become mattery and acquire yellowish-grey or greenish colouring. The matter dries up into the form of melichrous crust. After the crust drops out, the erosion appears, surrounded with delaminated horny masses of epidermis. Subjectively, the process is accompanied by light itching.

Impetigo vulgaris is mainly localized on the skin of face at the sites of natural orifices, such as nostrils, mouth, palpebral fissures, in women it can sometimes be localized on the hairy part of the head as well. The process seldom causes inflammation of regional lymph nodes.

The evolution of disease lasts 10 to 14 days, after that a temporary hyper pigmentation remains on the skin at the lesion sites. The possible are the lesions of mucous membranes of nostrils.

The treatment is predominantly topical and includes the application of 1-2% boric-tar ointment or naphthalani unguentum. The healthy skin areas are regularly wiped around the foci with 1% salicylic alcohol. Ointments with antibiotics are applied (mupirocin).

Chronic ulcerative pyoderma *(pyodermia chronica ulcerosa)* gradually transforms into ulcerative vegetating form. It is caused by the mixed strepto-staphylococcal infection. For the development of chronic ulcerative pyoderma the essential is, on the one hand, the reducing of pathogenicity of the disease agents and immunological response of the organism, and on the other hand, the weakening of the resistance of the microorganism, that leads to prolonged soft course of the disease. Its manifestations are localized exceptionally on the lower legs, and very seldom on the lower part of femora. In the setting of slightly edematous areas of hyperemia the phlyctenas appear, rapidly covered with thick crusts, beneath which the ulcers with callous undermined edges occur. After the dropping of the crusts off, the bottom of the ulcers becomes naked, it is covered with necrotic masses and soft grey granulations with significant amount of purulent and serous-purulent discharge. The process spreads over periphery, covering large sites of the skin. Around the ulcers there are pustular multiple small erosions. There is painfulness at palpation.

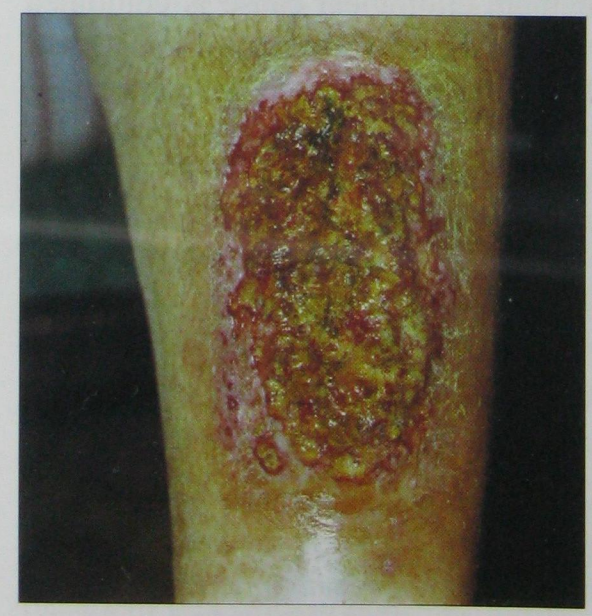

Fig. 4.12. Chronic ulcerative vegetating pyoderma of lower leg.

Chronic ulcerative vegetating pyoderma *(pyodermia chronica ulcero-vegetans)* is characterized by the appearance over the ulcer periphery of vegetations, which are verrucous growths, which can appear over the entire surface of the lesion. The bottom of the ulcers is bleeding. Vegetations can

enlarge along the periphery as well; in this case the process acquires serpiginous character. When pressing at the sites of lesion, the drops of dense matter are released from small fistular openings. The disease lasts for months or years and ends with formation of uneven hyperpigmented scars with small islets of unaltered skin.

It is necessary to distinguish chronic ulcerative vegetating pyoderma from serpiginous nodular syphilide and verrucous form of skin tuberculosis that requires additional examination of the patient.

When there are ulcerative purulent processes, the antibiotic therapy is appointed. In case of intense mattery discharges, the lotions with 2% solution of rivanol, solution of microcide (at a dilution 1:1) are appointed, and in the period of remission 2% gentian violet ointment.

Pyoderma, of the type of ecthyma vulgaris, sometimes is located independently on the genital organs, resembling syphilitic chancre, hence the name of the disease of chancriform pyoderma *(pyodermia shancriformis)*. It is very seldom, that chancriform pyoderma is localized on the face, lips and eyelids. The disease is caused by staphylococcus aureus. The disease is typical for untidy persons, who do not look after hygiene of the body, as well as the subjects with the presence of narrow preputial ring, when smegma accumulation causes maceration of the skin of the penis with further suppuration.

Clinically, chancriform pyoderma manifests as the appearance of clearly limited superficial ulcer of round to oval form with overlapped edges, located on the inflammatory infiltrate the size up to 2 cm. The bottom of the ulcer is fleshy red, sometimes covered with necrotic masses or purulent releases. It is painful at palpation. As a rule, with chancriform pyoderma there is regional lymphadenitis the size from kernel of a cherry to a nut, painful on palpation. The disease lasts from one to three months and ends with the cicatrization.

In all cases chancriform pyoderma should be differentiated with syphilitic solid chancre. In solid chancre the subjective sensations (pain, burning, itching) are absent, the infiltrate is of dense-elastic, but not soft consistency, does not overlap ulcer, the acute inflammatory phenomena are absent. As a rule, there are no purulent discharges. Regional lymphadenitis is also painless on palpation, of dense-elastic consistency. In some cases the clinical picture of chancriform pyoderma reminds syphilitic solid chancre so much, that the diagnosis is possible to be set only on grounds of the results of multiple tests for Treponema pallidum and data of serological reactions.

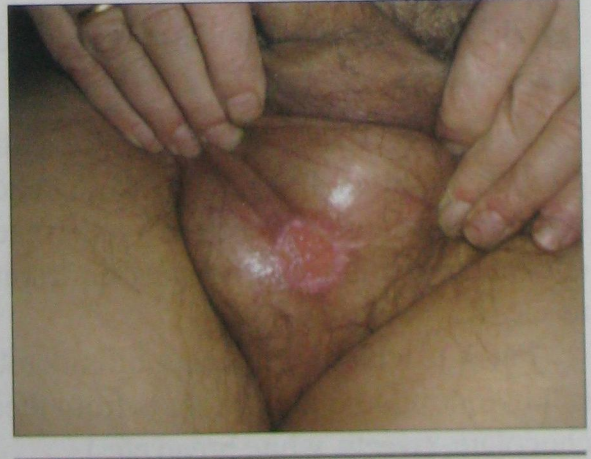

Fig. 4.13. Chancriform pyoderma.

For the treatment of chancriform pyoderma the bathes with a weak

solution of potassium permanganate, lotions with a physiological solution of sodium chloride or 2% solution of boric acid are applied to the full cicatrization. At the stage of differentiation with syphilitic solid chancre no antibiotics should be applied both topical, and for general therapy.

The group of chronic mixed pyoderma includes also pyogenic granuloma or ***botryomycoma*** *(botriomycoma)*, which is benign fungiform tumor-like growth, caused by Staphylococcus aureus. Botrycoms is most frequently localized on the skin of vermilion border, nose, ears, on the fingers and toes. Its appearance is preceded by the injuries, cuts, pricks, by means of which pyococci penetrate into the skin. Clinically, botryomycoma manifests as small-lobular tumor on the peduncle the size of a pea to a hazelnut, of round form and soft consistency. Botryomycoma has got intense red colouring and a large number of vessels, which are easily injured and bleed. Botryomycoma with time can be partially necrotized or form an ulcer with discharge of insignificant amount of seropurulent liquid.

Botryomycoma can exist without treatment for a long time, not disappearing by itself. The treatment consists in surgical (or with electrocoagulation) removal of the tumor. Sometimes botryomycoma can recur, so it is recommended to remove not only tumor itself, but its base as well, at the same time applying antibiotic therapy up to the absolute regress of the disease.

Pyoallergids are secondary allergic diseases, which are caused by the sensitization of patient organism to pyococci and their byproducts in the presence of purulent skin diseases with chronic course. Pyoallergids are most frequently observed in case of streptococcal infections (superficial chronic diffuse streptoderma, intertriginous streptoderma and other).

Pyoallergids appear mainly at the sites remote from the focus of pyoderma and are symmetrically located. Clinically, they most frequently remind eczematous reaction, as the small miliary papules and microvesicles appear on the bright red background which has not got clear boundaries, and is covered with fine scales. In the result of itch the scratches (excoriations), covered with small hemorrhagic crusts, appear on the surface of pyoallergids. And dense blisters can appear in palpation on the palms and plantae.

The treatment of pyoallergids is analogous to that conducted in case of allergic skin diseases.

Prophylaxis of pyodermae. Preventive measures are essential in treatment of purulent diseases. Physically healthy and weather-beaten people even in adverse conditions seldom suffer from pyodermae, which attack in general people, who are colds-prone, suffering from gastrointestinal disorders, increased sweating, alcohol abuse.

Patients with furunculosis, hidradenitis, ecthyma, multiple abscesses, strepto-staphylodermae should exclude from their diet food rich in carbohydrates (honey, chocolate, sweets, white bread etc.)

Personal hygiene of the skin is important for the prophylaxis of pyoderma. The preventive measures, aimed at the preventing of pyodermae spread, include the timely detection and medicamental sanation of people suffering from chronic nasal staphylococcal carriage. This type of nasal infection is directly connected with the risk of appearance of pyodermae and pyoseptic complications in dermatological and surgical practice. Both patients and medical staff can be the carriers of Staphylococcus aureus, as well as the personnel of child care centers and other categories of persons. To eliminate staphylococcal carriage (eradication of staphylococci), the nasal ointments with antibiotics are applied, the ointment of mupirocin, in particular, by smearing of each nasal opening twice a day during 5 to 7 days.

Children with pustular skin diseases in children's groups must be immediately isolated and treated up to moment of disappearance of all clinical manifestations of disease. The staff of nurseries and kindergartens, suffering from anginas, herpetic eruptions, acute respiratory diseases of the upper airway, as well as pyodermae, must not be allowed to work.

1. There are the following types of staphylococcal pyodermae, except:

A. Ostiofolliculitis
B. Carbuncle
C. Neonatal pemphigus
D. Hydratenitis
E. Hard chancre

2. Staphylococci most frequently affect:

A. Hair follicle
B. Smooth skin
C. Nails
D. Vermillion border skin
E. Skin of the palms and plantae

3. The diagnosis of ostiofolliculitis is set in case of:

A. Presence of small superficial stressed cone-shaped pustules, pierced with a hair in the centre
B. Presence of purulent necrotic inflammation of hair follicle and surrounding connective tissue
C. Presence of small pustules, not connected with a hair
D. Presence of nodule with central necrotic core
E. Presence of phlyctenae

4. Vesicle-pustulosis of children is an inflammatory disease, which appears:

A. In the pinafore stage of existence
B. Not until 12th year of life
C. is congenital
D. at the age from 2 to 6
E. at the age of 7

5. Multiple abscesses (pseudofurunculosis) are observed in:

A. Infants and children at early age
B. Children at the age of 6 to 10
C. persons of ripe years
D. Teenagers
E. Persons of advanced years

6. Ecthyma vulgaris is:

A. Profound streptococcal infection
B. Infectious process involving pilosebaceous apparatus
C. Inflammatory process of sweat glands
D. Inflammatory superficial staphylococcal process
E. Superficial streptococcal infection

7. In epidemic neonatal pemphigus the blisters are located:

A. Exclusively on the hands
B. Exclusively on the legs
C. Over the surface of the body
D. Exclusively on the hairy part of the head
E. Exclusively on the skin of buttocks

8. Children with pustular skin diseases in maternity hospitals and children's groups:

A. can be treated without isolation
B. should be isolated immediately and treated up to the moment of disappearance of all rash elements
C. should be isolated immediately and wait for self-healing
D. should be isolated for the first two weeks
E. should be isolated for the first six days

9. In ecthyma vulgaris:

A. Deep tissue necrosis covers not only derma, but subcutaneous fat as well
B. Deep tissue necrosis covers only derma
C. Necrosis is absent
D. Purulent infiltration is located only in epidermis
E. Process evolves without trace

10. Pathological process in profound folliculate regresses with the formation:

A. Cicatrix
B. Hemorrhagic spots
C. Eschar
D. Passes without trace
E. Lichenification

Answers to the quiz of the first level of complexity
1 – E; 2 – A; 3 – A; 4 – A; 5 – A; 6 – A; 7 – C; 8 –B; 9 – A; 10 – A

Answers to the quiz of the second and third levels of complexity
1a – A; 2a – C; 3a – A ; 4a – B; 5a – A

Task 1. A father led a boy at the age of 5 to visit a doctor with complaints of rash on the face, which appeared six days ago. *Physical examination*: there are several blisters on the face skin the diameter of 0,5-1,5 cm with thin flabby tectum, filled with muddy serous exudate, they are placed on hyperemic, infiltrated base. At the same time, erosions with release of serous-purulent liquid and thick crusts of melichrous colour are observed in the lesion foci.

a) Set clinical diagnosis:
- **A**. Impetigo vulgaris
- **B**. Infantile eczema
- **C**. Simple vesicular herpes
- **D**. Contact dermatitis
- **E**. Candidasis

b) Make the scheme of examination and treatment.

Task 2. An infant of 1 month is under hospital treatment with the diagnosis of enteritis. A pediatrician called dermatologist for consultation concerning rash on the back and buttocks, the temperature is 38 °C. *Physical examination*: there are multiple solid knits on the skin of the napex, back and buttock, ranging in size from a pea to a hazelnut of rich red color, from some knits dense matter of yellowish-green colour is discharged.

a) Set a preliminary diagnosis:
- **A**. Hidratenitis
- **B**. Furunculosis
- **C**. Multiple abscesses (pseudofurunculosis)
- **D**. Pemphigus vegetans
- **E**. Cutaneous tuberculosis

b) Perform differential diagnostics and make treatment schedule.

Task 3. A mother led to outpatient attendance a child of two years with complaints of eruption on the skin of the face. *Physical examination*: on the face skin in the area of mouth there are phlyctenae with the diameter ranging from 1 to 3 cm with muddy contents, over the periphery of which the narrow bright red strip of inflamed skin, erosions, and crusts of yellow color is observed.

a) Set a preliminary diagnosis:
- **A**. Streptococcal impetigo
- **B**. Acne vulgaris
- **C**. Eczema of face
- **D**. Dermatitis
- **E**. Herpes zoster

b) Make the scheme of examination and etiopathogenetic treatment schedule.

Task 4. An infant of 7 days is in nursery with eruption on the skin of trunk, upper and lower extremities. The temperature is 37,5 °C. *Physical examination*: on the skin of the trunk, upper and lower extremities (except palms and plantae), buttocks there are blisters with the diameter of 1,0–1,5 cm with muddy contents and flabby tectum. The skin in the areas of blisters location is not infiltrated, slightly hyperemic.

a) Set a preliminary diagnosis:
- **A**. Varicella
- **B**. Epidemic pemphigus
- **C**. Herpetic infection
- **D**. Pemphigus syphiliticus
- **E**. Infantile eczema

b) Offer the tactic of examination and treatment scheme.

Task 5. A patient at the age of 26 complains of the presence of eruption on the skin of face, itch, burning sensation, pain in the indicated area. He is suffering for year and a half. *Physical examination*: on the skin of face in the area of chin there are infiltrated foci of bright red color, covered with pusteles and crusts with multiple erosions. Submandibular lymph nodes are slightly enlarged, freely movable, and not glomerate with surrounding tissue.

a) Set a preliminary diagnosis:
- **A**. Coccogenic sycosis
- **B**. Parasitic sycosis
- **C**. Eczema
- **D**. Contact dermatitis
- **E**. Acne vulgaris

b) Perform differential diagnostics.

5 TOPIC | Psoriasis

Psoriasis *(psoriasis vulgaris)* is a genetically determined chronic autoimmune polysystemic disease of multifactor nature, characterized by the benign epidermal hyperproliferation, papulosquamous eruptions, staging, often severe clinical course and involvement of musculoskeletal system and internal organs in pathological process with corresponding morphological and functional changes.

TRAINING AND EDUCATIONAL PURPOSES

- To highlight the current medical judgments of psoriasis etiopathogenesis
- To identify the factors contributing to the development of the disease
- To determine classification characteristics of this dermatosis
- To determine its typical clinical manifestations
- To understand differential diagnostics of psoriasis
- To understand general principles of medical and preventive measures at this disease
- Define the principles of medical examination of psoriasis patients

TO KNOW:

- modern views on etiology and pathogenesis of psoriasis;
- risk factors of disease development and progression;
- clinical manifestation, evolution of rash, the stages of clinical course;
- diagnostic phenomena of psoriasis;
- clinical types and principles of classification of psoriasis;
- systemic signs of disease;
- features of differential diagnostics of psoriasis;
- histopathological signs of psoriasis;
- treatment algorithm and indications concerning medical-labour expert commission.

TO BE ABLE TO:

- correctly make examination and collect anamnesis of psoriasis patient;
- set clinical diagnosis and define disease severity index;
- run diagnostic tests and use additional examination methods, which confirm the diagnosis;
- make a differential diagnostics of psoriasis with the diseases with the similar clinical presentation picture;
- make a plan of recommendations for treatment and prevention of relapses for psoriasis patients.

Historical data. In ancient Greece the terms *«alphos», «lepra» or «psora»* were used to indicate psoriasis. Cornelius Celsus (30–40 A.D.) was the first who described the clinic of psoriasis in the book *«De medicina»*. The term *«psoriasis»* was firstly applied by the Roman doctor Claudius Galen to designate eyelids and eye corners lesions with scaling and the scrotal skin lesions with itching and excoriations. Only at the beginning of the XIX century Robert Willan distinguished two diseases, namely: *«lepra graecorum»*, or discoid psoriasis (lesion in form of round spots), and *«lepra psora»* – figured psoriasis. In 1841 Ferdinand Hebra combined these two forms into one – *«psoriasis»*, having indicated, that the presence of various forms of the rash in the course of psoriasis gave no grounds for distinguishing them as separate diseases.

Epidemiology. Around 2-3% of the world population suffers from psoriasis. The disease is spread across all geographical latitudes among people of different nationalities and races. Although it often appears between the ages of 10 and 25 years, there are the cases of psoriasis occurrence both at the age of 4 to 7 months and at the age of 80 years. Psoriasis affects both sexes equally, but among children it occurs more frequently in girls, and among adults – in men. Disease is less common among Eskimos, Japanese, Africans, and South American Indians. In the general structure of the skin pathology psoriasis specific gravity achieves 8 to 15%, i.e. nowadays it remains an urgent problem of dermatology and common disease of population.

Etiopathogenesis. For 200-year study history of psoriasis the aspects of pathogenesis (from the dermatosis with violation of keratinocytes proliferation to T-cell autoimmune disease), clinic (from skin disease to polysystemic disease) and treatment (from ectylotics to highly selective drugs) have been repeatedly revised. A psoriatic disease as autoimmune genetically determined disease has been thoroughly investigated only during the last decades.

Hereditary conception of psoriasis pathogenesis, based on the registration of disease cases among the members of the same family, is generally accepted. According to the different data, familial psoriasis occurs in 5-40% of cases and more. Close relatives of the probands on the father's side suffers more frequently than the ones on the mother's side. The literature describes the cases of psoriasis in twins, with that, the highest concordance is observed among monozygotic individuals. The recent researches established the multifactorial character of inheritance of psoriasis, at which the high risk of disease is observed in the carriers of histocompatibility antigens HLA A1, A9, A11, A28; HLA B13, B 17, B27, B38, B39; HLA CW2, CW3, CW4, CW6. Due to the fact, that the hereditary transmission of psoriasis of the majority of psoriasis patients cannot be established, there exists the suggestion, that the disease is not inherited but disposition to it, which in a number of cases is realized as a result of complex interaction of genetic factors and adverse environmental effects.

However, although a family history is common, there is often no clear-cut inheritance pattern. What does seem clear is that the ***cascade of changes probably***

result from an interaction between T-cells and keratinocites, with the involvement of various cytokines and chemoattractants – notably interleukins 1 and 8, tumor necrosis factor-alpha, E selectin and intercellular adhesion molecule-1. It is now considered to be due to T-lymphocytes mediated disease of abnormal keratinocyte proliferation in genetic predisposed subject.

According to the modern ideas, ***immune system disorder*** and the development of weak immune response of the psoriasis patients is caused by the antigenic mimicry, genetic factors controlling the process of normal differentiation of immunocompetent cells, or disorder of neurohumoral mechanisms. The characteristic feature of immunogram of these patients is the reduction of T-lymphocytes, change of T-helpers to T-suppressors ratio due to significant increase of T-suppressors, the increased level of immunoglobulin IgG, IgA, components of complement C_3, C_4 that indicates the activation of the autoantibody formation processes, circulating immune complexes and, accordingly, the suppression of defense mechanisms. On this basis, it was concluded, that the pathogenesis of psoriasis is largely autoimmune. The leading pathogenetic link of immune disorders in case of psoriasis is deviation of cytokine profile by Th1-type with increased level of interleukins (IL-8) and tumor necrosis factor as the key mechanism of disease development.

Clinical picture and course. Psoriasis vulgaris is characterized by monomorphic eruption, based in most cases symmetrically on the extensor surfaces of the extremities and the hairy part of the head. The nails and musculoskeletal system is often affected. Psoriasis can be accompanied by itching of different intensity (usually in progressing stage) and feeling of tissue contraction.

The primary element of rash is epidermic-dermic inflammatory papule. Psoriatic papule is round with distinct boundaries, of pink-red colour of different intensity: the recent elements are bright red and the existing longer ones are more faded. The surface of rash elements is covered with silvery white (silver color is connected with

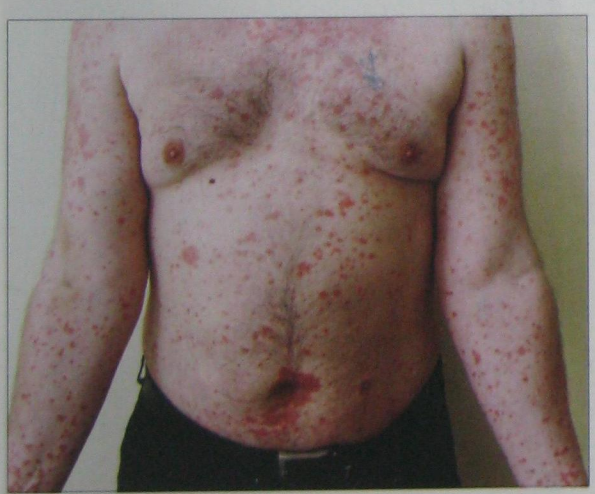

Fig. 5.1. Guttate psoriasis.

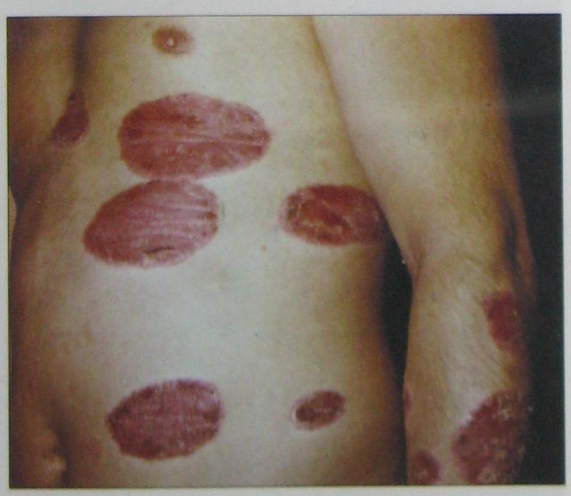

Fig. 5.2. Large-plaque psoriasis (pseudoatrophy Voronov's rim can be seen around papules).

the presence of air bubbles) furfuraceous and fine scales, which are easily removed during scraping. On the recent papules the scales are located in the center, and narrow bright circle remains on their periphery. Subsequently, the scaling increases, covering the entire surface of rash element. At the beginning the papules have correct rounded shapes and diameter of 1-2 mm. With the progression they are enlarged, form plaques, often achieving significant size and acquiring all possible shapes. Therefore, depending on the sizes of rash elements, the following clinical forms of psoriasis are distinguished:

- *Punctate psoriasis* (papules ranging in size from millet grain to match head);
- *Guttate psoriasis* (elements ranging in size from 3-4 mm to 7-10 mm);
- *Psoriasis nummularis* (papules from 10 mm to 30 mm);
- *Plaque psoriasis* (papules in size up to 6-7 cm);
- *Large-plaque psoriasis* (isolated plaques with size up to 15 cm and more);
- *Diffuse-plaque psoriasis* (dense confluent lesions, covering separate areas of body, such as back or abdomen, extremities);
- *Erythrodermic psoriasis* or *psoriatic erythroderma* (the patient's skin is the entire affected surface).

According to the infiltration rate of the plaques there are *moderate, marked and severe* (with presence of fissures) pathological skin process.

Depending on the extension of this process there can be *localized*, when the isolated plaques can be visualized on the skin of scalp or extensor surfaces of elbows, knees and other body parts, and *disseminated* or *extensive psoriasis* with numerous elements in different parts of the body.

Psoriatic erythroderma (*erythrodermia psoriatica, seu psoriasis universalis*) develops in the result of the effects of adverse exogenous factors, including stress situations and irritating treatment. Psoriatic erythroderma affects 1,5-3% of people with psoriasis, these people are often noted with reduction of immunological indices

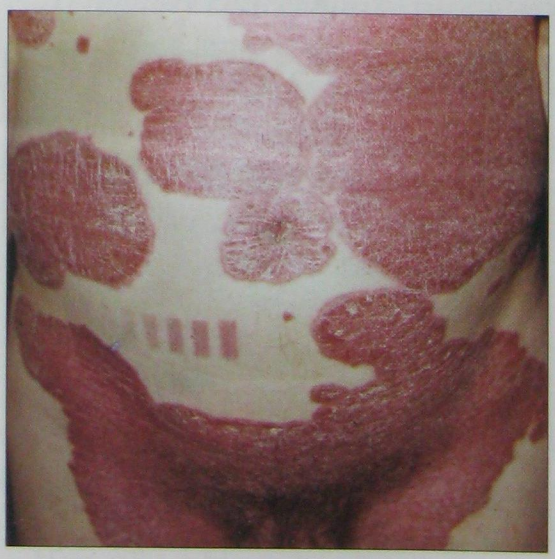

Fig. 5.3. Diffuse-plaque psoriasis.

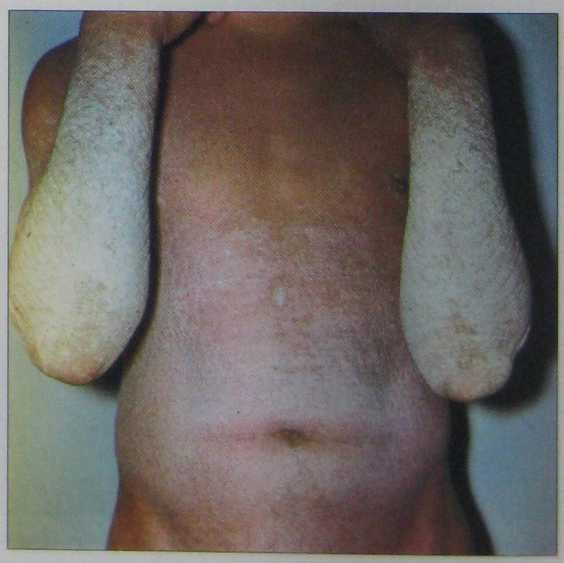

Fig. 5.4. Psoriatic erythroderma.

and high allergic reactivity. And often, the cause of erythroderma cannot be determined. The entire skin surface acquires bright red color with brown shadow. Edema and infiltration are not equally expressed on different parts. There is sharp skin flaking (furfuraceous desquamation in the hairy part of the head, and lamellate one in other parts), increasing of lymph nodes, possible temperature rise, nails destruction, occurrence of purulent paronychia. Such process without advance psoriatic anamnesis can be difficult to differentiate from other primary and secondary erythroderma.

For the diagnostics of psoriasis a range of symptoms, which include psoriatic triad and isomorphic reaction *(Koebner's phenomenon)*, is significant. A psoriatic triad includes three consecutive phenomena, in case of scraping of psoriatic papule, these are *phenomenon of stearic spot*, characterized by intense desquamation, when with light scraping in the result of dyshesion of keratinized plates, the affected skin area looks like porphyrized stearic drop; *phenomenon of terminal film* appears in the result of the further scraping of papule surface and squamosa erosion, when the wet surface of the spinous layer of epidermis is exposed; *phenomenon of pinpoint bleeding* (Auspitz' phenomenon), determined by the traumatization of capillary vessels of alpine papillae after light scraping of terminal film. Isomorphic reaction (*Koebner's phenomenon* or *psoriasis traumatica*) involves appearing of psoriatic rash in the areas of physical or chemical lesions of skin cover. It takes on the average 7-9 days after skin injury to the isomorphic reaction development.

There are three stages in the course of dermatosis, these are *progressive*, *stationary* and *regressive* ones. The *progressive stage* is characterized by the appearance of new papules, peripheral enlargement of old elements, the presence of inflammatory peripheral rim of papules increase, psoriatic triad, Koebner's phenomenon, the formation of new plaques due to fusion of papules or enlargement of old elements, sometimes accompanying by itching. For *the stationary stage* the

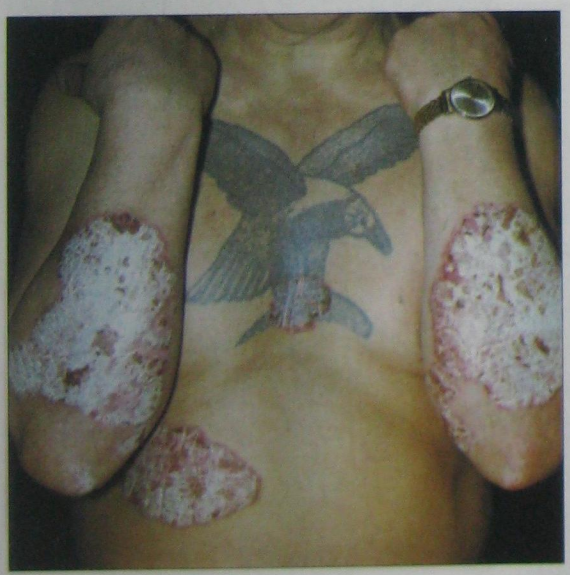

Fig. 5.5. Neglected large-plaque psoriasis.

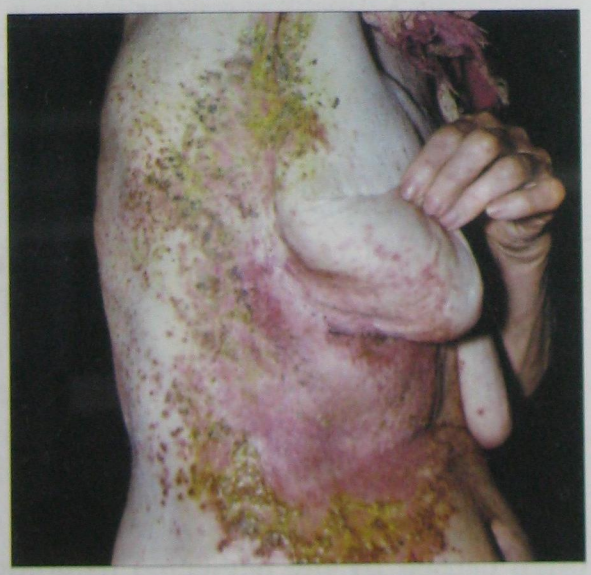

Fig. 5.6. Exudative psoriasis.

itching is uncharacteristic or weekly expressed, the new elements do not appear, peripheral rim of growth is absent, the surface of papules is entirely covered with silvery white scales, which are easily peeled, Koebner's phenomenon does not occur, pseudo atrophy Voronov's rim appears around the papules (slightly wrinkled white shiny ring up to 5 mm). In *the regressing stage* the elements of psoriasis are resolving and fading, the desquamation reduces and stops. Whitening of rash elements in the central part gradually leads to the appearance of the secondary eruption rests of various configurations in the form of hypo-, more rarely hyper pigment spots. With the disappearance of psoriatic eruptions, the remission is defined. The spontaneous remissions are marked approximately in 20% of cases, usually in the worm season.

Psoriasis is characterized by the process recurrence, appearing under the influence of various factors (climate, neuropsychic traumas, endocrine disorders, exacerbation of chronic infection). On grounds of climate and meteorological factors the *winter* (exacerbation in cold season), *summer* and *off-season,* or mixed, types of dermatosis are distinguished. The winter type of psoriasis occurs much more often than the other types.

The nature of popular elements determines typical course of psoriatic process on the skin. Accordingly, they distinguish between *psoriasis vulgaris (common)* and *atypical psoriasis*. Psoriasis vulgaris is characterized by typical popular rash, on the grounds of location they distinguish between linear psoriasis *(psoriasis linearis),* gyrate psoriasis *(psoriasis gyrata)*, figured or geographical psoriasis *(psoriasis geografica seu figurata),* and annular psoriasis *(psoriasis annularis)*. With atypical course of skin pathological process, the signs of other skin diseases are defined in the setting of characteristic clinical picture of psoriasis. Of atypical clinical forms of psoriasis, the most frequent are *follicular, verrucous (papillomatous*), ostraceous *(rupioides), intertriginous*, *exudative*, *eczematous, pustular, plaque, seborrheic, reverse, irritating* etc.

With follicular psoriasis *(psoriasis follicularis)* the presence of small conical papules, located in the sebaceous follicular areas, is noted. Papillomatous, or verrucous, psoriasis *(psoriasis papillomatosa seu verrucosa)* is characterized by significant infiltration and verrucous enlargement of rash elements. Rupioides psoriasis (*psoriasis rupioides*), in its turn, is presented by lamellate crusty scales on the eruption surface, giving them a conical shape, "snail shell" like. Rupioides and verrucous psoriasis are very often caused by the neglected pathological process *(psoriasis inveterata)*, in which in the

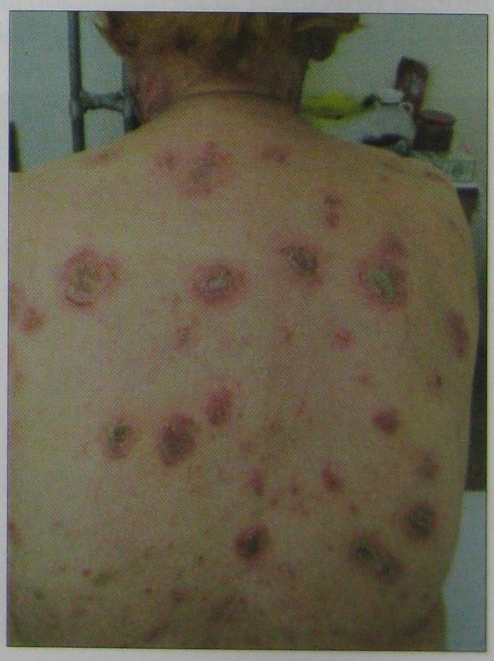

Fig. 5.7. Pustular psoriasis.

basis of pathological foci, there is significant infiltrate with massive scales, which are difficult to remove, and, as a rule, not fully. Asbestoslike psoriasis is a kind of neglected psoriasis. Intertriginous psoriasis *(psoriasis inversa, plicarum)* develops in nursing infants, suffering from severe diabetes, rheumatism, and in people, suffering from obesity, in advanced age and with high sensibilization. It is characterized by atypical localization on the flexor surfaces of the extremities, joints, on the skin of inguinofemoral and other folds, axillary hollows, on the palms and plantae. Its manifestations usually are nummular exudative plaques of purple red colour with sharp boundaries, insignificant desquamation, wet and macerated surface. It is often accompanied by itching and burning sensation. It is possible, that the given form is, in fact, exudative psoriasis, the clinical picture of which has changed somehow due to localization of the process in the skin folds. With exudative, or wet, psoriasis *(psoriasis exudativa seu humida)* the elements of rash are impregnated with exudate with the subsequent formation of yellowish-grey loose scaly crusts. With eczematous psoriasis *(psoriasis eczematoides)* the phenomena of exudation, crust, scratches layer and the pathological process looks like the eczematous one.

Two types of pustular psoriasis (*psoriasis pustulosa, seu suppurativa*) are distinguished; these are *Cymbush* type and *Baber's* type. The first type can appear primarily without typical picture of dermatosis and develops more frequently secondarily in the result of transformation of vulgar or intertriginous psoriasis. It is severe general illness, accompanied by temperature rise, general ailment, leukocytosis. First, there appear numerous disseminated erythematous and erythematous-edematous foci, which spreading over the entire skin cover, form various figures and diffuse bright red lesions. In this setting there are disseminated pustular elements, which shrivel into yellowish-brown crusts and crusty scales. Dermatosis can have malignant course with lethal outcome. With second type of

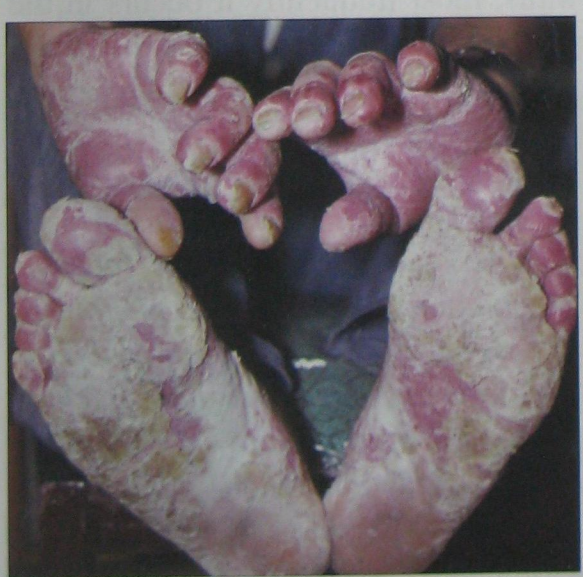

Fig. 5.8. Psoriasis of palms and plantae.

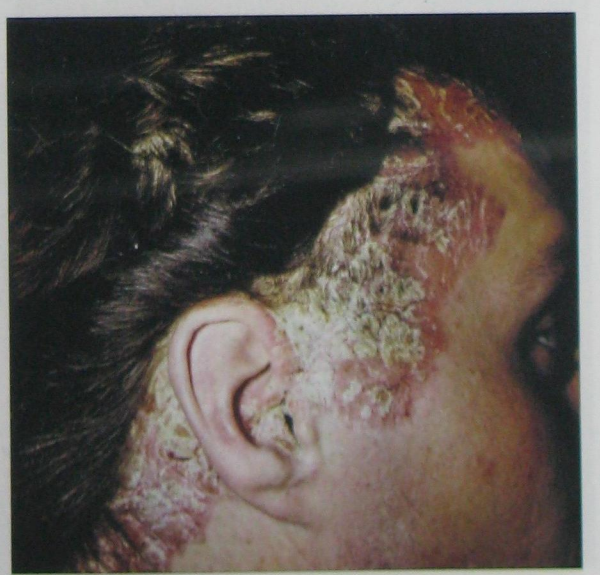

Fig. 5.9. Scalp lesion with psoriasis.

pustular psoriasis, symmetrically located pustular and psoriasiform foci, of small size at the beginning, develop in the area of palms and plantae in the erythematous setting. The content of pustules is sterile. Process generalization sometimes is possible. Along with the described lesions in the areas of palms and plantae, the typical psoriatic eruptions can be observed in the other areas of skin cover.

Often in dermatological practice the plaque psoriasis *(psoriasis exanthematica)* is observed, which is characterized by weakly expressed infiltration of elements, looking not like papules, but spots, that is similar to toxicoderma. With seborrheic psoriasis *(psoriasis seborroica)* the rash is localized on the seborrheic sites, herewith the scales adhere and acquire the yellowish colour due to secretions of the sebaceous glands, resembling clinically seborrheic eczema. With localization of the rash on the flexor surfaces of extremities, the inverse psoriasis or flexion psoriasis *(psoriasis inversa seu flexuratum)* is distinguished. The psoriasis of palms or plantae *(psoriasis palmaris et plantaris)* is ranked to atypical forms in the case, if psoriatic plaques acquire hyperkeratotic character, simulating clavuses formation *(clavi psoriatici)* or resembling callous eczema *(eczema tyloticum)*.

Diffuse or sharply limited layerings of scales can occur in the hairy part of the head, which often cover smooth skin, though the hair in never damaged. Psoriasis starts from the hairy part of the head approximately in a quarter of cases, and is combined with the eruptions in the other parts of the skin in more than 60% of cases. Scalp lesion is often of focal nature, occurring in the hindhead, temples, parietal area in the form of sharply raised plaques.

Approximately 25% of psoriasis patients have their nails affected; this pathology has got the early and late forms. Among the early forms of psoriasis of nail plates there are *punctate onychodystrophy,* or *pitting symptom* (characterized by punctate parakeratosis of proximal matrix), *symptom of oil stains* (presence of yellowish-brown subungual stains, mostly, in nail walls, most frequently it occurs in the progressive stage of psoriatic diseasea). With the late stages of the nail psoriasis more frequent are *hypertrophic onychodystrophy,* or *onychogryphosis* (in the form of «bird's claw» in the result of hyperkeratosis subungual), *atrophic onychodystrophy* or *onycholysis*, *transverse and longitudinal sulci on the nail plates, subungual petchiae* (Leventhal's symptom), ponychium defect *(Popov's symptom)*.

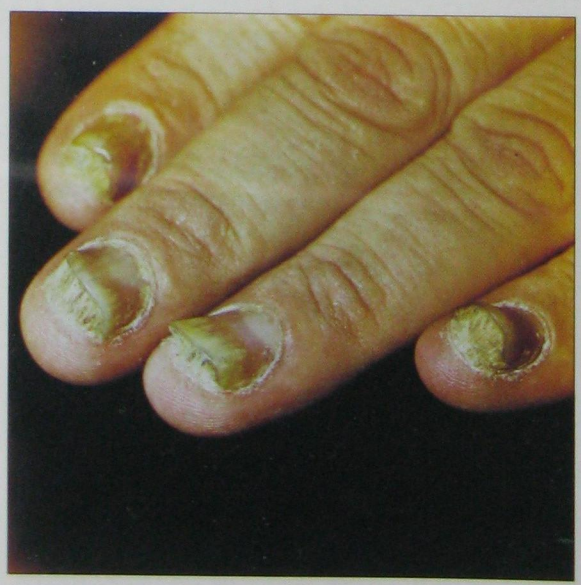

Fig. 5.10. Nails disorder with psoriasis.

10-12% of psoriasis patients have got additionally to the skin eruptions, progressive changes of joints from

periarticular injury (enthesitis, tendovaginitis) to arthritis with frank osteoarticular changes, that is crtified radiologically. Most of patients have got the joints lesion occurring in three-five years and more after the first psoriatic eruption. Symmetric multiple lesions of small peripheral joints and bones (osteoporosis, mainly in pineal glands, and the joint space narrowing) with the gradual involvement in the process of large joints, sometimes spine is characteristic. With the severe forms there occurs dispersion of end phalanges of hands and feet, and appearing of exostoses (osteophytes).

The development of pathological process during psoriasis can occur according to two types. The first type supposes the early manifestation of the disease (with peak at the age of 16-20). It is characterized by the large area of lesion, torpid course and frequent relapses. The second type has got later signs (peak at the age of 40-50). With the first type, the hereditary link is evident (in 44% of cases), whereas in the second type this connection is set only in 1% of cases.

Pathomorphology. Hyperkeratosis, parakeratosis, acanthosis in the inter-papillary areas of Malpighian layer and thickening of its above-papillary sites, intercellular edema, spongiosis, accumulation of neutrophils in the intercellular spaces, penetrating from dermis (Munro micro abscesses) is observed in epidermis. The horny layer over papule is thickened, loosened; it consists mainly of parakeratotic cells with elongated rod-like nuclei. There are numerous gaps between the plates, filled with air, in the result of which the scales are silver. Hyperkeratosis in the old elements is more pronounced, than parakeratosis. The nuclei in the horny layer occur regardless the age of the papule. Granular layer disappears or only interrupted series of its cells remains. In the severe cases, the shiny layer is absent. Malpighian layer over the papillae is thickened, epidermal outgrowths elongated. Acanthosis is sharply pronounced in the spinous layer between the papillae. Capillaries of the papillary layer are expanded, greatly curled and overfilled with the blood. Inflammatory infiltrate of lymphocytic histiocytic origin is observed around the vessels.

Electron microscopical study observes intense coloring of nuclei of basal layer, indicating increased proliferative activity of the cells of epidermis. The cell cycle of conversion of basal keratocytes in the horny scales reduces dramatically (up to four days, when the norm is 28).

Differential diagnostics. The skin manifestations of psoriasis should be differentiated primarily from the diseases, for which the characteristic rash

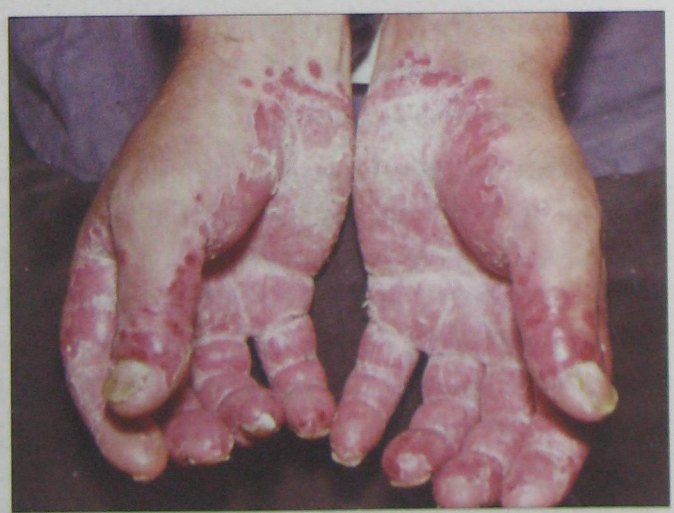

Fig. 5.11. Psoriasis arthropica.

element is a papule, covered with scales, namely, lichen planus, parapsoriasis, pink Gibert's lichen, atopic dermatitis, secondary syphilis.

In contrast to psoriasis, in case of *lichen planus*, the papules are polygonal squamous; desquamation on the surface of the rash elements is weakly pronounced; the color of efflorescence is purplish-red on the skin and pearl on the oral mucosa; waxy shine of eruption surface and the presence of umbilication in the center of papule is characteristic; texiform pattern on the surface of papule (Wickham's striae) is localized most frequently on the oral mucosa and on the skin of the flexor surfaces of forearms and lower legs; there is pronounced itch in the rash areas and negative psoriatic triad.

In case of *parapsoriasis*, the presence of characteristic phenomena is typical, such as wafer (a round scale is removed from the surface of the papule), occult desquamation (it is evident during scraping of surface of papule), collarette (the central site of papule is covered by greyish-brown scales, which are flaked off peripherally), purpura symptom (it is caused due to intense scraping of rash elements).

Pink Gibert's lichen is characterized by the presence of herald patch on the trunk, distribution of the daughter, mainly spotted, rash along the cleavage lines, symptom of "crumpled tissue paper".

Atopic dermatitis differentiates by the presence of erythema of dull color, pronounced lichenification, excoriations, localizations of eruptions on the skin of face, neck, upper chest, flexor surface of knee and elbow joints, allergological anamnesis.

Psoriasiform syphilides are characterized by pale red with copper color of rash elements, the absence of peripheral growth, the presence of Biett's collarette around papules, the absence of psoriatic triad, detection of pale treponema in the rash elements, positive serological reactions.

The differential diagnostics of atypical clinical forms of psoriasis should be carried out as well. Besides, nail psoriasis is often differentiated from onychomycosis on the basis of detection analysis for pathological fungi in the material studied; psoriatic erythoderma from toxicodermas, skin lymphomas and arthropathic psoriasis from the range of chronic arthritis. Arthropathic psoriasis is more frequently differentiated from rheumatoid arthritis.

Treatment. Pathogenetic treatments of psoriasis include systemic and topical treatment, skin care and additional methods of therapy, such as physiotherapy, balneotherapy, climate, psychological, herbal therapy etc. (Table 5.1.)

Systemic treatment. The volume of therapeutic interventions with psoriasis is determined by the main parameters of skin (stage, type, duration, course, etc.) and articular (lesions amount and symmetry, clinical anatomic and clinicoroentgenological variants of course etc.) pathologic processes and the degree of systemic manifestations.

Systemic treatment is aimed at the elimination of dominating manifestations of associated lesions, reduction of systemic symptoms of psoriasis and the use of

Table 5.1.
Agents for treating psoriasis

Topical	*Systemic*
Emollients	PUVA (psoralen +ultraviolet A)
Tar	Rethinoids
Salicylic acid	Cytotoxic, e.g. methotrexate, azathioprine, hydroxicarbamide (hydroxiurea)
Topical steroids	Systemic steroids * (see precaution below)
Dithranol (anthralin)	Cyclosporin
Vitamin D analogues (e.g. calcipotriol, tacalcitol)	'Biological' (cytokines): various monoclonal antibodies
Tazarotene	
Ultraviolet radiation	

preventive and curative interventions in order to improve functional state of skin and articular apparatus in psoriatic processes.

Systemic treatment is aimed at the reduction of psoriatic process activity by slowing of cutaneous syndrome progression and destruction of bone structure of articular apparatus. Systemic suppressive therapy of psoriasis is recommended with moderate and severe degree of cutaneous and articular sickness syndrome and includes the use of:

- glucocorticosteroids;
- disease modifying drugs (DMD) – cytostatic immunosuppressants (methotrexate, cyclosporine), sulfasalazine, gold preparations, leflunomide, aromatic retinoid;
- preparations of biological effects – monoclonal antibodies to tumor necrosis factor and its soluble receptor (infliximab, adalimumab, etanercept);
- other biological agents (anakinra, alefatsepta, efalizumab, abatacept).

Prescription of systemic *corticosteroid hormones* in the therapy of psoriasis requires comprehensive study. It should be kept in mind that systemic therapy with use of glucocorticosteroids frequently results in destabilization of psoriasis with formation of torpid, severe and atypical forms of dermatosis. Accordingly, the indications for use of such therapy in psoriasis are only generalized arthritis with pronounced exudative components, malignant form of arthropathic psoriasis, sharply pronounced organ manifestations, the combination of articular syndrome and atypical or extensive cutaneous psoriatic process (pustular psoriasis, psoriatic erythroderma, and extensive exudative psoriasis), intolerance to disease modifying drugs. Disease

modifying drugs (DMD) are the preparations of pathogenic effect with immunosuppressive property and capable of inducing remission of psoriasis or reducing the rate of development of articular destruction and cutaneous pathologic process. Curative effect of DMD appears in one to two months and more after the start of treatment, what is connected with the necessity of their accumulation in organism. In psoriasis those DMD are used, as a rule, which are widely used in rheumatology. Not only methotrexate, cyclosporine, sulfasalazine, leflunomide and the preparations of biological effects are dominating among them, but also colchicine, derivatives of fumaric acid, mycophenolate mofetil, somatostatin, aromatic retinoids.

Retinoids, which are already used about 30 years in dermatological practice, are characterized by high efficiency in moderate and severe course of psoriatic disease, psoriasis of hairy part of the head, arthropathic psoriasis and with psoriatic lesion of nail plates. The developments of the recent years have led to the appearance of new aromatic synthetic analogue of retinoid acid – acitretin, mechanism of action of which lies in inhibiting the proliferation of epithelial cells, normalization of keratinization processes and stabilization of membrane structures of cells, including liposomes.

Prolonged use of SMD and DMD gives the possibility to control the activity of psoriasis and its main syndromes, slow down the rate of disease progression, contribute to preservation of working capacity of patients and improve their quality of life. The use of DMD in therapy has a positive effect on the course not only of the pathological articular process, but cutaneous syndrome, as well, that provides for reduction of use of external therapy.

Topical therapy. Among many methods of psoriasis treatment, the preparations of external therapy and skin care products are essential. Rationally found external therapy for psoriasis is of great and often key importance. For the majority of patients with limited plaque form of psoriasis, this choice of therapy under condition of elimination of main initiating agents becomes the only. With other, more severe clinical forms and extensive pathological process, the external therapy is rather ponderable addition to the systemic one.

External treatment is carried out first of all considering the process staging. Local therapy is also divided into non-suppressive and suppressive. Non-suppressive treatment of psoriasis provides for use of skin care means (local means with softening and/or moisturizing effect, which do not contain the active substances – emollients), and means, which include salicylic acid (2% in progressive stage, 4% – in stationary stage and 10% – in regressive stage) or urea (2-10%), tar preparations, as well. The use of urea and salicylic acid is connected with their cheratolic, antimicrobial and keratoplastic properties. The best studied pharmacological agents of psoriatic suppressive treatment are topical glucocorticosteroids (TGCS), analogues of vitamin D_3, topical retinoids (TP), hydroxiantrons.

Topical glucocorticosteroids are important means of external therapy of psoriasis due to their rapid anti-inflammatory effect and pronounced wide range of actions. The strongest TGCS of the IV class of activity such as clobetasol propionate in the form of

ointment and cream can be used in the severe recurrent course of psoriasis during short course (one to two weeks), after that it is advisable to prescribe the other, less strong TGCS, in particular, of the III or the II class (Locoid, Eloson, Momederm, Moleskin, CelestodermV, Cutivate, Laticort, Flucinar etc). However, prolonged use and application of corticosteroids on the large surface, inhibition of the function of adrenal cortex and the development of undesirable side effects are possible. For such a reason, thorough selection of TGCS is essential, with optimal efficiency and low possibility of occurrence of local and systemic side effects. It especially concerns the prescription of TGCS for children with psoriasis. In this aspect such TGCS as fluticasone propionate (Cutivate), hydrocortisone butyrate (Locoid), methylprednisolone aceponate (Advantan) have the advantage. Combined therapy of TGCS combined with salicylic acid (LorindenA) or urea (Betasalic) is appointed with the pronounced skin flaking.

Emollients are the key skin care products for the patients, suffering from psoriasis, providing restoration and protection of the horny layer of epidermis, maintaining hydro lipid balance and preventing transepidermal water loss.

By origin the emollients can be hydrophilic, lipophilic and combined (amphiphilic). These means can contain various compounds of mineral and organic origin, these are the higher fatty acids, triglycerides, ceramides, phospholipids, squalene, cholesterol, liquid paraffin, white petrolatum, urea, glycerol, etc. Depending on production technology emollients can be presented by simple emulsion in the form of «water in oil» or «oil in water», laminar emulsions, liposomes/microspheres, nanoparticles and other forms.

For the local therapy of moderate and moderately severe psoriasis *synthetic retinoids* (Tazarotene) and analogues of vitamin D_3 (Calcipotriol, Tocalcitol) in the form of ointment, cream, gel are used.

Phototherapy is one of the effective treatments of psoriasis. The following forms of light therapy are recommended:

- Natural insolation (heliotherapy);
- Ultraviolet irradiation – selective phototherapy (wave length of 280-320 nm, dose of 0,05-0,1 J/cm^2 in a day, during the period up to two months);
- PUVA-therapy (systemic and local);
- Re-PUVA-therapy (retinoids + PUVA).

Photochemotherapy is used in case of moderately severe and severe forms of psoriasis. However, it must be taken into account the possibility of side effects development (itching, erythema, blisters, stimulating of malignant course of neoplasms) and the risk of skin cancer. At the Department of Dermatology and Venereology of Bogomolets National Medical University, zonal UV-irradiation by means of quartz lamps according to the schedule, developed by Professor I.I. Pototsky, is widely used, ensuring the achieving of significant clinical effect in the treatment of different forms of psoriasis.

Additional methods of treatment of psoriasis involve physiotherapy, balneal and climate treatment, phytotherapy, psychosocial therapy, surgical treatment and others.

Physiotherapy is widely used in dermatological practice as stage treatment of psoriasis patients. Complex methods of treatment are worked out, involving the effects of ultrasound, magnetic and laser therapy and phonophoresis on the affected sites.

Psychosocial therapy is important in the treatment of psoriasis, as it is often necessary to consider the possible disease impacts on social, emotional and psychic aspects of each patient. In case of serious patient sufferings, significant deterioration in his life quality, the consultation with specialist in psychosomatics and psychotherapy should be recommended. Psychosocial therapy can be used only in case of patient wish and consent.

Balneal and climate treatment has a positive effect both on cutaneous and articular syndromes of psoriatic disease. Health resort balneal treatment (hydrogen sulfide, radon, salt-water baths, seabathing) is recommended In intercurrent period, in particular, in the sanatoriums of Nemyrov, Synyak, Khmelnyk, Solotvyno, in Crimean resorts and others.

Phytotherapy, treatment by means of herbal preparations must be considered as an addition to the main therapy during stabilization of pathological psoriatic process in the form of baths, phytoapplications, wraps, ointments use etc.

Since psoriasis is of polyetiological nature and there is no absolutely explicit method of its treatment for today, the main rule of a doctor must be individual approach and selection of the most effective therapy, which reduces the disease activity, accelerates manifestation of clinical effect of treatment, prolongs remission of not only cutaneous but articular pathological process as well.

Therapeutic regimen and skin care. In order to avoid the development of isomorphic reaction, hydrotherapeutic procedures without strong direct irritant effect should be favoured. After a shower (bath) the skin must be moisturized by applying emollients. It is recommended to live a healthy lifestyle, avoid alcohol abuse and smoking. The diet of psoriasis patients should support the correct acid-base balance in organism with a predominance of alkaline reaction. The positive reinforcement is also essential, as negative emotions contribute to exacerbation of psoriatic process.

1. What is the most typical localization of eruptions in psoriasis process?

A. Flexor surfaces of extremities
B. Extensor surfaces of extremities
C. Interdigital folds
D. Skin around physiological ostia
E. Mucous membranes of genitals and oral cavity

2. What symptom is not used to confirm a psoriasis diagnosis?

A. Psoriatic triad
B. Koebner's phenomenon
C. Auspitz' symptom
D. Phenomenon of pintpoint bleeding
E. Phenomenon of apple jelly

3. Which clinical sign is not characteristic for the progressive stage of psoriasis?

A. Trend to fusion
B. Presence of psoriatic triad
C. Silvery white scales in the center of papule
D. Koebner's phenomenon
E. Silvery white scales over the entire surface of papule

4. What is characteristic for psoriatic papule?

A. Yellowish red color
B. Umbilication in the center of papule
C. Oval form
D. Surface covered with white scales
E. Shiny surface

5. Clinical types of psoriasis do not include:

A. Seborrheic
B. Foliaceous
C. Exudative
D. Follicullar
E. Rupioides

6. What is identified in case of psoriatic erythroderma?

A. Pronounced infiltration of rash elements with warty growth
B. Continuous fusion of rash elements with infiltration and edema
C. Pustular rash with increased temperature and lymph nodes enlargement
D. Ulceration of rash elements
E. Articular and edema syndrome without cutaneous symptoms

7. Psoriatic lesion of nails is characterized by the following, except:

A. Symptom of pitting
B. Symptom of oil stain
C. Symptom of apple jelly
D. Onychogryphosis
E. Onycholysis

8. What of the following is not occurred?

A. Jerky appearance of pustules on the places of eruptions or visually healthy skin, not connected with hair follicles
B. Sterile pustules
C. Sudden onset with fever, leukocytosis and increased ESR
D. Hypertrophic growth of papules on the palms and plantae
E. Possibility of disease transformation into another form of psoriasis or disability

9. Clinically peripheral arthropathic psoriasis is presented by:

A. Lesion of three joints of one finger
B. Tenosynovitis of separate fingers
C. Sausage-shaped configuration of fingers
D. Arthritis of distal interphalangeal joints
E. All of the above

10. What of the following is not qualified as pathomorphological characteristics of skin with psoriasis?

A. Hyperkeratosis
B. Acanthosis
C. Acantholysis
D. Parakeratosis
E. Munro micro abscesses

Answers to the quiz of the first level of complexity
1 – B; 2 – E; 3 – E; 4 – D; 5 – B; 6 – B; 7 – C; 8 – D; 9 – E; 10 – C
Answers to the quiz of the second and third levels of complexity
1a – A; 2a – A; 3a – E; 4a – A; 5a – B

Task 1. A patient at the age of 20 has disseminated papular rash of bright red color, of the size from the millet to lentil grain on the extensor surfaces of extremities, back, and hair part of the head, the surface of the elements in the center is covered with whitish scales. Applying the method of scraping the symptoms of stearin spot, terminal film and pinpoint bleeding are identified.

a) Which diagnostic test was used to confirm the diagnosis?
- **A**. Psoriatic triad
- **B**. Koebner's phenomenon
- **C**. Balser's test
- **D**. Phenomenon of falling the probe
- **E**. Phenomenon of apple jelly

b) Which histopathological processed are associated these symptoms with?

Task 2. A patient at the age of 45 has numerous inflammatory rashes consisting of pink-red papules, covered with whitish scales in the center, on the skin of the hair part of the head and trunk, appearing gradually during the period of two weeks. The eruptions of this nature were observed a few years ago in wintertime, and gradually disappeared without treatment by the end of spring.

a) What is the most probable preliminary diagnosis in this patient?
- **A**. Extensive psoriasis, progressive stage
- **B**. Extensive psoriasis, psoriatic erythroderma
- **C**. Extensive psoriasis, stationary stage
- **D**. Limited psoriasis, psoriatic arthropathy
- **E**. Limited psoriasis, stationary stage

b) Determine the complete clinical diagnosis with appointment of treatment.

Task 3. A woman is complaining about periodic appearance of eruptions during the year in the area of elbow and knee joints, which she associates with frequent nerve strains. During preliminary outpatient examination the diagnosis of psoriasis is set. At presentation: rash consists of numerous plaques, surface of central part of which is covered with easily falling-away silvery scales.

a) Determine medical tactic.
- **A**. Sedative therapy
- **B**. Antihistamine therapy
- **C**. Aevit appointment
- **D**. Application of 2% salicylic ointment
- **E**. All of the above is correct

b) Conduct a differential diagnostics of the indicated disease.

Task 4. A woman at the age of 52 is complaining about the rash, swelling, pain and stiffness of hands and feet movement. She has been sick for more than 15 years, relapses occur in the wintertime. *Physical examination*: numerous plaques with diameter of 2 cm. are observed on the skin of the hair part of the head, trunk, upper and lower extremities, which in some places are merged in large foci. The rash elements are rose-red with silvery white scales in the center, which easily fall-away. Small joints of the hand and feet are edematous, their mobility is limited. There is pitting on the nail plates of some fingers.

a) Set a preliminary diagnosis:
- **A**. Extensive, arthropathic psoriasis
- **B**. Extensive psoriasis, psoriatic erythroderma
- **C**. Extensive psoriasis, stationary stage
- **D**. Limited, arthropathic psoriasis
- **E**. Limited psoriasis, stationary stage

b) Suggest the examination plan and treatment scheme for this patient.

Task 5. A patient has got rash on the skin of chest and extremities in half and a month after suffering a shingles. *Physical examination*: rose-red papules of various sizes covered with white scales, which do not reach the healthy skin, leaving peripherally narrow pink line, are visualized on the skin of trunk, extensor surface of forearms and lower legs. Dotted papules with white scales are observed on the right lower leg along the scratch.

a) Put the preliminary diagnosis:
- **A**. Lichen planus
- **B**. Extensive psoriasis, progressive stage
- **C**. Extensive psoriasis, stationary stage
- **D**. Secondary syphilis
- **E**. Tuberculosis cutis lichenoides

b) Which diagnostic tests are necessary to make for setting a clinical diagnosis?

6 TOPIC

Lichen planus

Lichen planus (*lichen ruber planus*) is a peculiar reaction of organism of undetectable etiology, which is manifested as monomorphic papular rash on the skin and mucous membranes. This skin disease is usually regarded as multifactorial. Endogenous and exogenous factors along with genetic anomalies can be essential in the formation and character of the course of pathological process with lichen planus.

TRAINING AND EDUCATIONAL PURPOSES

- To determine the possible conditions and triggers of appearance of lichen planus
- To perform classification and define general peculiarities of various forms of lichen planus on the skin and mucous membranes
- To interpret clinical picture and course of lichen planus
- To generalize the principles of treatment of lichen planus

TO KNOW:

- etiological, pathogenetic and epidemiological features of lichen planus;
- clinical forms of lichen planus and current peculiarities of its course;
- diagnostic criteria of disease manifestations on the skin and mucous membranes;
- principles of preventive and curative interventions and preventive medical examination of the patients with lichen planus.

TO BE ABLE TO:

- correctly collect anamnesis of patients with lichen planus;
- make a differential diagnostics with dermatoses, which have similar clinical presentation;
- prescribe rational treatment for a patient with lichen planus.

Etiology of lichen planus cannot be regarded as finally established. In the development of dermatosis a significant part is assigned to infectious factors (viruses), toxic-allergic effects, as well as neurological and immune disorders. The researchers, who speak in favour of infectious nature of dermatosis, make reference to the good results of treatment with use of antibiotics, cases of familial disease, as well as identification of intracellular viral inclusions in these patients. The theory of neurogenic genesis of the disease is confirmed by frequent cases of lichen planus appearance after stressful situation, effectiveness of hypnosis or reflex-segmental therapy in some patients, and the location of rash sometimes along the nerves. Endocrinal theory of disease appearance is based on the hormonal and metabolic disorders in organism. A genetic predisposition to such pathology, transmitted in an autosomal-dominant pattern of inheritance is established.

Pathogenesis of lichen planus has got a lot of links, the interaction of which is diverse and not completely studied. According to the current judgment, the main organ, where there appears a pathological picture, is a skin, as it is the place, where the pronounced dystrophic and inflammatory reactions occur. The reasons and mechanisms of pathological skin changes in lichen planus are studied insufficiently. Currently, changes in an immune system, including those of autoimmune processes, are proved to be significant in the development of lichen planus.

Epidemiology. Lichen ruber planus (LRP) occurs quite often. According to the data of most researchers, the people at the age of 30 to 60 years prevail among the patients with lichen planus. Lichen planus in children is rare. In general structure of dermatological morbidity, lichen planus constitutes from 1 to 1.5%, it makes from 11 to 30-35% among the disease of oral mucosa. Lichen planus occurs in all countries and in different climatic geographical areas, the incidence of disease varies over a wide range.

Classification. Lichen planus of skin and mucous membranes is characterized by a great variety of atypical forms that significantly complicates diagnostics.

Clinical forms of lichen planus:

– *typical (classical or usual) form;*

– *atypical forms:*

- linear lichen planus *(lichen ruber linearis seu lichen ruber striatus)*;
- annular lichen planus *(lichen ruber anularis);*
- lichen rubber monoloformis (Wise-Reindisease, Kaposi's disease) *(lichen ruber planus monitiformis Kaposi);*
- lichen planus of the scalp *(lichen ruber planus capillitii);*
- lichen planus of the palms and soles *(lichen ruber planus palmarum et plantarum);*
- erythematous form *(lichen ruber erythematosus Kaposi);*
- hypertrophic (verrucous) form *(lichen ruber hypertrophicus seu verrucosus);*

- follicular form *(lichen ruber follicularis seu spinulosus);*
- lichen planus obtusus (Unna) *(lichen ruber obtusus Unna);*
- atrophic form *(lichen ruber planus atrophicus);*
- bullous or pemphigoid form *(lichen ruber bullosus seu pemphigoides);*
- pigmentary form *(lichen ruber pigmentosus);*
- psoriasiform lichen planus *(lichen ruber planus psoriasiformis Broca);*
- tropical lichen planus *(lichen planus tropicalis).*

It is accepted to distinguish between six forms of lichen planus on the mucous membrane of the mouth and vermillion border:

- typical;
- exudative-hyperemic;
- erosive-ulcerative;
- bullous;
- hyperkeratotic;
- atrophic.

Clinical picture. Typical form of lichen planus is characterized by monomorphic rash, formed by lenticular inflammatory shiny polygonal papules, on the surface of which there can be observed pathognomonic for the disease Wickham's striae opal-like white and grayish dots and stripes. Striae becomes more visible, if the surface of papules is moisten with water, plant or vaseline oil. Its formation can be explained by uneven granulosis. There is umbilication in the center of some papules. Merging with each other, small papules can form plaques with distinct boundaries of bluish discoloration, covered with fine plaques. In advanced stage of disease the positive Koebner's phenomenon (the appearance of rash in the sites of even insignificant skin traumatization) is observed, as well as itching. The rash is localized predominantly on the skin of inner forearms, wrist and ankle joints, sacrum and externalia area. The rash can spread throughout the entire skin cover (except face). With the regression of the process the secondary pigmentation remains on the sites of the papules. Sometimes there occurs cicatrical alopecia on the hairy part of the head.

In 25–70% cases of lichen planus, together with skin lesion there occur lesions of oral mucosae (cheeks, tongue, gingivae, palate) and externalia, which can be isolated or combined with skin lesion. Firstly, there appear small papules of greyish-white color, sharply defined on a pink background of mucosa, then the plagues form. The papules on the mucosae do not have typical shine, the infiltrate can be moderately expressed, and the elements hardly rise above the mucosal surface. They are mainly localized on the internal surface of cheeks on the line of the contact of teeth (molars), where they form whitish figure, similar to frond.

Change of nail plates in lichen planus is characterized by the formation of sulci, grooves, sites of opacification; the nails can become thinner, or they can be even destroyed in part or in whole, in this connection there are two the most common types of nail changes in this dermatosis, these are onychorrhexis and onycholysis.

Thus, the main clinical morphological primary element of rash in lichen planus is lenticular dermo-epidermal inflammatory papule, with the following characteristics for this dermatosis:

- polygonal outsides;
- umbilication in the center;
- absence of tendency to peripheral enlargement;
- presence of so-called Wickham's striae, which becomes visible in the deep of papules after their surface are moisten with water or oil;
- bluish-red (or lilac) pearly papules and polished shine of their surface with side lightning.

The course of dermatosis. Typically the localized forms of dermatosis are of long-term chronic course with periodic exacerbation. Sometimes the process can begin acutely, with fever, rapid generalization of the rash, skin edema and erythema is possible, erythroderma with fine (unlike psoriatic) desquamation may appear. Acute lichen planus can rather quickly regress, but mainly it becomes chronic. Quite often in the time of paracme the rash is not everywhere regressing, remaining on the lower legs and genitalia.

Diagnostics. Diagnostics is based on the clinical and histological data.

Pathomorphology. The main pathomorphological signs of typical lichen planus are hyperkeratosis, granulosis, acanthosis, vacuolar dystrophy of the basal layer, diffuse band infiltrate in the upper dermis, intimately adjacent to epidermis, the lower boundary of which is blurred by the infiltrate cells.

Differential diagnostics. Differential diagnostics is carried out with psoriasis, toxic-derma, neurodermatitis, verrucas plantar, papular syphilides. For clarification of diagnosis a skin biopsy is usually made.

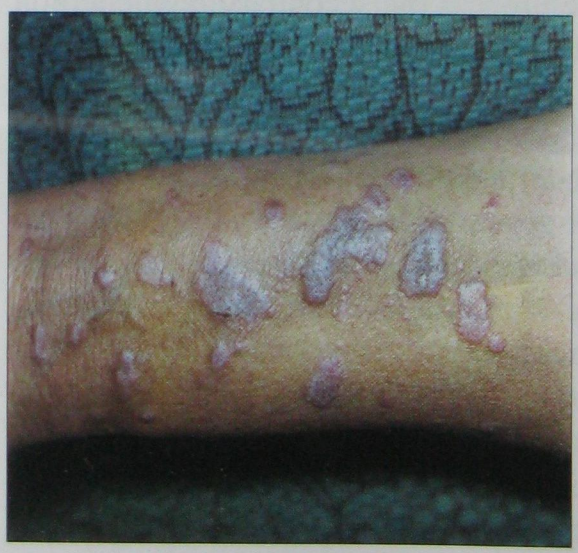

Fig. 6.1. Lichen planus.

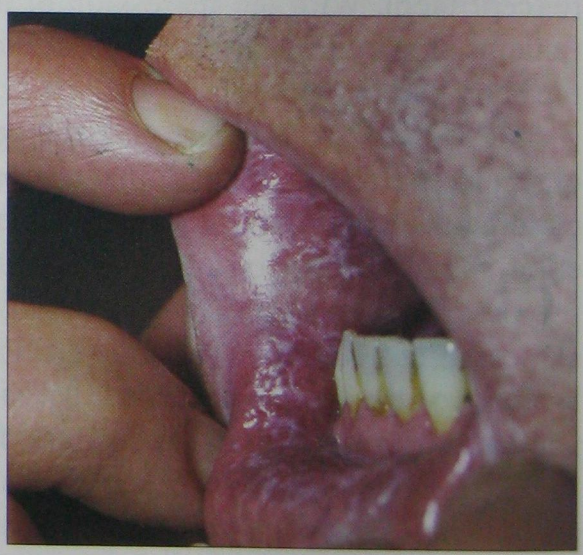

Fig. 6.2. Lichen planus of oral mucous membrane.

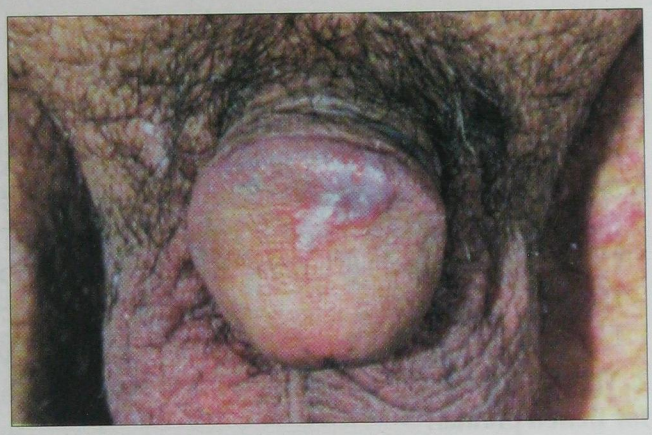

Fig. 6.3. Lichen planus.

Treatment. Currently, due to absence of a common conception of etiology and pathogenesis of lichen planus there are numerous methods of treatment of this disease. Taking into account the complexity of pathogenesis of lichen planus, success is possible only in the complex pathogenetic individualized therapy with use of modern methods. Thus, it is important to take into account the conditions, enabling disease appearance, risk factors, accompanying pathology, the presence of foci of focal infection, previous treatment. Traditional treatment involves antibiotic therapy, sedatives and antihistamines, topic corticosteroids, vitamins (A, D, E), retinoids (vitamin A derivatives), with use of exogenous interferon and interferonogenes, phonophoresis with sedative and anti-inflammatory drugs, cryomassage and cryodestruction (especially with verrucous form).

Retinoids (tigason, neotigason, etretinete) reduce the intensity of inflammatory reaction, influence the state of cell membranes and normalize the processes of proliferation. Retinoids are more effective in lesion of oral mucosa and vermillion zone. In the therapy of lichen planus the analogues of vitamin A carotenoids are used as well, especially in atypical forms, including erosive ulcerous, and in lesions of oral mucosa and genitalia as well.

Of topical corticosteroids the preparations of strong and very strong action are used in complex therapy of lichen planus. In case of severe lesion the appropriate therapy should be started according to the principle of "powerful start", using the drugs of the IV power category of action with short courses of administration (one-two weeks) with the following transition to the TGCS of the III or II classes.

Prognoses of lichen planus is favorable. However, the malignant transformation of papules is described in lichen planus. Its erosive ulcerous form on the oral and lips mucosa is considered to be precancerous condition. In this relation, the methods of treatment connected with irritation and cauterization should be avoided. In the long course of erosive ulcerous forms of lichen planus, hardly answering the treatment, it is necessary to carry out a histological study in order to detect malignification.

1. Indicate the clinical manifestations, common for psoriasis and lichen planus:

A. Polygonal popular elements with impression in the center
B. Frequent mucosae lesion
C. Isomorphic Koebner's reaction
D. Very strong itching
E. Paints in the joints

2. Which form of the following is not typical for lichen planus?

A. Exudative hyperemic
B. Hypertrophic
C. Typical
D. Palmoplantar
E. Intertriginous

3. What is a patient with lichen planus complaining of?

A. Temperature rise
B. Itching
C. Dyspepsia
D. Paints in the joints
E. Catarrhal signs

4. Clinical symptoms of lichen planus are:

A. Localization of the rash on the oral mucosa
B. Monomorphic rash
C. Long duration of disease (several years)
D. Appearance of rash in the areas of mechanical trauma
E. All of the above is correct

5. What clinical characteristics are typical of papules in lichen planus?

A. Polygonal
B. With waxy shine
C. Violetish
D. With umbilication
E. All of the above is correct

6. Specify the areas of rash localization in lichen planus:

A. Hairy part of the head
B. The front surface of the tibia
C. Elbows and knees
D. Neck
E. Palms and plantae

7. Which clinical forms are not observed in lichen planus?

A. Anular
B. Pemphigoid
C. Atrophic
D. Verrucous
E. Dyshidrotic

8. Which clinical forms of lichen planus are not observed on the vermillion border and oral mucosa?

A. Typical
B. Pigmentary
C. Exudative hyperemic
D. Hyperkeratotic
E. Erosive ulcerous

9. In lichen planus the most frequent localization of rash on oral mucosa is:

A. Contact line of teeth
B. Hard palate
C. Soft palate
D. Tongue
E. Mouth floor

10. Fort the treatment of lichen planus the following drug is not used:

A. Antimycotic agents
B. Immunomodulators
C. Retinoids
D. Vitamines
E. Antihistamines

Answers to the quiz of the first level of complexity
1 – C; 2 – E; 3 – B; 4 – E; 5 – E; 6 – B; 7 – E; 8 – B; 9 – A; 10 – A
Answers to the quiz of the second and third levels of complexity
1a – A; 2a – E; 3a – D; 4a – D; 5a – D

Task 1. A patient after past influenza has got itching rash on the trunk and extremities. *Physical examination*: there are lenticular papules of bluish red color with umbilication in the center on the sites of the trunk, flexor surfaces of the forearms and extensor surfaces of the lower legs. A linear grouping of rash elements in the areas of skin trauma attracts attention.

a) The described clinical picture corresponds to:
- **A.** Lichen planus
- **B.** Psoriasis
- **C.** Herpes zoster
- **D.** Herpes simplex
- **E.** Eczema

b) From what dermatoses should these presentations be differentiated and how?

Task 2. A patient at the age of 27 is complaining of rash on the trunk, upper extremities and itching. She associates her disease with nervous overstrains. *Physical examination*: there are polygonal papules with shiny, like waxed surface on the trunk and on the flexor surfaces of forearms. Umbilication occurs in the center of some elements. There are whitish papules on the oral mucosa.

a) What dermatosis should be considered in this case?
- **A.** Eczema
- **B.** Psoriasis
- **C.** Herpes zoster
- **D.** Herpes simplex
- **E.** Lichen planus

b) What therapeutic and preventive measures should be carried out?

Task 3. A patient at the age of 43 is complaining of itching rash on the sites of both lower legs. *Physical examination*: there are brownish red plaques covered with dry horny wartlike masses, tightly adhering, on the flexor surfaces of the lower legs; polygonal lenticular papules with umbilication in the center and shiny like waxed surface are sometimes observed.

a) To which disease does the described clinical picture correspond?
- **A.** Eczema
- **B.** Psoriasis
- **C.** Allergic dermatitis
- **D.** Lichen planus
- **E.** Mycosis of the skin

b) Which methods of therapy should be prescribed for the treatment?

Task 4. A patient after past otitis has got rash on the oral mucosa appeared. *Physical examination*: there are strip plaques of the whitish opal color on the cheeks and palate, isolated papules of bluish red color with umbilication in the center on the flexor surface of the forearms.

a) Which diagnosis can be set?
- **A.** Eczema
- **B.** Psoriasis
- **C.** Allergic dermatitis
- **D.** Lichen planus
- **E.** Mycosis of the mucosae

b) What are the features of the tactics and follow-up of this patient?

Task 5. A patient of 45 after prosthodontic treatment has got whitish papules appeared on the oral mucosa, which gradually grouped, forming the irregular figures, similar to frond. A patient has not self-medicated.

a) What is the most probable character of this disease?
- **A.** Eczema
- **B.** Mucosal candidiasis
- **C.** Allergic dermatitis
- **D.** Lichen planus
- **E.** Leukoplakia

b) What are the peculiarities in the scheme of examination of this patient?

7 TOPIC

Fungal Infections of Skin and Mucous Membrane

Fungal infections of skin and mucous membranes (mycosis) is a nosological concept that combines a number of diseases of skin and its appendages and human mucus membranes, which are caused by pathogenic and opportunistic fungi.

TRAINING AND EDUCATIONAL PURPOSES

- To understand the relevance of mycotic skin and mucous membranes lesion; the ways and conditions of mycosis infection.
- To understand basic essentials of epidemiology and pathogenesis of mycotic lesions of skin and mucous membranes.
- To classify mycotic lesions with due account for the localization of pathological process, the peculiarities of pathological manifestations, prevalence, lesion depth, the relation to the skin appendages.
- To single out characteristic clinical features of mycotic lesions of skin and mucous membranes.
- To generalize the principles of diagnostics, therapy and prophylaxis of mycotic lesions of skin and mucous membranes.

TO KNOW:

- Modern classification of mycotic lesions of skin and mucous membranes;
  The general flow and clinical manifestations of typical forms of mycotic lesions of skin and mucous membranes and possible complications;
  The principles and methods of diagnostics of mycotic lesions of skin and mucous membranes;
  The principles of treatment and prophylaxis of mycotic lesions of skin and mucous membranes.

TO BE ABLE TO:

- Correctly collect an anamnesis of a patient with mycotic lesions of skin and mucous membranes;
- Correctly conduct clinical examination of the patient with mycotic lesions of skin and mucous membranes;
- Conduct differential diagnostics of typical manifestations of mycotic lesions of skin and mucous membranes with diseases that have similar clinical picture;
- Assign a rational complex treatment to the patient with mycotic lesions of skin and mucous membranes;
- Define and take necessary measures for the prevention of mycotic lesions of skin and mucous membranes and their complications.

Epidemiology. Mycoses (fungal infections) of skin, its appendages and mucous membranes constitute a large group of lesions caused by the action of specific micro-organisms i.e. fungi that modern science included in a separate kingdom called *Fungi* or *Mykota*.

Biodiversity of fungi in nature is rather wide. Based on the taxonomists' data, there are nearly 1.5 million species of fungi, of which only 4% have been studied, to include over than five hundred registered species of fungi that are pathogenic to humans and animals.

The risk to humans and the need for commitment to antimycotic therapy against fungal infections is caused by:

the development of pathological changes in the skin, hair, nails and internal organs and breach of their functions;

prolonged chronic course, which is accompanied by serious complications (erythroderma, abscesses, erysipelas, elephantiasis, granulomatous and other neoplastic processes, etc.);

the formation of mycotoxicosis;

intolerance of antibiotics due to the presence of common antigenic determinants of fungi and antibiotics that are based on molds;

the development of secondary immunodeficiency;

prolonged duration of fungal infections, when fungi contribute to the development of allergies and can induce such diseases as eczema, atopic dermatitis, asthma, vasculitis, vasomotor rhinitis, etc.

Based on WHO data for different years, fungal infections occupy the first place in the structure of registered infectious diseases.

Opportunistic fungi cause so-called opportunistic fungal infection. Their development requires serious disorders in common resistance and especially in the immune status of the organism. In recent decades, the importance of opportunistic fungi and opportunistic fungal infections that they cause has increased dramatically in the context of HIV infection.

The sources of fungal diseases infection may steam from a sick person or an animal, as well as from plants and soil. Sometimes the cause of the spread of infection is a healthy-looking person, who is classified as an asymptomatic carrier of pathogenic fungi.

Conditions and channels of infection. Basic conditions for the development of mycoses are the presence of pathogenic fungus as well as various factors that reduce the body's defense in general and the skin in particular and, thus contribute to human disease. Only a relatively small number of pathogenic fungi – the agents of dangerous fungal infections, can cause disease in humans without any additional conditions.

The reasons leading to the development of fungal diseases are conventionally divided into two groups – the exogenous and endogenous. The first basically determines physiological decrease relative to the protective function of the skin against fungal infections, while the second adversely affects the human body as a whole and reduces its resistance.

Exogenous factors are rather diverse, where the leading role is played by superficial and deep trauma. Increase of skin moisture, maceration of horny layer of epidermis associated therewith and occurrence of surface erosion reduces the resistance of epidermis and creates confortable conditions for the penetration of fungi. Abundant sweating results in disruption of physicochemical properties of the skin, in particular in reduction of its acidity.

A contributing factor in the development of fungi is a combination of excessive humidity and temperatures.

Hypothermia and hyperthermia, certain anatomical defects (plastypodia, narrowness of feet interdigital folds), peripheral blood circulation disorder (acrocyanosis, eritrocyanosis, Friedlander's disease), trophic disorders (thinning of the skin, xerosis, callosity, keratoses) play a significant role in the development of fungal infections of skin.

The dynamics of some fungal diseases is influenced by seasonal factors.

Contamination of fungal infection may be contributed by certain habits and national traditions (smearing of hair with vegetable oil, common hats).

Endogenous factors that reduce the body's defenses against infection include intercurrent diseases, especially cancers of different origin, autoimmune diseases; metabolic diseases and hormonal disorders (hypo-and disproteinemiya; impaired glucose metabolism, impaired functioning of sexual and adrenal glands, thyroid gland; hypo-and avitaminosis; dysbiosis; malnutrition; gastrointestinal tract diseases; long-term use of antibiotics, corticosteroids, immunosuppressive and cytotoxic drugs). In recent years, greater importance has been gained by primary and secondary immunodeficiencies (especially the latter, in particular HIV-infection).

A certain role in the development of fungal diseases is played by the age, gender, ethnicity, socio-economic conditions, as well as genetic predisposition of some people to infection by fungi. Children and elderly people are more susceptible to it. Scalp mycoses are mainly observed in children, while adults often suffer from fungal infections on the feet and onychomycosis.

There are two main ways of infection and spread of fungal diseases i.e. direct and indirect. *Direct infection* occurs through direct contact of a healthy person with the sick one. Indirect infection occurs more often and is performed through various items contaminated with infectious material (skin flakes, nails particles, hair etc.) containing pathogenic fungi.

The spread of fungal diseases is largely dependent on the type of agent, source of infection, clinical presentation and localization of lesions.

Direct infection occurs via the contact with lesions on the skin, in common bed, during sexual intercourse, by kissing, as well as in public baths or bath if the latter is used by both sick and healthy people.

Direct infection often occurs in people who have domestic or professional contact with sick animals, whereat the animals are not only the source of infection, but also the carriers of pathological material from sick to healthy people.

The possibility of indirect fungal infection is caused by extremely high survival of pathogenic fungi in infectious material, whereat the fungi are not only able to exist for a long time in the environment outside the body of the patient, but also, under certain conditions, are able to reproduce on different subjects.

Infection can occur in hairdressing when you have you haircut, shave, manicure etc. Schools and kindergartens are the most common sites of fungal infections of children. Infection is contributed by the accumulation of children, their close contact with each other and sharing of infected objects. In the kindergartens fungal infections, especially microsporosis, often lead to epidemic outbreaks. From school or kindergarten the infection can get into the family. Most often, patients infect linen, slippers, socks, stockings, handkerchiefs and gloves.

A laundry can also be the source of spread of fungal infections. Poor laundered, not boiled and dried linen, which preserved fungi, can cause infection of healthy people.

Significant role in the spread of fungal infections, especially fungal infection caused by *Trichophyton rubrum*, is played by the foci of infection in the family.

Pathogenesis. Fungi are members of microbiota, and therefore have the properties of both animals and plants. The relativeness of fungi with animals is conditioned by the presence of chitin in the coat and that fungi are involved in nitrogen metabolism (splits the urea) and carbohydrate metabolism (splits the glycogen). Besides, fungal cells contain cytochromes that are involved in the process of breathing. The relativeness of fungi with plants is conditioned by the mode of eating (not through the absorption of dietary components, but through their absorption via the entire wall of the fungus) and their unlimited growth in the medium.

The development of fungal diseases of skin is determined by several factors i.e. pathogenicity and virulence of the pathogen and the state of human and environmental conditions that may contribute to infection and impact the course of the disease.

Clinical manifestations of mycoses, on the one hand, are caused by the destruction of horny layer of epidermis, hair and nails due to the growth and reproduction of fungi therein, and on the other hand – by diverse degrees of inflammatory reaction in dermis.

There are three main types of direct pathogenic effects of fungi on the human body i.e. *intoxication*, *sensitization* (mycetogenic allergy) and *infection* (the development of fungal infections of skin and other organs).

Many fungi are the causes of specific sensitization of human body, which has a peculiar form of allergic disease and induces different allergic reactions. Mycetogenic sensitization can manifest by general symptoms (e.g. by the symptoms of hay fever). Almost all pathogenic fungi cause specific sensitization of the patient's organism, which is accompanied by a kind of allergic complications, including secondary allergic rash.

Classification of mycotic lesions of skin and mucous membranes

It should be noted that today there is no any classification of these lesions that could be ideal and standard for everyone. At the same time, all over the world they use different classification of mycotic lesions of skin and mucous membranes, which determine only individual aspects of clinics, microbiology and pathology of fungal infections, but none of them, can solve a complex problem of diagnostics of these diseases.

In accordance with the latest recommendations of WHO Commission on terminology of nosologic forms of diseases and their causative agents, the names of infectious diseases are primarily determined on the basis of gender of their causative agent (e.g. rubromycosis, candidiasis, malassezia etc.).

In many foreign textbooks and manuals they use the classification of mycoses, which is based on the characteristics of localization of pathological process:

- scalp mycosis – *tinea capitis*;
- smooth skin mycosis – *tinea corporis*;
- face skin mycosis – *tinea faciei*;
- beard and mustache growth site mycosis – *tinea barbae*;
- mycosis of large folds of the body – *tinea cruris*;
- feet and hand mycosis – *tinea pedis et manuum*;
- onychomycosis – *onychomycosis, tinea unguium*.

This classification is useful from a practical point of view, but it does not take into account etiological peculiarities of the agents and pathogenetic features of the process.

There is a well-known classification of Professor Arievich A.M., modified by Professor Sheklakov N.D., under which they distinguish between the following classes of mycoses:

1. *Keratomycoses*: tinea versicolor, nodular piedra, tinea-imbricata.
2. *Dermatomycoses*: jock itch, mycosis caused by rubrophytosis, trichiphytia, microsporia, favus.
3. *Candidiasis*: superficial candidiasis of skin and mucous membranes, visceral candidiasis, chronic generalized (granulomatous) candidiasis;
4. *Deep (visceral, systemic) fungal infections*: histoplasmosis, coccidioidomycosis, blastomycosis, cryptococcosis, geotrichosis, chromomycosis, rhinosporidios, aspergillosis, penicilliosis, mucormycosis.

At the same time, recent microbiological studies have demanded to highlight some of the diseases that have traditionally been considered in a group of fungal infections as a separate group of pseudomycoses (surface forms – erythrasma, inguinal trichomycosis and deep forms – actinomycosis, micromonosporosis, nocardiosis, mycetoma) for the reason that their pathogens have been allocated to the special microorganisms occupying an intermediate position between fungi and bacteria.

Some authors' inclusion of pseudomycoses (as well as the name itself) into the aforementioned classification is hardly feasible and is only a tribute to the tradition.

Also, certain changes have taken place in regard to nosological term «tines versicolor», which is recognized today as one of the many clinical forms of skin *Malassezia*.

For clinical practice, classification of disease should contain data on the pathogen, form of infection (acute or chronic), the peculiarities of pathological manifestations (clinical, morphological and pathophysiological) – systemacity, type, prevalence, depth of lesion, relation to skin appendages (hair, nails), localization and epidemiology.

Clinical classification of mycotic lesions of skin and mucous membranes

1. Keratomycoses (nodular piedra, tinea-imbricata)

2. Dermatomycoses (epidermomycoses)

2.1. Tinea pedis

2.1.1. Intertrigo form

2.1.2. Squamous form

2.1.3. Dishydrotic form

2.1.4. Acute form

2.1.5. Mixed forms

2.2. Smooth skin dermatomycoses (Tinea corporis)

2.2.1. Smooth skin microsporosis

2.2.2. Smooth skin trichomycosis

2.2.3. Rubromycosis

2.2.4. Clinical types of smooth skin mycosis

2.3. Dermatomycosis of hands (Tinea manuum)

2.3.1. Squamous (hyperkeratonic) form

2.3.2. Dishydrotic (eczematoid) form

3. *Trichomycoses*

3.1. Surface forms (scalp microsporosis, trichomycosis)

3.2. Deep forms (pustular form, infiltrative-purulent form)

3.3. Favus: surface (pustular) and deep (infiltrative) forms

4. *Onychomycoses*

4.1. Subungual onychomycosis distal-lateralis

4.2. White surface onychomycosis

4.3. Proximal subungual onychomycosis

4.4. Total dystrophic onychomycosis

5. *Candidiasis*

5.1. Skin candidiasis

5.2. Mucosal candidiasis

5.3. Chronic (granulomatous) candidiasis of skin and mucous membranes

6. *Skin Malassezia*

6.1. Surface (furfuraceous) non-inflammatory forms of Skin Malassezia

6.2. Follicular forms
6.3. Inflammatory forms
6.4. Neoplastic forms
7. Deep mycoses
7.1. Subcutaneous mycoses
7.2. Opportunistic deep mycoses
7.3 Endemic deep mycoses
7.4. Visceral and systemic mycoses
9. *Allergic and paraallergic reactions in fungal diseases*

Laboratory diagnostic methods. Clinical picture of fungal diseases of skin is very polymorphic, so in all cases, the diagnosis should be confirmed by laboratory tests.

Laboratory diagnostics of fungal infections consists of several stages. In clinical practice they usually limit themselves with microscopic and culture studies of contaminated material. If necessary, these methods are supplemented by immunological, molecular, histological studies and infection of experimental animals. In some skin mycoses an important supporting role in the diagnostics is played by luminescence method.

Immediately prior to sampling pathological material the lesion should be treated with 96% alcohol or xylene solution. It is better to take material from fresh but already fully developed lesions. Skin scales should be scraped off from the periphery of lesions. In patients with fungal infections of scalp, the hair is usually pulled with epilation forceps.

Scrapings from surface lesions of affected nail plates shall be done with scalpel, while thickened nail plates should be cut with scalpel or nail clippers; subungual hyperkeratosis material can be obtained using a dissecting needle.

Liquid pathological material is collected aseptically in a sterile container, while skin scales, nails and hair particles – to the sheets of ordinary or soft parchment paper.

Mucous discharge to inoculate is taken with absorbent cotton swab which is then placed in a dry sterile tube or a tube with 2 ml of medium. For microscopy purposes, white-grayish plaque from mucous is taken with Volkmann's spoon or a glass slide.

If necessary, pathological material is transported to the laboratory in a special container or metal boxes. Received material is tested within one hour after sampling and at room temperature storage or within not more than three hours when stored in a refrigerator at 4°C.

Keratomycoses

Keratomycoses (*keratomycosis*) is a collective term, in which modern mycology combines mycoses affecting superficial part of horny layer of skin or hair cuticle. These mycoses include nodular piedra and tinea-imbricata.

Nodular piedra (synonym: piedra – from Spanish piedra, which means «stone») is a hair cuticle disease that is accompanied by the formation of white (*piedra alba*) or black (*piedra nigra*) nodules on the hair.

Etiology and pathogenesis. The causative agent of white nodular piedra is *Trichosporon Beigellii*, while of black nodular piedra – *Trichosporon Hortai*, which belong to yeast-like fungi. The development of the disease is favored by warm climate, wash of hair with standing water, sour milk and processing with mineral oil.

Epidemiology. White nodular piedra is sporadically met in Central Asia, the Caucasus, but mostly occurs in the UK, Hungary, Spain, France, USA and Japan.

Black nodular piedra is distributed mainly in Central and South America, Africa, India, China, Burma and Thailand. The disease is hardly infectious, but in endemic areas, its outbreaks are possible. It can be transmitted via brushes and combs.

Clinical picture. Subjective feelings are absent. White nodular piedra affects scalp, hair in the beard and mustache and in the genital area. Black nodular piedra affects only hair on the head. Multiple small, but very hard nodules are formed on the hair shaft (up to 20-30 nodules on each hair). They have irregular oval spindle-shaped form and almost completely cover the hair. Sometimes they merge, forming a short, in black piedra (1-2 mm) and long, in white piedra (up to 10 mm or more) sheath consisting of tightly bonded mycelium and spores of the fungus. Hair, in nodular piedra, does not break off.

Diagnostics. The diagnostics is based on clinical picture with due account for the anamnesis and the dynamics of symptoms. In complicated cases, the diagnosis can be confirmed by microscopic and cultural studies.

Treatment. Often, there is only local treatment of white and black nodular piedra. The patients are advised to shave the affected area, apply an antiseptic (such as hot solution of mercuric chloride 1:1000-1:2000, 0.01% sublimate vinegar or 2% solution of salicylic acid). You can also assign imidazole shampoos, lotions, creams.

Tinea-imbricata (synonyms: tokel, tropical circular mycosis) is a tropical fungal infection, characterized by the appearance of small brown spots on the trunk and limbs, which are eccentrically increasing in diameter and are covered with imbricate scales accompanied by mild itching.

Etiology. The causative agent of tinea-imbicata is *Trichophyton concentricum* – an anthropophilic cutaneous fungus, which does not hit the hair.

Epidemiology. Tinea-imbricata is a typical epidermomycosis of tropical countries, especially common in areas with high humidity. Epidemic foci are concentrated in Africa, Southern India, China, Indonesia, Vietnam, Laos and the Pacific Islands.

Factors contributing to the emergence of this disease include lack of hygiene of the body, excessive moisture and related maceration and stratification of epidermis. It is especially contagious in adults. Children get sick less often. Routes of infection may be both, the direct contact with patients and household items, especially bed clothes and underwear.

Clinical picture. Tinea-imbricata is characterized by skin itching, which is particularly enhanced in hot days with excessive sweating, as well as with consumption of alcohol, salty foods and swimming in the sea water.

The process is mainly localized on the trunk, extensor surfaces of extremities, buttocks, and rarely in other parts of the body. The disease begins with the appearance on the trunk of non-inflammatory small spots of light brown color, sometimes surrounded by barely perceptible narrow inflammatory aureole. Progressively as the eccentric growth and increase in size, in the center of each spot there appears, at first, small and then more noticeable desquamation.

In fully-formed elements of rash the central zone is slightly discolored and edged with an aureole of hanging scales, which in its turn is surrounded by dusky brown ring of hyperpigmentation. In a few months after infection the patient's skin is covered with numerous round plaques having eccentrically located rings of desquamation. They are often compared to the tiles or fish scales. The process can cover large areas of the body, where only face and scalp remain intact. The spread of the disease to the feet may cause hyperkeratosis and painful cracks when walking. As a rule, there are no abnormalities in general condition of the body.

Treatment. Usually they indicate daily warm bath with further processing of lesions with a pumice stone and use of various keratolytic ointments, creams or emulsions with salicylic acid. In acute cases, azole antifungals and terbinafine, topically, within two weeks are indicated.

Prophylaxis. To prevent recurrence of the disease and reinfection it is necessary to take a complex of sanitary and anti-epidemic measures, which include personal hygiene, detection and treatment of infected people, disinfection of clothing, underwear and household items.

Dermatomycoses (epidermomycoses)

Dermatomycoses are infections of skin and its appendages caused by cutaneous fungi. *Cutaneous fungi* are a group of filamentous fungi which attack dead skin keratinocytes. Dermatomycoses are divided into: *epidermomycoses* (infection of horny layer of epidermis), *onychomycoses* (nail infections) and *trichomycoses* (hair infections).

Etiology. There are more than forty species of cutaneous fungi, ten of which cause dermatomycoses. There are three genera of cutaneous fungi i.e. *Microsporum, Trichophyton, Epidermophyton*.

Epidemiology. Depending on the relation of cutaneous fungi with environment, they are divided into *anthropophilic* (transmitted from person to person via everyday objects and in contact), *zoophilic* (transmitted from animals in contact with a person or objects in use) and *geophilic* (found in soil).

Pathogenesis. Cutaneous fungi synthesize keratinase, which allows them to break down keratin, grow and develop in dead skin keratinocytes. The resistance to cutaneous fungi is determined by the state of cellular immunity and fungicidal activity of neutrophils.

Diagnostics. Dermatomycoses should be diagnosed with due account for:

- the clinical picture;
- fluorescent diagnostic data using a Wood's lamp (in microsporosis of hair affected by *Microsporum*, which gives greenish glow);
- the results of microscopic analysis of pathological material conducted to detect fungus elements (mycelium, spores etc.);
- the isolation of causative agent in the culture with its subsequent identification on Sabouraud 's medium (glucose-peptone agar-agar).

Treatment. External(local) therapy is effective only with smooth skin lsions. In case of affecting the hair or nails, it is ineffective.

Topical products: terbinafin, ketoconazol (gel, spray, cream) miconazole (cream), clotrimazole (cream), etc.

More effective are *systemic medications* that are indicated for the treatment of dermatomycoses, trichomycoses, onychomycoses in case of large lesions, inefficiency of topical products and significant inflammatory response. Systemic medications are those that are administered in different ways in the blood system and affect lesions via blood. *Systemic products*: griseofulvinum, ketoconazol, itraconazole, terbinafine, fluconazole.

Griseofulvinum has a fungistatic activity. It is mainly used in trichomycosis, especially microsporosis. Side effects: headache, epigastric syndrome, photosensitivity, hepatotoxicity. It requires regular blood, urine tests and hepatic function control.

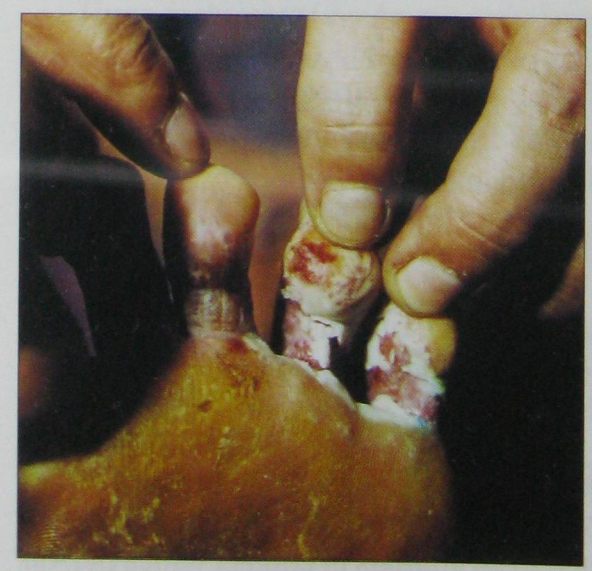

Fig. 7.1. Tinea pedis (intertrigo form).

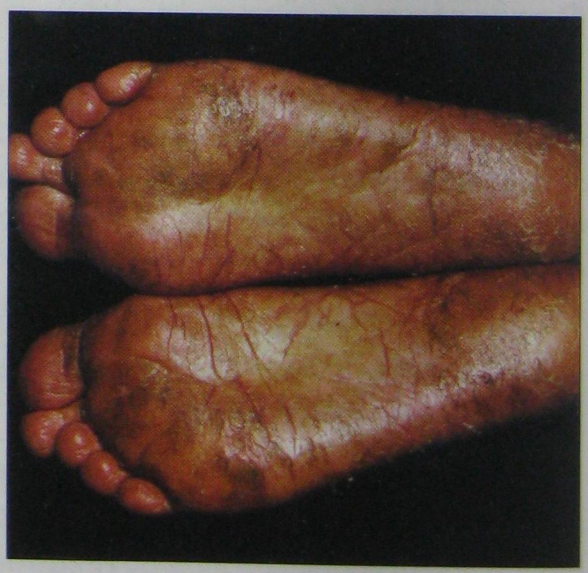

Fig. 7.2. Tinea pedis (squamous-hyperkeratotic form).

Ketoconazol has a fungistatic and partially fungicidal activity. It is used in treatment of dermatomycosis, candidiasis and malassezia. Side effects: epigastric syndrome, hepatotoxicity. It requires regular hepatic function control.

Itraconazole has a fungistatic and fungicidal action. It is used to treat dermatomycosis, candidiasis, malassezia, any cause of onychomycosis and deep mycoses. Side effects: epigastric syndrome, increased levels of hepatic enzymes.

Terbinafine has a fungistatic and fungicidal action. Amongst all systemic antifungals, it is the most effective against cutaneous fungi. Side effects: epigastric syndrome, aplastic anemia.

Fluconazole has a fungistatic and fungicidal action. It is used to treat superficial and deep candidiasis, cryptococcosis and tinea versicolor.

Tinea pedis is a chronic fungal infection of feet caused by cutaneous fungi.

Etiology. The agents of tinea pedis are: *Trichophyton rubrum, Trichophyton mentagraphytes, and Epidermophyton floccosum.* There is possible superinfection by yeast-like and mold fungi, to include staphylococcus microbiota.

Classification. Tinea pedis can be of intertrigo, squamous, dishydrotic, acute and mixed forms.

Clinical picture. *Intertrigo form* is manifested by maceration, desquamation, peeling of epidermis, cracks, erythema in the fourth, rarely in the third interdigital fold. In case of acute course of the disease it is often accompanied by bacterial (Gram-negative) flora with unpleasant odors.

Squamous form is characterized by well-demarcated erythema, mucous desquamation, keratinization first on the soles, in the interdigital folds, then on the lateral surfaces of feet and toes. With the affection of rear side of foot the lesions are outlined by typical inflammatory demarcated scalloped shape border. By moccasin type, the affection of feet can be integral.

Hyperkeratotic form is manifested by dense hyperkeratotic layers resembling corns.

Dishydrotic form is characterized by rash of vesicles and blisters on the instep that contain clear fluid and accompanied by itching. There is possible bacterial infection with formation of pustules. The course of the disease is not stable and is characterized by exacerbations in spring and autumn.

Acute form is manifested by erythema, edema, maceration, the formation of vesicles and blisters with considerable desquamation, erosion and

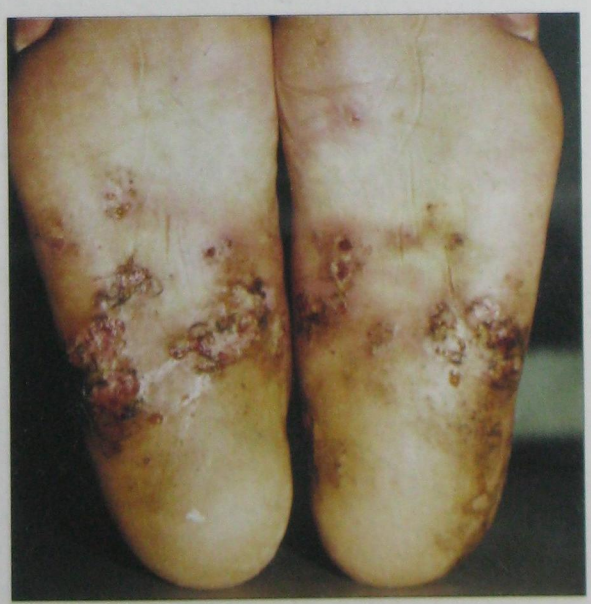

Fig. 7.3. Tinea pedis (dishydrotic form).

ulceration. Accession of a secondary infection may cause deterioration of health and fever, impetigo, lymphangitis, lymphadenitis, odor, itching and pain.

Differential diagnostics. Interdigital candidiasis is different from intertrigo tinea pedis by the appearance of bright red erosion with macerated whitish horny layer on the edges, often joined by candida onychia and paronychia. Squamous form of tinea pedis should be differentiated from psoriasis, tyioticum eczema, neurodermatitis, while dishydrotic and acute forms from impetigo, allergic contact dermatitis and dishydrotic eczema.

Treatment. Systemic therapy is prescribed for common forms, lack of effectiveness of external medications in dishydrotic and acute forms along with hyposensitizing drugs and antihistamines. Usually they prescribe itraconazole and terbinafine.

In acute inflammatory forms, external therapy involves, first, foot bath or lotion with aniline dyes, creams with antifungals in conjunction with antibiotics or corticosteroids, after remission of acute events – combined anti-inflammatory, anti-fungal, anti-bacterial creams (Lorinden, Oxycortum, Pimafucin, Pimafucort etc.).

In squamous forms it is recommended to apply creams and ointments with anti-fungal agents for two to four weeks.

In hyperkeratotic form first they usually prescribed dressings with creams or ointments containing antimycotics and keratolytics, and then – antifungal ointments and creams.

Prophylaxis. *Primary prophylaxis* is the use of individual rubber footwear during visits to public baths and pools, after which you should wash your feet with Myramistinum or Benzoyl peroxide solution. *Secondary prophylaxis* involves washing and treatment of feet with Myramistinum or Benzoyl peroxide solution, the use of antifungal creams, powders, boiling of cotton socks and disinfection of footwear with 20% formalin solution.

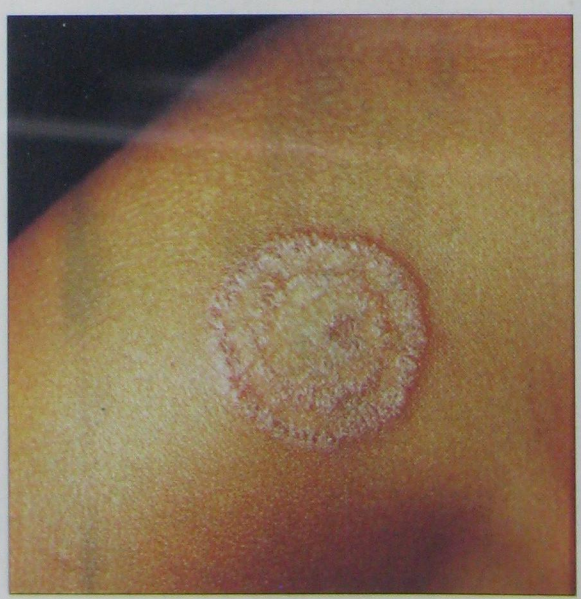

Fig. 7.4. Smooth skin microsporosis.

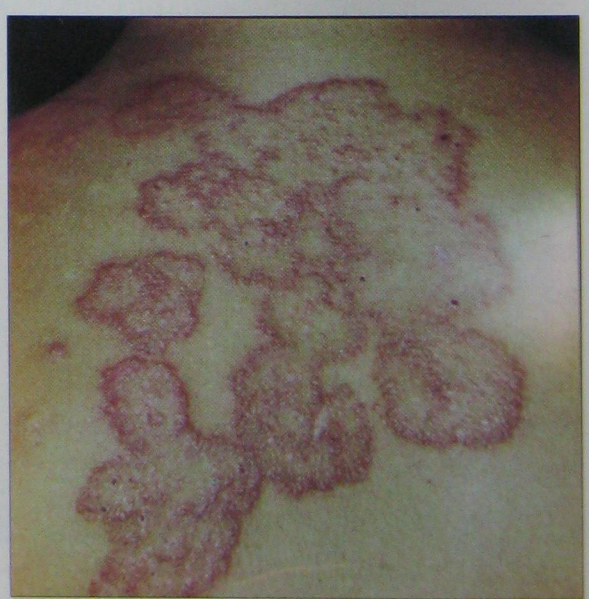

Fig. 7.5. Smooth skin trichomycosis.

Smooth skin dermatomycoses is a nosological concept that combines the affection of any part of the body, including rear sides of hands and feet, except for the soles, palms, scalp and axilla.

Etiology. The causative agents of smooth skin dermatomycoses are: *Trichophyton rubrum, Microsporum canis, Epidermophyton floccosum, Trichophyton mentagrophytes, Microsporum audouinii*.

Clinical picture. Clinical picture of smooth skin dermatomycoses depends on the type of pathogen.

Smooth skin microsporosis. On private parts of the body there are small lesions in the form of medallions with minor inflammatory phenomena, papules and vesicles, possible peripheral «ring in the ring» growth.

Smooth skin trichomycosis. In open areas of skin that are prone to friction and trauma, there appear annular lesions with clear margins and inflammatory border at the periphery, typical desquamation and hyperpigmentation in the center. If chronic, the clarity of foci is often lost. Trichomycosis and microsporosis are often combined with scalp affection.

Smooth skin rubromycosis is the most common form, naturally combined with feet rubromycosis and onychomycosis, and least with rubromycosis of folds and palms. There can be any localization of lesions; it is asymmetrical; often the disease begins on large folds, palms and soles. It is characterized by peripheral growth and fusion centers with the emergence of coherent site of moderate erythema and desquamation with general scalloped border.

Clinical types of smooth skin mycosis are: classic ringed with flaking and medallions; psoriasiform; verrucous; deep infiltrative dermatomycosis caused by zoophilic and geophilic fungi.

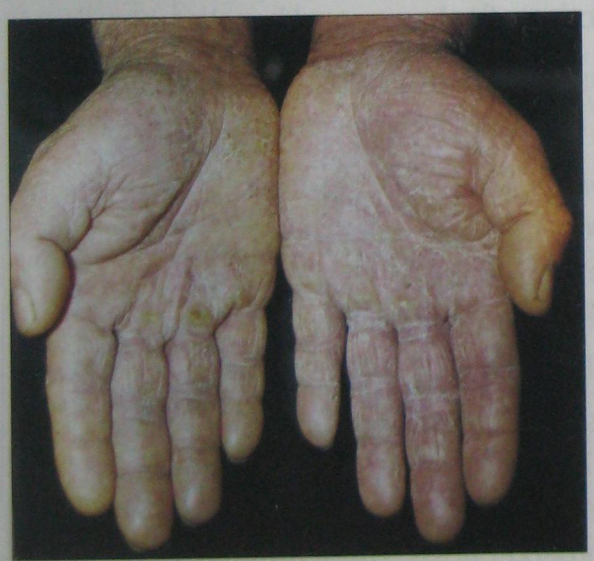

Fig. 7.6. Palm rubromycosis.

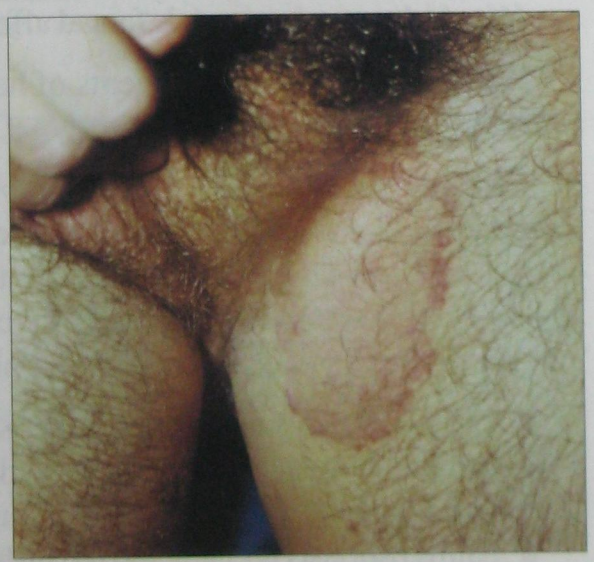

Fig. 7.7. Inguinofemoral rubromycosis.

Differential diagnostics. Differential diagnostics of smooth skin dermatomycosis is conducted with:

- seborrheic dermatitis, which in contrast to smooth skin dermatomycosis, is localized on the scalp, face, seborrheic areas in the form of low inflammatory yellowish-pink spots with fuzzy boundaries, and is accompanied by itching;
- contact dermatitis, differing by the appearance of erythema after the exposure to irritants obligate, edema, vesicles, cracks, erosion, and then followed by lichenification, excoriation, burning;
- psoriasis, which is characterized by skin lesions in different locations and the appearance of bright red papules, with silvery white scaly lesions on the surface, clear boundaries, a characteristic symmetrical peripheral location, merging of elements, and observation of three psoriatic phenomenon when scraping;
- limited neurodermatitis, which is characterized by the appearance of lichenification on the skin, flat papules on the periphery, hyperpigmentation, lack of vesicles and oozing and significant itching;
- white lichen manifested by confluent pale pink spots of irregular shape, with small scales on the surface and a slight itching;
- microbial eczema, which, unlike smooth skin dermatomycosis, is characterized by the appearance of round-shaped foci with sharp edges of different localization and size, slight oozing, exudation on the background of erythema and purulent crusts on the surface.

Treatment. Systemic antifungal therapy with Terbinafine or Itraconazole is assigned in common forms, in case of relapses and lack of effective local therapy. External treatment is carried out by means of creams and ointments with an antifungal action

Prophylaxis. Prophylaxis includes examination of organized children's groups and control of household contacts during six weeks, treatment of fungal infections and prevention from contact with people and animals sick for mycosis.

Hands dermatomycoses is fungal affection of palms caused by cutaneous fungi.

Etiology. The causative agents of hands dermatomycoses are: *Trichophyton rubrum, Microsporum mentagraphytes, Epidermophyton floccosum.*

Clinical picture. There are *squamous* (hyperkeratotic) and *dishydrotic* (eczematous) forms of hands dermatomycoses. In *squamous form* the skin on palms becomes yellowish-red with desquamation more pronounced in areas of folds; appears hyperkeratosis, sometimes – cracks, pain, itching. In *dishydrotic form*, you may observe erythema, microvesicles, itching, burning.

Differential diagnostics. In comparison with hands dermatomycosis:

- contact dermatitis is characterized by the appearance of erythema, edema, vesicles and cracks after the exposure to irritants obligate;
- palms psoriasis is characterized by hyperkeratosis, slight inflammation of skin around the periphery, bright red papules or pustules and nail infections;

- limited neurodermatitis is characterized by the appearance of lichenification, hyperpigmentation, flat papules and itching;
- dishydrotic eczema is characterized by a symmetrical process of vesicle deep in the skin filled with clear liquid and itching.

Treatment. Is similar to the treatment of tinea pedis.

Prophylaxis. Is similar to the prophylaxis of tinea pedis.

Trichomycoses

Trichomycoses are diseases that occur when hair is affected by cutaneous fungi. Depending on the location there are scalp and beard and mustache mycoses.

Etiology. The causative agents of trichomycoses are: *Microsporum canis, Microsporum ferrogineum, Microsporum aundouinii, Trichophyton violaceum, Trichophyton verrucosum, Trichophyton Shoenleinii.*

Clinical picture. Clinical features of trichomycosis depend on the type of pathogen and the depth of penetration of pathological process.

1. Surface forms of trichomycoses:

a) *scalp microsporosis* is characterized by the appearance of one or more lesions with clearly defined boundaries between 2 and 5 cm in diameter; smaller "drops" between 1 and 2 cm in diameter on the periphery; the hair on the site of lesions is broken off at the one and the same level i.e. above the surface of the skin for 4-6 mm; the skin is reddened, covered with grayish small scales. There is possible exudation with crusts, pustules and itching,

b) *scalp trichomycosis* manifests in the appearance of multiple isolated foci of up to 2 cm in diameter, with clear boundaries of irregular shape; the affected hair breaks off at a height of 2-3 mm above the surface of the skin; hair fragments are bent like a hook. Sometimes, hair is broken at the skin level, where it remains «stubs» or «black spots». Vesicles, crusts, pustules can be observed in the lesions. In chronic

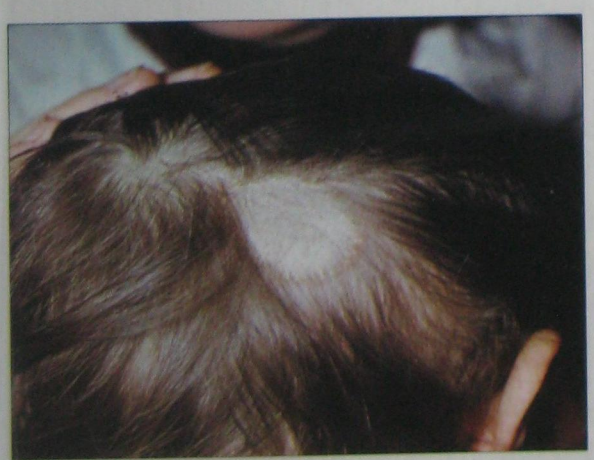

Fig. 7.8. Scalp microsporosis.

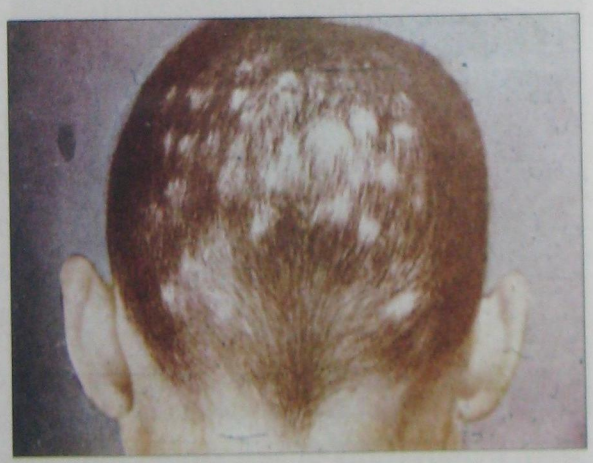

Fig. 7.9. Scalp trichomycosis.

trichomycosis adults may have focal or diffuse unobtrusive scaling, small atrophic lesions and «black spots»;

c) *favus (scald head)* causes the formation of a red spot on scalp skin , followed by the appearance of papules. In the center of papules there appears yellow pustule, which forms a skutula (fungal colony) in the form of a dry saucer-shaped dirty gray or yellow crust of 1-3 cm in diameter. Hair in the lesion become ash-gray, it is easily pulled out and does not break off. Later, the lesion is covered with solid crust, which has an unpleasant «mouse» odor. After skutula rejection, there is a scarring alopecia on its place. There are also atypical forms of favus without skutuls i.e. squamous and impetiginous.

2. Deep forms of trichomycoses:

a) *surface (pustular) form* is manifested by the presence of folliculitis, pus, crusts, which coalesce to form plaques (barber's itch);

b) *deeper (infiltrative-purulent) form* occurs on the scalp or on the face in beard, mustache, eyebrows areas; it is characterized by the appearance of pustules and formation of kerion – a painful purple-bluish infiltrative node of 6-8 cm in diameter. The kerion has a rounded shape, clear boundaries and bumpy surface. Then the node softens, yellow pus (a symptom of honeycomb) releases when pushing the hole, the hair is easily removed, it drops out and alopecia begins. The disease may be accompanied by such common conditions as fatigue, fever, malaise, headache and regional lymphadenitis. Dermatomykida appear in the form of patches, papules, eczematids, erythema nodosum.

Differential diagnostics. Unlike trichomycoses:

- staphylococcal impetigo is manifested by follicular pustules up to 5 mm, with hyperemia aureole or crusted papulo-pustules;
- abscess is characterized by the emergence of red unclearly-defined foci of irregular shape with tight edema, skin infiltration, hemorrhagic purulent exudate, pain, common symptoms of intoxication;

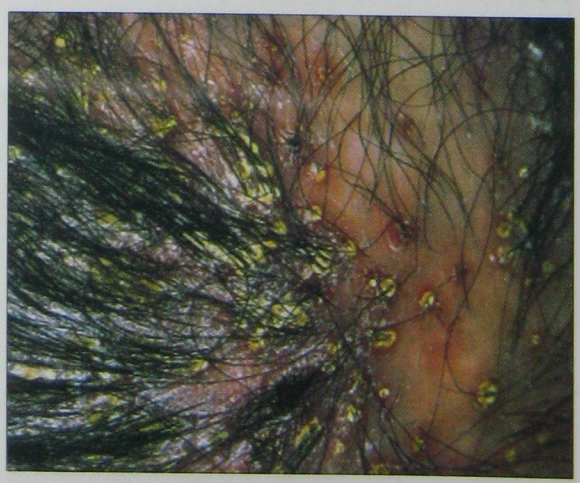

Fig. 7.10. Favus.

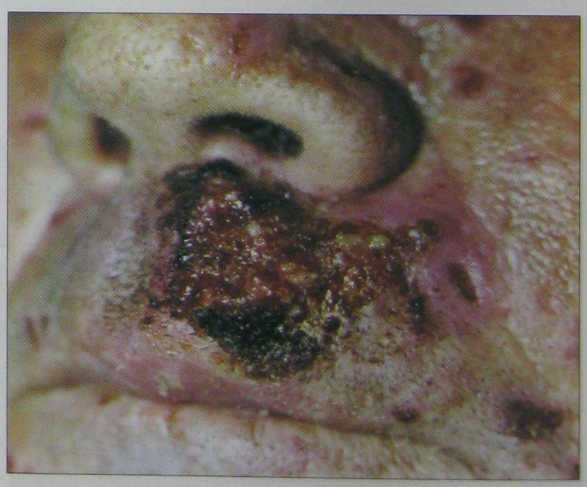

Fig. 7.11. Infiltrative and purulent (deep) form of trichomycosis.

- a boil arises where there is the hair shaft, around which there is a bright red infiltrate with tight-elastic consistency without clear boundaries, swelling, pain; then there is a softening that releases purulent-necrotic core that is removed together with a pus after breaking through; after regression boils remain scars;
- carbuncle, in contrast to deep forms of trichomycosis, is manifested by tight and painful infiltration with pustules and boils, followed by ulcers with uneven bottom and undermined edges, which then remain a scar;
- seborrheic dermatitis and malassezia eczematids differ from surface trichomycoses by inflammatory yellowish-pink spots on the face, scalp, in the folds and seborrheic areas, blurred focus, greasy scales, itching;
- psoriasis is characterized by skin lesions in different locations, especially scalp and nails, the appearance of bright red papules, with silvery white scaly lesions on the surface, clear boundaries and observation of three psoriatic phenomenon when scraping;
- limited neurodermatitis differs from surface trichomycosis by the appearance of lichenification on the skin, flat papules, hyperpigmentation and itching;
- alopecia areata is a hair loss that occurs for no apparent reason, without inflammatory changes in the skin, lesions are round, total baldness is possible and the hair is easily pulled out;
- vulgar (staphylococcal) sycosis occurs in men and is characterized by the formation of multiple drain folliculitis on the face in the beard and mustache areas on the background of erythema and edema, and chronic course.

Treatment. Systemic antifungals such as terbinafine, ketoconazole, griseofulvinum are applied. Locally, ointments and creams with antifungals are prescribed. In case of layering of a staph infection they use local combined antibacterial agents, while in severe course – systemic antibiotics or GCS. In case of kerion formation, it is recommended to make applications with antiseptics and remove hair manually.

Prophylaxis. Prophylactic methods include investigation of collective and household contacts during six weeks and washing head with ketoconazol shampoo.

Onichomycoses

Onichomycoses are infectious multifactorial diseases of nails, the causative agents of which are cutaneous, mold and yeast-like fungi.

Etiology. In the majority of cases onichomycoses are caused by antropophilic fungi: *Trichophyton rubrum, Trichophyton mentagraphytes, Trichophyton violaceum, Epidermophyton floccosum, Candida and zoofilnymi fungi Trichophyton verrucosum.*

Clinical picture. Depending on the localization of pathological process there are:

a) *subungual onychomycosis distal-lateralis* – on the free or side edge of nails of the first and fifth toes there appears a white spot, which soon becomes black, nail

plate loses its transparency and becomes thinner, can be separated from the nail bed, crack and crumble;

b) *white surface onichomycosis* – in the proximal part of the nail plate of the toes there appears a milky-white spot, which is growing and can take the entire area of the nail, leading to its destruction; the affected part is easily removed with a curette, the disease may be associated with subungual onichomycosis distal-lateralis;

c) *proximal subungual onichomycosis* – under rear nail fold of the toes there appears a white spot that extends distally;

g) *total dystrophic onichomycosis* is observed in case of loss of connection between a nail and its bed.

Differential diagnostics. Unlike onichomycosis:

- nail psoriasis is characterized by dotted deepening (a "thimble" symptom), loosening and thickening of nails, onichogriphosis, subungual hyperkeratosis and other psoriatic symptoms on the skin;
- trauma is manifested by nail changes resulting from subungual haematoma, nail destruction and affection of soft tissues;
- candidial onychia and paronychia are manifested by inflammation of nail folds, pus, change of a nail on the fixed edge, pigmentation, destruction, cracks, hyperkeratosis and are to be confirmed by laboratory microbiological tests.

Treatment. *Systemic therapy* includes application of Griseofulvinum during 4–12 months in hands onichomycosis and during 12–18 months in feet onichomycosis; of Itraconazole – during 2 months and 3 months respectively (by way of pulse-therapy courses); of Terbinafine – during 1,5 months and 3 months respectively and of Fluconazole – during 4–6 months and 6–12 months. *External therapy* is performed by smearing nails with 5% of iodine solution and topical antifungal preparations.

Prophylaxis. It is necessary to keep hygiene of nails and hands and feet skin, especially when staying in the area abundant for fungi (in the swimming pools, sauna,

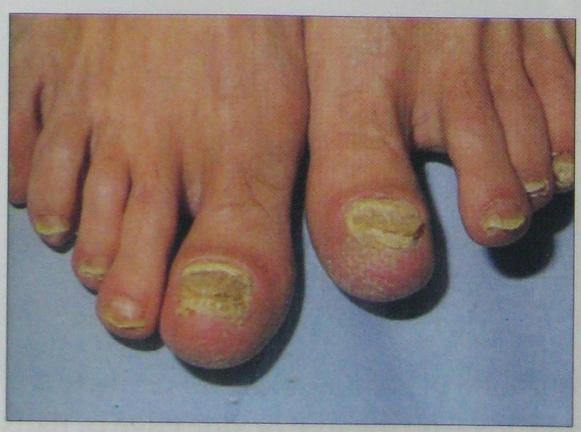

Fig. 7.12. Onichomycosis.

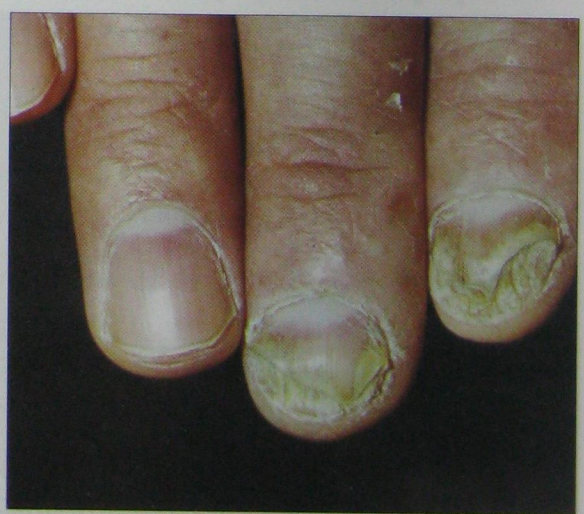

Fig. 7.13. Onichomycosis.

public shower rooms, on the beaches etc.). Also it is necessary to disinfect footwear with 25% of formaldehyde solution. It is recommended to wear antifungal socks.

Candidiasis

Candidiasis is a group of infections of skin, its appendages and mucous membranes caused by fungi of Candida genus.

Etiology. The main causative agent of candidiasis is *Candida albicans*, seldom – *C. tropicalis, C. parapsilosis, C. glabrata* etc. These belong to normal flora of oropharynx, gastrointestinal tract and vaginal area. They are yeast-like fungi that reproduce by budding and form pseudomycelium. *Candida* quickly grows on nutrient media, and after 48 hours, forms characteristic colonies.

Epidemiology. Exogenous candidiasis is rare. Candidiasis is primarily an endogenous infection. Due to unfortunate circumstances, own Candida goes into a pathological condition and cause candidiasis. Such risk factors include age, endocrine pathology (especially diabetes), impaired immunity, HIV infection, tuberculosis, prolonged use of corticosteroids, cytotoxic agents, anti-metabolites, immunosuppressants, oral intake of broad-spectrum antibiotics, especially without medical supervision.

Classification. Candidiasis is classified as follows.

1. *Skin candidiasis*: interdigital candidiasis; large folds candidiasis, small folds candidiasis, Candida folliculitis, diaper dermatitis, bandage dermatitis, candidiasis of nails and nail borders, paronychia and onychia, chronic candidiasis.

2. *Mucosal candidiasis*: a superficial mucosal candidiasis (stomatitis, glossitis, sore throat, thrush, atrophic candidiasis, candida leukoplakia, angular fissure), genital candidiasis (balanitis, balanoposthitis, vulvitis, vulvovaginitis), deep mucosal candidiasis (esophagitis, tracheitis, bronchitis, cystitis).

3. *Chronic (granulomatous) candidiasis of skin and mucous membranes*.

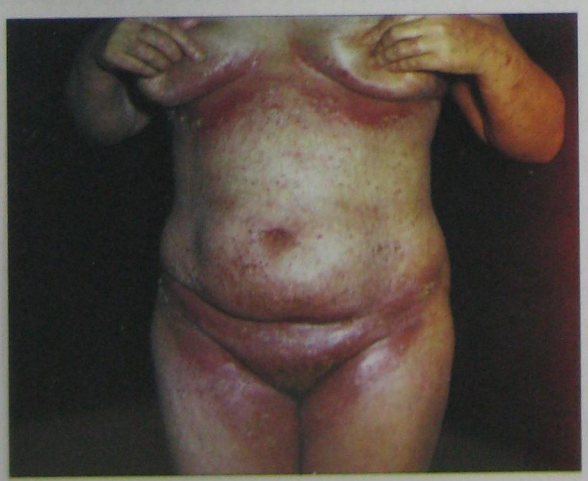

Fig. 7.14. Skin folds candidiasis.

Skin candidiasis is clinically divided into:

1. *Candidiasis of folds*. First appear erythema and phlyctena. Then on the place of phlyctena appears pink-red erosion, oozing, fusion followed by formation of a locus with scalloped edges, the periphery of which is covered with small «subsidiary» phlyctena. Patients complain of itching and burning.

2. *Intertrigo candidiasis* occurs in the interdigital skin folds of feet and hands in those who are in frequent contact with

the water. First appear phlyctena, on the places of which there is erosion and cracks, oozing. Then, there appear lesions covered with white bloom with a border of exfoliating epidermis. It is often associated with Candida paronychia and onychia.

3. *Diaper dermatitis* occurs in children in the buttocks, perineum and inguinal folds and on the inner thigh. It is manifested in the form of erythema, swelling, phlyctena, papules, erosions, oozing and lesions with peeling epidermis.

4. *Bandage candidiasis* occurs under occlusive plaster cast or in bedridden patients on the skin of a back or buttocks. It is manifested by erythema, edema, phlyctena, papules, erosion, ulceration, oozing.

5. *Candidiasis folliculitis* occurs in the same places as bandage candidiasis, and is characterized by the appearance of small papules delineated near the hair follicles.

6. *Candida paronychia and onychia* is typical of women – housewives, laundresses, dishwashers, waitresses etc. Initially, near the nails, there appears erythema, swelling of the skin around nail borders. When pressed, creamlike pus is released, palpation is painful. One may observe degeneration of the nail plate, discoloration and onycholysis.

Diagnostics. It is necessary to carry out a microscopy of pathological material after it being treated with alkali or the investigation of native or Gram stained preparation on the subject of finding both *Candida* cells and kidney cells and the presence of pseudomycelium, also they count CFU (colony forming units). To identify *Candida* species and enzyme activity it is necessary to carry out an inoculation on Sanuro medium or wort agar.

Differential diagnostics. Differential diagnostics should be performed with:

- seborrheic dermatitis and malassezious eczematid in which there are areas of inflammatory patches of yellowish color with fuzzy boundaries, in the folds and seborrheic areas, fat scales and itching;
- psoriasis, which in contrast to candidiasis, is characterized by skin lesions of various locations, the emergence of bright red papules with a silvery-white scales

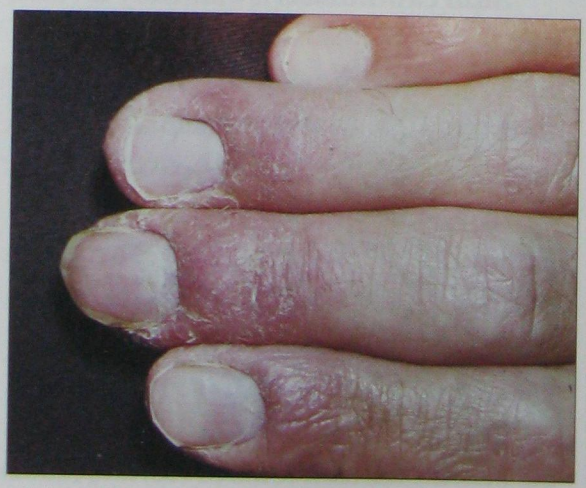

Fig. 7.15. Candida paronychia.

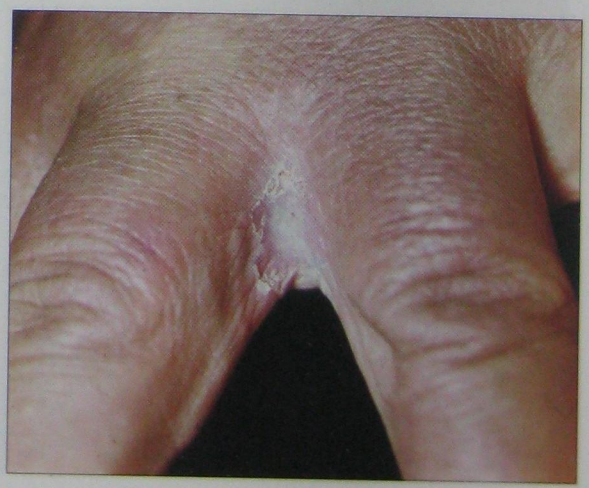

Fig. 7.16. Intertrigo candidiasis.

on the surface, characterized by clear lines, with the observation of three psoriatic phenomenon when scraping;

- dermatomycosis that differs by spots with clear boundaries, desquamation on the surface with peripheral growth, a fold with papules and vesicles on the edge, positive for cutaneous fungi;
- simple contact dermatitis occurs after exposure to obligate irritants and is characterized by the appearance in the impact place of erythema, swelling, vesicles, erosions, oozing, subjectively felt burning sensation;
- staphylococcal folliculitis is characterized by inflammation of the follicles with the appearance of purulent crusts and hair in the center;
- staphylococcal felon has a more pronounced inflammatory reaction, erythema, pain, pus;
- onichomycosis, unlike Candida onychia and paronychia, is characterized by nail infections on the free edge without inflammation of the nail fold, subungual hyperkeratosis, nail destruction and positive tests for cutaneous fungi.

Treatment. First, you need to pay attention to trigger factors (diabetes, immunodeficiency, chronic infection, vitamin deficiency, metabolic disorders, receiving of corticosteroids, cytotoxic drugs, antibiotics etc.).

General therapy involves administration of itraconazole 200 mg per day for 3-7 days, fluconazole – 150 mg per day for 1-3 days, in case of torpid process – 150 mg per day once a week for 2-3 weeks. Also they assign restorative drugs, immunomodulators and vitamins.

Topically they use 1-2% solution of methylene blue or 1% brilliant green, fucorcinum; creams with nystatinum and its derivatives. Smear the lesions until they completely disappear and then apply the cream for another week.

Prophylaxis. Preventive measures are aimed at keeping skin clean and dry. The folds should be daily treated with talc containing miconazole. In case of prolonged use of antibiotics and corticosteroids it is recommended to go through the preventive course of 150 mg of fluconazole per day once a week.

Mucosal candidiasis. The given pathology is clinically divided into:

1. *Superficial mucosal candidiasis* (stomatitis, glossitis, sore throat, thrush, atrophic candidiasis, candida leukoplakia, angular fissure):

- stomatitis, glossitis, pharyngitis and thrush are located on the tongue, buccal mucous and are manifested by the appearance of multiple white plaques 1-2 mm in size on an erythematous background, which are easily removed with dry gauze;
- in atrophic candidiasis there are smooth red lesions of atrophy;
- candida leukoplakia is characterized by dense white plaques, which are difficult to remove;
- with angular fissures, the corners of the mouth are covered with erythema, cracks, white coating, and pain.

2. *Genital candidiasis* (balanitis, balanoposthitis, vulvitis, vulvovaginitis)

- vaginitis and vulvovaginitis are characterized by hyperemia, swelling of mucous membrane of the vagina and cervix, white cheesy discharge, itching and burning, pain, exacerbation of these effects before menstruation;
- with balanitis and balanopostitis, first the glans penis and foreskin are covered with erythema, papules, phlyctena, then they are followed by erosion, pain, burning, swelling, cracks.

3. *Deep mucosal candidiasis* (esophagitis, tracheitis, bronchitis, cystitis).

Diagnostics. The diagnostics is similar to the one for skin candidiasis.

Differential diagnostics. Differential diagnostics should be conducted with:

- leukoplakia, which is characterized by the appearance of one or more sites of a whitish-gray color of different shapes and sizes without significant inflammation, sometimes joined by papillomatosis, hyperkeratosis, erosions and ulcers;
- lichen planus, which in the mucous manifests itself by the fusion of white (pearl) papules, Wickham's net and umbilical impression in the center of papules (if located on the skin), the formation of arcs, rings, waves in the form of a fern leaf (if located on the buccal mucosa);
- secondary syphilis, which unlike candidiasis, is characterized by patches of red circular shape with a smooth surface and sharp edges, and the pea size papules of red color with bluish tint, painless, dense, smooth surface, eventually becoming opal-white; sometimes there are erosions and ulcers; serological reactions are positive; manifestation of secondary syphilis are observed on the skin;
- other, primarily sexually transmitted infections, are sometimes similar to candidiasis, but do not have the typical clinical presentation, thus laboratory tests are required.

Treatment. *General therapy* involves the use of Fluconazole, Ketoconazole, Itraconazole, Pimafucin.

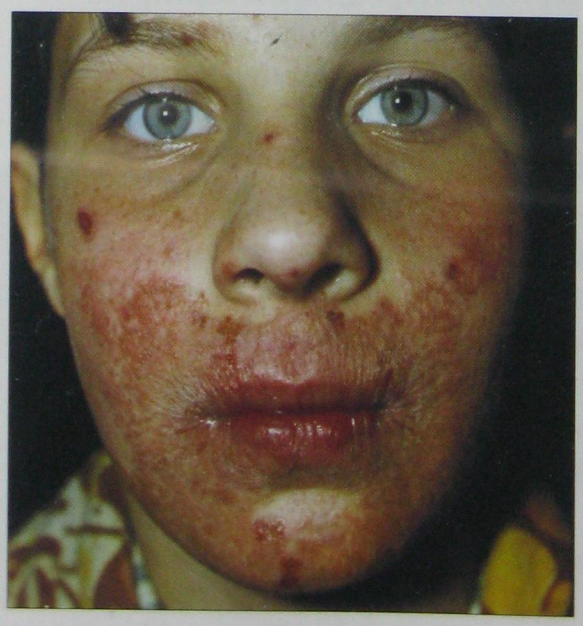

Fig. 7.17. Chronic candidiasis.

Externally it is recommended to apply as follows:

a) in glossitis, stomatitis, pharyngitis – nystatinum or clotrimazole in lozenges;

b) with angular fissure – 1.2% alcoholic solution of methylene blue or on a pre-dried surface;

c) with vaginitis, vulvovagintis – vaginal tablets Tergynan (to enter 1 tablet deep into the vagina at bedtime for 10 days) or vaginal capsules Lomexin (1 capsule at night inside the vagina for 3-6 days); clotrimazole are applied by softly rubbing or smearing the skin and genital mucous 1-2 times a day for 6-12 days.

Chronic (granulomatous) candidiasis of skin and mucous membranes. Clinically this disease is characterized by lesions of erythema with marked infiltration and hyperkeratosis, vegetations (called granulomas) tended to increase and then group. Often this disease affects children. In the mouth, there are widespread erythema with infiltration, erosion and angular fissures. One may observe paronychia, affection of palms. In some patients, such candidiasis is associated with HIV infection, immunodeficiency.

Treatment. *General treatment* includes the use of fluconazole, itraconazole.

Skin Malassezia

Skin Malsssezia (*malasseziosis cutis*) is a disease of skin and its appendages, which is caused by yeast-like lipophilic fungi of *Malassezia* genus.

Etiopathogenesis. According to the decision of the International Committee on Taxonomy of Fungi *Malassezia* genus is one of the fifteen genera of yeasts-like fungi that make up *Cryptococcaceae* family, which is part of a group of imperfect yeasts and refers to an anamorphic basidiomycetes.

The main factors of skin malassezia pathogenesis are disorders in proliferation and differentiation of epithelial cells; changes in the chemistry and physical properties of sebum; metabolic disorders (such as melanin disorder) caused by the impact of metabolic products of the fungus on to the host cells; immune disorders; inflammation and development of sensitization.

The source and the transmitter of the infection can be people, animals and plants. The spread of some forms of skin malassezia depends on the age, sex, profession etc.

Clinical picture. There is a diversity of clinical manifestations of skin malassezia, which is caused not so much by the kind of fungus, as by the patient's organism reactions (degrees and types of reactivity) that vary in a wide range – from asymptomatic carriage to systemic manifestations of infection.

Skin malassezia in patients is manifested not only as one nosology entity, but as a combination of two, three or even four entities.

Clinical forms of skin malassezia:

a) surface (scaly) non-inflammatory:

- Daria's Kerosis is a common hypertrophy of horny layer with a trend towards slight desquamation, coloring of skin into dirty yellow, dark brown or grayish color, openings of sebaceous glands pores (skin glands mouths' keratosis) and changes in the chemistry of sebum;

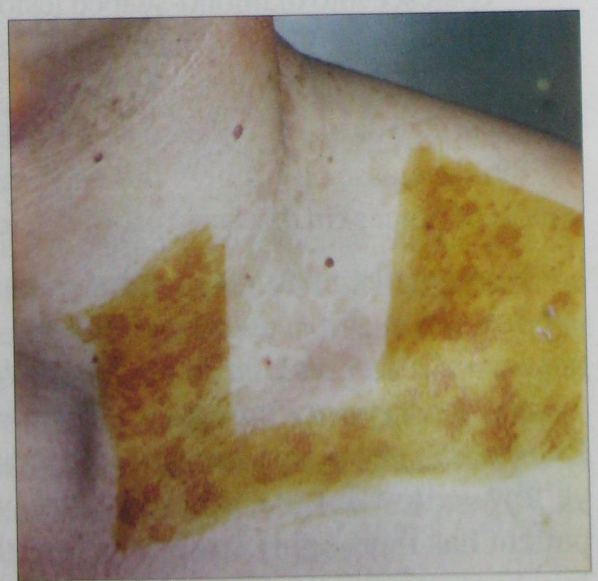

Fig. 7.18. Tinea versicolor (Balzer's test).

- simple common and limited pityriasis of trunk and limbs skin – unclearly delineated areas of defurfuration of non-inflammatory trunk and limbs skin with several layers of white or yellowish, dry or greasy scales or without them;
- simple pityriasis capitis of newborns (gneiss) and adults (dandruff) – unclearly delineated defurfuration of non-inflammatory areas of scalp, with several layers of white or yellowish, dry or greasy scales or without them;

b) follicular:

- comedones («black spots») are the formation of sebaceous plugs in the hair follicle consisting of fat and horny scales and have the form of black point;
- non-purulent malassezious folliculitis of trunk and limbs skin – a chronic dermatosis, which is manifested by small follicular inflammatory papules with pike-shaped scales on the surface etc.;

c) inflammatory (inflammatory and allergic)

- tinea versicolor – a dermatitis, which is characterized by the appearance on the skin of neck, torso and shoulders of slightly scaly spots of pink or yellowish-brown color;
- pitiriasiformis malassezia of smooth skin and scalp (pitiriasiformis Daria's eczematid of smooth skin) – clearly defined, red or reddish-yellow spots with scaly surface different in size, dry or fatty, circular or often ring-shaped;
- seborrheic dermatitis of scalp, trunk and extremities – chronic dermatitis, which is localized in seborrheic areas and is characterized by clearly defined common inflammation lesions, the surface of which is covered with fatty yellowish scales, also exudation phenomenon is observed;

d) neoplastic:

- confluent and reticulated papillomatosis – a dermatosis that is characterized by the appearance in the chest, epigastric and interscapular areas, as well as on the back of the hands, of small red quickly darkening and coarsen papules that coalesce into diamond-shaped lesions;
- granulomatous skin malassezia – an erythematous, with plaque and nodular granulomatous infiltrate of the walnut size with rough weeping pus, often vegetating surface or ulcers covered with thick scales, crusts.

Skin malassezia triggers the appearance of acne, eczema or complicates the course of other skin diseases (psoriasis, rosacea, atopic dermatitis etc.).

Diagnostics. The diagnostics is based on clinical picture, anamnesis and determination of pseudomycelium and yeast-like form of fungus in *uncoated, potassium alkali cleared or methylene blue-stained preparations*.

If you carry out fluorescent diagnostics of colored lichen, when viewing the smooth skin and scalp lesions via the light of Wood's lamp, you'll observe golden-yellow, yellow-brown or brown glow, which is considered to be typical (if the patient has thoroughly washed before the test, the results of this study may be falsely negative).

Baltzer's iodine test is used to determine latent desquamation when diagnosing pityriasis (tinea) versicolor – the spots are smeared with 2-5% alcohol solution of iodine; due to loosening of horny layer, the solution within rash areas is absorbed intensively and the spot becomes more contrast than the surrounding healthy skin. It should be noted that with leukoderma, which remains after tines versicolor, Baltzer'a test is negative, especially in persons, who were exposed to UV radiation. This test is also positive in Fox's impetigo.

Differential diagnostics. Unlike skin mallasezia:

- ichthyosis simplex is a chronic long proceeding dermatosis; at first there appears dryness, desquamation on the skin; soon it increases forming thin white scales across the surface;
- dry streptococcal impetigo is characterized by the appearance of pale pink irregular shaped spots with fine scales on the face, upper extremities, trunk, mainly in children and women;
- Devergie's lichen is a hereditary dermatosis, which is characterized by the appearance of pointed conical red papules around the follicle with a whitish-gray scales, which coalesce and form a generalized rash (erythroderma); one may observe "grater" syndrome and hyperkeratosis on the palms and soles; nails and har change;
- contact dermatitis caused by tar and oils occur in the event of prolonged exposure of the skin to these stimuli; the latter becomes dry with slight desquamation; first appears follicular hyperkeratosis and then it is followed by purulent folliculitis;
- at toxicoderma, after taking some medication, on the 7-10 day there appears itching and polymorphic rash prone to mergers; in 10-14 days all symptoms disappear even without treatment;
- lichenoid tuberculosis is characterized by the appearance of small papules on the trunk up to millet grain round and yellowish-brown in color; they are disseminated, sometimes grouped, with scales; other manifestations of tuberculosis are also important, thus additional tests are recommended;
- small-papular syphilid causes the appearance of small brown-red round or conical shape papules on the skin near the mouths of hair follicles; the surface is covered with scales or horny spikes, "grater" syndrome is observed; serological tests are positive, other manifestations of syphilis are present;
- bacterial folliculitis begins with genital inflammatory follicular pustules, in the center of which there is a hair, around – pink-red aureole and infiltrate in the recess, the affected area is painful; then a pustule dries up, purulent-hemorrhagic crust appears and pigmentation takes place;
- acne vulgaris – first they appear in the form of comedones on the face, back, then there is inflammation and the formation of papules; pus accumulates, results in formation of acne that breaks up and forms a crust, which is followed by the appearance of atrophic scar; acne vulgaris is typical of puberty;

- candidosis begins with the appearance of erythema, then phlyctena, erosion with scalloped edges, subsidiary pustules mainly in the folds; tests for *Candida* fungi are positive;
- Unna disease means the appearance on the scalp, the face, seborrheic areas of pale pink spots with yellow tint without clear boundaries of any size that fuse and greasy scales appear on their surface; the patient feels itching;
- Gibert's disease is characterized by the appearance of pink «mother» spot followed by pink spots of various sizes on the body that are not merged and have clear boundaries; first spots resemble crumpled paper then desquamation and itching start;
- psoriasis is characterized by skin lesions at various sites, especially the scalp, nails, the appearance of bright red papules with clear boundaries and silvery-white scales on the surface;
- vitiligo is characterized by the appearance of depigmented patches of various sizes and configurations in any area, often confluent, with bloodshot aureole; subjective feelings do not arise;
- dermatomycosis appears in the form of inflammatory patches of different sizes of round shape with sharp edges and desquamation on the surface, as well as with peripheral growth; a fold with papules on the edge; fungi tests are positive;
- atopic dermatitis is characterized by the appearance on the trunk, limbs and in the folds of lichenified and infiltrated areas of the skin that are merged; there are also small flat shiny papules, hyperpigmentation and hemorrhagic crusts;

The course and prognosis. Skin malassezia runs chronically, with relapses and stage-based transformations. The prognosis if favorable provided that the treatment is done properly and timely. But it is difficult to prevent re-infection via carriers or infected household items. Some forms of skin mallasezia are contraindications for certain types of professional activities.

Treatment. It is important to consider together all manifestations of skin malassezia and the nature of concurrent diseases in each patient, as well as their characteristics, since different forms of malassezia require different methods of treatment and may worsen or become complicated in case of irrational therapy.

Combination therapy:

- systemic antifungal therapy – oral intake of azole antifungals (fluconazole or topical causal treatment (application of selenium disulphide preparations to damp skin and hair daily for 10-14 days and then a cream, lotion or shampoo with other antifungal ketokonazole azoles once a week).

Prophylaxis. To prevent the relapse and re-infection, it is necessary to take a complex of sanitary-antiepidemic measures, which include: identification and treatment of infected people, disinfection of clothing, linens and household items.

Deep mycoses

Deep mycoses include subcutaneous mycoses, opportunistic and endemic deep mycoses. *Subcutaneous mycoses* affect dermis, deep muscle tissue and sometimes bones. Infection is caused by skin injury and penetration of a causative agent contained in soils and plants therein. This is the way how chromomycosis, sporotrichosis, eumycetoma and other disease appear. *Opportunistic deep mycoses* are infectious diseases caused by opportunistic fungi on the background of severe immunodeficiency. They affect visceral organs and deep tissues. Their causative agents are quite common soil fungi that infect humans by spores that enter the body via medical instruments during blood transfusion or via contamination of wounds and dressings. A part of opportunistic fungal infections are AIDS-associated infections (cryptococcosis, candidiasis, aspergillosis etc.). *Deep endemic mycoses* (respiratory) are caused by dimorphic fungi, which parasitize in the soil of certain geographic regions. These mycoses are represented by histoplasmosis, blastomycosis, coccidioidomycosis, endemic penecilliosos and manifest as an acute pneumonia with abortive course. Causative agents of these diseases do not transmit from person to person, but infection is poaaible via work with a particular culture.

The most spread deep fungal diseases are: chromomycosis, sporotrichosis, eumycetoma and pseudomycosis (actinomycosis).

1. The causative agent of tinea versicolor is:

A. *Microsporum* genus fungi
B. *Trychophyton* genus fungi
C. *Malassezia (Pityrosporum)* genus fungi
D. *Candida* genus fungi
E. *Rhodotorula* genus fungi

2. When viewing skin lesions of tines versicolor via Wood's lamp their color is:

A. Bright green
B. Golden yellow
C. Dark blue
D. Violet
E. White

3. Which of the below stated disease is real mycosis?

A. Mycosis fungoidea
B. Trichomycosis axillaris
C. Actinomycosis
D. Erythrasma
E. Candidiasis

4. Which of the below stated preparations is not an antimycotic agent meant for local therapy of mycoses?

A. Ketoconazole
B. Metronidazolum
C. Clotrimazole
D. Sulfur ointment
E. Nystatinum

5. Which of the below stated disinfection matters is reasonable to use against mycoses?

A. Ethyl alcohol
B. Formalin
C. Medicinal soap
D. Benzyl benzoate
E. Paracetamol

6. To conduct mycological test for cutaneous fungi they use inoculation for:

A. Sabouraud's medium
B. Peptone water
C. Muller broth
D. Price's medium
E. Bordet-Gengou potato blood agar-agar

7. The causative agent of actinomycosis is:

A. *Microsporum audouinii*
B. *Trichophyton mentagrophytes*
C. *Epidermophyton floccosum*
D. *Actinimyces bovis*
E. *Malassezia pachydermatis*

8. Which of the below stated do not belong to clinical forms of onichomycosis?

A. Subungual distal-laterallis
B. Interdigital
C. White surface
D. Proximal subungual
E. Total dystrophic

9. Which of the below stated is not a highly contagious infection?

A. Rhinosporidiosis
B. North American blastomycosis
C. Histoplasmosis
D. Coccidioidomycosis
E. Rubromycosis

10. Which of the below stated is typical of scalp trichomycosis?

A. In place of the lesions all hair is broken off at a height of 4-6 mm from the skin surface
B. The hair is broken off at a height of 2-3 mm from the skin surface; also there are "stubs" or "black spots"
C. Presence of skutula
D. Nikolsky's symptom
E. Hardy's symptom

Task 1. A farm worker complains of the appearance of painful rash on the scalp. *Objectively*: there are three well-defined infiltrated circular focus of 3-4 cm in diameter are found on the scalp; the surface is covered with crust, after removal of which there is pus in the hair follicles. Submandibular and cervical lymph nodes are enlarged to the size of a plum and are painful.

a) What preliminary diagnosis can be set?
- **A.** Scabies
- **B.** Scalp microsporosis
- **C.** Surface scalp trichomycosis
- **D.** Scalp psoriasis
- **E.** Infiltrative-purulent scalp trichomycosis

b) Which is the most possible course of the given disease?

Task 2. The dermatologist has been addressed by 62 years old patient complaining of change of color and thickening of nail plates on the feet. The patient has been ill for more than 10 years. *Objectively:* nail plates on all feet fingers are thickened and of dirty yellow color. In the course of microscopic examination of nail plates material treated with alkali solution, there were defined mycelium threads.

a) What diagnosis can be set?
- **A.** Panaricium
- **B.** Onichya candida
- **C.** Nail psoriasis
- **D.** Dystrophy of nails
- **E.** Feet rubroonichomycosis

b) Which dosage forms and pharmaceutical combinations would be reasonable to use for local therapy in this case?

Task 3. Four months old child's mother is complaining of reddening of the baby's skin between buttocks and inguinal region, which appeared on the 16th day after birth. *Objectively:* the skin between buttocks and inguinal folds of a baby is bright red, the lesions are big and clearly bordered and the surface is wet, covered with easily removable white film. Pseudomycelium was found during microscopic tests.

a) What diagnosis can be set?
- **A.** Diathesis
- **B.** Eczema
- **C.** Cutaneons diphtheria
- **D.** Toxicodermia
- **E.** Candidiasis

b) What pathogenetic feature the physician should pay attention to?

Task 4. Thirty one year old patient addressed the doctor with complains of rashes on the feet. She associates the disease with increased sweating. *Objectively:* the soles, side surfaces of fingers are covered with erythematous, vesicular elements, some of which have merged and formed lesions.

a) What diagnosis can be set?
- **A.** Restricted neurodermatitis
- **B.** Contact dermatitis
- **C.** Psoriasis
- **D.** Dishydrotic form of tinea pedis
- **E.** Erythrasma

b) What is the examination plan for this patient?

Task 5. Nineteen year old patient addressed the doctor with complaints of rash in inguinal area and itching. *Objectively:* inguinal-hip folds are covered with circled sharply delineated pin spots; their peripheral part is elevated and is surrounded with an area of vesicular elements.

a) What diagnosis can be set?
- **A.** Lichen ruber planus
- **B.** Big folds epidermomycosis
- **C.** Pemphigus vulgaris
- **D.** Scabies
- **E.** Eczema

b) Make up a treatment plan. What peculiar features it has?

Answers to the quiz of the first level of complexity
1 – C; 2 – B; 3 – E; 4 – B; 5 – B; 6 – A; 7 – D; 8 – B; 9 – E; 10 – B

Answers to the quiz of the second and third levels of complexity
1a – E; 2a – E; 3a – E; 4a – D; 5a – B

8 TOPIC Dermatitis and Eczema

Dermatitis and Eczema are considered diseases caused by the immune response of the body to various allergens, which is accompanied by damage to its own tissues.

TRAINING AND EDUCATIONAL GOALS

- To form the modern concept of the allergodermatoses' etiopathogenesis
- To identify ways and likely conditions of the organism sensibilization
- To distinguish the factors affecting the course of allergodermatoses
- To generalize the characteristics of the allergodermatoses' clinical course
- To establishes the general principles of differential diagnosis of allergodermatoses
- To determine a rational treatment strategy of patients with allergic skin diseases

8.1 DERMATITIS

Dermatitis is considered an inflammatory lesion of skin caused by a direct effect of various exogenous factors.

TO KNOW:

- etiopathogenic characteristics of dermatitis;
- features of classification and clinical manifestations of dermatitis;
- meaning of 'monosensibilization' and 'polysensibilization';
- comparative characteristics of simple and allergic dermatitis;
- principles of treatment and prevention of dermatitis.

TO BE ABLE TO:

- properly collect patient history on dermatitis;
- make a diagnosis on clinical grounds;
- run diagnostic (skin) tests to confirm the diagnosis;
- make a differential diagnosis;
- assign individual pathogenetic treatment.

Etiology. The cause of dermatitis may be mechanical, physical, chemical and biological exogenous factors. Depending on the nature of skin exposure, the stimuli may be subdivided into unconditional (obligate) and conditional (optional).

Unconditional or *obligate* factors are the stimuli which may cause inflammation of patient's skin subject to considerable strength and duration of exposure: *mechanical* (friction, prolonged pressure), *physical* (UV radiation, high or low temperature, electric current, ionizing radiation), *chemical* (concentrated acids, bases, salts), and *biological* ones (some plants such as nettle, parsnip, etc.; insect bites). Due to exposure to skin of the unconditional factors, the patients may have easy contact dermatitis.

Conditional, or *optional* factors (allergens) – are the stimuli of physical, chemical or biological nature, to which patients become hypersensitive (sensibilization). Due to their exposure to skin, allergic contact dermatitis advances.

Classification. They distinguish between *simple contact dermatitis and allergic contact one*. A variety of simple contact dermatitis is an *artificial* dermatitis, which can occur due to excessive use of external dosage forms (with vigorous rubbing them or imposing the occlusive dressings, etc.), the use of too high concentration of solutions, wrong physical therapy appointment (too high doses of UV radiation, etc.).

For the duration of the flow, dermatitis may be of *acute* (up to two months) and *chronic* (more than two months) nature.

Development of chronic dermatitis is promoted by both exogenous (meteorological factors – long-term sun exposure, high humidity, strong winds; drafts; use of tight shoes or clothes) and endogenous factors (presence of vegetative-vascular disorders, endocrine diseases, metabolic disorders, decreased immune responsiveness, disorder of microcirculation and the like).

Simple contact dermatitis

Etiology. Simple contact dermatitis can occur in any person as a result of exposure to skin of unconditional stimuli. Mechanical factors (friction) may cause diaper rash, limited hyperkeratosis, when exposed to high temperatures – burns (*combuscio*), low – frostbite (*congelacio*) or freezing (*perniones*); when exposed to sunlight – solar dermatitis (acute or chronic), ionizing radiation – radiation dermatitis; if skin contacts with chemical agents – acute contact dermatitis, with biological factors – phytodermatitis. In this case, the clinical picture of simple contact dermatitis first of all depends on the irritating factor – its concentration, temperature, duration of exposure, etc., as well as on the individual characteristics of the patient's skin.

Clinical picture. The features of clinical manifestations of simple contact dermatitis are an acute onset after exposure to the irritant, absence of the latent period, rapid flow, lack of sensibilization. The lesion focus occurs in the region of contact of the pathogenic factor with the skin, has clear boundaries, precisely

reproducing the place of its contact with the skin, with no tendency to dissemination and diffusion of the process on the focus periphery. Patients are concerned about feeling pain and burning in the affected area. Typical signs are also considered continuous nature of the foci, monomorphic eruption, rapid regression of inflammatory processes in the skin after exposure to stimuli is over.

Depending on the nature, strength (concentration) and duration of the stimulus and individual characteristics of the patients' skin, there are following clinical forms of simple contact dermatitis:

a) erythematous – there are hyperemia and swelling of the skin in the foci;

b) vesiculobullous – appearance on the erythematous background of vesicles and bullous elements drying out along with formation of crusts or destroying with the formation of erosions, which are further epithelized;

c) necrotic ulcerous – development of tissue necrosis and ulceration with subsequent scarring.

With the development of chronic simple contact dermatitis (due to prolonged pressure, friction, sun exposure, exposure to ionizing radiation, etc.), there moderate hyperemia, dryness, peeling, hyper- or depigmentation, telangiectasia may be observed in the lesion foci; thickening and lichenification or, conversely, thinning with loss of skin elasticity, cracks may advance.

Differential diagnosis. The diagnosis of simple contact dermatitis is not complicated, it is made on the basis of complaints (pain, burning), the history data (rapid development of inflammation of the skin at the first contact with the irritant), characteristic clinical signs (development of dermatitis in contact with the irritant, clear boundaries of the foci, continuous nature of the lesion, monomorphic eruption). Simple contact dermatitis should be differentiated from epidermolysis bullosa (occurs in early childhood, has long relapsing course, some secondary changes in the skin areas of previous lesions); streptoderma (primary rash elements in the form of phlyctens, there are festering scabs); acantholytic pemphigus (often begins with the mucous membrane of the mouth, has a torpid course, positive Nikolsky's symptom and Asboe-Hansen sign, in smears from the bottom of erosions there are acantholytic Tzanck cells).

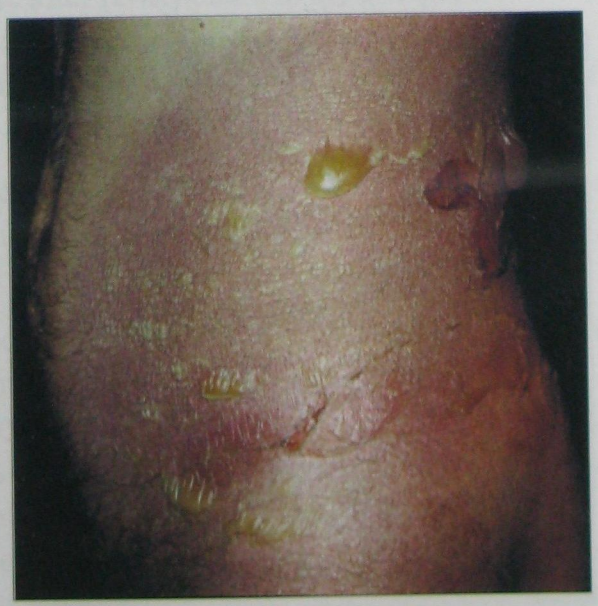

Fig. 8.1. Simple contact dermatitis (vesiculobullous form).

Treatment. The first step is to stop the exposure of etiological factor to the patient's skin. For small in size and mild forms of simple contact dermatitis, they use only external anti-inflammatory

therapy: if there an edema and sharp hyperemia in the focus – cool (2-3% aqueous solution of boric acid), corticosteroid creams. With the development of bullous elements, they should be pierced, smeared with aqueous solutions of aniline dyes; for moisting surfaces soaking therapy shall be prescribed; on erosive areas an epithelizing ointment shall be applied.

In the chronic form of a simple contact dermatitis during the exacerbation, they use corticosteroid ointment, and in the period of recourse – softening salicylic containing and absorbable ointments (with tar, sulfur, naphthalan, ichthyol).

For common skin lesions, the presence of multiple bullous elements and disorders of general condition of the patient, it is indicated a systemic corticosteroid therapy; to prevent secondary pyoderma, antibiotics or sulfa drugs shall be prescribes, to accelerate epithelialization – bio timulants, B vitamins, vitamins A and E, nicotinic acid.

Allergic contact dermatitis

Etiology and pathogenesis. Allergic contact dermatitis is caused by exposure of the skin to optional factors (allergens) that cause inflammation of the skin only in individuals with hypersensitivity to these factors (sensibilization, predominantly of monovalent one). According to WHO, in recent decades, the world has been recording an increase in the incidence of allergic diseases of the skin as a result of sensibilization of the population of chemical environmental factors. The number of patients with allergic contact dermatitis in the overall number skin pathology comprises more than 20%.

The most common causes of allergic contact dermatitis are considered chemicals (nickel, chromium salts, etc.); some industrial chemicals; household chemicals, insecticides; cosmetics; metal jewelry containing nickel and molybdenum; synthetic

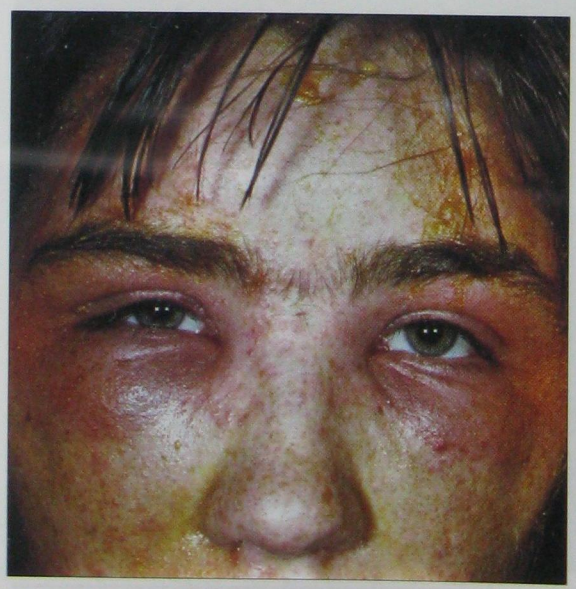

Fig. 8.2. Allergic contact dermatitis (after application of mascara to eyes).

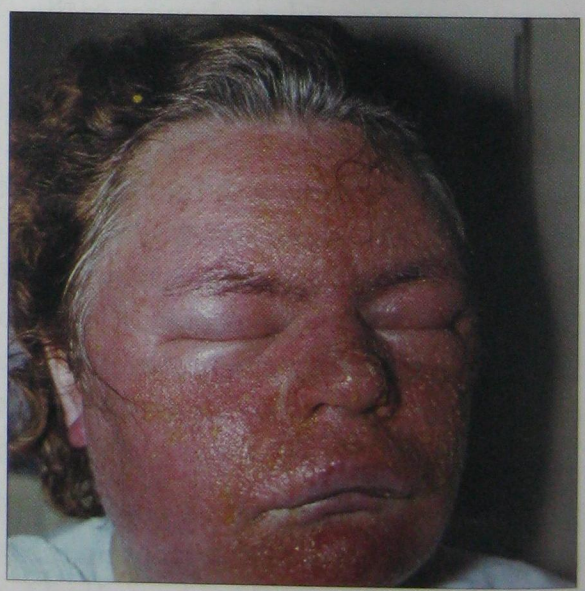

Fig. 8.3. Allergic contact dermatitis (after the application of cosmetics).

clothing; external medicine, and so forth as well as physical (sunlight) and biological (ragweed and other plants) factors.

The basis of histopathological processes in the skin upon contact allergic dermatitis is comprised in immunological delayed reaction. Period of sensibilization to exogenous factors can last from days and months up to years, which is largely dependent on the patient's genetic susceptibility to allergic reactions, and the level of immune reactivity of the nervous, endocrine and other systems of the patients.

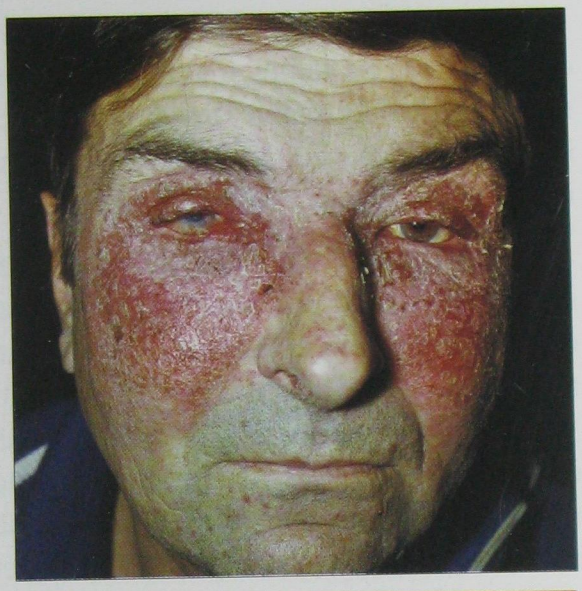

Fig. 8.4. Allergic contact dermatitis (after application of eye drops).

Clinical picture. Features of clinical manifestations of allergic contact dermatitis are considered its development after re-exposure of skin to exogenous factors (allergen), subacute onset (presence of the latent period – sensibilization period) and subacute or acute course.

The lesion occurs in the area of contact with the allergen, however, in contrast to the simple contact dermatitis, it has fuzzy boundaries with a tendency to spread the process beyond allergen contact with the skin. Lesions are of discontinuous nature, rash areas are alternates with those of healthy skin. The rash is characterized by polymorphism (patches, papules, vesicles less). Patients suffer from itching in places of rashes.

Contact allergic dermatitis is characterized by long duration after cessation of exposure stimulus and tendency to relapse after re-exposure to the allergen.

The diagnosis of allergic contact dermatitis is based on the patient's history (relapse after exposure to the allergen), clinical manifestations, and positive results of allergy skin tests.

Pathomorphology. There are intercellular edema in the epidermis, hyperplasia and hypertrophy of endothelial and perithelial vessels, narrowing of their lumen, perivascular infiltration.

Differential diagnosis. The diagnosis of allergic contact dermatitis is not complicated, it is made on the basis of complaints (itching of the rash), the data history (development of dermatitis after repeated exposure to the allergen), characteristic clinical signs (development of dermatitis in place of contact with the stimulus, fuzzy boundaries and discontinuous nature of the lesions, polymorphism of the rash). Contact allergic dermatitis should be distinguished from eczema, which is characterized by the formation of polyvalent sensibilization, presence of microvesicles and microerosions (symptom of serous wells) with phenomenon of oozing lesion, often has a multifocal nature of the skin lesions.

Contact allergic dermatitis should also be differentiated from the scabies, which is characterized by itching mainly in the evening and at night, symmetric localization of rash in typical for scabies areas, paired localization of rash elements, and presence of burrows and pathognomonic symptoms (Cesar, Hardy-Gorchakov, Michaelis) identification of same rashes in the family.

Treatment. First, it is required to determine the etiologic factor and to eliminate its effect on the skin. Patients are encouraged to hypoallergenic diet (to exclude the obligate food allergens, extractives substances, acute-irritating dishes, alcohol). Drug treatment should be comprehensive. With a limited process and moderate clinical signs of the disease, antihistamines (Cetirizine, Cetrine etc.) and corticosteroid creams or ointments of moderate impact shall be prescribed. In severe clinical manifestations of dermatosis, intense itching, sleep disturbances, complaints of neurotic nature, it is recommended an injectable form of antihistamines, sedatives (3% solution of sodium bromide, valerian extract, tincture of peony), vitamins A, E and C. In the event of multiple skin lesions or presence of the torpid form, systemic glucocorticoid medications (prednisone, dexamethasone, betamethasone, triamcinolone) on short-term courses should be administered.

Preventive measures. The patient should avoid repeated exposure to allergens, in case of confirmation of a professional nature of allergic factors, the suitable employment is required. Rational skin care, eliminating dryness by applying a number of emollients is recommended.

8.2 ECZEMA

Eczema is a chronic relapsing allergic skin disease that occurs on the background of a polyvalent sensibilization of the body and manifests through the inflammatory processes in the superficial layers of skin with the appearance of polymorphic rash and intense itching.

TO KNOW:

- etiopathogenic characteristics of eczema and its varieties;
- features of classification and clinical manifestations of eczema;
- stages of development of eczematous process;
- differential diagnosis of this disease;
- principles and features of dermatological tests helping to identify the allergens;
- principles of treatment, prognosis and prevention of relapse of eczema.

TO BE ABLE TO:

- properly collect patient history on dermatitis;
- make a diagnosis on clinical grounds;
- run diagnostic tests to confirm the diagnosis (Brocq scraping, skin tests);
- make a differential diagnosis;
- assign individual pathogenetic treatment.

Epidemiology. Eczema is one of the most common dermatitis; share of eczema in the structure of dermatological disease is 20 to 34%. Both men and women suffer from eczema most often at the age of 30-60 years.

Etiopathogenesis. Eczema is considered as *polyetiological* allergic disease, which is caused by the impact of a complex set of exogenous and endogenous etiologic and pathogenetic factors. Among the exogenous causes of eczema they distinguish *exogenous allergens* of chemical and biological, and environmental factors – risk factors of environmental, climatic, physical, occupational, psychological, hygienic, social plan. Endogenous pathogenic factors are important in the development and course of eczema – family history, changes in immune responsiveness, disorder of the nervous and endocrine regulation, presence of chronic foci of infection, impaired microcirculation, concomitant diseases of the digestive system, metabolic disorders, etc. They contribute to the formation of *endo-* and *autoallergens*.

Most often, patients associate debut and relapse of eczema with exposure to chemical agents (detergents and disinfectants, petroleum lubricants, construction materials, cosmetics, external dosage forms), trauma, microtrauma and burns of skin, use of trophallergens (milk, eggs, mushrooms, honey, chocolate, canned and smoked products, citrus fruits, etc.), exacerbation of varicose symptoms, nerve trauma or nerve strain, carried over colds, etc.

Clinical picture. Eczema occurs acutely, but subsequently acquires the features of chronic dermatosis with a tendency to relapse and resistance to treatment. The disease develops due to combined effect of exogenous and endogenous factors in patients with congenital or acquired predisposition to it.

There is no standard classification of eczema, on clinical manifestations and course there are following clinical forms of eczema: *true* (idiopathic), *microbial* (paratraumatic, varicose, mycotic, nummular), *seborrheic*, *professional* and *infantile*. According to the course of disease, one may subdivide *acute*, *subacute* and *chronic* eczema stages.

True (idiopathic) eczema occurs mainly after exposure to chemical agents encountered in industrial activity and in the home, or after use of certain cosmetics and drug products, as well as after eating trophallergens, endured neuropsychiatric stress or no apparent reason. Lesions are characterized by symmetry with the localization on the face, arms and legs with alternating areas of healthy and diseased skin. The foci have fuzzy boundaries with a tendency to spread, characterized by the current and evolutionary polymorphism of rash, severe itching. In the setting of severe erythema, there are multiple exudative papules, microvesicles, point erosion ('serous wells') with moist varying intensity. After drying appear the serous crusts, occurs desquamation. Around the main foci and in remote areas, allergids as eritemato-squamous and papules vesicular rash appear. In patients with long chronic dermatosis phenomenon of infiltration, thickening, congestive hyperemia, lichenification, dryness, peeling, cracks may be observed in the lesions.

Dyshidrotic eczema (clinical variety of true) occurs predominantly on the sides of the fingers, palms and feet, has a subacute course. In the lesions in the setting of low hyperemia, may appear small (pinhead) dense vesicles resembling the grains of cooked rice, and characterized by intense itching, sometimes burning.

Eczema keratosis (coarsen, tilotice) is a true manifestation of chronic form of eczema with localization in the palms of hands with the transition to the side and rear areas. Foci are characterized with clear boundaries, moderate erythema and severe infiltration with phenomena of lichenification, on the surface there are deep painful cracks with bloody crusts and big lamellar peeling. Resistance to treatment is marked. To exclude rubromycosis the patients are subject to mycological study of scaly crusts on mycology.

Among the clinical varieties of microbial eczema, the often recorded paratraumatic eczema, which occurs around nonhealing wounds after injury (micro traumas, cuts, burns of skin) due to sensibilization to pyococcus. In most patients, dermatosis is characterized by asymmetry, localization in the open areas of the skin (hands, arms, legs, face, neck). Lesions have clear limits with trimming like lamination of epidermis along the edge of the focus. At the core of the lesion in the setting of erythema and edema, it is observed a moderate oozing of point erosions, multiple seropurulent crusts, and on the periphery – pustular elements. On the lower extremities, the lesions are characterized by hyperemia of bluish tinge. In the case of exacerbation of chronic microbial eczema, the allergid eritemato-squamous and papular or vesicular papules elements appear around the principle focus and in remote areas.

Varicose eczema is a kind of microbial eczema, which occurs predominantly in elderly patients with chronic venous insufficiency during exacerbation of thrombophlebitis. Lesions are localized on the lower legs and feet, often around venous ulcers, characterized by clear boundaries, swelling of the skin, bluish tint, severe oozing, itching and feeling of painfulness, burning sensation.

Mycotic eczema is a kind of microbial eczema, which develops in the setting of course of a longlasting foot mycoses, confirmed by clinical and laboratory data. Lesions first appear on the skin of the feet, then spread to the lower legs, which is accompanied by a sharp itching, erythema, edema, vesiculation, weeping, painful cracks and erosions in the interdigital folds.

Intertriginous form of eczema is diagnosed mainly at persons of hypersthenic built, often in the setting of concomitant diabetes mellitus. Lesions in these patients are located in the major

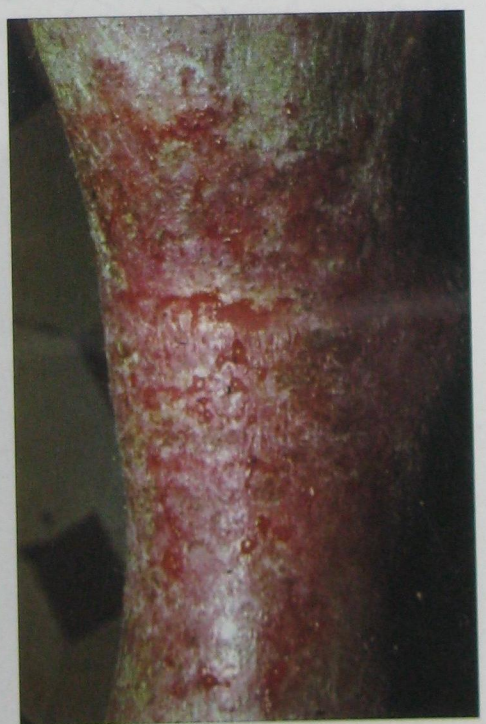

Fig. 8.5. True eczema.

folds (armpit, groin, under the breasts in women), have fuzzy boundaries, characterized by all the signs of eczematous process – presence of hyperemia, edema, polymorphous rash, oozing, might be accompanied by widespread allergids. Mycological study should be conducted in order to eliminate candidiasis of the folds.

Nummular eczema occurs mainly during the cold season, often for no apparent reason, sometimes – after suffering a cold. Its development is associated with sensibilization to the patient's chronic foci of infection. The patients have multiple symmetric clearly demarcated round or oval lesions of coin size preferentially localized on the extensor surfaces of the extremities, at least – on the back and sides of the torso. The skin in the foci has signs of hyperemia and infiltration, on the surface appear miliary papules, vesicles, occasionally – a small soak, and serous and serous purulent crusts, scaly crusts.

Foci of **seborrheic eczema** are localized mainly on seborrheic areas of the skin (scalp, postaural folds, face, sternum, interscapular region of the back), accompanied by intense itching. In the setting of erythema, in areas there are layers of scaly crusts, under which some moist erosive surface with moderate soak may be found, and on the edge of the lesions one may see an inflammatory rim with clear boundaries, covered with scales and crusts (seborrheic 'crown'). In the area of ears and postaural folds, the skin is flushed, swollen, wet, with diffuse oozing, in the folds of auricles – painful cracks. In the sternum area, the patients may have seborrheic dermatitis – round yellow and pink erythematous patches covered with greasy yellowish scales.

Infantile eczema develops at an early age against atopy as a manifestation of the exudative phase of atopic dermatitis. Often infantile (atopic) eczema is localized on the face and scalp, although it may be common. In areas, there are signs of true, microbial and seborrheic eczema in various combinations.

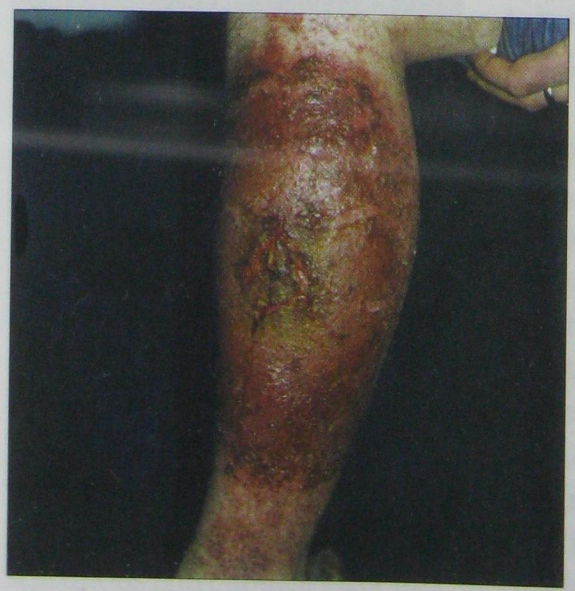

Fig. 8.6. Microbial eczema.

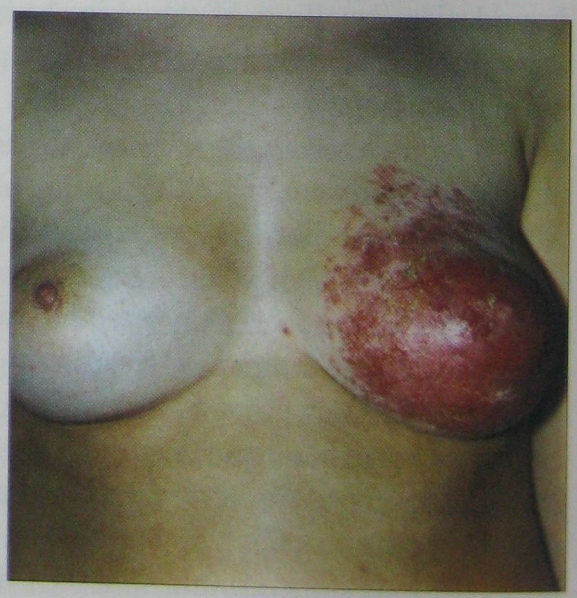

Fig. 8.7. Microbial eczema.

Occupational eczema is caused by exposure to sensitizing substances in conditions of production with primary localization on exposed skin (back of the hands, arms, face, neck, sometimes legs, feet), has clinical manifestations of true eczema. In areas against hyperemia and edema, multiple vesicles, oozing lesions may be observed. The course is continuous, regression of clinical symptoms occurs after removal of contact with the allergen. Diagnosis shall be made by occupational pathologist on the basis of history, clinical picture, disease course, and skin allergy tests.

Pathomorphology. In the acute phase of eczema, an intercellular edema of Malpighian layer, spongiosis with vesiculation may be detected on the skin. In the dermis, especially in the papilla, there is a notable expansion of blood vessels with perivascular, predominantly lymphocytic infiltration. In the chronic stage, acanthosis, parakeratosis and hyperkeratosis, and a powerful perivascular infiltration of cell (lymphocytes, histiocytes and tissue basophilic cells) may be revealed.

Diagnosis of eczema with typical clinical manifestations is not complicated. Polymorphic elements with microvesicles and microerosions with formation of 'serous well', oozing, typical localization of the foci, significant pruritus, duration of flow, tendency to relapse – are characteristics of it.

Differential diagnosis. Eczema is differentiated with allergic dermatitis (the process is limited, microvesicles occur occasionally, no oozing), atopic dermatitis (the dominant is infiltrative component of inflammation with marked lichenification, no polymorphism of primary morphological elements, oozing lesions with typical 'serous wells' are not observed), scabies (itching mainly in evening and night, symmetrically localized rash in typical for scabies areas, pair location of the rash, presence of burrows and pathognomonic symptoms of Sezary, Hardy-Gorchakov, Michaelis, revealing a similar rash in the family), Duhring dermatosis herpetiformis (characteristic

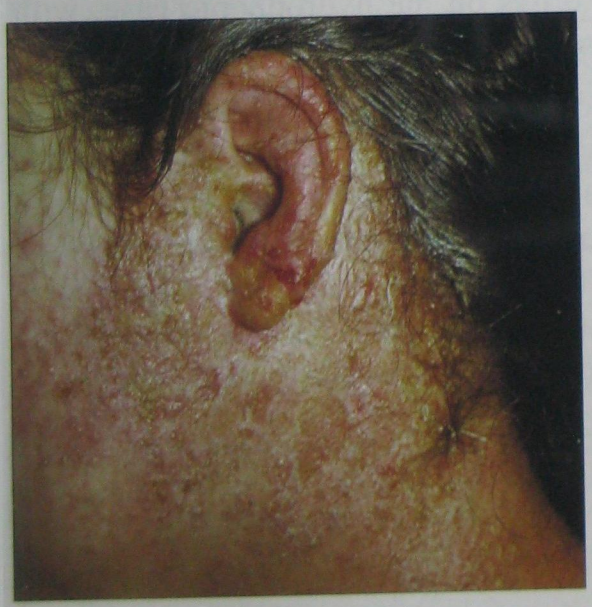

Fig. 8.8. Seborrheic eczema.

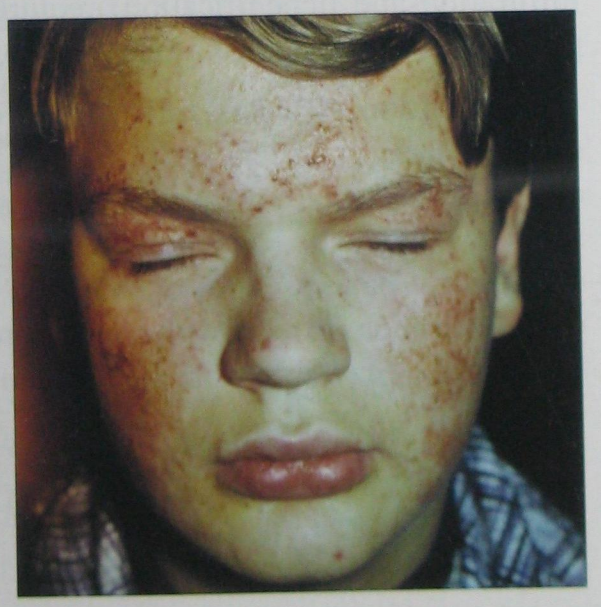

Рис. 8.9. Infantile eczema.

grouping of rash, eosinophilia in the blood and in the content of bubbles, positive Jadassohn test – new elements appear after the applicative test with 50% potassium iodide ointment). Squamous hyperkeratosis form of rubromycosis of hands and feet differs from chronic hyperkeratosis eczema with mucosa-like desquamation of the epidermis in the folds of skin, in cultural studies of scaly crusts fungus may be detected.

Treatment. The treatment program includes a hypoallergenic diet, systemic and topical treatment of dermatosis, and correction of neural and neuroendocrine disorders, sanitation of foci of chronic infection, limiting contact with water.

Hypoallergenic diet involves eliminating the obligate food allergens, extractive substances sharp-irritating dishes, alcohol, limiting of salt and carbohydrates.

Systemic therapy of patients with eczema includes antihistamines (chloropyramine, clemastine, loratadine, desloratadine, etc.). In the case of common and torpid forms of eczema the patients shall take glucocorticoids for systemic use (prednisolone, dexamethasone, betamethasone, triamcinolone). In case of sleep disorders, neurotic disorders, psychotropic drugs shall be prescribed: tranquilizers (diazepam, nitrazepam), sedatives drugs. The external treatment for eczema patients shall be administered differentially. In the acute stage of eczema with symptoms of oozing, the cold lotion with 2% solution of boric acid and 0.25% solution of silver nitrate is indicated. In severe forms (acute and subacute stages), the topical corticosteroids on a hydrophilic base containing mometasone furoate, fluticasone propionate, clobetasol propionate, etc. shall be prescribe. The oozing is over in severe dry skin, the moisturizing drugs contain fluocinolone acetonide shall be administered. If complicated with infection (fungal, bacterial, fungal and bacterial), combined corticosteroid drugs containing substances with antibacterial and antimycotic activity should be applied. Chronic forms of the disease (severe infiltration and lichenification) require drugs on the hydrophobic base, with prednisone, urea, betamethasone valerate, salicylic acid.

Prevention. Affected areas require constant care, which involves the use of neutral creams, emulsions and ointments, which, if necessary include corticosteroids and keratolytics, and emollients. In varicose eczema, the surgical removal of varicose veins, use of elastic bandages, rational treatment of venous ulcers may become obligatory. Patients are encouraged to hypoallergenic diet. They have contraindications to work with production allergens irritating the skin, and polluting substances, as well as to work in high humidity and high psycho-emotional stress.

8.3 TOXIDERMIA

Toxidermia or **toxic and allergic dermatitis** are diseases of skin, frequently of mucous membranes, mostly in the form of acute inflammation, caused by an allergic or toxic-allergic effects of substances penetrated into the patient.

TO KNOW:

- etiopathogenic characteristics of toxidermia;
- features of classification and clinical manifestations of toxidermia;
- meaning of 'monosensibilization' and 'polysensibilization';
- comparative characteristics of toxidermia;
- principles of treatment and prevention of these diseases.

TO BE ABLE TO:

- properly collect patient history on toxidermia;
- make a diagnosis on clinical grounds;
- make a differential diagnosis;
- assign individual pathogenetic treatment.

Epidemiology. The term 'toxidermia' is considered conditional one, since in most cases the basis for their development are allergic mechanisms. The most common are *drug toxicoderma*, second in frequency is *food toxicoderma*, *occupational toxicoderma* is rarely seen. In foreign medical literature, the concept is limited only with medication exanthemas.

Etiopathogenesis. Development conditions of toxidermia are:

1) penetration of allergen, substances into the bloodstream,

2) presence of immunity to these substances, which may be congenital in the form of idiosyncrasy or acquired as a monovalent or polyvalent sensibilization

The causes of toxicoderma are *drugs* (antibiotics, sulfonamides, nonsteroidal anti-inflammatory drugs, analgesics, vitamins, etc.), *food* (food allergens or substances formed during their long-term storage or damage, as well as food additives – preservatives, dyes) *industrial and household chemicals*. Drug toxicodermatoses are the most frequently observed in clinical practice. Severe forms of toxicodermatoses often may cause a death.

The main mechanism of toxidermia development is considered *allergic* one, rarely – *toxic* one. Drug allergy is the result of penetration into the body of minimum amount of the drug; its manifestations do not conform the pharmacological action of drugs. Allergic skin damage is realized through immunological mechanisms (B- and T-immunity) in the form of various types of ***allergic reactions*** (on humoral or cell type): *type I, anaphylactoid* (allergic reactions of immediate type) – caused by IgE, most often occurs with the introduction of drugs and manifested, as a rule, as a urticarial rash (urticaria, angioedema, anaphylactic shock); *type II, cytotoxic* – caused by IgG and IgM or complement and is often manifested as a purple rash; *type III, immune deposit* – caused by circulating immune complexes, and often manifests itself in the form of vasculitis and urticaria; *type IV, cellular* (delayed allergic reaction) – caused by T-cells and occurs mainly in the form of papular rash.

They also distinguish ***non-immune mechanisms*** of acute drug toxidermia development – *idiosyncrasy* (congenital intolerance to certain drugs), and *polypragmasy* (concurrent use of large amounts of drugs from different groups, metabolism products of which may increase the allergenic effects of each other).

Clinical picture. Clinical manifestations of toxidermia with few exceptions are considered non-specific. One and the same substance can cause different patients have different symptoms. One and the same clinical picture may develop under the influence of different chemical substances. However, for most toxidermia are characterized by the following *common features*: acute onset and rapid course, patient complaints of itching skin rash; spread and symmetry of the rash; rash is localized not only on the skin, but also on the mucous membranes; disseminated nature of the rash location; the rash frequent polymorphism; the presence of urticaria elements; presence of intoxication syndrome, etc.

Depending on the nature of the rash, *macular, papular, urticaria, vesicles bullous, pustular,* toxicodermatoses may be distinguished.

According to severity, toxidermia may manifest themselves in:

I. Mild forms characterized by itching, limited urticaria, fixed erythema, multiforme exudative erythema, the reaction on lichen planus type.

II. Moderate forms in which there are generalized urticaria, angioedema, bullous toxicoderma, hemorrhagic vasculitis, bromide and iodide acne, common toxic and allergic dermatitis.

III. Severe forms means the appearance of Lyell's syndrome, Stevens-Johnson syndrome, bromoderm, ioderma, systemic lupus erythematosus, syndrome of dermatitis herpetiformis.

Mild forms of toxidermia

The patient complains of ***itching*** after using drugs, there are no primary morphological elements rashes on the skin, secondary elements in the form of bloody crusts and excoriations may occur.

Limited urticaria. Characterized by the appearance in some areas of urticarial elements (blisters) pink-red in color, protruding above the skin, patients suffer from severe itching.

Fixed erythema *(sulfanilamide)*. Erythematous patches (single or multiple series) red in color appear on the skin of the trunk, extremities, genitals, mucous membranes, when getting a long time the secondary brown pigmentation is preserved; no subjective feelings; in case of repeated exposure to a provoking factor, the rash occurs on the same areas (hence the name 'fixed erythema'), as well as on new ones.

Erythema multiforme. Polymorphic rash of swollen spots or papules of right round shape coin-sized with a bluish sunken center; on the surface may appear vesicles or bullous elements.

The reaction on lichen ruber planu types. There is a common symmetrical rash of typical papules (flat polygonal with pearlescent shine and navel-like sunken centre) patients suffer from severe itching.

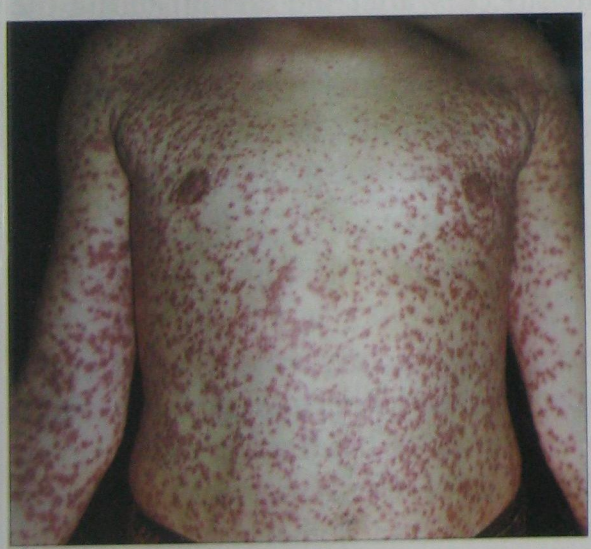

Fig. 8.10. Drug toxicodermatosis.

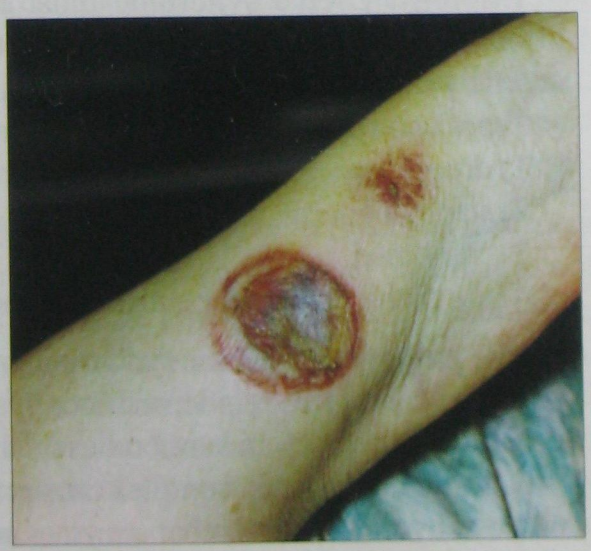

Fig. 8.11. Bullous fixed toxicodermatosis (on barbiturates).

Toxidermia of moderate severity

Generalized urticaria. Urticaria is characterized by multiple elements on large areas of skin, sharp itching, ephemeral elements.

Angioedema. There is a limited asymmetrical swelling of hypodermis; sense of increasing the organ size, asphixy, no itching.

Bullous toxicodermatosis. Disseminated bubbles with erythematous borders appear on the skin, often on the mucous membranes; erosion may form after their explosion, and upon drying – crusts, after healing the secondary spots may be preserved for a long time.

Hemorrhagic vasculitis. One may observe disseminated purpura, petechiae, ecchymosis on the skin, after their recourse – secondary hyperpigmental pots.

Bromine and iodine acne. May appear on the seborrheic areas due to accumulation of bromine or iodine drugs in the body; rash looks like pustules, acne elements.

Common toxic and allergic dermatitis. They are characterized by the appearance on the skin of the trunk and limbs of common symmetric polymorphic rash (spots, blisters, papules); sudden itching, burning skin, body temperature may increase to low grade, fever, indigestion, weakness, myocarditis.

Severe toxicodermatoses

Lyell's syndrome or ***acute toxic epidermal necrolysis*** is considered a severe form of toxidermia, manifested through acute generalized inflammation of the skin and mucous membranes with exfoliation of the epidermis, visceral injuries, intoxication. Lyell's syndrome arises in the setting of the polyvalent sensitization as a result of adequately re-prescribed and appropriately administered medicines. Manifestations of the disease are caused not so far by the pharmacological properties of drugs, but by many features of patient's immune system and his constitutional and genetic predisposition.

Etiology. Lyell's syndrome most often occurs after the administering the sulfonamides, antibiotics, barbiturates, thiamine, serums, etc., and might also be caused by chemicals, poor quality food, infectious factors (staphylococcus of various serotypes), and therefore the following forms of the syndrome may be distinguished:

1) drug,
2) staphylococcal,
3) combined,
4) idiopathic (where the cause is unknown).

The pathogenesis of the syndrome is complex and poorly studied. It is believed that at the heart of toxic epidermal necrolysis is an immediate cytotoxic response with the binding of antigen and antibodies on the surface of the basal epidermal cells, and formed antigen-antibody complex causes the lability of cells' lysosomal membranes, release of hydrolases and other enzymes and destruction of epithelial cells.

Clinical picture. The body temperature rises suddenly, the patients complain of malaise, drowsiness, headache, possible vomiting and diarrhea. On the skin, there is a

painful erythema which is spreading rapidly, there are bullous elements appear, one may observe a rapid exfoliation of the epidermis (the 'symptom of wet laundry' – wrinkled epidermis, gathered in folds and peeled); sharply positive Nikolsky's sign. A characteristic feature of toxic epidermal necrolysis is soreness of affected and unaffected skin. On the skin and mucous membranes there are widespread erosions; it is possibly an exfoliation of the mucous membranes of respiratory tract and internal organs.

Research data: a general analysis of blood shows iron-deficiency anemia, eosinophilia, agranulocytosis, pancytopenia, probable leukocytosis, increased ESR.

Positive allergological tests: intradermal, intraoral, drip, scarification, sublingual.

Positive immunological tests: PHT (passive hemagglutination test); direct and indirect degranulation of basophils or mast cells; neutrophils damage indicator; IFA for the detection of antibodies of class IgG, IgM, IgE; RIML (reaction of inhibition of leukocyte migration).

Pathomorphology of skin: epidermal necrosis with its subsequent exfoliation between the epidermis and dermis without defeat of the dermis itself. Staining the preparation in case of medical syndrome, they might detect cuboidal cells with large nucleus, in the case of staphylococcal – large epithelial cells with a small core.

Stevens-Johnson syndrome (form of bullous multiforme exudative erythema) – heavy, form of toxidermia, which manifests itself in the form of bullous multiforme exudative erythema with lesions of the skin and mucous membranes of at least two organs.

Etiology. Multiforme exudative erythema – a disease which is based on vascular lesions of the dermis with characteristic repeated changes in the skin and mucous membranes. They distinguish its infectious-allergic (idiopathic) form, which occurs in 93% of cases, and infectious-toxic one. A 'small' form (Gebre type) caused by a viral or mycoplasma infection, and 'large' (actually Stevens-Johnson syndrome) as a manifestation of drug toxidermia (because of penicillin, streptomycin, tetracycline, sulfonamides, griseofulvin, barbiturates, acetylsalicylic acid, chlorpromazine, codeine, vaccines, etc.) are also distinguished. Multiforme exudative erythema can also be observed in diseases of the connective tissue, malignant tumors, etc. The **pathogenesis** is not fully understood, hypothermia and focal infections are of big value. Dermatosis is understood as hyperergic reaction to drugs, infections and toxic factors, aimed at the keratinocytes.

Clinical picture. Diseases begins suddenly and acutely with fever and disorders of general condition of the patient. The process of the skin is spread, it damages not only the skin, but also the mouth, eyes, genitals, anus. Bronchitis, pneumonia, myocarditis, nephritis, diarrhea, arthritis, otitis media, paronychia might also develop. On the skin one may observe a patchy papular bullous rash with elements of considerable size, which is characteristic of the exudative multiforme erythema. On mucous membranes numerous bullous elements might appear, which are followed by the formation of large-sized erosions, massive hemorrhagic crusts may form on the lips on the erosion surface. The development of catarrhal or purulent conjunctivitis, ulcerative keratitis and uveitis is quite probable; that is can lead to vision loss. On the

genitals and in the perianal area, one can observe bullous elements, painful erosion that can affect urination and bowel movement difficulty. Simultaneously, the process involves the internal organs with development of pneumonia, bronchial pneumonia, pleurisy, myocarditis, glomerulonephritis, diarrhea, damage to the cardiovascular system and liver with symptoms of general intoxication.

Data of laboratory studies: a general analysis of blood indicates leukocytosis, stab shift, lymphopenia, eosinopenia.

Pathomorphology. Histologically it is possible subdivide dermal, mixed epidermal-dermal and epidermal types of skin lesions. Dermal changes are characterized by swelling of the endothelium of blood vessels and perivascular lymphohistiocytic infiltration with neutrophils and eosinophils, edema of the papillary layer, extravasations of red blood cells. Epidermal changes are accompanied by epithelial cell necrosis, spongiosis, vacuolar degeneration of the basal cells, the formation of bullous elements, basal membrane is intact. Histological changes in hemorrhagic rash look like anaphylactoid purpura as a result of degenerative disorders of the endothelium of the capillaries and perivascular accumulation of neutrophils and eosinophils.

Treatment. The treatment program regarding toxidermia patients includes stopping the impact of drugs as etiological factors and the removal of these substances residues from the body.

Systemic therapy involves the use of enterosorbents (polyvidone, dioctosmectite), detoxification therapy (polyvidone, sodium chloride, potassium chloride, calcium chloride, magnesium chloride, sodium bicarbonate, furosemide, 0.9% sodium chloride solution); antihistamines. With widespread and severe toxidermia, the patients are prescribed glucocorticoids for systemic use (prednisone, triamcinolone, dexamethasone, betamethasone), with marked edema – diuretics (furosemide, hydrochlorothiazide), and complications of secondary infection – antibiotics to choose from (cephalexin, erythromycin, azithromycin, doxycycline, ciprofloxacin, moxifloxacin, levofloxacin, etc.).

External therapy is prescribed depending on the nature of the rash. On erythema, urticaria, bullous and papular rash, steroid creams or ointments shall be applied.

Prevention. The causative agent of the disease is identified with the help of specific biophysical and immunological tests; the risk factors contributing to the development of sensitization shall be eliminated if possible; sanitation of lesions of focal infection shall be made;

Rehabilitation measures are generally aimed to prevent sensibilization to allergens, to eliminate them and treat the diseases, caused the development of toxidermia, to sanitate the focal lesions of chronic infection.

8.4 ATOPIC DERMATITIS

Atopic dermatitis is considered a chronic allergic skin disease that occurs in people with a genetic tendency to atopy, has a relapsing course and age features of clinical manifestations. Typical clinical signs of atopic dermatitis are eritemato-squamous, eczematous and lichenoid eruptions, arising as a result of hypersensitivity to the specific (allergens) and non-specific stimuli. The relevance of atopic dermatitis is defined by intense itching, presence of common skin rashes, often relapsing disease course that is the cause of a neurotic state of the patient, violation of his working capacity, reducing the quality of life and social maladjustment.

TO KNOW:

- the definition and epidemiology of atopic dermatitis;
- its etiopathogenetical features;
- age-related clinical features and classification of atopic dermatitis;
- principle and additional criteria of clinical manifestations of the disease;
- principles of treatment and prevention of atopic dermatitis.

TO BE ABLE TO:

- properly collect patient history on atopic dermatitis ;
- make a diagnosis on clinical grounds;
- make a diagnostic tests to confirm the diagnosis (identification and evaluation of dermographism);
- make a differential diagnosis;
- assign individual pathogenetic treatment.

Etiopathogenesis of atopic dermatitis is multifactorial and not fully understood. In addition to genetic determinism – (two genes were identified in the chromosomes, which are responsible for the abnormal hyperproduction of IgE), central and autonomic nervous system damages, dysfunction of the gastrointestinal tract, of hepatobiliary system in particular, presence of helminths and other foci of chronic infection (ENT-organs, teeth), and changes in immune responsiveness (decrease in subpopulation of T-suppressor cells, reduced phagocytosis), microcirculatory disorders, etc. are of great value in the pathogenesis of atopic dermatitis.

Clinical picture. The first clinical manifestation of atopic dermatitis occur on the skin of three months old children (early weaning, or coming to artificial feeding), and in older children. First rash usually occurs on the face (in 65% of children), scalp (34%), elbows and / or knees and on the extensor surfaces of the extremities. In the course of atopic dermatitis, there are three age periods with different clinical manifestations on the skin: the first period (up to two years) – during infancy (exudative stage); the second period (from two years to adolescence) – infiltrative stage; and the third period (juvenile and mature age) – lichenification manifestations.

Classification. In different age periods, one can observe different clinical manifestations of atopic dermatitis. Depending on the type of rash character, it is possible to distinguish the following clinical forms of atopic dermatitis:

- eritemato-squamous;
- vesicle crusted (eczema);
- eritemato-squamous with low lichenification;
- pruriginous;
- lichenoid.

Eritemato-squamous form is more likely to occur in infants at the age of four months and children under two years old and characterized by the appearance of erythematous patches with edema and desquamation on the face, neck and scalp. In their centers one might see microvesicles with weeping, crust and crust-scales by type of seborrheic, true or microbial eczema – it is a ***vesicle crusted form***. Then, symmetrical rash may spread to the trunk and extremities. In the period of acute dermatosis, the child is restless, not sleeping, itching the skin. Atopic dermatitis is characterized by alternating exacerbations and remissions, but even in remission behind the ears and near the lobes one can observe deep cracks,

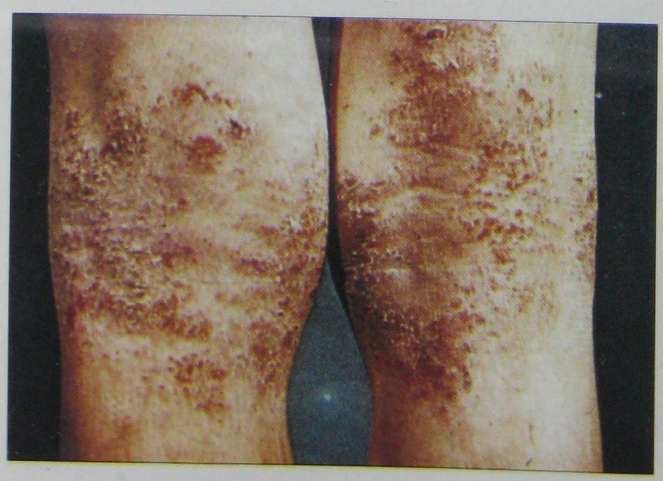

Fig. 8.13. Atopic dermatitis.

which get wet and become infected. Dermographism in most patients (80%) remains red.

Eritemato-squamous form with low lichenification (from two years to puberty) is characterized by appearance of eritemato-squamous foci in areas of typical lesions, and lichenoid papules on the skin of the flexor surfaces, large folds, neck, where the skin is infiltrated, becomes dry, compacted and covered with scaly scales, excoriations and hemorrhagic crusts.

At ***pruriginous form*** (from adolescence to adulthood) amid the signs of atopic dermatitis, which manifest as mild or clinically erased eritemato-squamous form with signs of xeroderma, one might observe dense pruriginous multiple papules or papule-vesicles, localized on the extensor surfaces of the extremities, usually legs, shoulders and upper arms, and resemble s prurigo rash (strophulus like). Patients suffer from severe itching, so on the skin frequently the signs of scratching and hemorrhagic crusts might be observed.

At ***lichenoid form***, which occurs in adults, in the setting of xeroderma one might observe flat polygonal lichenoid follicular papules, the color of which varies from normal skin to pink-gray. They are located on the flexor surfaces of the extremities with subsequent formation of foci of lichenification, over time, these areas become pigmented. In some patients, the lesions become of widespread or even generalized nature with the formation of atopic *diffuse dermatitis (diffuse neurodermatitis)* involving the skin of perineum, genitalia, and gluteal and infrabuttock folds. In these cases, the disease course is severe, general condition of patients gets worse, the biopsy skin itch leaving a linear scar may occur, the secondary infection often joins. Dermographism is resistant, white. The course of diffuse atopic dermatitis is torpid; it responds poorly to treatment, the remission is of temporary nature. Patients suffer from insomnia and chronic fatigue, psychic and emotional exhaustion.

Diagnostic criteria and assessment of severity of atopic dermatitis. The diagnosis of atopic dermatitis shall be made when the patient has three major and at least three additional clinical criteria.

I. Major criteria:

1) itchy skin;

2) typical morphology and localization of skin rashes: in infants – eczematous rash on the face and extensor surfaces of the extremities, in older children and adults – lichenification on flexor surface of the limbs;

3) chronic relapsing course;

4) atopy in a personal and family history.

II. Additional criteria:

1) xerosis (dryness) of the skin;

2) ichthyosis, mostly of palms;

3) immediate hypersensitivity reaction in skin tests with allergens;

4) localization of cutaneous process on the hands and feet ;

5) cheilitis;

6) nipple eczema;
7) susceptibility to infectious skin lesions due to impaired cellular immunity;
8) dermatosis beginning in early childhood;
9) erythroderma;
10) relapsing conjunctivitis;
11) Dennie-Morgan fold;
12) keratoconus (conical protrusion of the cornea);
13) anterior subcapsular cataracts;
14) cracks behind the ears;
15) high level of IgE in the blood serum;
16) darkening of the areas around the eyes;
17) pallor or erythema of the face;
18) tinea alba (white lichen);
19) food sensitization;
20) intolerance to wool and lipid solvents;
21) perifollicular localization of the rash;
22) influence of emotional factors on the disease course;
23) white dermographism or delayed blanching.

To assess the severity of atopic dermatitis it is required to determine in SCORAD index in points by the formula: SCORAD Index = A / 5 + 7xB / 2 + C, where A – the rate of process spreading on the skin (as a percentage), B – rash manifestation intensity indicator (erythema, edema/papule, crust/weeping, excoriation, lichenification, dry skin from 0 to 3 points), C – indicator of the subjective state of the patient (pruritus and sleep disturbance from 0 to 10 points). The severity of atopic dermatitis index SCORAD: 20 points – an easy, 20-40 points – moderate, more than 40 points – severe.

Methods of laboratory diagnostics. General blood analysis indicates eosinophilia, elevated levels of immunoglobulin E; immunologic blood study – reducing the total number of E-rosetting lymphocytes and the relative number of T-suppressor subpopulation of lymphocytes, impaired immunoregulatory index; disimmunoglobulinemia, colonization of the affected and surrounding skin with Staphylococcus aureus, etc.

Pathomorphology. In the epidermis – hyperkeratosis, acanthosis, parakeratosis; in the dermis – edema and perivascular infiltration of lymphoid cells and neutrophils. In long-term course of the dermatosis, the dermal papillae are swollen, twisted, sclerosal, with marked changes of elastic and collagen fibers, increased number of capillaries.

Differential diagnosis. The diagnosis of atopic dermatitis is not complicated, and shall be made on the basis of complaints (itching, sleep disturbance), history data, characteristic clinical signs (major and additional criteria). Atopic dermatitis should be differentiated from scabies (itching occurs mainly in the evening and at night, paired

arrangement of elements of the rash; it is localized in typical scabies areas, there are burrows and pathognomonic signs of Hardy-Gorchakov and Sezary, such a rash might be revealed in the family), from streptoderma (primary rash elements in the form of phlyctens, there are purulent crusts), from eczema (often begins at an older age, and there are multiple microvesicles and microerosions with the symptom of 'serous wells' and the phenomena of weeping; often the skin lesions are of common multifocal nature without characteristic for atopic dermatitis additional clinical criteria), from limited neurodermatitis (dermatosis starts, according to history, in adolescence or adulthood without a rash of early childhood, no characteristic of atopic dermatitis additional clinical criteria).

Treatment of atopic dermatitis should be individualized, taking into account the patient's age, clinical symptoms, degree of activity and severity of disease. Therapeutic measures are aimed at eliminating the inflammatory process, as well as the extension of the state of clinical remission and relapse prevention.

First of all, patients are encouraged to conduct elimination activities in the home – avoid contact with pets, daily wet cleaning of premises, etc. Patients must follow a hypoallergenic diet for exceptions obligate food allergens, extractives, sharp-irritating foods, carbohydrates, alcohol and restriction of salt.

In period of exacerbation patients shall receive comprehensive medical treatment: detoxification drugs (Neohaemodes, Rheopolyglucinum, Reosorbilact, etc.), antihistamines (Chloropyramine, Clemastine, Loratadine,), with severe clinical manifestations of dermatosis – their injectables forms, stabilizers of mast cell membranes (ketotifen), psychotropic drugs (in sleep disorders, presence of neurotic disorders) for one to four weeks before the normalization of emotional states, sedative drugs. In severe atopic dermatitis and resistance to other therapies the patients shall be administered immunosuppressive agents (cyclosporine, systemic glucocorticosteroid drugs).

External treatment of atopic dermatitis involves the use of aniline dyes, topical calcineurin inhibitor, including the drug tacrolimus ("Protopic"). In severe sharp-inflammatory manifestations (acute and sub-acute stages), topical corticosteroids on a hydrophilic base containing mometasone furoate, betamethasone valerate) shall be prescribed, and after cessation of exudation in severe dry skin – moisturizing drugs containing fluocinolone acetonide. Correct daily skin care (cleansing and moisturizing it) is of great value. The products containing emollients shall be applied. The patients with atopic dermatitis are also indicated physiotherapy techniques – selective phototherapy, PUVA therapy

Preventive measures. To prevent relapses of atopic dermatitis, topical calcineurin inhibitors might be applied, in particular – tacrolimus in the form of ointment. Such patients are advised to respect the hypoallergenic diet with the exception of extractive foods and refined carbohydrates, and continuous care of the skin (its cleansing and moisturizing), which requires the use of indifferent emollient containing drugs. Questions for self-control of the first level of complexity

1. Characteristic feature of simple contact dermatitis is:

A. Fuzzy boundaries of the lesion
B. Clear boundaries of the lesion
C. Occurrence only in sensitized patients
D. Occurrence in 12-74 hour after exposure to a certain factor
E. Possible generalization of the process.

2. It is not observed in allergic contact dermatitis:

A. Rash on the place of influence of a certain factor
B. Clear boundaries of the lesion
C. Occurrence in sensitized patients
D. Subacute onset and course
E. Possible recurrence after re-exposure to the allergen.

3. Which of the following is not used for the treatment of allergic contact dermatitis:

A. Glucocorticoids containing creams and ointments
B. Antihistamines
C. Hyposensitizing drugs
D. Vitamins
E. Photosensitizers

4. Toxicodermatosis is not characterized by:

A. Acute onset
B. Symmetry of rash
C. Polymorphic rash
D. Development of lichenification
E. Possible lesion of mucous membranes.

5. Fixed erythema is not characterized by:

A. Occurrence as a result of taking sulfonamides, antibiotics etc.
B. Occurrence due to exposure to obligate irritants
C. Rash of several red spots 2-5 cm in diameter
D. Localization on the skin, genital and oral mucosa
E. After recourse pigmentation remains for a long time.

6. Which of the following is not applied for the treatment of toxicodermatosis:

A. Enterosorbents
B. Photosensitizers
C. Antihistamine drug
D. Detoxification drugs
E. Glucocorticoid creams

7. Lyell's syndrome is characterized by:

A. Isomorphic Koebner response
B. Auspitz triad
C. Nikolsky's symptom
D. Meshcherskiy-Besnier's symptom
E. Jadassohn's symptom

8. Which of the following form is not characteristic for eczema :

A. Idiopathic
B. Microbial
C. Herpetiform
D. Occupational
E. Seborrheal

9. Which of the following does not belong to the main diagnostic criteria for assessment of the severity of atopic dermatitis:

A. Skin itch
B. Ichthyosis, mostly of palms
C. Typical morphology and localization of skin rashes: in infants – eczematous rash on the face and extensor surfaces of the extremities, in older children and adults – lichenification on flexor surfaces of the extremities
D. Chronic relapsing course
E. Atopy in personal and family history.

10. Primary morphological element with urticaria is considered:

A. Vesicle
B. Bulla
C. Macule
D. Boss
E. Knot

Task 1. The plumber came to the doctor complaining of redness, burning sensation, pain in the right forearm. Some rash appeared in 10-15 minutes after contact with hot pipes. *Objectively*: on the skin of the right forearm extensor surface there is a severe focus with clear boundaries of 3x6 cm; there is skin hyperemia in the focus, some individual bullous elements with clear content.

a) Make a diagnosis:
- **A.** Dermatitis herpetiformis Duhring
- **B.** Allergic contact dermatitis
- **C.** Eczema
- **D.** Simple contact dermatitis
- **E.** Pemphigus vulgaris.

b) What should be the behavioral tactics of the patient?

Task 2. 32-years old female patient consulted the dermatologist complaining of some rash on the skin of neck and chest, accompanied by itching. Rash appeared after taking aspirin and kept on the skin for about two hours and then without any action gradually disappeared appearing at new areas. *Objectively*: there are multiple palm-sized urticaria elements. The patient has red dermographism; eosinophilia is revealed in the blood.

a) What kind of skin disease can be thought of in this case:
- **A.** Urticaria
- **B.** Allergic dermatitis
- **C.** Eczema
- **D.** Simple contact dermatitis
- **E.** Atopic dermatitis

b) Make a differential diagnosis of the disease.

Task 3. A boy of 7 years complains of feeling unwell, pain on swallowing, painful rashes on the lips and in the mouth, itchy rash on the face and body, which appeared three days after receiving analgin because of acute otitis. *Objectively*: the general state of the child is severe; body temperature is 38.6 °C. On the skin of the lips and oral mucosa, there are common erosions with massive hemorrhagic crusts. On the face and body – multiple erythematous spots and edematous papules with deep bluish center, on the surface of which there are vesicles and blisters; the elements tend to be drained. Nickolsky's sign is negative.

a) What kind of emergency prehospital care should be provided to the child:
- **A.** To prescribe corticosteroids orally, canceling all other drugs
- **B.** To prescribe corticosteroids parenterally, canceling all other drugs
- **C.** To prescribe corticosteroid ointments, canceling all other drugs
- **D.** To prescribe antihistamine drugs, canceling all other drugs
- **E.** To prescribe purgatives, canceling all other drugs

b) What preventive measures are required in this case?

Task 4. Female patient, 37 years old, employed as a manipulative nurse, complains of feeling unwell, weakness, headache, muscle and joint pain and skin rash, accompanied by severe pain and burning sensation. Some rash appeared two days after receiving ampicillin because of sore throat. *Objectively*: general condition of the patient is severe; body temperature is 39.2 °C. The entire skin is bloodshot. On the trunk and extremities, there are multiple bullous elements, erosion, fragments of epidermis observed. The skin is painful on palpation. Nikolsky's sign is positive.

a) What kind of emergency pre-hospital care should not be provided to the child:
- **A.** Stop taking ampicillin
- **B.** To prescribe corticosteroids parenterally
- **C.** To prescribe enterosorbents
- **D.** To refer the patient to the intensive care unit for further treatment
- **E.** To refer the patient for consultation and supervision of the allergist

b) Make a differential diagnosis of the specified disease.

Task 5. 34 years old woman consulted the dermatologist about the skin lesions of hands and lower third of the forearm as a significant

swelling, redness, vesiculation, weeping. Got ill six months ago. The disease appeared after four months use of 'Lotus' washing powder. In the past, the patient has no skin diseases.

a) What is the most likely diagnosis in this patient:

A. Contact allergic dermatitis
B. Simple contact dermatitis
C. True eczema
D. Toxicodermatosis
E. Microbial eczema

b) What diagnostic tests should be carried out to establish the clinical diagnosis?

Answers to the questions of the first level of complexity
1 – B; 2 – B; 3 – E; 4 – D; 5 – B; 6 – B; 7 – C; 8 – C; 9 – B; 10 – B
Answers to the questions of the second and third levels of complexity
1a – D; 2a – A; 3a – B; 4a – E; 5a – C

9 TOPIC

Professional dermatosis

Professional dermatosis is the skin disease caused by impact of production factors.

EDUCATIONAL AND TRAINING PURPOSES

- To determine the general progress and clinical findings of different professional dermatoses
- To determine the diagnostic criteria and terms of occurrence of professional dermatoses
- To analyze the principles of therapy and preventive treatment of professional dermatoses

TO KNOW:

- Etiopathogenesis of professional dermatoses;
- Classification of professional dermatoses;
- Clinical peculiarities of their modern progress;
- Diagnostic criteria of professional dermatoses, skin test meaning;
- Methods and principles of medical and preventive measure for the persons who have professional dermatosis.

TO BE ABLE TO:

- Collect anamnesis of the person who has professional dermatosis;
- Analyze clinical findings and results of laboratory research;
- Hold differential diagnostics on diseases with similar clinical findings;
- Put on reasonable treatment for the people who have professional dermatoses;
- Recommend the necessary preventive measures against professional skin diseases.

Most of the professional dermatoses are caused by chemical agents.
Professional dermatoses are divided into:

I. Professional dermatoses of chemical etiology

1. Professional dermatoses caused by obligate irritants: simple contact dermatitis, epidermitis, chemical burns, skin and mucous ulceration, onychia and paronychia.
2. Allergic professional dermatoses: allergic dermatitis, eczema, toxicodermia, uticaria.
3. Professional dermatoses caused by hydrocarbon compounds: toxic melanodermia, folliculitis, acne, limited hyperkeratosis.
4. Dermatoconiosis.

II. Professional dermatoses caused by impact of physical factors

1. Professional dermatoses caused by thermal, actinic, mechanical factors: freeze injuries, burn injuries, perfrigeration, photocontact dermatitis, cheilitis.
2. Professional signs (stigmae): temporary – colour staining, callosity, pigmentation, cracks, excoriation; and stable – penetration (contamination), scars, telangiectasia, nail changing.

III. Professional dermatoses of infectious and parasitical etiology

1. Erysipeloid, paravaccinia, mycoses.

Professional dermatoses of chemical etiology

Simple contact dermatitis is mainly an acute skin inflammation arisen directly at the place of irritators' activity and is clearly spread within the boundaries of its activity. The degree of response manifestation depends on the etiological factor, exposition period, localization and individual sensitivity.

Etiopathogenesis. The cause of occurrence is the impact of the irritators of chemical etiology (concentrated acids, alkalies, paints, lacquers), physical factors (mechanical impact, freeze injuries, burn injuries etc.) and biological factors. Simple contact dermatitis develops without incubation period.

Clinical findings. Clinical findings of the simple contact dermatitis are skin redness, edema, vesicular and bullous eruption which causes after cutting off the numerous erosions and madescence. The lesion focus is clearly distinguished from the unaffected skin area. It is localized in the place of contact with irritator.

Diagnostic. Diagnosis of the simple contact dermatitis is based on anamnesis data and clinical findings, inspection of the working conditions and detection of the contact with irritators of chemical etiology. It is differentiated with the allergic dermatitis, eczema, toxicodermatosis.

Treatment. It is necessary to eliminate the skin contact with the working chemical factor which caused the contact dermatitis. The affected area shall be rinsed with water. In case of small affected areas and mild form of the simple contact dermatitis

only the local anti-inflammatory therapy shall be applied, in particular cold lotion with 2% boric acid solution, Rivanol/Furacilin solutions as well as the madescence ceases.

Epidermitis is the skin inflammation with chronic progress which occurs as the result of frequent impact of organic solvents, coolants, solutions of oxides, alkalies and other degreasing agents.

Clinical findings. Clinical findings of the epidermitis are skin dryness, peeling, there may be deep cracks without clear inflammation and infiltration. The subjective symptoms are itchiness and soreness. Affection is mainly localized in hands.

Diagnostic. Diagnosis is based on anamnesis data and clinical examination, fact of contact with production factors.

Treatment. Detoxication, antihistamines (Cetrine, Aerius, Claritin etc.) and local agents (indifferent and corticosteroid pastes) are used.

Chemical burns occur at the working place as the result of skin contact with obligate irritants (acids, alkalies, salts of heavy and alkaline metals etc.).

Clinical findings. According to the clynical progress there are four stages of disease: I – erythematic, II – vesiculobullous, III – sphacelous, IV – necrosis of all skin layers.

Diagnostic. Diagnosis is based on anamnesis data, peculiarities of clinical findings, results of laboratory research complex, fact of contact with chemical irritants.

Treatment. Impact of the production factor shall be detected and eliminated; the place of impact of chemical irritants shall be washed with water under pressure. The acid residues shall be neutralized by 2% soda solution and in case of alkaline burns by 2% acetic/citric acid solution. Detoxication, keratoplastic and steroid crиmes and antihistamines are used in treatment.

Prevention. Sanitary standards for manufacturing areas and work hygiene rules shall be observed. Personal prevention means are also used.

Allergic professional dermatoses

Allergic professional dermatitis results in the repeated contact of skin areas with irritants in unfavourable working conditions. In such case univalent sensibilization to one of the impact factors is shown.

Etiopathogenesis. The allergens can be different chemical agents used in work (paints, lacquers, solvents, oils and lubricants, medical drugs, nickel and chrome salts, insecticides etc.).

On the repeated contact with the allergen the sensibilizing lymphocytes flow towards the allergen excitation focus followed by lymphoquin release and leukocyte involvement which causes skin inflammatory reaction as the result of release of inflammatory mediators by the abovementioned cells.

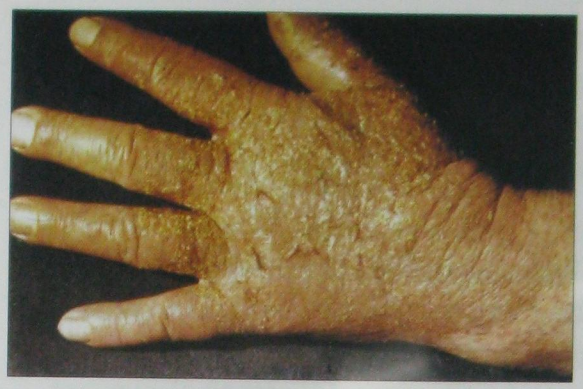

Fig. 9.1. Professional eczema.

Clinical findings. The process is widely spread, with the trend to peripheral growth. Eruptions are polymorphous. Clinical findings are erythema, edema, papula, microvesicles, cometimes vesical eruptions which cause anabrosis in case of opening them up. The affections are localized in hands, forearms, face. The subjective symptom is middle itchiness.

Diagnostics. Diagnosis is based on anamnesis data, clinical findings, results of laboratory research and skin samples which is compulsory fir determining the allergen and making a diagnosis of the allergic professional dermatitis.

Treatment. The repeated contact with irritants shall be eliminated. The antihistamines and sedation medications are used. In severe cases the glucocorticosteroid hormones are used. External therapy depends on the clinical findings: anabroses are cured by aniline ink water solution and then by indifferent pastes or corticosteroid pastes and creams.

Prevention. Sanitary standards for manufacturing areas and work hygiene rules shall be observed.

Professional eczema is a chronic skin disease (often recurrent) caused by contact with sensibilizing occupational irritant. The details of professional eczema are described in the chapter *Dermatites and Eczema*.

Professional dermatoses caused by hydrocarbon factors

Toxic melanodermia results in long-term work contact with oil and coal products.

Clinical findings. The general intoxication is shown on face, neck, arms, rarely on body and legs, with erythema, pigmentation, follicular hyperkeratosis, telangiectasia and peeling. The typical colour of skin is brownish grey, the skin becomes rough and wears thin.

Treatment. The production factor shall be detected and the contact thereto shall be eliminated. In order to stabilize the general condition the detoxification therapy, antihistamines, B and C vitamins, external corticosteroid pastes or creams are used.

Folliculitis and acne. Engineers, tractor drivers suffer oil, resinous, tar, chloric folliculites due to contact with machinery lubricant, oil, petrol, petroleum oil and in case of direct permanent contact with oil-soaked clothes.

Typical localization of affection: hands, forearms, thighs, stomach.

Clinical findings. Black spots occur in hair follicle mouths; further the development of inflammatory nodules with transformation to furuncles is possible.

Treatment. Contact with production factor shall be eliminated. The general treatment provides antibiotics, sulfanilamides and anti-inflammatory drugs; in case of folliculate transformation to furuncles the antihistamines (Cetrine, Aerius, Claritin etc.) are used, 1-2% salicyl or boric spirit, pure ichthyol are applied externally, physiotherapeutic procedures (ultraviolet irradiation) are also used.

Limited hyperkeratosis develops on skin after long-term contact with coal tar and oil products. Its main feature is increased keratosis of epidermis at the highly contaminated skin areas: face, back of hand, thighs, forearm extensor surface.

Clinical findings. Solid pea-size flat round grayish verrucas occur. No subjective symptoms.

Treatment. The production factor shall be detected and eliminated. A and E vitamins, keratolytic pastes and creams are used.

Dermatoconiosis is resulted in impact of chemical, mineral, plant and animal powdery substances which occur in work conditions and cause mechanic or chemical irritation on penetration to skin and blocking excretory ducts of oil and sweat glands. Affection is localized in opened skin areas.

Clinical findings. The colour skin changes, the non-inflammatory and erythematic spots, follicular papules, excoriations occur. The subjective symptom is itchiness. The disease is differentiated with atopic dermatitis.

Treatment. Depends on the clinical findings, like for common dermatoses.

Professional dermatoses caused by physical factors

Freeze injuries occur under long-term impact of low temperatures, under the contact with coolants etc. It may cause both local and general hypothermia. According to the clynical progress there are four stages of freeze injuries: first stage is characterized by stagnant cyanochrous colour and edema of the affected skin areas accompanied with feelings of tingling and itchness; the second – by appearance of vesicles filled with serous or haemorrhagic fluid in the setting of stagnant cyanochrous skin; the third – by necrosis of the affected skin areas and deep soft tissues; the fourth – by necrosis of soft tissues and bones.

Treatment. At the responsive stage in case of first degree freeze the diseased person shall get warm in the room with the temperature of 18-20 °C followed by rubbing the affected skin areas by 5% tannin or boric spirit and applying the warm aseptic dressing. In case of second degree freeze the affected areas shall be cured with spirit, the vesicle tectorium shall be cut off and the lotion with disinfecting solution shall be applied. In case of third and fourth degree freezes the surgical stationary treatment is indicated.

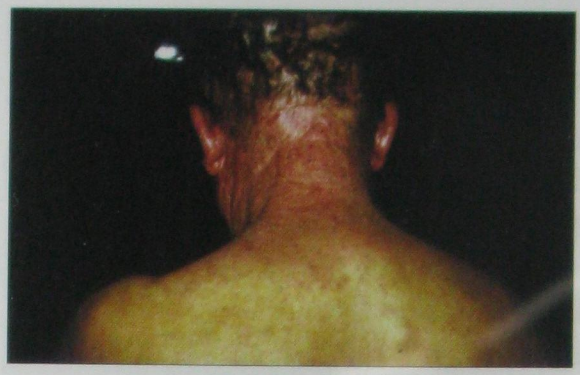

Fig. 9.2. Photocontact dermatitis.

Perfrigeration is caused by impact of cold jointly with humidity (at the water transport, in fishing and forestry industry, agriculture). Its clinical findings are the limited edema, solid or soft consistence and cyanochrous reddish colour of the affected skin areas.

Photocontact dermatitis occurs under the impact of sun rays at the persons who work in the open space (house builders, agricultural workers) or who work with welding machine under the impact of artificial radiation and at the persons who contact with photodynamic substances (tar, creosote, sulfanilamides, mercury agents, aromatic hydrocarbons, manganese and iron salts, oil and coal tars). Sun dermatitis usually occurs in spring and summer.

Etiopathogenesis. As the result of direct impact of ultraviolet rays on skin the DNA structure destroys and the inflammation mediators are histamine, prostaglandins, interleukins which cause pain and edema.

Clinical findings. On the open skin areas (face, neck, arms) the hyperaemia and edema occur in the setting of itchiness, soreness (first degree), large amount of vesicles appears (second degree). General condition disorder (high temperature, weakness, anxiety, sleep disorder, loss of appetite). In three or four days inflammation is replaced by peeling and the stable brown pigmentation develops which prevents recurrence.

Diagnostic. Diagnosis of photocontact dermatitis is based on anamnesis and clinical examination. Differential diagnostics is held with protoporphyria and drug-induced phototoxicity.

Treatment. Insolation and contact with photosensitizer shall be avoided. B vitamins are indicated in standard doses, coolants, glucocorticosteroid pastes and creams are applied. Series of cosmetological means are used for skin care.

Prevention. Solar radiation shall be avoided, the reasonable clothes made of solid protective fabric shall be worn, photoprotective pastes and creams (Anthelios XL SPF 50+, Anthelios AC SPF 30 etc.) shall be used.

Cheilites occur as the result of impact of meteorological, mechanic, chemical and biological factors in working conditions.

Clinical findings. Inflammation of vermilion zone is shown in the form of dryness, peeling, scabs, cracks of mucous and skin around the mouth.

Treatment. The basic treatment is elimination of impact of etiologic factors. The B vitamins are used, the lotions with anti-inflammatory and disinfecting solutions are

used in case of acute events. After decrease of inflammatory events the corticosteroid creams are spread on the affected areas.

Prevention. Impact of factors which cause the disease shall be eliminated.

Burn injuries are caused by impact of high temperature on skin.

Clinical findings. There are four stages of burns: I – erythema and edema accompanied with itchiness and soreness, II – erythema and large vesicles filled with serous or haemorrhagic fluid, III – derma necrosis, IV – necrosis of all skin layers.

Treatment. In case of limited first degree burns the anti-inflammatory, anti-allergic and antipruritic means are applied on the affected surface. In case of limited second and third degree burns the skin around the burnt area is cured with spirit, the physiological solution or hydrogen dioxide is applied on its surface, the vesicle tectorium is cut off and the sterile dressing with disinfecting solution is applied on the diseased area; later pastes and creams with glucocorticoids are used. In case of deep fourth degree burns the surgical treatment is indicated.

Professional signs (stigmas)

There are temporary and stable professional stigmas. *Temporary* stigmas are:

1. **Colour staining** is caused by penetration of colouring agents inside skin, hair and nails. Its stability depends on the depth of penetration and colouring agent features (typical of lacquerers, painters etc.).
2. **Callosity** is thickening of skin corneous layer (hyperkeratosis) which is caused by long-term friction or pressure (typical of refining miners, diggers etc.).
3. **Pigmentation** is skin colour changing up to brown caused by crusting and solar radiation (typical of seamen, fishermen etc.).
4. **Cracks** is the skin integrity disorder with loss of skin viscoelasticity caused by impact of mechanic factors, crusting, freeze injury (typical of solderers, tinsmiths etc.).

Stable professional stigmas are:

1. **Penetration (contamination)** is occurrence of stable dim-grey skin colour as the result of contact with metal dust jointly with mineral oils (typical of miners, steelworkers, combat engineers etc.).
2. **Scars** are caused by small injuries in working conditions due to penetration of hot or molten metals, hot liquids, steam onto skin typical of mill roller, moulders).
3. **Telangiectasia** is a stable expansion of surface blood vessels which appears on the opened skin areas (typical of hot shop workers, steelmakers, glassblowers, coal heavers, cooks, smiths etc.).
4. **Nail changing** is the result of mechanic and chemical impact (typical of watchmakers, laundresses, enamellers etc.). Clinical findings are cracks in nails, nail malacia, thinning, tarnish. In order to prevent their affection by mycotic infection (onychomycosis) the water-repellent gel *Nailexpert by Wartner* is recommended to be applied locally. The unique formula of such product creates a special pallet on the

nail surface, creates conditions unfavourable for fungus existence and prevents infection of healthy nails.

Professional dermatoses of infectious and parasitical etiology

Erysipeloid *(erysipeloid Rosenbach)* is the most widely spread professional skin disease of infectious etiology.

Etiology. Caused by swine erysipelas *(Rhusiopathiae suis).*

Epidemiology. It is the most widely spread among the meat factory, leather/fur industry, canning\fish factory workers. Developent of the disease is caused by injuries and penetration of the irritant through the affected skin. Rodents are the main infection carriers. The disease usually does not transmit from the ill person to the healthy one.

Clinical findings. The incubation period lasts from several hours till two or five days.

The affections are mainly localized on the skin of hands and fingers. At the place where the irritant penetrates inside the limited acute painful swell occurs with dark red erythema (further replaced by cyanochorus blue-grey colour), papules and vesicles may appear. The process trends to spread over the finger joints. High temperature, weakness, shiver, enlargement of regional lymph nodes are possible. The disease lasts from two till six weeks and may become chronic with repeated recurrences.

Diagnosis. Diagnosis is based on the anamnesis, clinical findings and the result of laboratory research complex.

Treatment. Antibiotics (erythromycin, penicillin), pastes and creams with antibiotics and corticosteroids are used.

Prevention. Sanitary standards for manufacturing areas and work hygiene rules shall be observed, deratization shall be held. The veterinary inspection of animals, meat and fish products, prevention of small injuries and proper cure of the affected skin are required.

Paravaccinia is the dairy-women's professional disease; zootechnicians, veterinary physicians and other workers who have contact with the infected cattle may also suffer it.

Etiology. Etiological agent is the cowpox virus.

Epidemiology. Disease for cows (rarely for ship and goats). As for the diseased animals the eruptions occur in utter and nipple skin. People get infected during the direct contact with such animals. The disease does not transmit from the ill person to the healthy one.

Clinical findings. The incubation period makes up three or four days. The affections are mainly localized on hands, forearms, face. The painful solid pea-size nodules with

hollows in the center appear in the setting of hyperthermia and edema. Further the blackish brown scab appears on the hollow and when it deops off the dark spot and the scar remains. The eruption lasts up to two months.

The progress is favourable in general.

Diagnostic. Diagnosis of paravaccinia is based on anamnesis, clinical findings and the result of laboratory research complex.

Treatment. In order to accelerate the nodule dispersion and to prevent the development of secondary infection the antivirus pastes and aniline dyes are used.

Prevention. Work hygiene rules shall be observed, the veterinary inspection of animals shall be held, small injuries shall be cured properly.

Professional mycoses (trichophytia, microsporia, favus, epidermophytosis, rubromycosis, candidosis, deep fungal diseases) occur mainly at the veterinary physicians, zootechnicians, vivarium employees who contact with animals ill on mycosis as well as hairdressers, bath men and medical employees contacting with those who are ill on fungal diseases.

Etiology. The disease is caused by different species of anthropophilic, zooanthropophilic and candidal fungi. The infection occurs due to non-compliance with the work hygiene rules, due to contact with the ill people and animals.

Clinical findings. It corresponds to the clinical symptoms of non-professional diseases of the similar features.

Diagnostic. Diagnosis is based on the anamnesis, clinical findings, the results of bacterioscopic and cultural research.

Treatment. It is necessary to eliminate the contact of the ill person with the production factor. The systematic and local antimycotic drugs are prescribed taking into account the etiology of mycosis, localization and severity of the affection.

Prevention. Sanitary standards for manufacturing areas and work hygiene rules shall be observed, the current disinfection shall be held, the staff shall be provided with working clothes and shall store it correctly; the deratization is necessary if there are any rodents. They should hold periodic medical inspections and take care of the proper treatment and of taking the individual preventive measures; personal, social and health education is required.

1. The professional dermatosis develops:

A. Under the contact with different irritants in work conditions
B. As the result of genetic predisposition
C. Under viscerapathology
D. As the result of inefficient treatment
E. Due to injuries

2. What skin tests are used in professional dermatoses:

A. Application tests
B. Spot tests
C. Scarification tests
D. Intradermic tests
E. Everything is right

3. What are the features of the simple contact dermatitis:

A. Absence of incubation period
B. Long-term incubation period
C. Prevalence of the affection
D. General condition disorder
E. Strong itchiness

4. What are the causes of the professional signs (stigmas):

A. Work with dyes
Б. Work in hot shop
C. Work with plants
D. Work in open space
E. Everything is right

5. What external irritator may cause the simple contact dermatitis:

A. Use of antibiotic pastes
B. Use of laundry powders
C. Contact with synthetic fabric
D. Use of cosmetics
E. Everything is right

6. The simple contact dermatitis is:

A. Acid burn
B. Dermatitis caused by the contact with jellyfish
C. Dermatitis caused by the contact with household detergent
D. Dermatitis caused by the contact with plant
E. Everything is right

7. What is the influence of the professional signs (stigmas) on working capacity:

A. Disorder of working capacity
B. Temporary disorder of working capacity
C. No disorder of working capacity
D. Physical disability
E. Temporary disorder of working capacity

8. What are the typical features of the professional eczema:

A. Hive localization on hands
B. Asymmetric eruption
C. Hive localization in knife pleats
D. Hive localization on mucous
E. Everything is right

9. What are the symptoms of the simple contact dermatitis:

A. The affection area corresponds to the boundaries of the irritator's activity
B. The affection area is beyond the boundaries of the irritator's activity
C. Presence of incubation period
D. Degree of dermatitis manifestation does not correspond to the impact of the irritator
E. Hive of the haemorrhagic origin

10. What are the main preventive measures of the professional toxicoderma:

A. Collection of allergological anamnesis
B. Treatment of concomitant diseases
C. Use of personal protection if necessary
D. Work hygiene observance
E. Everything is right

Task 1. The worker of the cement plant visited a dermatologist with complaint on itchiness, redness, edema, madescence of hand skin. The affection boundaries are unclear; there is no eruption in other skin areas. He has been ill for almost one year with periodic recovery during vacation.

a) Choose the most probable diagnosis:
- **A**. Allergic professional dermatitis
- **B**. Chronic ulcerative pyoderma
- **C**. Seborrheic eczema
- **D**. Microbial eczema
- **E**. Professional eczema

b) Make a differential diagnostics.

Task 2. The 56-year old patient who had a permanent contact with ultraviolet radiation visited a dermatologist. He has complaint on roughening of the opened skin areas, crack and scars thereon.

a) What diagnosis may be suspected:
- **A**. Professional stigmas
- **Б**. Lupus erythematosus
- **C**. Chronic dermatitis
- **D**. Allergic dermatitis
- **E**. Toxicoderma

b) Make a diagnosis, choose the methods of treatment and preventive measures.

Task 3. The 46-year old driver visited a dermatologist with complaint on hand skin affection after engine repair work on freeze. The subjective symptoms are itchiness, feeling of tingling, burning, pain. The objective symptoms are: stagnant cyanochrous colour of skin, edema.

a) What is the provisional diagnosis:
- **A**. Perfrigeration
- **B**. First degree freeze injury
- **C**. Second degree freeze injury
- **D**. Psoriasis
- **E**. Professional eczema

b) Make a differential diagnostics.

Task 4. The 52-year old woman who worked as a yard-keeper visited a dentist. She has complaint on lip affection, itchiness in this area on eating spicy food. Has been ill for three months. Objective symptoms: dryness, peeling, cracks, scabs on the vermilion surface.

a) What is the provisional diagnosis:
- **A**. Freeze injury
- **B**. Lichen acuminatus
- **C**. Cheilitis
- **D**. Syphilis of the secondary stage
- **E**. Psoriasis

b) Determine the criteria of differential diagnostics of the present pathology, put on medication

Task 5. The 25-year old woman visited a dermatologist. She has been working in a greenhouse as flower cultivator. She has been ill for two weeks. Clinical findings are: erythematic spots, follicular papules, excoriation on the opened skin area. The subjective symptom is itchiness.

a) Choose the most probable diagnosis:
- **A**. Allergic professional contact dermatitis
- **B**. Melanoderma
- **C**. Limited hyperkeratosis
- **D**. dermatoconiosis
- **E**. Eczema

b) Name the causes of the abovementioned disease, make a differential diagnostics, put on medication.

Answers for first level self-control questions
1 – A, 2 – E, 3 – A, 4 – E, 5 – E, 6 – A, 7 – C, 8 – A, 9 – A, 10 – E
Answers for second and third level self-control questions
1a – E, 2a – A, 3a – B, 4a – C, 5a – A

10 TOPIC Bullous dermatosis

There are non-communicable dermopathies which most components of hives is a bulla belongs to bullous dermatosis. There are following dermatosis belongs to them:

1. Pemphigus Vera (acantholytic):
   a) pemphigus vulgaris;
   b) pemphigus vegetans;
   c) pemphigus foliaceus;
   d) seborrheal pemphigus.
2. Nonacantholytic pemphigus of oral mucosa.
3. Pemphigoids
   a) bullous pemphigoid;
   b) cicatricial pemphigoid;
   c) pemphigoid gestationis.
4. Dermatitis herpetiformis:
   a) Duhring disease;
   b) Sneddon-Wilkinson disease.
5. Genetic bullous dermatosis
   a) Gougerot-Hialey-Hailey disease;
   b) Group of bullous epidermolysis.

EDUCATIONAL AND TRAINING PURPOSES

- Determine possible conditions and triggers for emergence of pemphigus vera and Duhring disease
- Examine classification and common characteristics of different forms of pemphigus vera and Duhring disease
- Examine common clinical course and clinical findings of pemphigus vera and Duhring disease
- Determine clinical findings of typical appearances of pemphigus vera and Duhring disease
- Determine principles of therapy for pemphigus vera and Duhring disease

10.1 Pemphigus Vera

Pemphigus Vera is a malignant autoimmune disease which declares itself as elaboration on uninflamed skin and mucous coats of bulla being developmental as a result of acantholysis and generalized for whole skin integument without appropriate treatment.

TO KNOW:

- classification of bullous dermatosis;
- modern conception of aetiopathogenesis of pemphigus vera;
- pathomorphological changes of skin at pemphigus vera;
- its' clinical forms;
- clinical laboratory diagnostic criterion of this disease;
- methods and principles of remedial measures and periodic health examination of patients with pemphigus vera.

TO BE ABLE TO:

- correctly determine the Nikolsky's sign;
- perform the differential diagnostics with dermatosis having similar clinical presentation;
- assign corresponding laboratory assessment required for diagnosis confirmation;
- prescribe balanced treatment for patient with pemphigus vera.

Aetiopathogenesis. Aetiology is not investigated fully but autoimmune mechanisms are very important in pathogenesis. The circulating autoimmune antibodies of lgG type related to intercellular substance of prickle-cell layer and membrane antigens of prickle epithelial cells were discovered in organism of patient with pemphigus. For the now, there is no clear vision of autoimmune antibodies' action mechanisms during pemphigus vera. The patients have been discovered autoimmune antibodies for albumins of skin keratinocyte cell-cell becoming a part by desmos and forming relations between cells – desmoglein-3 (DSG3) and desmoglein-1 (DSG1).

Interaction of autoimmune antibodies with DSG1 and DSG3 leads to acantholysis (breaking of cytoadherence between keratinocytes) resulting in formation of clefts filled up with subcutaneous water inside the epidermis and mucous coats and appearance of bulls. As far as epidermis growths the bulls are opens resulting in formation of anabrosis on skin and mucous coats.

The difference in damages being observed during pemphigus vulgaris and during pemphigus foliaceus is aligned with distinction in localization and density of DSG1 and DSG3 expression. Desmoglein-3 is expressed in deep layers of epidermis and in epithelium of mucous coats while desmoglein-1 is expressed in facial layer of skin keratinocytes. The expression of these desmogleins is observed mostly in cells of laminated epithelium. It depends on human's age and changes in accordance with allocation of cells in epidermis. It is understandable that bundling of autoimmune antibodies with DSG3 molecules on the surface of keratinocytes plays decisive role in process of acantholysis. Great significance in acantholysis induction the apoptosis has induced by failure of cells normal functioning because of lost contact with adjacent cells.

It is proved that dominant class of autoimmune antibodies at pemphigus the lgG antibodies are of lgG_4 subclasses mostly, more rarely these are antibodies of lgG_1 and lgG_2 classes. The patients with active form of disease have the immunoglobulins of lgG_1 and lgG_4 classes as main autoimmune antibodies. The patients in disease-free survival the dominant antibodies are antibodies of lgG_1 class but they also have antibodies of lgG_4 class since its dilution is much less than at recrudescence of disease. The result of antibodies action is an attenuation of intercellular substance and destroying by desmos i.e. loss of connection between epidermal cells (acantholysis). As a result of acantholysis the acantholytic bullas typical of Pemphigus Vera are appear in epidermis (in distinction to pemphigoid).

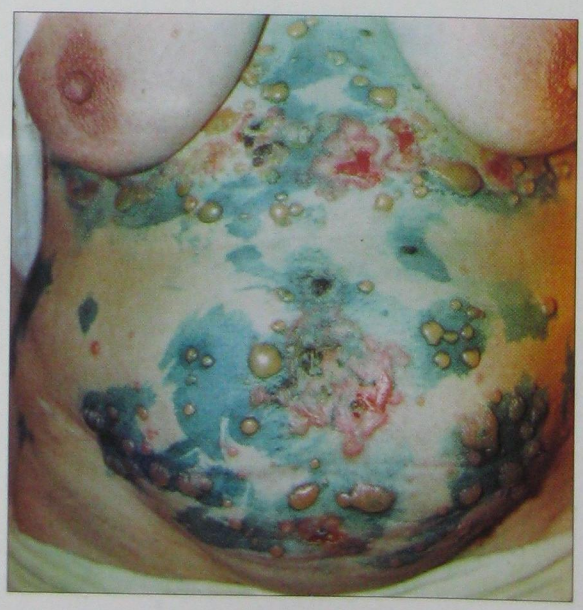

Fig. 10.1. Pemphigus vulgaris.

Clinic findings. There are four clinical forms of pemphigus vera which are vulgaris (common), seborrheal (pemphigus erythematosus), vegetans and foliaceus.

Pemphigus vulgaris occurs more often in comparison with other types of pemphigus vera. As a rule, the disease begins from damage of oral mucosa and throat which could exist separately during few months. Bulla breaks fast and transforms into painful bright-red or covered by whitish deposit of anabrosis bordered by pieces of epithelium (residues of bulla cover). Then bulla appears on skin. Bulla are located on externally unchanged skin and they full of transparent serosal content.

After existence during few days the bullas are open having been left anabrosis of bright red color. Sometimes their content becomes opacity or suppurative. Common condition of patients is satisfactory at first but it becomes worse gradually. The weakness appears and a low-grade fever. Anabrosis are epithelized slowly.

Pemphigus seborrheal (erythematosus) occurs on seborrheal areas of skin (face, hairy part of the head, cervix, dorsum). It is characterized by occurrence of spots and soft surface bulla with thin tegmentum which are fast-transformed into scaly crusts. After crusts being removed the wet eroded surface is open. The formation of bulla could be hidden resulting in impression of primary appearance of crusts which reminds seborrheic dermatitis.

As distinct from pemphigus vulgaris the course of disease is long-standing and relatively nonmalignant. The common condition of patients is agglutinin aggravates at condition of prominent prevalence rate of process only.

Pemphigus vegetans is characterized by such differential characteristic as appearance on the bottom of bulla of enlargements in form of vegetations which overhang over the faying surface. Typical places of hives localization are outer genital organs, big sentinel piles, inner surface of hips, oral mucosa.

Pemphigus foliaceus declares itself by bulla which has soft rugate tegmentum, layered scaly furfur and crusts. The differential characteristic of this form is absence of regeneration under crusts and formation of new bulla on place of cicatrization.

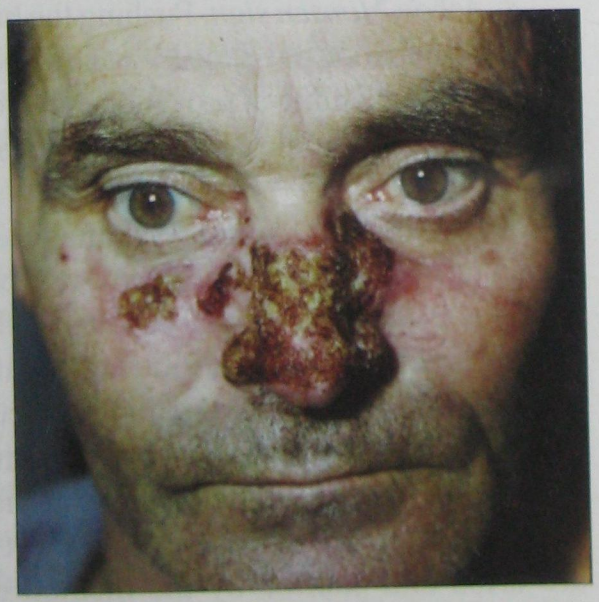

Fig. 10.2. Pemphigus seborrheal.

Diacrisis. The criteria of diagnostics for pemphigus vera include clinical and laboratory evidences of presence of acantholysis in epidermis and epithelium of mucosa coats.

There are seven most criteria distinguished:

1. Typical clinical picture of damage: the bulla on unchanged skin,

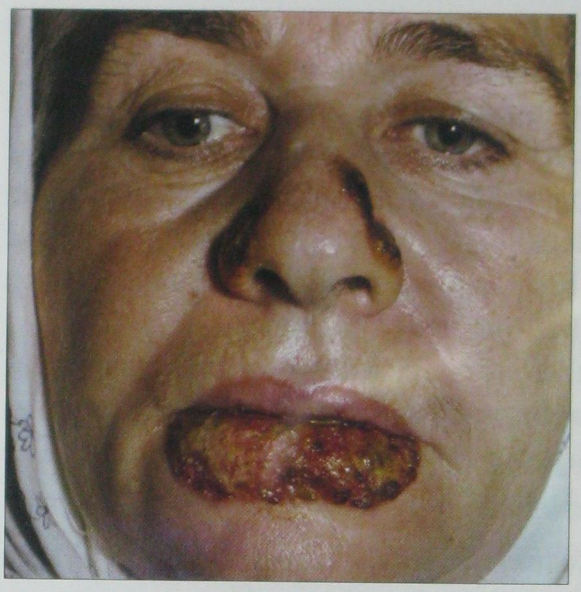

Fig. 10.3. Pemphigus vegetans.

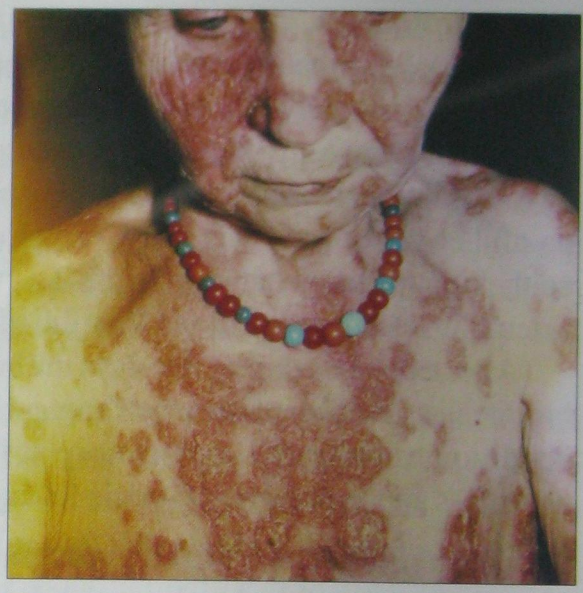

Fig. 10.4. Pemphigus foliaceus.

long-standing anabrosis on skin and on unchanged oral mucosa, conjunctiva, nasal mucosa, genital organs. There are residues of bulla tegmentum could be observed on the periphery of anabrosis.

2. Nikolsky's sign on externally unchanged skin. At weak tension by finger of outwardly healthy skin near the bulla and sometimes far from them the lamination of epithelium surface layers occurs with formation of anabrosis. This phenomenon is considered to be most informative at diagnosis of all types of pemphigus. However, it is not pathognomonic.

3. Nikolsky's edge sign. While pulling by forceps of pieces of bulla tegmentum the lamination of epithelium out of borders of visible anabrosis occurs.

4. Asboe-Hansen sign. Pushing the bulla leads to increasing of its square.

Determination of Nikolsky's and Asboe-Hansen's signs are important diagnostic clues for establishing of provisional diagnosis. However, they are non-specific. The cytological, histologic and immunomorphologic approaches are used for diagnosis confirmation.

5. Cytological approach of diagnostics (cytodetection by Tsank) provides obtaining of impression smears from the bottom of fresh anabrosis. For this the object carrier is used which is solidly applied to the surface of fresh anabrosis. For getting of smears from anabrosis on mucosa coat of bony and soft palate and throat the mediated approach of its obtaining is used. The soft scraping of anabrosis surface is performed by blunt depressor or by nutrient spoon avoiding visible damage of surface and escape of blood after which the material being taken from the bottom of anabrosis is applied carefully to the object surface in form of smear. Obtained smears are dried and painted by the method of Romanovsky-Gimza. During further microscopic evaluation of specimens the Tsank's acantholytic cells are determined. This is a changed cells of spinous layer which were subject to acantholysis,

degenerated and thus obtained morphological and tinctorial properties which differs them from normal cells of this layer:

- they are round (oval), disengaged, and less than normal epidermal cells by size;
- the nucleus of acantholytic cells are painted intensively;
- there are two or three small nucleus could be discovered inside the enlarged nucleus;
- cytoplasm of cells is extremely basophilic and is painted unregularly; the light-blue area is formed around the nucleus and the condensation of painting in view of intense blue border occurred on periphery;
- under pemphigus the acantholytic cells could form a symplast cells which have several nucleus.

6. Histologic approach of examination is one of the most and obligatory while confirmation of pemphigus vera diagnosis. It is necessary to biopsies the fresh bulla or marginal layer of anabrosis with pinch of undamaged skin. The most earlier histologic changes in epidermis under pemphigus vera are vacuolar degeneration and disappearance of cell bridges in bottom part of spinous layer. Because of acantholysis the clefts appears inside the epidermis and then a bulla located suprabasally (i.e. over basal layer of cells, intraepithelial). The typical histologic characteristic under pemphigus vera is also discovering of separate changed prickle cells which after loss of connection with each other are left attached to the layer of unchanged basal cells.

Immunomorphologic approaches play decisive role in diagnostics of pemphigus vera. Even at early developmental stages the approach of direct ELISA on frozen skin sections allows to discover the deposits of class G immunoglobulines and complement localized in intercellular space of epidermis (greenish fluorescence). The approach of indirect immunofluorescence in blood and bulla liquid allows to discover high dilutions of autoimmune antibodies (lgG) in relation to proteins of elements by desmos. The height of their dilutions is straight correlate with heavy of section for pemphigus vera.

Treatment. Till now, the causation of pemphigus vera is not known so the treatment of this group of diseases is left pathogenetic and directed to depression of fusion of autoimmune antibodies to the proteins of epidermis spinous cells' desmosomal linkages. The main remedy of the *systemic medication* of patients with pemphigus vera are the glucocorticosteroid hormones. They are assigned independently (monotherapy) or together with cytostatic (combination therapy) – with azathioprine or metatrexate, cyclophosphamide, cyclosporine A. Combination therapy is implemented during treatment of pemphigus which is resistant to the high doses of glucocorticosteroids and in case of necessity of its daily dose decreasing (loading or maintenance).

The treatment of patients with pemphigus vera should be started from loading doses of glucocorticosteroids, preferably from prednisolone or prednisone. The dose of prednisolone of 100-120 mg per day is an adequate loading dose allowing to stop the forming of bulla and to enforce the epithelialization of anabrosis.

When the glucocorticoid therapy is assigned it is necessary to remember that efficiency of treatment is increased if daily dose of hormone is divided in accordance

Table 10.1.
Differential diagnostics of clinical forms of pemphigus vera

Evidences	Pemphigus vulgaris	Pemphigus vegetans	Seborrheal pemphigus	Pemphigus foliaceus
Location	Visible mucosa coats, more often in oral cavity; skin of head and body; could appear in skin folds	Mucosa coats, more often oral one, big skin folds, places of transition of skin into mucosa coats	Face, hairy part of the head, mammary gland, dorsum, rarely the oral mucosa coat could be damaged	Skin of head, mammary gland, dorsum. The damage on a face could have a shape of butterfly
Clinical implications	Soft unstable bulla on unchanged skin and mucosa coats, long-standing anabrosis under crusts (out of folds), pigment spots	Soft bulla opened fast, on unchanged mucosa coat and skin; stable anabrosis with whitish (pultaceous) vegetative surface, enlarged and interlocked	Rash is represented by weak infiltrated erythematic patches covered by furfur and lamellar crusts removing of which leads to cropping out the surface anabrosis. There are thin-shelled soft bulla could be observed on erythematic background.	The mucosa coats are not damaged. Erythematic sites of damage tend for peripheral growth, covered by lamellar furfur removing of which leads to discovering of anabrosis. Separate flat bulla on erythematic background. The peripheral growth, interlocking of sites of damage, and forming of exfoliative erythrosis are typical.
Nikolsky`s sign	+	+	++	+++

Table 10.1. (cotinued)

Evidences	Pemphigus vulgaris	Pemphigus vegetans	Seborrheal pemphigus	Pemphigus foliaceus
Cytodetection (impression smear)	Acantholytic cells	Acantholytic cells, eosinocytes	Acantholytic cells are discovered rarely	Same
Histodiagnosis	Intraepidermal, suprabasal acantholysis determines the origin of fissured cavity paved by spinous (acantholytic) cells	Intraepidermal, suprabasal acantholysis, fissured suprabasal cavities, pseudoepitheliomatous hyperplasia, acantholytic cells, numerous eosinocytes	Intraepidermal, suprabasal acantholysis, leukocytic infiltration of papillary dermis	Intraepidermal, suprabasal acantholysis determines the origin of fissured cavities under horny layer and at level of granular layer. Small inflammatory leukocytic infiltrate in dermis
Direct IF	Deposits of lgG and C_3-complement on intercellular linkages level of epidermis` spinous layer cells	Same	Deposits of lgG and C_3-complement in intercellular spaces and directly under epidermis	Same
Non-Direct IF	Antibodies against intercellular substance of epidermis (lgG)	Same	Same	Same

with physiological rhythm of provisional cortex secretion hormones. Maximum dose of glucocorticosteroids (usually it is a two thirds of daily dose) is being used after meal in morning and one third more at day. The therapy should be started after complete clinical examination of the patient and verification of diagnosis (biopsy of damaged skin, immune histochemical tests). Unfortunately, there are no such drugs which will decrease selectively the formation of autoimmune antibodies of pemphigus only without simultaneous depression of biosynthesis of many other protective antibodies. A variety of other undesirable effects (*Exogenetic (medicamental) Cushing syndrome*, immunosuppression, water-salt balance failures, hypoproteinosis, intestine issues and other) is opposed to the same anti-inflammatory and antiproliferative action of glucocorticosteroids.

There are water and alcohol solutions of aniline colorants used for local therapy as well as aerosols, unctures and creams containing glucocorticosteroids.

Course and prognosis. The course is chronicity with intermitting remissions and recrudescence. The using of glucocorticosteroids and immunosuppressants rapidly improves the prognosis. Cause of death for the now is generally results of continuous treatment by glucocorticosteroids and immunosuppressants.

10.2 Duhring disease

Duhring disease is a long-lasting dermatosis of unknown causation which is characterized by continuous anticipate course with attack-like appearance of pruritic polymorphous eruption on skin of body and extremities.

TO KNOW:

- classification of bullous dermatosis;
- modern conception of aetiopathogenesis of Duhring disease;
- pathomorphological changes of skin at this disease;
- clinical forms of Duhring disease;
- its' clinical laboratory diagnostic criterion;
- methods and principles of remedial measures and periodic health examination of patients with Duhring disease.

TO BE ABLE TO:

- correctly determine the Nikolsky's sign;
- perform the differential diagnostics with dermatosis having similar clinical presentation;
- assign corresponding laboratory assessment required for diagnosis confirmation;
- prescribe balanced treatment for patient with Duhring disease.

Aetiopathogenesis. The hypersensibility of organism to the vegetable protein (protein containing in grain varieties) which is appear in intestinal canal during digestion process and to the halogens (iodine, fluorine) plays role in pathogenesis. The provoking factors are inflammatory processes in gastrointestinal tract, lumbricosis, malignant tumors, using of halogens, and contagious diseases.

Duhring disease is more often progresses with individuals who have pathematology of small bowel with intestinal malabsorbtion – malabsorbtion syndrome (gluten entheropathy) which is conditioned by absence of tolerance to gluten. It is considered that gluten plays a role of antigen of protein nature and initiates the production of lgA-antibodies inside the gastrointestinal tract. The result is that reboiling of graminoids containing gluten leads to forming of immune complexes which causes to damage of mucosa coats of small bowel with progression of villous atrophy and intestinal tract parafunction. Through the damaged papillae of mucosa the immune complexes goes to general circulation penetrating to the papillary dermis and fixing there. Most of patients with Duhring disease have deposits of lgA-antibodies in structure components of dermal papillae at area of dermoepidermal junction of skin damaged sites and around. The deposits of lgA-antibodies is often allocated in view of granules on the top of the papillary dermis and inside them and it is more rarely allocated in-line, along the basement membrane. The antibodies circulating to the gluten and its fraction which gliadine is or circulating immune complexes are discovered in blood.

Clinic findings. As regular, the disease begins from occurrence of pruritus, heat or tingling which could be accompanied by general uneasiness, rise of temperature and usually outrun the occurrence of hives for several hours, days or even months. The hives is usually symmetric, could be at any site of skin integument, but favorite places are extensor surfaces of extremities, skin of body and buttock.

Hives at Duhring disease is characterized by true pleomorphism. Following initial components could be separated:

- *erythematic* spots and papule which being interlocked and grouped forms figures with different contours (herpetiform groups);
- strained *vesicles* on edematous warp having tend to grouping and herpetiform allocation; after being opened they form anabrosis on edematous background on the periphery of which the pieces of vesicles tegmentum are observed; The crusts are formed on the anabrosis surfaces, the epithelialization occurs fast under them, often the areas of hyperpigmentation are left;
- *strained bulla* with diameter of 0.5-2 cm and more with solid tegmentum and at first with serosal and then with opacity content; bulla appear as a rule on slightly edematous erythematic but could appear on externally unchanged skin; in number of cases the soft bulla discovered with thin tegmentum; after bulla being opened the anabrosis forms on its places without tendency for peripheral growth, have relatively fast epithelialization and then left stable hyperpigmentation; bulla on oral mucosa coat appears extremely rare.

Morbid anatomy. During Duhring disease the following is discovered histologically:

- subepidermic bulla in content of which the eosinocytes are prevail;
- microabscesses located on the tops of dermal papillae and containing neutrophils, eosinocytes, polymorphonucleocytes and deposits of fibrins;
- oedema and infiltration of derma which is stipulated by angiectasis and accumulation of perivascular eosinocytes, neutrophils and destroyed nucleolus («nuclear dust»).

Diacrisis. Diacrisis of Duhring disease is based on following clinical information:

1) continuous pruritus of different gradation, sometimes esthesia of heat, tingling and painfulness appears;

2) true polymorphism of hives which could be accompanied with false polymorphism;

3) herpetiform grouping of hives components;

4) chronicity which is interrupted by complete or incomplete remissions of three months to one year duration and more.

The content of eosinocytes in blood and bulla liquid where it is far exceeds is determined for diagnosis confirmation. The patients with Duhring disease are differed by hypersensibility for iodic medical product so the three-glass test with 50% potassium iodide is used which is performed in two options – epicutaneous and per os. Analysis is considered to be positive in case of in-patient recrudescence. The negative Nikolsky's sign and absence of acantholytic cells in bulla content are typical.

Treatment. The lifelong gluten-free dietary intervention is indicated. The abuse of wheat, corn, barley, oats and millet i.e. bread, alimentary products, manna, oat and wheat grits is excluded. There are mushes from buckwheat, rice or corn grits allowed. Moreover, the products which the presence of iodine is considered to be in are excluded (seafood).

The most effective modes of therapy for Duhring disease now are medical products of sulfonic range – diamino diphenyl sulfone (DDS), dapsone, aulosulfone, diucifone, and sulfapyridine. Usually, the DDS or diucifone is assigned by 0.05-0.1 g twice per day in cycle of 5-6 days with interval of 1-3 days (3-5 cycles and more). Course dose depends on effectiveness and tolerance of a drug.

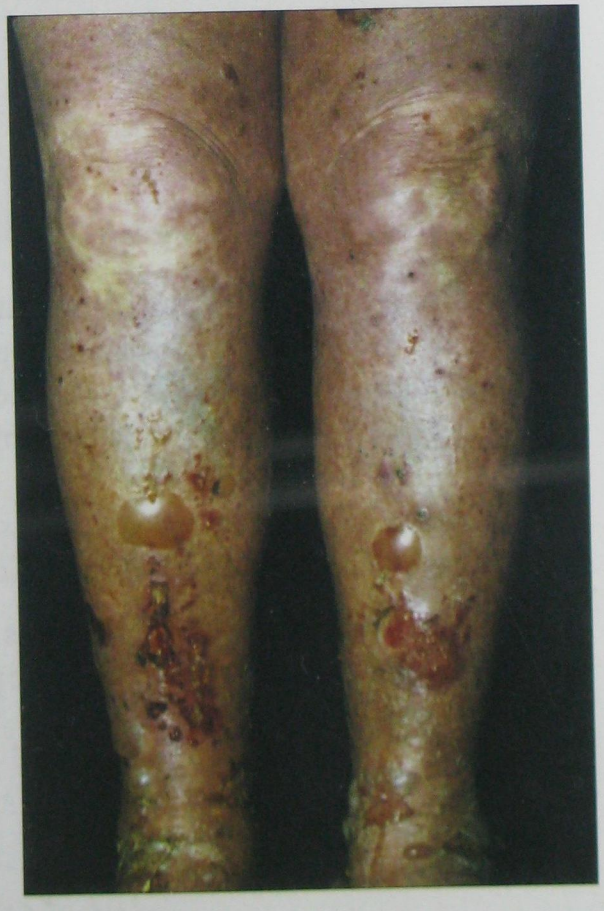

Fig. 10.5. Duhring disease.

Table 10.2
Differential diagnostic characters of pemphigus vera and Duhring disease

Indicants	Pemphigus vera	Duhring disease
Age	Children ill very rare	Any age
Start of disease	At first the oral mucosa is damaged and then a skin	The skin is damaged but the oral mucosa is not
Bulla`s nature	Soft unstrained pear-shaped bulla	The bulla ar e strained and have round shape
Location of hives	Non-grouped	Grouped
Anabrosis	Major	Small
Allocation of bulla in relation to epidermis	Intraepidermal	Subepidermal
Nikolsky`s signs	Positive	Negative
Hansen`s symptom	Positive	Negative
Three-glass test	Negative	Positive
Tsank cells	Discovered	Not discovered
Presence of eosinocytes in bulla and peripheral blood	Absent	Present
Pruritus of skin	Non-typical	Very strong
Process of epithelialization of anabrosis	Slow	Fast
Presence of antibodies	Antibodies of lgG class	Antibodies of lgA class

There are hyposensitizaton and antihistamine drugs, vitamin therapy (vitamins of B, A, E and C group), disintoxication therapy and antioxidants (folic acid, methionine, Retabolil and others), anxiolytics and anxiety medications also indicated.

While intake the sulfones the erythropoiesis stimulating agents are assigned (vitamin B_{12}, hemostimulin).

The spirits of aniline colorants are used *for local therapy* as well as aerosols, creams and unctures containing glucocorticosteroids.

Course and prognosis. The course is chronicity, anticipate with decreasing of Duhring disease's evidences intensity. The prognosis is relatively advantageous.

1. There are following types of pemphigus vera separated:

A. Vulgaris
B. Vegetans
C. Foliaceus
D. Seborrheal
E. All of mentioned above

2. For treatment of pemphigus the most effective are:

A. Antibiotics
B. Sulfanilamides
C. Glucocorticosteroids
D. Choloroquine
E. Ftivazide

3. The test provided for confirmation of Duhring disease is about tolerance to:

A. Sulfanilamides
B. Antibiotics
C. Glucocorticoids
D. Iodine
E. Procaine

4. The most important thing for confirmation of pemphigus vera diagnosis is:

A. Discovering of acantholytic cells in the impression smears
B. Eosinophilia in anabrosis of bulla surface
C. Leukocytosis in peripheral blood
D. Discovering of LE-cells
E. Frank trombocytopenia

5. Typical location of rashes at pemphigus vulgaris is:

A. Body, oral mucosa coat
B. Hairy part of the head, body
C. Palms and feet
D. Genitals and face
E. Genitals and oral mucosa coat

6. The following are typical for Duhring disease:

A. Polymorphism of rashes
B. Leukocytosis
C. Damage of oral mucosa coat
D. Discovering of acantholytic cells in impression smears
E. Positive Nikolsky's sign

7. The Tsank cells are:

A. Acantholytic cells
B. LE cells
C. Stem cells
D. Specialized ganglion cells
E. Thymic cells

8. What symptom has special diagnostic consideration at pemphigus vera:

A. Symptom of «apple jelly»
B. Symptom of «stearic spot»
C. Nikolsky's sign
D. Besnier's symptom
E. Kebner's symptom

9. Nikolsky's sign means that:

A. The peeling is increase during erasion of components
B. Lamination of top layer of epidermis when mechanical influence
C. When pressing the bosselation the slowly disappeared pitting is left on its surface
D. Presence of Wickham's mesh
E. Discharge of blood from enlarged vases at strong press

10. Leading pathological process of pemphigus is:

A. Dyshidrosis
B. Acantholysis
C. Apoptosis
D. Hyperkeratinization
E. Spongiosis