

**Dermatitis**  
**Cheilitis**  
**Stomatitis**  
**Toxicodermia**  
**Eczema**

# Dermatitis

- Dermatitis is an acute inflammation of the skin caused by the direct exposure to various external agents
- Dermatitis is characterized by some morphological changes of the skin at the site of exposure and a relatively quick resolution after the irritant termination
- Irritants can be obligatoty (unconditional) and facultative (conditional)



# By its nature obligatory factors may be

- 1. Mechanical (*pressure, friction, traumas, injuries, wounds*)
- 2. Physical (*high and low temperatures, UV, x-ray, radiation*)
- 3. Chemical (*injuries caused by strong acids and alkalis*)
- 4. Biological (*nettle and some types of plants, insects, caterpillars' jellyfish*)



# Facultative factors may be

1. Biological (*poisonous ivy, poisonous plants, sumac cowparsnip insects*)
2. Chemical
  - a) drugs for external use (*ointments with antibiotics, analgesics*)
  - b) metals and their salts (*cobalt, nickel, chrome*)
  - c) cosmetics (*paints, varnishes, creams*)
  - d) polymeric materials (*epoxy resins, glue polymeric dentures*)
  - e) industrial and household chemical substances (*chemicals*)



# Classification

- The following two forms of contact dermatitis are distinguished: simple and allergic
- Simple contact dermatitis and allergic contact dermatitis differ in etiology
- Obligate factors lead to the development of simple contact dermatitis
- Facultative factors lead to the development of allergic contact dermatitis



# Pathogenesis of simple contact dermatitis

- Simple contact dermatitis has only one causative factor
- It arises after the contact with an external factor that destructs the skin
- The alteration is the first stage of inflammation caused by the external factor

# Simple contact dermatitis

- Simple contact dermatitis is not a skin disease
- It is a surgical pathology caused by burns, sunburns, mechanical damages, radiation and so on
- Each person with simple dermatitis presents with inflammation after the contact with an irritant (e.g. tight footwear, sunburn, intertrigo)
- The clinical picture of simple contact dermatitis corresponds to the degree of burns, frostbite and depends on the strength and duration of exposure to the irritant



# The clinical picture of simple contact dermatitis

- The clinical manifestations of simple contact dermatitis are erythema, edema, blister, erosion, ulcer, necrosis









# The main symptoms of simple dermatitis are

1. Simple contact dermatitis arises after the action of an irritant in a few minutes or hours (an acute onset)
2. It depends on the nature, strength, and duration of exposure to the irritant (*Intensive inflammation is caused by the action of strong and long irritants; light dermatitis is caused by the action of weak and short Irritants*)
3. Complete cure at the initial state (*dermatitis is cured by a mustard plaster application in 24 hours*)
4. A particular place of inflammation (*a damaged area depends on exposure to an irritant*)
5. Absence of allergic reaction
6. Subjective sensations (*pain, burning*)
7. Resolution of inflammation (*the organism is completely restored*)
8. Topical treatment

# Treatment depends on the severity of the inflammatory phenomena

1. Avoid contact with an irritant
2. Use only topical treatment in mild skin lesions
  - In the case of erythema different ointments are used (*D-panthenol, emollients*)
  - Blisters are pinned up with a needle and a dry dressing is applied on the skin
  - In the presence of erosive and ulcerative lesions, aniline dyes and epithelizing drugs are used
3. In severe forms of simple contact dermatitis (*for example, extensive deep burns with skin necrosis*), surgical options are recommended.

# Allergic contact dermatitis

- Allergic dermatitis is a widespread allergic disease
- Allergic contact dermatitis is a form of contact dermatitis that is the manifestation of an allergic response caused by contact with a substance; the other type being irritant contact dermatitis



# Contact allergic dermatitis

- Contact allergic dermatitis is a result of allergen-specific immunologic reactions, which requires presensitization with the offending agent
- Acquired immediately-delayed hypersensitivity develops in individuals with altered reactivity, pathology of the neuro - endocrine system
- Allergic dermatitis is mediated by T - cells and characterized by the development of immunologic memory to the allergen
- Sensitization usually results from repeated use or exposure to an allergen, which is a chemical in smaller concentrations capable of triggering the characteristic immune response
- The inflammation manifested is called contact sensitivity or contact sensitization and can result from contact with numerous agents
- Allergic dermatitis more often affects women and persons with light skin and hair pigmentation

# The clinical picture

- The clinical manifestations of contact allergic dermatitis are erythema, edema, microvesicles, and papules resulting from a delayed reaction in the papillary layer of the dermis
- Immunologically this reaction is termed as eczematous reaction and morphologically - spongiosis







# Diagnostic criteria

- Skin exposure to different facultative irritants
- Polymorphic rashes (*erythema, edema, microvesicles, papules, erosion, moist crust*)
- The rash is more likely to be more widespread on the skin
- Allergic contact dermatitis is acute if it lasts for 2 months
- Allergic contact dermatitis results from prolonged exposure to irritants in a few days



# Treatment starts with

1. Eliminating or avoiding the source of irritation
2. Prescription of antihistamine drugs, desensitizing drugs (*calcium chloride*)
3. Administration of corticosteroids in severe forms
4. Topical treatment (*prescription of topical corticosteroid creams to lessen inflammation and relieve irritation*)

# Outcomes in allergic contact dermatitis are

- 1. Recovery
- 2. Development of allergic eczema









# Cheilitis

# itis

- The cheilitis is a benign inflammatory disease of the lips
- This inflammation may include the perioral skin (the skin around the mouth), the vermilion border, or the labial mucosa





# Cheilitis is classified as

- true (*exfoliative, glandular, actinic, meteorological, contact*)
- symptomatic which are one of the symptoms of diseases of the mucous membrane or of the skin

*(atopic, eczematous, macrocheilitis with Melkersson-Rosenthal syndrome with lupus erythematosus and lichen planus)*

- The following two types of contact cheilitis are distinguished: *simple and allergic*

# Simple contact cheilitis

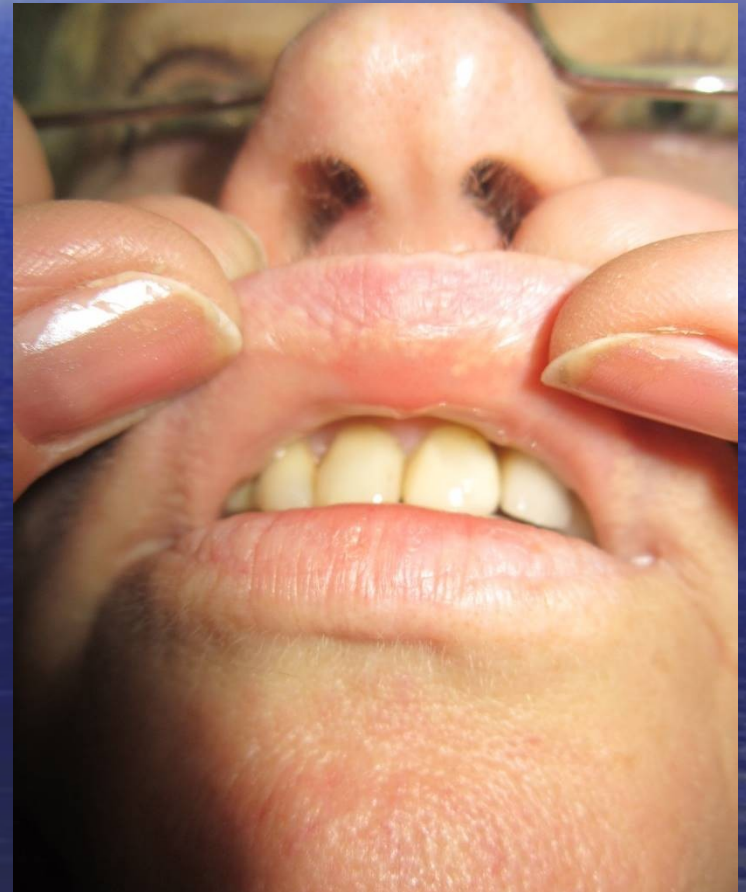
- Simple contact cheilitis is caused by obligate irritants (*high and low temperature insolation*)
- It is characterized by hyperemia, dryness of the skin, peeling fissure





# Allergic contact cheilitis

- Allergic contact cheilitis is more often caused by chemicals leading to sensitization
- It can be cosmetics lipsticks, medicines
- The clinical picture is characterized by the appearance of target erythema on the red border of the lips
- Vesicles resolve quickly and weep erosion develops in case of a pronounced inflammatory reaction
- Patients complain of itching and burning
- Sometimes a slight peeling is noted







# The treatment of cheilitis

- The treatment of cheilitis includes elimination of etiological factors
- Administration of local treatment  
(*corticosteroid ointments, gels*)
- In case of allergic cheilitis, antihistamines and vitamins are recommended

# Stomatitis

- Contact stomatitis is an acute inflammation of the oral mucosa resulting from direct contact with an exogenous irritant
- There are two types of contact stomatitis: simple and allergic





# Simple contact stomatiti s

- Simple contact stomatitis can be caused by **mechanical** factors (*pressure from a poorly made prosthesis*), **physical** (*high temperatures resulting in burns of the oral mucosa*), **chemical** (*drugs, for example, resorcinol*)
- The clinical picture depends on the strength and duration of exposure to irritants
- It can vary from the appearance of erythema to the formation of blisters, painful erosion and necrosis



# Allergic contact stomatitis

- The causes of allergic contact stomatitis are the materials most often used in dentistry (*external medicines, toothpastes, elixirs*)
- Its development is based on sensitization
- The clinical picture is characterized by the appearance of erythema, edema, vesicles, erosion, and petechiae
- The treatment of contact stomatitis is to eliminate irritating factors and the use of local treatment (*aniline stains epithelizing drugs*)



# Toxicodermia

- Toxicodermia is a common toxic and allergic disease with predominant manifestations on the skin and mucous membranes, resulting from exogenic and endogenic factors which penetrate into the skin by the hematogenic way
- These substances are considered to be allergens, but in some cases, causative substances may act directly on such parts of the skin as blood vessels, mast cells, melanocytes and so on
- Generally, the etiology of toxicoderma turns out to be allergic



# Exogenous factors are

- **Medicines** (*antibiotics, analgesics, vitamins, vaccines*). Medicinal toxicodermia is a more common form of the disease
- **Food products** (*citrus, seafood, spices, eggs, honey, nuts, strawberry, chocolate, coffee, etc*)
- **Industrial and household chemical substances** (*aerosols, inhalants, chemicals, etc*)
- These substances get into the body through the alimentary canal and respiratory airways
- Medicines can cause toxicodermia by parentally injection penetration through the skin.



# Endogenous factors

- Gastrointestinal tract disorders
- Neoplasms
- Liver diseases
- Kidneys pathology
- Thyroid gland disorders
- Helminthic invasion

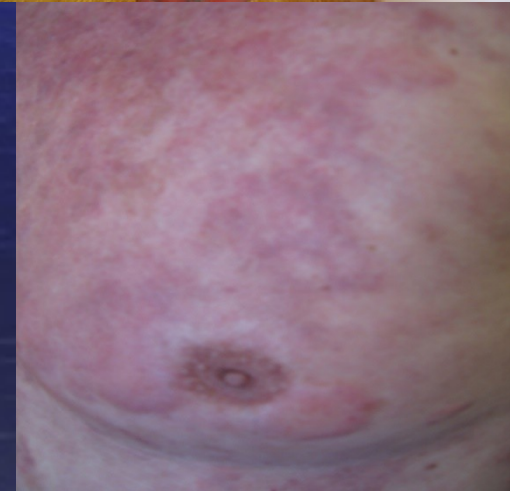


# The pathogenesis of toxicodermia

- The pathogenesis of toxicodermia is based on an allergic reaction
- Antibodies appear in the blood serum in allergic toxicodermia
- Toxic and allergic components are seen in the blood serum
- The allergic reaction may be of delayed type, immediate type or mixed type
- Contact allergic dermatitis contributes to allergic reactions of delayed type

- Toxicokodermia is characterized more often by polymorphic, less often by monomorphic eruptions of inflammatory character, manifesting against the background of a general state disorder
- Microvesicles, as in contact allergic dermatitis, appear in the reaction of delayed type
- Allergic reactions of immediate type may have various clinical manifestations
- Vascular spots such as petechial manifest due to immune response

- Proliferative processes result in the formation of papules
- If immune complexes are fixed on the surface of the deep blood vessels, nodes are formed
- The fixation of immune complexes on the melanocytes leads to the formation of hyperpigmented spots
- The influence of mast cells may cause urticaria
- If immune complexes are in the basal membrane, blisters or vesicles may be noted
- Accumulation of some substances such as halogens in the sebaceous glands may cause pustules







- The clinical picture of true polymorphism is characterized by different primary elements (*vesicles, papules, spots, urticarial, blisters, pustules, nodes*)
- Rashes are common and sometimes generalized
- Toxicoderma may be limited and common
- There are several clinical forms of toxicoderma

# Urticaria

- Acute urticaria is an allergic reaction of immediate type which is characterized by the appearance of urtica on the skin and severe itching
- It lasts no more than 24 hours



# Quincke's edema

- A serious complication of urticaria is rapidly developing edema of the deep layers of the dermis, subcutaneous tissue and submucosal layer, which is called Quincke's edema or angioneurotic edema
- Quincke's edema is more often characterized by fullness and soreness than itching
- Erythema is not observed
- Quincke's edema usually resolves during 72 hours





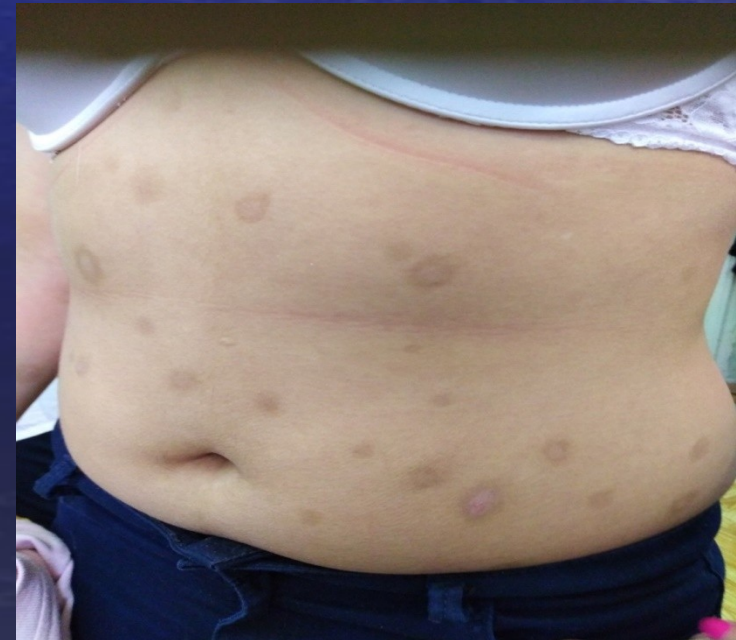
# Fixed erythema

- The cause of fixed (sulfonamide) erythema is the intake of sulfonamides or analgesics
- The presence of erythematous foci of a round or oval shape in size of 2-3 cm is a characteristic of sulferithema
- A blister may appear in the center of erythema
- In a few days they turn to brown and in 7-10 days the eruption resolves.
- Erythema recurs at the same site and leaves a persistent pigmentation
- The mucous membranes can be affected.



# Toxic melasma

- The cause of toxic melasma is the intake of analgesics
- It is characterized by the appearance of brown spots





# A generalized form of toxicodermia

- A generalized form of toxicoderma begins after several hours or several days after the drug intake
- The clinical picture of a generalized form of toxicoderma is distinguished by polymorphic rashes: *vesicles, papules, erythema, blisters, urticarial, pustules, and nodes*
- Lesions of the skin and mucous membranes may be noted
- The symptoms of intoxication may develop (*fever, malaise, weakness, anorexia*)
- The subjective sensations are itching, burning





# Iododerma and bromoderma

- Iododerma and bromoderma (iodide and bromide or halogen acne) arise from the intake of bromide and iodide preparations
- Halogens are excreted by sebum
- Exposure to the skin of bromine and iodine preparations that change the sebum chemistry contributes to the activation of staphylococcal infection and the appearance of acne
- The clinical picture is characterized by the appearance of edematous red pustules with vegetative surface purulent crusts, sometimes symptoms of intoxication may develop



# Lyell's syndrome

- Lyell's syndrome (*toxic epidemic necrolysis*) is an allergic reaction
- In its severity it is compared to anaphylactic shock
- The mortality for toxic epidermal necrolysis is 25–30 % in inadequate treatment
- It develops in a few hours or days after the first intake of the drug
- It arises sharply and is characterized by the development of epidermal necrolysis and necrolysis of the mucous membranes of the mouth
- Rashes are common



# Lyell's syndrome

- The general condition of patients is extremely severe
- The phenomena of intoxication and dehydration are expressed
- These symptoms are often accompanied by coma
- At first, diffuse erythema appears
- Then within 12 hours a massive exfoliation of the epidermis takes place
- It resembles burns of the 2nd degree
- Blisters of various sizes appear and the massive painful erosions develop



# Lyell's syndrome

- The most characteristic lesion of the mucous membranes of the mouth, nose in the form of hyperemia, edema followed by the development of large blisters, which, rapidly opening, form extremely painful extensive erosion and ulceration with the remnants of the blister covers
- The symptoms of Nikolsky and Azbo-Hansen are sharply positive
- Lyell's syndrome is accompanied by a rise in body temperature of 38-40 ° C and dystrophic changes in internal organs: the heart, kidneys, liver
- Leukocytosis and acceleration of ESR are presented in the blood



# Treatment of toxicodermia

- Elimination of allergen (*diuretics, laxatives, gastric lavage*)
- Detoxification (*infusion therapy: physiological solution, saline solutions*)
- Enterosorbents (*lactofiltrum, enterosgel, polyphepan*)
- Antihistamines (*suprastin, tavegil, cyterisins, clarithin, loratadine*)
- Hyposensitizing (*calcium, gluconate, sodium thiosulfate*)
- Locally: lotions, powders, shaken fluids, corticosteroid ointments and creams

# Treatment of Lyell's syndrome

- Treatment of Lyell's syndrome includes:
- large doses of systemic corticosteroids (*prednizolon 100-120 mg*)
- symptomatic treatment (*analgesics, antibacterial drugs, infusion therapy*)
- plasmapheresis, hemosorption



























# *Eczema*

- Eczema is a chronic noncontagious inflammatory disease of the skin, neuroallergic in nature characterized by various eruptions, a burning sensation, itching, and a tendency to recur
- A variety of factors, both exogenous and/or endogenous are conducive to the development of eczema leading to damage to the epidermis
- Eczema is the most common dermatoses of allergic origin
- It accounts for up to 40% of all cases of acute and chronic skin pathology



- Eczema is caused by a complex of neuroendocrine metabolic infectious-allergic vegetative-vascular and hereditary factors
- Immunogenic characteristics of the body (linked with HLA, B22 and HLA, Cw1 antigens) play an important role in the development of eczema



# Etiology and pathogenesis of eczema

- Changes in the central nervous system
- Imbalance between the sympathetic and parasympathetic parts of the autonomic nervous system (with a predominance of parasympathetic influences)
- Changes in the endocrine system
- A variety of gastrointestinal tract disorders
- Metabolic disorders of the trophic tissue

# Exogenous and endogenous triggers

- Exogenous triggers are chemical substances, bacterial agents, physical factors, medicines, food products
- Endogenous triggers manifest microorganism antigenic determinants from the foci of chronic pathological infection (*helminthiasis*) of the internal organs (*most often gastrointestinal tract*)

# Clinical forms of eczema

- Several forms of eczema are distinguished, depending on the cause, site, and nature of the inflammation
- They are:
  - *true eczema*
  - *microbial eczema*
  - *seborrheic eczema*
  - *professional eczema*



# True

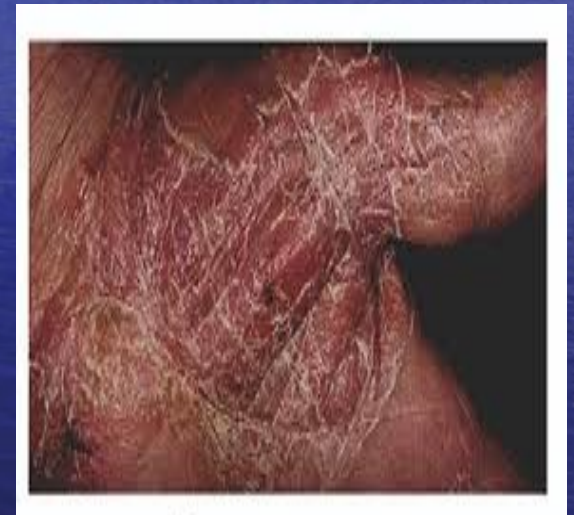
## eczema

- True eczema is marked by a sudden onset
- multiple sites of inflammation including the nails
- symmetrical location of the foci, which have indistinct contours;
- reddening of the skin in the affected area
- Other symptoms are the formation of vesicles, some of which change into pustules or moist erosions (*hence the obsolete name "weeping lichen"*), and later the formation of scales and crusts



- There are three stages of eczema

- *weeping (moist):*
- *eczema squamous*
- *eczema crustosum*





# The microbial form of eczema

- The microbial form of eczema develops as a result of an immediately-delayed hypersensitivity to staphylococci and their metabolic products
- It is often associated with malnutrition of the skin due to the vascular pathology (*varicose of veins, thrombophlebitis and endarteritis*)
- The activation of pathogenic flora and a decrease in local immunological protection lead to microbial sensitization and the development of an eczematous process

# Microbial eczema

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- Most often, the development of microbial eczema is observed against a background of streptococcal or staphylococcal infection, as well as skin lesions by fungi (*in the form of scratches, abrasions, trophic ulcers, mycoses (fungal lesions), purulent wounds, eczematous lesions of an allergic nature*)



# The clinical picture

- The clinical picture of microbial eczema begins with the appearance of pustules followed by edematous erythematous spots with papule

- vesicles weeping, and serous- purulent crusts

- The boundaries are clear at the edges of the fringe of the exfoliating epidermis
- Ostiofolliculitis, folliculitis, impetigo can appear in a circle
- The foci are asymmetric
- Rashes are located more often on the lower extremities
- In case of irrational treatment, microbids may appear
- They are polymorphic elements similar to the type of foci in true eczema localized far from the main focus and constitute to the development of a secondary allergic reaction





# Seborrheic eczema

- Seborrheic eczema is a chronic skin disease, based on a violation of the secretory function of the sebaceous glands, detected in the sebaceous glands-rich areas of the skin
- The average incidence of the disease among the population is 3-5%
- Genetic factors, hyperproduction of the sebaceous glands, pathological changes in the function of the sebaceous glands, bacteria found in the mouths of the hair follicles and sebaceous glands, stress, and allergic reactions play a significant role in its development



# The clinical

## picture



- A lesion occurs on the skin of the scalp and the skin of the face in the area of the eyebrows and nasolabial folds may be affected, flexion of the limbs, large body folds may be involved in the process of spreading
- Rashes occur in places characterized by a high content of sebaceous glands - the face scalp, chest, interscapular region, large folds
- Symptoms are most often represented by the presence of inflammatory reddened and slightly infiltrated foci with irregular outlines, with yellowish scales and crusts on a hyperemia background
- It is accompanied by itching

# Professional I eczema

- The etiological factors of professional eczema are occupational and domestic irritants
- The dynamics of the disease (*passes during the vacation*) is of great importance
- It is due to the usual contact with the allergen
- Its clinical picture is similar to true eczema



- Clinically, mainly erythematato-vesicular manifestations are found in the acute, subacute periods and chronic course

● acute



● subacute



● chronic





# The acute stage

- It begins with the appearance of red itchy spots, the formation of small vesicles with serous fluid on the pinhead
- After opening of vesicles, erosion is formed
- "Serous wells" of Meshchersky appear
- The formation of erosion contributes to extensive weeping erosive surfaces
- An isomorphic reaction is very typical (*the appearance of new elements of rash on apparently healthy skin when it is irritated*)



# The subacute stage

- The subacute stage is characterized by the presence of papulo-vesicular elements, drying of erosions, absence of edema, weeping and a pronounced inflammatory reaction



# The chronic stage

- The chronic stage of eczema is characterized by foci of congestive erythema with lichenification infiltration with the presence of scales and crusts on their surface
- Foci persist for a long time even with the disappearance of skin lesions



# Treatment of eczema

- Obligatory sanitation of foci of infection
- Drugs to normalize the functions of the gastrointestinal tract
- Diet restricted in easily digestible carbohydrates (*white bread, sweets*), extractive substances (*saturated broths*), citrus fruits, colored carbonated drinks, fatty, spicy, smoked dishes
- Antihistamines (*suprastin, tavegil, clarithin, diazolin, peritol, phencarol, cetrin*)
- Hyposensitizing agents (*calcium gluconate, sodium thiosulfate*)
- Sedative therapy (*preparations of valerian, bromine, peony, tranquilizers*)
- In severe swelling and exudation for 2-3 days, diuretics (*furosemide, hypothiazide*)
- In microbial eczema - antibacterial drugs; in mycotic eczema - antimycotics; in seborrheic eczema - drugs that regulate fat metabolism (*methionine, lipamide, essentielle, purified sulfur*)
- For common, severe, torpid forms - systemic corticosteroids (*prednisone 20-50 mg*)

# Eczema treatment

- Solutions of antiseptics for the treatment of damaged surfaces and *compresses* (2% *boric acid*, 0.25% *silver nitrate*), in microbial eczema – *uratsillin*, *rivanol*
- Ointments with drying effect (*zinc*, *Lassard paste*, *aniline dyes*)
- Creams and ointments with corticosteroids (*Elocom*, *Celestoderm*, *Advantan*)
- In chronic eczema, ointments with *naphthalan*, *ichthyol*, *tar*

# Other methods of treatment

- Electrosleep, acupuncture, hypnotherapy, bromotherapy, valeriana root tranquilizers, antihistamines
- Ultraviolet irradiation
- Electrophoresis
- Bath with pine needle extracts















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