongiosis, parakeratosis, acanthosis and derma.

CHILDREN'S ECZEMA

Children's eczema begins with more ecxudative diathesis-appropriate changes, mainly because young mothers themselves break the diet, breastfeed the child, then their mistakes in complementary feeding the child develop and provoke this disease. In close relatives, parents or both of them have had various allergic diseases, errors in the child's diet quickly lead to the development of eczema.

The child is preceded by monovalent sensitization, followed by various foci of chronic infection, gastrointestinal diseases and the right-wrong treatment against

them, which reduces the immu-nobiological forces of the body and leads to the development of polyvalent sensitization. The disease first distorts the child's quality of life, bringing him to mental and physical weakness than his peers.

In the clinical course of the disease, on the other hand, there are various stones-malars characteristic of the eczema of adults, which the child may show signs of chin, microbial and seborrheic eczema. Children quickly develop a secondary infection, the lymphatic nodes enlarge, and can then develop into a clinical manifestation diffuse neurodermitis (atopic dermatitis).

Case.

1. Elimination of allergens that cause the disease.
2. Allergen-free, low-salinity and low-fluid diet.
3. Anti-inflammatory treatment (antihistamine and desensitizing drugs)
4. Tranquilizer and sedative preparations.
5. Identify diseases in the internal organs and treat them.
6. Corticosteroid drugs for a short time.
7. Local treatment is given according to the rules of local treatment, taking into account the indivi-dual sensitivity of the disease in each patient, depending on the type and clinical course of the disease (listed in the general part of the book, appendix No. IX).

Bounded NEURODERMITIS
(NEURODERMITIS SIRCUMSRINTA

Etiopathogenesis. The main pathogenetic factors in the origin of the disease are an increase in skin sensitivity to irritants, which is caused by an increase in nerve endings as well as hyperplasia of the epidermis in response to mechanical influences. As a result of this, a strong itching appears in the furnace as a result of an extremely weak effect, while such cases are not observed in the area of healthy skin.

Borderline neurodermitis is observed alone or in the case of several itchy foci of lichenization. Li-xenization foci are a characteristic symptom of borderline and diffuse neurodermitis. Limited neurodermitis will last for several decades if drug-induced skin itching does not stop the irritation.

The disease is often observed in adults and occurs the same in both sexes and lasts from a few weeks to several years. The main complaint of patients is itching of the skin. The Ja-enjoyed hearth becomes a kind of "erogenous zone", as patients take a break from kashinish-s. Therefore, the sleeping patient also has instinctive skin irritation. If the furnace is located in the foot area, patients will rub the furnace with their heels. Over time, constipation becomes a habit, which patients themselves do not notice. The need for tanning arises for trivial reasons: during the

time of dressing and undressing, during the period of urinating or rinsing cosmetics, before the touch of clothing and before sleep (the place-the skin on the bed warms up for a while, and the heat calls for a feeling of itching).

Clinic. On the skin, there is mainly a lichenization furnace, which looks like they are made up of small, dense elastic nodules. Dandruff in the oven will be strongly underdeveloped. When palpating, the skin is compacted, thickened and roughened, and when examined, the skin is less noticeable on the skin, the skin is tightened, the skin is hyperpigmented, and traces of straight – line Kashima are observed on the surface-excoriations, bloody scales. Slowly rubbing with a cotton swab-a strong itching is observed; this reflex is not observed on healthy skin. The Shape of the kilns is round, suyri, oblong (lengthening in the direction of contraction) and the border is usually clear. The furnaces are located in a recessed, observed in the case of one or more furnaces. Border neurodermitis most often settles on the back surface of the neck (women), on the hairy part of the head, on the curving surface of the knee joint, on the wrist fold, on the writing surfaces of the wrists, on the vulva, in the groin areas, in the perianal area, on the skin of the groin. Dermographism is white.

Anamnesis is made on the basis of clinical manifestations and comparative diagnoses.

1. Treatment of the disease is very difficult. The patient is constantly warned that the skin does not rub or wrinkle.

2. Occlusive dressings that are laid at night have a very good effect: these types of dressings protect the skin from tanning-peeling and relieve the rubbing of drugs.

3. Local-corticosteroid, rubbed with mazlar, over which dry marlya (gauze) binds. The method of injection of corticosteroids into foci is also considered to be effective methods. Triamcinolone is applied in the amount of 3 mg of µg mL, can invoke a high amount of skin atrophy. If the patolo-GIC furnace is located on the limbs, a mixture of black oil, zinc oxide, and a medium-strength corticosteroid is applied under the dry binding. Typically, corticosteroid mazes are rubbed and tied over.

But without rubbing the drug, binding is also an effective method, the cause is prevention of constipation. In addition to gauze ties, synthetic ties are also used. In some cases, applying hydrocolloid to the skin of the furnace first after a corticosteroid ointment will also give a good result. The bandage is placed for 1 Week. Corticosteroid plastic also has a good effect, it is put on 1 day. Zinc-gelatin binding. The gauze is impregnated with Unna paste, then large lichenization, which is tied to the cloth, the binding is placed for 1 Week.

ATOPIC DERMATITIS

Atopic dermatitis, or diffuse neurodermitis, is a skin multifactorial allergic disease that is chronic, reversible, accompanied by severe gills – swelling and specific rashes.

Etiology. Autosamal dominant the presence of a congenital predisposition to various allergic reactions.

Pathogenesis. From the reasons why the disease periodically recurs:

1. Exogenous and endogenous allergens.
2. Emotional and stress factors.
3. The skin becomes dry.
4. Infections and functional diseases of the internal organs.
5. The heat is hot and the body's tendency to sweat.

Clinic. The first signs of the disease begin at the age of a baby, at the time of giving the child additional food, and most often on his face (diathesis), acute inflammation in the skin joints or derma-tit-specific rashes with seborrhea appear. In the near term (2-3 years), identify the allergen that affects the child-nib, if it is not eliminated, polyvalent sensitization develops, and the patient develops a neurodermitis or a clinical-appropriate rash of eczema.

Diffuse diffuse diffuse neurodermitis is very aggressive, leaving only the entire skin of the patient dry for a short time without calming or constantly stopping, concealing it with non-sharp signs of inflammation (licheniphika-TSIA), traces of constipation and flaking. But the most severe damage to the inflamed foci is mainly concentrated on the inner surface of the face, neck, thigh and the arms, leg flexors. Of course, secondary infection is added to the traces of constipation, and the patient's lymphatic glands become larger. White dermographism is detected.

Most scientists in foreign countries do not distinguish between diffuse neurodermitis and atopic dermatitis. Signs that distinguish atopic dermatitis from other neurodermatoses:

Itch (PRURIGO

Itching (pochesuxa) is an acute or chronic, relapsing disease that is accompanied by severe itching of the skin. More common types of itch in practice are pediatric itch (Prurigo infantum), adult itch (Prurigo adultorum), and nodular itch (Prurigo nodularis

Etiology. Factors that cause itching in children and the development of the disease-the reasons for atopic dermatitis or children's eczema are reminiscent. In children, it is more maladaptive feeding, exudative diathesis and various intoxication, defects in the Endocrine, internal organs and si-stem.

Clinic. On the writing surfaces of the arms and legs, small crumbs, nodules and nodules-blisters appear, which are strongly itchy on the abdomen and buttocks. Erosion on the nodules, bloody hives appear in kashigan, and secondary infection

may develop. The general condition of the patient changes, a nervous state arises, and the disease continues to be chronic, disrupting the quality of life of the patient. The patient, on the other hand, causes children to lag behind their peers in development and, if left untreated, adult itching or atopic dermatitis.

Nodular itching (Prurigo nodularis), is a disease that occurs mainly in the calf of women, accompanied by limited, severely itchy nodules and is rare.

Clinic. The disease begins with severe itching, and as a result of constipation, nodules appear on the skin of the bol-dir part of the foot. The size of the nodes is on average 1 centimeter, the consistency is dense-solid, does not merge with each other. Itching is exacerbated, and New traces of tanning, bloody crusts and pigmented scars around the skin constantly appear. Nodes can be stored a lot of time, hypertrophied and enlarged around.

Pathogistology: infiltrate consisting mainly of mucosa, thickening of the granular floor, acanthosis, lymphocyte in the derma, fibroblast, histiocytes. Infiltrate is observed to lack collagen and elastic fibers, hyperplasia of nerve fibers, and thickening of the Schwann shell.

Anaphylactic shock

Anaphylactic shock is a systemic manifestation of an allergic reaction, accompanied by the separation of IgE-antibodies and immune mediators, leading to damage to vital tissues and organs.

Etiology. The main cause of anaphylactic shock (ASh) is the contact of the highly sensitized organ-m with specific antigens and allergens.

The main causes are medication (antibiotics, sulfonamides, analgesics, vitamins, etc.), certain foods and chemicals; insect bites (especially Wasps); diagnostic and therapeutic treatments with allergens. In many cases, as a result of hypersensitivity to medications, the patient may also develop severe rashes typical of Layell syndrome or Stevens-Johnson syndrome.

Pathogenesis. The basis of ash is a rapid type of allergic reaction, which is formed in a highly sensitized organism.

Therefore, the susceptibility to shock reaction is formed during the period of sensitization, and during that period the IgE-type reagin antitana is formed. They attach to their basophilic and fat cells with their Fc-fragments, leading to the separation of granules with rapid type mediators from them.

Clinic. Various organ and tissue damage is observed in ash. The main clinical types of ash are manifested as follows:

with a pleasant loss of collapse (in a severe course of shock), various degrees of arterial hypotension and garanging (in a lighter course);

pain in the abdomen, donkey, itchy skin as a result of hiccups or bronchospasm in the rapid deterioration of the breath (asphyxia).

Shock can occur in relatively light, medium-heavy and heavy levels. There is a mildly short prodromal period, which can range from a few minutes to an hour. These are the type-symptoms of an allergic reaction: itchy skin, rash of the donkey type, erythematosus, petechial and papulose elements, skin hyperemia, a feeling of overheating, spastic cough, dizziness, nausea, difficulty breathing-shi. Some have facial skin hyperemia, lip cyanosis, depression.

In the middle severe course, before shock, there is weakness, fear, facial hyperemia, a feeling of overheating, pain in various loka-lysations, rashes, cough, vomiting, followed by rapid pleasant loss. Cold sticky sweat forms on the forehead, some of them foam into the mouth. Blackberries do not react to light by expanding. Involuntary defecation and urination, spastic uterine contractions and the arrival of bloody vaginal secretions are observed.

Tonic and clonic contractions are observed as a result of cerebral ischemia and swelling of the serous layers.

As a result of the activation of the fibrinolytic system, bleeding from the nose, stomach and intestines can occur.

In the severe course of ash, rapid disruption of the central and vascular system occurs within a few seconds or minutes. The skin coating is sharply pale; foam appears on the chest, hands, lips – cyanosis, mouth-on the skin. The forehead is covered with cold and sticky sweat. The ventricles are dilated, there is no reaction to light, the cervical veins are dilated. Breathing is noisy, arrhythmic, in auscultation a "dumb" lung is heard.

Are the main causes of death in anaphylactic shock:

acute cardiovascular failure caused by stasis, thrombosis, microcirculation disorders and vascular collapse;

asphyxia of the hiccup as a result of Quincke's edema;

thrombosis of veins and blood transfusions to the organs necessary for life

Laboratory and instrumental examinations

In anaphylactic shock, the indicators of hemodynamics, microcirculation and metabolism are disrupted. Usually arterial pressure, circulating blood pressure decreases, which is checked at the expense of central venous pressure (5-12 CM water column in the norm), blood clotting and hematocrit increase. Hyperlactemic acidosis, reduced hydrocarbons in arterial blood (in the norm 22-25 mmol/l), there will be a deficit of bases (more than 5 mmol/l). Lactate levels exceed 1.6-2.8 mmol/L. In intense periods of shock, erythremia, leukocytosis, increased Soe, thrombocytopenia, middle eosinophilia.

During the period of shock, diuresis decreases; in kidney damage, proteinuria, hematuria, leukocyturia, cylindrical.

Diagnosis.

In typical cases, diagnosis is based on Anamnesis and clinical signs. Diagnosis becomes drastically more difficult in cases where Anamnesis cannot be collected.

Treatment.

The following stages are distinguished in the treatment of ash:

1) primary rapid therapy phase;

2) stage of therapeutic treatment of ckilamchi.

Primary rapid therapy phase.

1. Stop the introduction of allergens.

The drug that called the shock is to put a jgut above the injection site. Jgutni every 10-15 min.da 3-4 min.ga emptied. A 0.3–1 ml 0.1% adrenaline solution is introduced where the Allergen enters.

2. Adrenaline injection.

Adrenaline is injected under the skin or between the muscles with a solution of 0.3–0.5 ml of 0.1%, then the injection is done twice at an interval of 20 min until a therapeutic effect. If the arterial blood bo-cable drops minimally, adrenaline is injected under the skin or between the muscles. In a sharp violation of breathing and a sharp drop in arterial pressure, a 0.5 ml 1% adrenaline solution is placed under the tongue, and a 3-5 ml 0.01% solution is injected intravenously in a 9 ml isotonic solution.

3. Filling the circulating blood volume.

Intravenously drip Ringer, isotonic sodium chloride solutions 1000 ml, polyglucine 400 ml are administered. If after 2-3 hours there is no result fresh frozen plasma (400 ml), albumin is made.

4. Ensuring airway permeability.

If there is no breathing or there is a sharp impairment, the patient is transferred to endotracheal intubation, artificial na-fas, and 100% oxygen respiration.

5. The use of vasopressor amines.

If the above treatments do not work and arterial hypotension is maintained, intravenous drops of dopmin (dopamine) are administered at a dose of 15-17 mcg/kg/min. 200 mg of the drug is dissolved in 200 ml of 5% Gluco-za or isotonic solution. In the case of persistent hypotension, a 1 ml 0.2% solution of norepinephrine is injected into the vein at a rate of 20-25 drops per minute in 250 ml of isotonic solution.

Stage of secondary therapeutic treatments

Application of glucocorticoids.

Glucocorticoids do not give significant changes in the first 6-12 hours, and therefore it is recommended to use them in elongated cases of ash. But since the weight of the reaction in advance and its stretching is difficult to say, glucocorticoids can be introduced in different periods. In the acute period, 240 ml of Prednisolone

is slowly injected into the vein within 5 min. This dose can be re-administered every 6 hours.

Antihistamine drugs.

Antihistamine drugs do not act quickly and can be a life-saving remedy. Antihistamine should be used after improving the hemodynamics indicators of drugs, since they show a hypotensive effect (especially pipolfen).

Administration between muscle: 1-2 ml 1% dimedrol; 1 ml 2% suprastin; 1 ml 2.5% pipolfen solution 3-4 times a day with or without control of arterial pressure per day: diazolin 0.1 g to 3 times a day.

Dimedrol 0.05 g to 3 times a day; suprastin 0.025 to 3 times a day.

Fencarol 0.05 to 3 times a day.

Antihistamine drugs block N1-histamine receptors, and additional N2-histamine receptor blockers are used if the anaphylaxis simp-roof is not lost or relapsed (ma-salan, simetedin 300 mg to V/in every 6 hours).

Eufillin application.

Eufillin is used to relieve bronchospasm if adrenaline is not effective at administration. Intravenously, 10 ml of 2.4% eufillin is administered in 10 ml of isotonic solution under slow AB control.

Application of sodium bicarbonate.

If hypotension is maintained despite the treatments used, thinking about metabolic acidosis is za-rur, since it reduces the effectiveness of vasopressor agents.

In this case, the acid-base balance is checked, and if the acid-base condition it is corrected by sending 4% sodium bicarbonate to 150-200 ml of intravenous drop-lip.

Application of penicillinase.

Once in ash caused by penicillin, it is necessary to administer 1,000,000 units of peni-tsillinase between muscles in 2 ml of isotonic solution.

In ASh resulting from Bicillin, penicillinase is yubo-administered 3 times per day from 1000,000.

Local treatment is carried out depending on the change in the skin and mucous membranes.

Prevention.

Patients who have undergone ASh as a result of penicillin are treated in the inpatient period of less than 10-12 days. In a milder ASh, the duration of observation may be less. It is necessary that Be-mor stand in the dispensary control in the allergological cabinet after discharge from the hospital. They should be given a PA that has information about allergens (insect bites, preparations) that call Ash. Specific gi-posensitization is recommended for patients after ash caused by insect

bites. Such patients need to constantly carry adrenaline with them in the seasons in which those insects are present.

General and local treatments for skin diseases

In the treatment of skin diseases, a complex (general and local) treatment is used, and this treatment will be aimed at eliminating exogenous factors and endogenous changes, as well as increasing the body's ability to fight the disease.

If the etiology of the disease is known, it is necessary to immediately eliminate the causative agent of the disease. For example, an ointment that has the property of killing Cana leads to a complete cure for scabies. However, often pathogenetic mechanisms also play a large role in the development of dermatoses (changes in the internal organs, nerves, endocrine and immune systems, changes in the reactivity of the body, etc.). In such cases, etiological treatment does not always work, the ijo-biy result is carried out only in cooperation with the etio - and pathogenetic treatment.

The treatment of dermatoses cannot be imagined without a local or skin exposure method, and when thought of more directly, they are also a general treatment method, the reason is that local agents have a general reflector effect on many nerve vessel receptors and the whole organism as a whole. Therefore, the principle of individual approach is very important in the treatment of mahal-Li, especially when choosing the method of treatment and Means.

When prescribing a treatment, it is necessary to identify the risk factors that have led to the disease and determine the method of treatment that is necessary for their correction, keeping them in mind. Masa-lan: identifying allergens in patients with allergodermatosis, in addition to quickly removing these allergens from the body, it is necessary to determine whether there is a hereditary predisposition, identify household and profes-rational sensitizing factors, determine the nature of nutrition and pathology of the gastrointestinal tract (intestinal microbiocinosis, pathology of the hepatobiliary system, viral, parasitic infections, etc.).

In each specific case, with the use of topical corticosteroid agents, the minimum amount required to reverse the acute course is once a day, with the appointment in the morning being effective. Usually, treatment begins with strong corticosteroid agents, can be applied in a short (week) period, and then go to a weaker, less potent one. In order to avoid unpleasant complications, the treatment stage must be short. It is necessary to avoid the appointment and long use of strong corticosteroid agents in breast age and young children, ay-specifically on the face, folds and in the intermediate area. To quickly sanitize various infectious complications, it is necessary to use additional local and general means. In these cases, it is necessary to treat all over the injured areas of the skin, using various corticsteroid agents or in combination with other topical agents.

In the treatment of skin diseases in the face area, elocom and 1% hydrocortisone are used, which does not store fluoride. Unlike synthetic corticosteroids, hydrocortisone does not call teleangiectasia, pe-rioral dermatitis, atrophy, and striae.

In rare cases, injections of corticosteroid suspensions are administered to wound foci-di, which provides a high concentration in chronic foci that are locally administered, resistant to corticosteroid. But, such injections often call skin atrophy and teleangiectasia in the foci. In order to avoid the risk of complications from this variety being observed, a dissolved triamcional acetonide suspension with a sterile saline solution is administered to the foci of the wound-conducting sides to a concentration of 2.5–10 mg/ml. High concentrations of corticosteroids can only be used in the treatment of keloids.

Thus, the treatment of skin disease is conditionally divided into general and local treatment.

Common treatments. In the process of treating dermatoses, it is necessary to pay attention to the methods of eliminating these internal CA-salinity and functional deficiencies, providing for the continuous connection of skin diseases with all internal organs, mainly the central nervous complex. General treatments are divided into several groups. This division is approximate and does not apply only to dermatolo-Gia.

Sedative therapy is a method of treating a violation of the function of the central nervous complex, calming down, and will be of great importance in the pathogenesis of various dermatoses (hypnosis, Electric Sleep, intravenous, under the muscle and the use of drugs for drinking).

Rp.: T-rae Valeriane 30.0 Rp.: Elenium 0.01
D.S. 20-30 drops, D.S. 1 tablet
After meals. 3 times a day.

Desensitizing therapy-used in the loss of various allergic skin reactions (sensitivity, irritability), (calcium-retaining drugs, autogemotherapy, stophilococcal anti-phagini, etc.k.)

Rp: Sol. Natrii thiosulfatis 30% -10.0 Rp.: Sol. Calcii chloridi 10% – 10.0
D.S. 5.0-10.0 per blue vein, slow D.S. to the vein, every day

Antihistamine drugs are widely used drugs that eliminate the biological substance histamine, which is of great importance in the pathogenesis of skin diseases (dimedrol, diazoline, CLA-rhitin, telfast, suprastin, pipolfen, etc.).

The drugs diazoline, Claritin and telfast are distinguished from other antihistamine drugs by the absence of a calming effect.

Rp.: Dimedroli 0,05 Rp.: Tab. Diasolini 0.1
Glucosae 0.2 D.t.d. № 30
M.f. pulv. S. 1 tab 2 mahal
S.1 por. 2 mahal.

Vitaminotherapy. It is widely used in certain dermatoses caused by hypo - and avitaminosis. Most are of Type A, V, S, RR and their types (peridoxalphosphate, carboamide, etc.k.) are more commonly used.

Rp.: Sol. Thiamini bromide 6% - 2.0 Rp.: Sol. Cobalamini 1% – 1.0
D.S. From 2 ml to muscle, D.S. From 1 ml to muscle,
kunora daily

Hormone therapy. Steroid hormones are used with caution, mainly when other general treatments do not give good results, in order to compensate for the lack of natural hormone or to achieve a specific pharmaco-logical effect (desensitizing, immunodepressive, allergic and anti-inflammatory). In some skin diseases, hormones are used in the group of basic treatment drugs (sore throat, run red, etc.).k.). Water-salt with hormone preparations (cortisone, prednisolone, dexamethasone, triamcinolone, etc.), potassium preparation-ri (orotate potassium, panangin, asparkam) and anabolic hormones (retabolil) are definitely given to prevent disruption of fat metabolism.

Steroid hormone drugs are a diverse, strongly affecting, effective drug for the body, and the patient has his own law of use in the treatment process-starting from a high amount before the recovery of the disease, reducing the daily dose little by little, determining and maintaining the minimum dose that the patient needs. The daily amount of the drug depends on the clinical course of the disease, individual effectiveness and patient weight.

Antibiotic therapy. Currently, antibiotics are also widely used in purulent diseases of the skin, dermatoses in which a double infection has occurred, while skin and mucous membranes are also widely used in silica, leprosy, most Zambo-fungal and venereal diseases. If the antibiotic is given for a long time, the patient should be given preparations that prevent albat-ta candidiasis.

Rp.: Streptomycini sulfatis 1.0
S.0.25% li 3ml novocaine
S.30 minutes before meals
dissolve in solution and 1 time in 1 day
Rp.: Rifampicini 0.15 Rp.:
D.t.d.N 50 in caps. Gelat.
drink 2 times a day

Nistatini 250.000 Ed
S.drink 2 capsules 4 times before meals

Chemotherapy is less commonly used for antibiotics. Some drugs have not lost their relevance even now. For example, in the treatment of leprosy – sulfones, in skin tuberculosis – a hydrocide of isonicotinic acid, in red yugiric – delagyl, plaquenil, bioxinol, etc.k.

Physiotherapy-gives good results in chronic, recurrent dermatoses. (UFO-ultra purple Rays, PUVA–therapy, Bukki borderline Rays, diathermia, ozokerite, uqlash, etc.k.)

Spa therapy-different, mainly used in strengthening clinical recuperation, distancing remission intervals. For example, mineral waters and healing mud with sulfur, Rhodium ore, bathing in the seas, etc.k.

Before starting a local treatment, it is necessary to determine the stage of the disease, find out that the cancellation cannot take one or another drug, and only then start.

Local remedies are used for the following purposes:

1. Cleaning and treating skin defects (erosion, wounds, cracks), protecting the skin.
2. Kill the causative agent (bacteria, fungi, parasites).
3. Relief of subjective sensation, baratrafing (aching, itching, pain, irritation).
4. To eliminate the inflammatory process, accelerate the healing of wounds and other injuries.

Types of drugs that are used locally.

When choosing drugs for local treatment (not only the patient young children are assigned agents of low exposure and low concentrations), it is necessary to take into account the area of \ u200b \ u200bThe location of the pathological hearth, its

prevalence, stage of the disease, the nature and depth of the inflammatory process, and how the patient was able to The Aller-GIC reaction and skin irritation depend not only on the account of the active component of the drug, but even on the part that forms its basis.

The types of drugs are estimated according to the degree of penetration into the skin as follows: septic drugs (prisipkas), moisturizers (solutions), rinsing applicators (bol-tushkas), pastes, oils, ointments, creams, compresses, plasters.

1. Sepsis and powder drugs have an anti-surface inflammation effect. They are used against itchy skin and to disinfect the skin. These different means absorb moisture (flood), sweat, reduce the force of friction, cool the skin. Indifferent (zinc oxide, talc, white clay) and dysinfective (iodoform, xeroform, dermatol) spraying drugs are used. According to Za-rurat this sepma is used by adding naphthalene oil, boric acid, menthol, retsorzine, antibiotic, sulfanilamides, etc. Sprinkle pills sprinkle thinly on the wound furnace-di.

2. The drug is used in the case of aqueous solutions of substances – moisturizing - soaking (primochka), wet builder's bandage and warming compresses.

Due to the evaporation of water in aqueous solutions, moisturizing sharply cools the skin and narrows the skin vessels. Therefore, moisturizing is used when acute inflammation of the skin, severe hyperemia, edema and nausea are observed.

“Get wet-get wet!"is known among dermatologists as its basic principle in recommending a topical treatment for a patient.

Soaking with chilled solutions of drugs (1% retsorzin, 2% boric acid, lead water, bite tincture, Burrow liquid, etc.) has an anti-inflammatory effect, narrowing the to-MIRS, reducing nausea and edema, suppressing itching, aching. For soaking, soak a 5-6 layer cloth (bandage, marli) with a solution of ice, lightly squeeze it, and then put it in the oven-di, the cloth is changed every 5-6 minutes and put (soak) 3-4 times a day for 1-2 hours.v

Wet drying ties (Marley is soaked in one of the above-mentioned solution of medicines and placed on the surface of the furnace, on the surface of which a thin cotton mat is tied) are applied similarly to the soaking method, but are replaced after drying, often alternating after 2-3 hours. Slow evaporated liquid, like soaking, cools the skin, only slower. As a result, acute inflammatory-nish symptoms are reduced. This method is used for acute subsurface inflammations accompanied by nausea. The drug, which is part of the moisturizing agent, according to the type of preparations, has a disinfectant, drying effect, and together it gives an anti-inflammatory effect.

3. Shaking-mixtures (bolts). 30% of them will have a powdery substance (zinc oxide, starch, talc, etc.) and 70% will have a water or fatty base (water,

glycerin, alcohol, vegetable oil). They are used in acute inflamed but not hydrated dermatoses, as an anti-inflammatory, skin-cooling and drying agent. Rinsing-according to the composition of the mixtures will be juicy, water-alcoholic and oily. The presence of a large amount of liquid (50-60%) increases the cooling specific-surface. Oily mixtures are prescribed if the patient's skin is dry or if it is necessary to influence the drug for a long time. To accelerate the drying of the skin and increase the healing effect, up to 10% alcohol or ichthyol, sulfur, menthol, anesthetics, etc.are usually added to the boltushka.

Before applying the Bolts, it is thoroughly rinsed and mixed, then a cotton swab is applied to the bi-lan skin. The liquid part of the boltushka quickly evaporates into the air, leaving a layer of ku-day on the skin. Boltushka is applied without a bandage 2-3 times a day, do-ri tools are added, which, as necessary, are actively acting.

4. Pastes are composed of an equal amount of powdery (talc, zinc oxide, etc.) and oily or oily (lanolin, Vaseline, etc.) substances. They have a deeper and more drying effect against inflammation than boltushkas, but are less active compared to ointments.

Pastes are applied to the skin once a day. When applied, it is applied in the direction of the hair and, once every 3 days, the vegetable oil is wiped off the skin using a dampened tapmon. The paste is applied to maintain the elasticity of the skin, various drugs (sulfur, black-oil, retsorzin, etc.) are added as needed.

5. Oils are mainly used to cleanse the injured skin area of packaging, Bran, drug residues, and as an ingredient in the preparation of oil bolts and ointments.

6. Mazes, ointments-be made on the basis of a recipe in formal or apothecary wax-kin. Mazes are the most commonly used type of Medicine in local treatment. Mazes have an oily base (vase-lin, lanolin, refined lard, naphthalene, etc. According to the state of the process on the skin and the required healing effect, they are applied directly to the skin and do not bind. In rare cases, the skin is rubbed with rubbing or rubbed into the Marli and tied. Mazs soften the skin, especially swollen skin. They are applied to the skin covered with thick packaging, lichenized or Bran. Applying mazes to the skin is uncomfortable for patients-while Liks call them, they are more likely to help the drugs absorb into the skin than creams.

7. Creams (which are emulsions in the form of oil in water or water in oil) are considered one of the local remedies that are widely used in dermatology. It is convenient to apply them, there will be no feeling of increased skin weight when applied to the skin.

8. Compresses are mainly used against skin inflammation and for the purpose of teasing the absorption of infiltrates. They have a deeper effect than mazes. For a compress (3% boric acid, lead water, 1% Burrow liquid), one of the medicinal

solutions is soaked and a bi-rose compressed cloth is placed in a furnace, over which it is tied by placing paper or Clayon that does not absorb water. It should also be borne in mind that warm moisture greatly softens the epidermis, which creates favorable conditions for the development of a secondary infection.

9. Healing soaps (the combination of oil with alkali) include the following types of soaps: sulfur-containing, ichthyol-containing, black-oiled, sulsen-containing, blue soap, etc. It should be borne in mind that in most skin diseases (for example: piodermitis), during the treatment period, it is recommended to interact with water, especially washing with soap. In such cases, it is enough to wipe the areas of healthy skin with weak alcohol solutions.

10. Aerosols have become popular in recent years for their convenience, sterility and high efficiency, and are widely used. Aerosols do not store fat. They are sprayed on the surface of the injured sides of the skin, while mixtures of small drug particles in air or gas. Aerosols are indispensable for patients with increased sensitivity to lanolin, Vaseline, and those who cannot lift maz, creams. In addition to antibiotics in the composition, corticosteroid-preserving aero-halls are very popular, they have an anti-itch, anti-inflammatory, anti-allergic and bacteriostatic taste.

Nowadays, official, that is, ready-to-use corticosteroid drugs are becoming more common and popular among the staff of Medicine, the people.

We cite retsep-Tura, some drugs that are widely used in the local treatment of skin diseases:

Shaking mixtures (bolts).
Rp.: Zinci oxydati
Talci veneti
Amyli tritici aa 10,0
Ol. Persicorum ad 100,0
M.D.S. chayqatib ishlatiladi

Pastalar

Rp.: Zinci oxydati
Amyli tritici
Vazelini
Lanolini aa 10,0
M.f. pastae
D.S. malham

Rp.: Ac. Salicylici 2,0
Zinci oxydati
Amyli tritici aa 25,0
Vazelini 50,0
M.f. pastae
D.S. malham

Rp.: Hydrargyri praecipitati albi 2% – 30,0
M.D.S.malham
M.f. ung.

Rp.:Ac.Salicylici 1,0
Ac. Benzoici 2,0
Vazelini ad 30,0
S.malham

Rp: Lanolini
Vaselini
Aq.destilli aa 20,0
M.D.S. krem

Plastirlar
Rp: Epilini 4,0
Aq.destilli 15,0
Lanolini 22,0
Cerae flovea 5,0
Emplostri plumbi 54,0
M.D.S.

Local drugs are divided into the following groups, depending on their composition: keratoltic, keratoplastic, antiparasitic, anti-itchy, used in depigmented spots.

Keratolytic drugs separate the mucosal layer from the skin level, more for this purpose salicylic acid, lactic acid, resortsin, etc.are used. These have the ability to kera-tholitic and powdery drop when more than 2% is prepared, and lower amounts have keratoplastic contribution.

Rp: Ac. Salicylici 12,0
Ac. Lactici 6,0
Vaselini ad 100,0
M.f. ung.
D.S.

Rp: Resorcini 3,0
Vaselini 30,0
M.f. ung.
D.S. malham

Keratoplastic drugs are low-content (concentrated) and are used to renew the mucosal layer, anti-inflammatory, disinfect and leave itching.

In dermatology, resin (derived from birch or pine, mojjevikali, etc.), sulfur, naphthalene, ichthyol, etc. are often used.

Rp : Ol .Cadini
Sulfurus precipitate aa 5,0
Vaselini 50,0
M.f. ung
D.S. malham

Rp: Ichtioli 3,0
Vaselini 30,0
M.f. ung
D.S. malham

Antiparasitic drugs: sulfur, mercury, iodine and others work in the loss of parasites-wished.

Medications used against itching – menthol, dimedrol, anesthetics, table vinegar, alcohol, citric acid-also include.

Rp: Novocaini 0,3
Anestesini 2,0
Lanolini
Vaselini
Aq. destill aa 10,0
M.f. ung
D.S. malham

The acting drugs are used in chronic inflammatory diseases of the skin, they affect the receptors of the skin and the central nervous complex.

Rp: Jodi puri 0.1
Kalij jodidum 1,0
Parafini 30,0
Chloroforum 70,0
M.D.S. mahalliy

Drugs used against pigmented spots are used against limited (small amounts) pig-hinge spots on the skin, in which a powdery drop is achieved.

Rp: Perhydroli 10,0
Sp. Vini rec 96%- 100,0
M.D.S. mahalliy

Sunscreen preparations include 5-10% salol, tannin, paraaminobenzoic acid ointments.

In some cases, more cryotherapy, eletrocoa-flowering, diathermocoagulation and laser light are used in place of decaying (destructive effect), incinerators(lyapis, three chloruxus kis-lotasi, lactic acid, manganese crystals, podophylline).

Local applicator
CORTICOSTEROID DRUGS

Corticosteroids are the most effective means in the local treatment of dermatoses, with an anti-inflammatory effect. Topical corticosteroid treatment is undoubtedly sa-marali and safe if the drug is used for correct and specific purposes, following the rules and principles of their use.

In some skin diseases (acne, some fungal and purulent diseases), a topical treatment with corticosteroids can lead to exacerbation of the disease. That is why it is advisable to use topical glucocorticosteroid drugs after the diagnosis is made clear.

Topical corticosteroid drugs are prescribed only for the purpose of treatment, and not for the purpose of prophylaxis of dermatoses. They are used in the form of creams, solutions, aerosols and plasters. The complexes that we present are also popular in nature.

Diagnosis of skin diseases and
General means of their treatment

General diagnostic methods. The diagnosis of skin diseases is based on the data of Anamnesis, the main symptoms of the disease (examination, laboratory) performed at the azmoishgah and on-site examination-SHS. Clearly assembled Anamnesis, methods for detecting rashes-help to determine the etiological and pathogenesis of the disease.

1. Anamnesis:

a) approximate short Anamnesis-will be a tool for detecting dermatoses;

b) accurate Anamnesis – it will be of great importance to accurately diagnose and determine the pathogenesis of the disease.

2. Examination of the skin and mucous membranes:

a) skin-fat separation;

b) general appearance, color of the skin;

C) stretch and elasticity;

g) dermographism (Red, White, different);

d) the structure of the subcutaneous fat tablet;

ye) pigmentation(discoloration;

j) visible mucous membranes;

The location of the rash (localization), bounded, multiple, diffuse (diffuse), (on the entire skin level and mucous membranes), symmetrical, asymmetrical, can be visible (appendix No. IV-V).

3.Common contributions of rashes:

a) inflamed;

(B) not yalligated.

4.Primary morphological elements:

a) large-small;

b) flat, uneven;

v) appearance (semi-round, conical, etc.);

g) color;

d) boundaries (clear, uncertain);

ye) top (smooth, oval, uneven, pole thrower, etc.);

j) peripheral growth(edge-to-edge distribution;

z) consistency: (soft – hardness) – harder, harder-elastic, soft.

5. Full characteristic of secondary rashes.

(a) chin or false polymorphic, monomorphous;

b) the location of the elements (added, separately);

v) Group (true, false).

6. Assessment of the general condition of the patient:

a) subjective complaints;

b) objective signs.

7. Examination of internal organs, nerves and other organs.

8. Conducting the necessary examinations and tests: (allergic tests, Minor fracture in leprosy, Nikolsky's sign in a purulent wound, tuberculosis runner – diascopia, a trio of Auspitts in psoriasis, etc.) (appendix VI).

9. Conduct General Azmoishgah inspections.

In the current era of health care, the general practitioner is gradually gaining a key place. It is necessary for a general practitioner to confront and treat various diseases in his practice by diagnosing them. Our goal to create this book is to enrich the knowledge of students and general practitioners with information about common skin venereal diseases and skin changes in internal organs with disease.

Doctors of all specialties: pediatrician, allergologist, therapist, surgeon, general practitioner and secondary medical officer during their labor activity, patients collide with pathological strangers on their skin. Therefore, they must also know the basics of Dermatology. The ja-pleasure of the skin can be inadvertently “found” unexpectedly or determined due to the main complaint of the patient. Since cardiologists “read” the electrocardiogram, it is necessary that each doctor

accurately and correctly “read” the dermatology “Alphabet”, that is, see rashes, refer or treat the patient to a dermatologist himself.

The main weapon of Dermatologists is their eyes. Dermatological diagnosis is based mainly on the examination of the skin, mucous membranes. Skin changes are detected through the unarmed eye, it is only necessary to know how to see and what to look for.

A correctly established diagnosis is the key to an effective treatment. We believe that through a prepared, miraculous booklet, it makes it easier for doctors of different fields to diagnose skin diseases that occur in their practice. Skin diseases are not easy to diagnose, the main thing is to be able to observe skin rashes, to be able to distinguish one from the other. That is why this book describes the short clinical manifestations of diseases, the principles of diagnosis and treatment using a large number of tables. We recommend that you carefully examine the rashes in each disease and compare them with each other, paying attention to the diagnostic signs presented in the text and tables. We think that from this book a practitioner or medical student will receive a lot of new, za-rur information, while specialist dermatologists will enrich their knowledge with new information.

In some cases, it is thought that doctors, despite all the conditions and conditions, in many cases do not pay enough attention to the study of skin diseases. In cases where the patient does not complain to the skin, the rash is not paid attention to a pronounced level. Such” unnamed“,” unknown " diseases are treated for months, in some cases with corticosteroids, antibiotics or cytostatics. As a result, there are cases of cosmetic defects, exacerbations of patients suffering for a long time, dispersion of the process, irreversible changes, the main thing is to diagnose internal severe hastans with an unforgivable delay.

Skin changes, which at first glance seem harmless, can be the only sign of severe diseases. For example, a bruise stain is a developing melanoma, and a small wound can be the initial waist – gout of the poison. These different skin changes can and must be detected in various preventive examinations. Otherwise it may be that time has passed for the doctor's treatment.

The patient is at an outpatient reception or in a hospital setting, regardless of the complaint the condition of his skin and mucous membranes must be examined by a doctor. Paying attention to any changes, determining what elements the rashes are composed of, a biopsy should be taken if necessary.

The member, whose skin condition is considered the most convenient to check, is also a source of information, which at one time has many necessary significance-Yat, only this information should not be thawed to take.

EPIDEMIOLOGY

According to the World Health Organization (WHO), skin and subcutaneous tissue diseases are among the most prevalent non-communicable diseases worldwide. It is estimated that **more than 1.9 billion people globally** suffer from skin conditions at any given time, accounting for approximately **20–30% of the world's population**. Chronic skin diseases constitute a substantial proportion of this burden due to their long-term course, frequent relapses, and resistance to therapy.

WHO data indicate that skin diseases rank among the **top ten causes of non-fatal disease burden** globally, measured in years lived with disability (YLDs). In low- and middle-income countries, dermatological diseases are among the **leading causes of outpatient medical visits**, reflecting their widespread distribution and socioeconomic impact.

Atopic dermatitis affects up to **15–20% of children** and **2–10% of adults** worldwide, with increasing prevalence in urbanized and industrialized regions. Psoriasis affects approximately **2–3% of the global population**, corresponding to more than **125 million people** worldwide. The disease occurs in all ethnic groups and geographical regions, with comparable prevalence rates reported in Europe, North America, and parts of Asia.

Eczema and chronic contact dermatitis are among the most common inflammatory dermatoses, particularly in occupational settings. According to WHO and International Labour Organization (ILO) reports, **occupational skin diseases account for up to 40% of all work-related illnesses**, especially in industries involving chemical exposure, healthcare, construction, agriculture, and cleaning services.

Seborrheic dermatitis affects approximately **1–5% of the general population**, with higher prevalence observed in infants, adolescents, and individuals with neurological disorders or immunodeficiency. Lichen planus has a global prevalence ranging from **0.2% to 1%**, with a higher incidence reported among middle-aged adults and a female predominance in mucocutaneous forms.

WHO epidemiological studies emphasize the strong association between chronic skin diseases and comorbid conditions. Up to **30–40% of patients** with chronic dermatoses have concomitant systemic diseases, including endocrine disorders, metabolic syndrome, cardiovascular diseases, gastrointestinal pathology, and mental health disorders. Psoriasis, in particular, is recognized by WHO as a **systemic inflammatory disease** associated with increased cardiovascular risk.

Geographical and climatic factors significantly influence the epidemiology of chronic skin diseases. Higher prevalence rates are reported in regions with extreme climatic conditions, high humidity, or low ultraviolet exposure. Seasonal exacerbations are common, with many chronic dermatoses worsening during colder months due to reduced sunlight, low humidity, and increased infectious triggers.

Socioeconomic determinants play a crucial role in disease distribution. WHO reports indicate that populations with limited access to healthcare, inadequate sanitation, overcrowded living conditions, and low health literacy experience a disproportionately higher burden of chronic skin diseases. Children, elderly individuals, and socially vulnerable populations are particularly affected.

In conclusion, WHO recognizes chronic skin diseases as a major global health challenge with significant medical, psychological, and economic consequences. The high prevalence, chronic course, and frequent comorbidities underscore the importance of early diagnosis, preventive strategies, patient education, and integrated long-term management to reduce disease burden and improve quality of life.

Table. Epidemiological Characteristics of Major Chronic Skin Diseases

Disease	ICD-11 Code	Global Prevalence	Age Characteristics	Key Epidemiological Features
Psoriasis	EA90	2–3% of the population	Peaks at 15–35 and 50–60 years	Systemic inflammatory disease; associated with

Disease	ICD-11 Code	Global Prevalence	Age Characteristics	Key Epidemiological Features
		(≈125 million people)		cardiovascular disease, metabolic syndrome, and psoriatic arthritis
Atopic Dermatitis	EB20	15–20% of children, 2–10% of adults	Mostly childhood onset	Strong association with asthma, allergic rhinitis; increasing prevalence in urban areas
Chronic Eczema (Contact Dermatitis)	EB10–EB12	5–10% lifetime prevalence	All age groups	High prevalence in occupational risk groups; frequent relapses
Seborrheic Dermatitis	EA80	1–5% of population	Infants, adolescents, adults	More common in immunodeficiency and neurological disorders
Lichen Planus	EA86	0.2–1%	Middle-aged adults	More frequent in females; mucocutaneous involvement common

PREVENTION OF CHRONIC SKIN DISEASES

Prevention of chronic skin diseases is a complex and multifactorial process aimed at reducing disease incidence, preventing exacerbations, and minimizing complications. Given the chronic and relapsing nature of many dermatoses, preventive measures play a crucial role in dermatological practice and public health.

Primary prevention focuses on eliminating or reducing exposure to etiological and risk factors that contribute to the development of chronic skin diseases. These include environmental irritants, chemical agents, occupational hazards, physical factors, allergens, and infectious triggers. Proper skin hygiene, rational use of cosmetic and hygienic products, avoidance of aggressive detergents, and protection of the skin from excessive ultraviolet radiation, extreme temperatures, and mechanical trauma are essential preventive measures.

Occupational prevention is particularly important in individuals exposed to chemical substances, detergents, solvents, metals, and repeated mechanical irritation. The use of personal protective equipment, adherence to occupational safety standards, regular dermatological screening, and timely elimination of harmful exposures significantly reduce the risk of occupational dermatoses, especially chronic eczema and contact dermatitis.

Secondary prevention is aimed at early detection and timely treatment of skin diseases to prevent chronicity and complications. Regular medical examinations, early diagnosis of inflammatory and allergic skin conditions, identification of provoking factors, and prompt initiation of appropriate therapy are key components of secondary prevention. Patients with a family history of chronic dermatoses or allergic diseases require special attention and dynamic observation.

An important role in the prevention of chronic skin diseases belongs to the management of comorbid conditions. Endocrine disorders, metabolic diseases, gastrointestinal pathology, chronic infections, and neuropsychiatric conditions may significantly influence the course of dermatoses. Timely diagnosis and treatment of

these conditions contribute to disease stabilization and reduce the frequency of exacerbations.

Lifestyle modification is an essential element of preventive strategies. A balanced diet rich in vitamins, trace elements, and essential fatty acids, adequate hydration, regular physical activity, normalization of sleep patterns, and stress management positively affect skin barrier function and immune regulation. Avoidance of harmful habits, such as smoking and excessive alcohol consumption, is also recommended.

Psychological prevention is of particular importance, as emotional stress and neurogenic factors play a significant role in the onset and exacerbation of many chronic skin diseases. Patient education, psychological support, stress-reduction techniques, and, when necessary, consultation with mental health professionals contribute to improved disease control and quality of life.

Tertiary prevention is aimed at preventing disease progression, relapses, and disability in patients with established chronic dermatoses. This includes long-term maintenance therapy, adherence to treatment recommendations, regular follow-up visits, patient self-monitoring, and education regarding early signs of exacerbation.

In conclusion, prevention of chronic skin diseases requires an integrated approach that combines medical, hygienic, occupational, lifestyle, and psychosocial measures. Effective preventive strategies not only reduce disease burden but also improve long-term outcomes and quality of life in patients with chronic dermatoses.

Table. Levels of Prevention of Chronic Skin Diseases

Level of Prevention	Main Aim	Key Measures	Target Population
Primary Prevention	Prevent disease onset	Elimination of risk factors, skin protection, hygiene education, occupational	Healthy individuals, risk groups

Level of Prevention	Main Aim	Key Measures	Target Population
		safety, avoidance of allergens and irritants	
Secondary Prevention	Early detection and prevention of chronicity	Early diagnosis, timely treatment, identification and elimination of triggers, monitoring high-risk patients	Patients with early or mild disease
Tertiary Prevention	Prevent relapses, complications, and disability	Maintenance therapy, long-term follow-up, patient education, rehabilitation	Patients with established chronic dermatoses

Test assignments

№1
Proliferative histomorphological changes include Which of those below:
hyperkeratosis
Spongiosis
acantholysis
cariorexis

№2
In which part of the skin, sweat glands are not observed:
lab's red hoshia
foot palm
armpit
hand palm

№3
In which part of the skin the sebaceous glands are not observed:
hand and foot palm
nasal lip triangle
breast
hairy part of the head

№4
Which element is a type of pustula:
flictena
excoriation
vesicle
Teleangiectasia

№5
Indicate the type of stain:
Roseola
acne
rupiah
ektima

№6

6. Which morphological element has an ephemeral property:
Urtica
pustula
Papula
tuberculosis

№7
Where are the" tizone sebaceous glands " located:
inner angle of the head and body of the penis
red lip hoshia
on the skin of the mammary gland
perianal Soha

№8
The" meibomium " sebaceous glands are located:
at the edge of the eyelids
on the skin of the ear Supras
in the skin of the genital Sox
red lip hoshia

№9
In which Sox of the skin the sebaceous glands open directly to the epidermis:
penis head part
nose skin
hairy cyst of the head
breast skin

№10
Acantholysis (acantholysis) is:
violation of inter-cellular connection of the thoracic floor
violation of the process of Decay
increased division of Thoracic floor cells
granular floor thickening

№11
Paraceratosis is an abnormal keratinization of epidermocytes, ... dystrophy-reliever:
granular floor
basal floor

thoracic floor
suction floor

№12
Granulez is the name of:
granular bracket thickening
muguz floor thickening
granular floor thinning
xujayra ichi shishi

№13
Anaplasia is the name of:
epithelocytic nucleus atypia in tumors
epithelocytic nucleus compression
cell cytoplasm granularity
violation of inter-cellular connection of the thoracic floor

№14
Spongiosis is the name of:
Intercellular edema on the floor of Malpigi
Muguz floor intercellular edema
Caseose breakdown of tissues
Vacuum degeneration on the floor of Malpigi

№15
Acanthosis is not typical:
sclerodermia
neurodermitis
Red flat ironclad
Psoriasis

№16
In which dermatoses acantholysis is observed:
bruise wound
contact dermatitis
ektima
Eczema

№17

In which dermatoses granules are observed:
red flat ironclad
common ironclad
Turniol
normal blister dermatosis

№18
Does not enter the functions of the skin:
dressing toxins
Absorption
Separation
thermoregulation

№19
The type of angular stomatitis is characterized by:
streptodermia
Candidiasis
Purulent wound
Staphylodermia

№20
Cancerous piodermia often begins:
from erosion
From pustula
From the dog
from the bubble

1. Lichenoid parapsoriasis is described by all above, except.
) scales similar to kpak, with small lichenoid brown red papules
brown pigmentation with primary telangiectasia
) small purpura
) severe itching
2. In the pathogenesis of Rosacea, the following factors have significant tension:
angioneurotic disorders
) insolation
) seborrheic dermatitis
) rhinophymes
xanthelasma

3. Seborrhea contributed to the development of all the diseases listed below

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) rosacea

) seborrheic dermatitis

) rhinophymes

xanthelasma

4. This trichodonosis

points. uz) knots in the hair shaft

) short hair

) spindle-shaped hair

) combed hair

5. With arthropathic psoriasis, it is recommended to buy all of the above

) penicillin and prednisolone

) drugs against nosteroid yaligation

) oxyferricorbon

D) detox tools

antioxidants)

6. Typical rash elements of red-like planus have the following characteristics, krome

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depressions in the center of the papula

) waxy shine

) Wickham's net on the surface of the papules

with primary localization on the face

7. Plaque exhibitions parapsoriasis is characterized by the following symptoms, in addition:

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) using anicized grotesque

preferential localization in the trunk and lower games

) lack of interest

) infiltration and general condition disorder

8. Hypothyroidism develops

) common myxedema

) nodular myxedema

) pretibial myxedema

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) none of the above

9. Telangiectatic form observed:

in childhood)

) only on Adult Subjects

) since birth

) during puberty

at the same frequency at any age

10. Includes historical changes to the people's granuloma:

) focal necrobiosis of collagen in the middle dermis

A member of the Ozlidep faction in the Ohio House of Representatives, he is a member of the Ozlidep faction in the Ohio House of Representatives.more

production) the upper part of the dermis is the expansion of the vessels, swelling of the endothelium, opening of the vessel wall, perivascular lymphoid and histiocytic infiltrates, possible types of erythrocytes

) breakdown and swelling of collagen in the middle part of the dermis

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11. The cause of dermatoses that develop in diabetes:

A'zrtxb) A'zrtxb) A'zrtxb) A'zrtxb

) metabolic disorder

microangiopathy).

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12. Describes the lobe of the multiple auricle:

) with psoriasis

with seborrheic dermatitis

) with mycotic eczema

) with streptoderma

with contact dermatitis

13. Non-budgetary pension fund under the Ministry of Finance of the Republic of Uzbekistan, extra-budgetary pension fund under the Ministry of Finance of the Republic of Uzbekistan, extra-budgetary pension fund under the Ministry of Finance of the Republic of Uzbekistan:

) age of patients

sharp development and rapid development of common nodule and tumor shaking

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atypical localization in the head, boyin, trunk, mouth

compounds with visceral lesions and increased lime

14. Hyperelastic skin (Ehlers - Danlos syndrome) is characterized by all of the above, except:

manifested from early childhood

Ozrtxb) gradually increases skin weakness with changes in hematomas, treatment and environmental innovations.

and the excessive appearance of the skin on the face

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15. Normally restricted neurodermatitis has 3 zones:

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) erythematosus

) average

) boundary line

) peripheral

16. Among the itching, the most turning course and skin names appear:

) strofulus

) Nodular itch of the Hebra

) pregnancy prurigo

) sunny prurigo

prurigo acute in adults

17. Atopic cheilite may be added:

self-harm

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bacterial infection

) candida infection

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18. Vidal simple chronic lichen:

) restricted-neurodermatitis

) like a bullet

) amyloid-like

) atopic dermatitis

19. In which dermatoses is the morphological element-seropapula observed?

) strofulus

) adult Horn

acantholytic dermatosis

20. Solar urticaria which is often diagnosed with impaired metabolism? Information about the Council of the Senate of the Republic of Uzbekistan
) mineral
) porphyrin
) carbohydrate
) lipid

ANSWERS TO TESTS

1. G
2. D
3. D
4. And
5. And
6. D
7. D
8. G
9. B
10. D
11. D
12. D
13. In
14. D
15. A, B, D
16. B
17. D
18. A, B
19. In
20. In

SITUATIONAL TASK

1) A 30-year-old patient complains of the appearance of rashes accompanied by severe itching. He considers himself sick for 10 days. The cause of the appearance of rashes cannot be determined. He took diphenhydramine, but had no effect from the treatment. Objectively: small shiny papular elements of pink-red

color on the skin of the flexor surfaces of the forearms and calves are detected in calves of cyanotic shade. The Shape of the papules is polygonal, with an umbilical depression in the center.

1. You have a suspicious diagnosis?
2. What diseases should be diagnosed differentially?
3. Principles of treatment?

2) a 20-year-old woman came to the doctor complaining of rashes on the front surface of her wrists, accompanied by itching. Sick for 2 weeks. The appearance of rashes is associated with strong emotional stress. The rashes are represented by a large number of polygonal papules of purple-blue color with a waxy shine, they form small plates in places, the surface of which resembles a stone bridge, in the center of the papules there is an umbilical cavity.

1. What disease can you think of?
2. What diagnostic phenomenon can confirm a Dubious Diagnosis?
3. How to prescribe treatment to the patient?

3) Patients B, 25 years old. Complains of the appearance of a rash accompanied by moderate itching. I was sick for 3 weeks, after hypothermia I noticed one spot on the skin of my chest. After washing in the shower a few days later, an abundant rash appeared on other areas of the skin. Lens: on the skin of the front wall of the chest, a round red dot measuring 2x4 CM, the bark has appeared. On the skin of the trunk, neck, upper limbs, thighs, a multi-rash is detected in the form of oval and irregularly shaped spots of pink color up to 2 cm in diameter. The spots are located along the line of tension of the skin, and a crumpled paper-like crust appears in the center of the spots.

1. You have a suspicious diagnosis?
2. What diseases should be diagnosed differentially?
3. Prescribe treatment.

4) A 27-year-old patient complains of itchy rashes on his body, on the front surface of the wrists and on the bottom of the legs. The rashes are represented by flat, small shiny papules with a polygonal outline of a blue-pink color, with an umbilical depression in the center of the papules.

1. What is your diagnosis?
2. To prescribe treatment to the patient.

5) During the examination in a 12-year-old child, many reddish papules with a clear border were found on the extensor surface of the upper limbs, scalp and body, the entire surface of which is covered with scales and bark . No itching.

1. Your Dubious Diagnosis.

2. What additional research is needed to make a final diagnosis?

3. Conduct differential diagnostics.

4. Prescribe general and local treatment.

6) A 25-year-old woman applied for admission with a complaint of redness, swelling and the appearance of rashes on the skin of the hands. 2 months sick. She works as a nurse in a treatment room where she has to do various injections. On the next vacation, the rashes completely disappeared, and after 1-2 weeks after returning to work, they appeared again.

1. What disease can be predicted?

2. Tactics of treating this disease?

7) The 35-year-old patient number 7 came to the doctor with a complaint of rash and itching on the skin of the hands. It is known from Anamnesis that similar rashes appeared several times during the year after washing with the washing powder "Lotus". On examination: pronounced erythema, edema, numerous nodules and blisters on the skin of the back of the hand, erosion, wet.

1. What disease can you think of?
2. How to prescribe treatment to the patient?
3. How to prevent the appearance of a similar skin lesion in the future?

8) A 45-year-old man, a cement plant worker, complained of injuries sustained by moderate itching on the skin of his hands. For the first time, the disease appeared about an hour after the start of work. He notes that his condition improved during the holidays. Objectively: places of congestive hyperemia and infiltration in the hands and wrists, against the background of which there are many small vesicular, serous shells, deep cracks in the hands.

1. What disease can be predicted?

2. How to conduct additional survey methods?

3. The main principle of prevention of acute diseases.

9) The 40-year-old patient No. 9 came with complaints accompanied by a 40-year-old patient, impotence, rash on the entire skin, itching. The appearance of the disease is associated with the intake of paracetamol for headaches. Skin rashes appeared 2 days ago and are gradually increasing in both volume and volume. On examination: on the skin of the body and limbs there are many erythematous spots of different sizes, with unclear boundaries, connective in places. Body temperature 37.8 °C.

1. Estimated diagnosis?

2. Principles of treatment?

3. Prevention of this disease?

10) A 30-year-old woman working as confectioner No. 10 complained of a wound accompanied by Burns and itching between the III and IV fingers of the

right hand. Objectively: without infiltration, irregularly drawn erosion is detected, bounded by the White Collar of the swollen cornea.

1. What disease can you think of?

2. What laboratory research should be carried out?

3. Make a treatment plan.

11) A 20-year-old woman came to the doctor complaining about the appearance of rashes on the front surface of her wrists, accompanied by itching. Sick for 2 weeks. The appearance of the rash is associated with strong emotional stress. The rashes are represented by a large number of polygonal papules of purple-blue color with a waxy shine, they form small plates in places, the surface of which resembles a stone bridge, in the center of the papules there is an umbilical cavity.

1. What disease can you think of?

2. What diagnostic phenomenon can confirm a Dubious Diagnosis?

12. How to prescribe treatment to the patient?

12) Patients C, 25 years old. Complains of the appearance of a rash accompanied by moderate itching. I was sick for 3 weeks, after hypothermia I noticed one spot on the skin of my chest. After washing in the shower a few days later, an abundant rash appeared on other areas of the skin. Lens: on the skin of the front wall of the chest, a round red dot measuring 2x4 CM, the bark has appeared. On the skin of the trunk, neck, upper limbs, thighs, a multi-rash is detected in the form of oval and irregularly shaped spots of pink color up to 2 cm in diameter. The spots are located along the line of tension of the skin, and a crumpled paper-like crust appears in the center of the spots.

1. You have a suspicious diagnosis?

2. What diseases should be diagnosed differentially?

3. Prescribe treatment.

13) At the time of examination in a 9-year-old child, many reddish papules with a clear border were found on the extensor surface of the upper limbs, scalp and body, the entire surface of which is covered with scales and bark . No itching.

1. Your Dubious Diagnosis.

2. What additional research is needed to make a final diagnosis?

3. Conduct differential diagnostics.

4. Prescribe general and local treatment.

14) A 19-year-old woman applied for admission with a complaint of redness, swelling and the appearance of rashes on the skin of the hands. 2 months sick. She works as a nurse in a treatment room where she has to do various injections. On the next vacation, the rashes completely disappeared, and after 1-2 weeks after returning to work, they appeared again.

1. What disease can be predicted?
2. Tactics of treating this disease?

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