Term	Topic content
Dermatitis	- is considered an inflammatory lesion of skin caused by a direct effect of various exogenous factors.
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). **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 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. **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.
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. **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 painfullness, 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 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.
Seborrheic eczema	is 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.
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 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 keratolyses, 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.
Toxidermia	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. **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: penetration of allergen, substances into the bloodstream; 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: /. ***Mild forms*** characterized by itching, limited urticaria, fixed erythema, multiforme exudative erythema, the reaction on lichen planus type. //. ***Moderate forms*** in which there are generalized urticaria, angioedema, bullous toxicoderma, hemorrhagic vasculitis, bromide and iodide acne, common toxic and allergic dermatitis. ///. ***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. **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 ofbromine 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 predispositioa.

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:

l) 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 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.