Dermatology. Venereology
Part 3

Textbook for 4-year dentistry students
(English medium)

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

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Дерматологія. Венерологія
Частина 3

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для студентів IV курсу
стоматологічного факультету
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A textbook Dermatology. Venereology for stomatological faculty students of medical university. The issues of etiopathogenesis, clinic, diagnostic, treatment and prevention of infectious skin diseases are shown in textbook.

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У навчальному посібнику розглядаються питання етіопатогенезу, клініки, діагностики, лікування та профілактики інфекційних захворювань шкіри.

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PYODERMAS

Definition: Pyodermas are skin lesions characterized by the presence of pustules. They account for most skin diseases and take first place among all dermatoses. They are quite common among all population groups but are registered rather more often among persons engaged in certain branches of industry, namely, construction, metallurgical and mining industries, in trans-port, etc., where they have become occupational diseases. From this standpoint, pyoderma control is of state importance and is a social problem of medicine. Pyodermas are also the most prevalent of all dermatoses encoun-tered in paediatric dermatological practice. Diverse manifestations of pyoderma occur primarily (as various independent nosological forms) or as a complication of other dermatoses, especially in patients with pruritic dermatoses (neurodermatoses, scabies, pediculosis).

Epidemiology: Various species of staphylococcus and streptococcus are the most common causative agents of pyoderma. Tho disease "may also be caused by the blue-pus bacillus, Proteus vulgaris, Escherichia coli, fungi, pneumococcus, gonccoccus, and many other micro-organisms. The extensive spread of staphylococci and strep-tococci in nature (on the clothes, in the house, at places of work, in street dust, etc.), on the skin of sick persons, and on the mucous membranes and skin of individuals who have no noticeable patho-logical changes (20 to 75 per cent of persons examined prove to be bacilli-carriers), the possibility of the transformation of non-patho-genic forms to pathogenic forms on the skin surface under definite conditions – all this makes the extensive prevalence of pyoderma understandable.

It is an established fact that staphylococci are almost always present on the skin of healthy persons (usually in the orifices of the hair sacs and in the ducts of the sebaceous glands), but the iden tified strains are pathogenic in only 10 per cent of them, whereas • among patients with pyoderma and those with a history of tho dis-ease the percentage of pathogenic strains increases sharply (to 90). Streptococci are found much less frequently on the skin of healthy persons (in up to 6 to 10 per cent) and are mainly localized in the skin folds.

Pathogenesis. The development of any form of pyoderma is determined not only by the pathogenicity and virulence of the cocci strain (although these arc factors are important), but also by various exogenous predisposing factors that alter the protective functions of the skin and decrease, in particular, its ability to resist the development of pyoderma.

The conducive factors for the development of pyodermas ir chil-dren are an imperfect physiological barrier, particularly increased moistness, looseness, and fragility of the epidermal horny layer, labile colloido-osmotic state, and high absorption capacity of the skin.

All pyoderma are divided into staphylococcal, streptococcal and mixed in accordance with the etiotiological factor.

Classification of pyoderma: There is no generally recognized classification of pyoderma. The most common and convenient in practical terms is the classification by etiological principle. According to this classification, distinguish staphylococcal, streptococcal and mixed (strepto-staphylococcal) skin lesions. In addition, each group is given surface and deep pyoderma, which can occur acutely and chronically. To superficial pustular lesions of the skin are those nosological forms, in which the epidermis and the upper layer of the dermis are affected. With deep pyoderma, the lesion can seize not only the dermis, but also the hypodermis.

Staphylococcal pyoderma:

- superficial – ostiofolliculitis, superficial folliculitis, impetigo staphylococcal bullous (in children), staphylococcal pemphigoid of newborns;
- deep – deep folliculitis, boils, acute furuncle, carbuncle, hydradenitis, multiple abscesses of infants.

Staphylococcal pyoderma, which occur chronically:

- superficial – sycosis vulgar;
- deep – furunculosis chronic (localized and common), folliculitis decalving.

Streptococcal pyoderma, acute:

- superficial – impetigo streptococcal, intertrigo;
- deep – ecthyma streptococcal, erysipelas.

Streptococcal pyoderma, which occur chronically:

- deep – chronic diffuse streptoderma.

Strepto-staphylococcus pyoderma flowing sharply:

- superficial – impetigo vulgar;
- deep – ecthyma vulgar.

Strepto-staphylococcal pyodermas are deep, flowing chronically (chronic atypical pyoderma):

- ulcerative chronic pyoderma and its variety – shankriform pyoderma;
- ulcerative vegetative pyoderma;
- abscessed chronic pyoderma and its variety – inverse conglobata acne.
- Staphylodermia acute, chronic.

Acute staphyloderma: ostiofolliculitis, folliculitis, furuncle, acute localized furunculosis, carbuncle, hydraadenitis, epidemic (staphylococcal) pemphigus of newborns, multiple abscesses in infants.

Chronic staphyloderma: vulgar sycosis, chronic furunculosis.

Streptodermia acute: impetigo – intertriginous, annular, bullous; acute diffuse streptoderma.

Chronic diffuse streptoderma, vulgar ectima.

Vulgar impetigo (staphyloderma and streptoderma).

Depending on the etiology, there are streptococcal, staphylococcal and mixed, mainly staphylostreatococcal skin lesions; downstream they are divided into acute and (rarely) chronic; according to the depth of the lesion – to superficial (mainly streptococcal) and deep, mostly staphylococcal or mixed.

Eruptions on the skin with pyoderma are polymorphic. The type of primary elements of the rash depends on the nature of the pathogen and the depth of the skin lesion.

Staphylodermas

Staphylococci can be divided into two clinically relevant groups: Coagulase-positive staphylococci (*Staphylococcus aureus*) producing both invasive and toxin-mediated infections. Coagulase-negative staphylococci (*Staphylococcus epidermidis*), causing variety of hospital infections.

Ostial folliculitis

Definition: Ostial folliculitis, or staphylococcal impetigo (L. impetus assault) sets in with redness and some pain around-the orifice of a follicle or sebaceous gland. A semispherical or conic swelling forms soon with a pustule in the center; the top of the pustule is yellow because of the pus that accumulates under it. A few days later the contents of the pustule dry up and a crust forms, the surrounding inflammation subsides and the process terminates without a trace or only a light pigmentation remains.

Pathogenesis: Ostial folliculitis is marked by the formation of a bulla in the epithelium of the orifice of the hair follicle directly under the horny layer. The cavity of the bulla is filled with fibrin, polymorphonuclear leucocytes, and a few lymphocytes. The Giemsa or Gram's stain demonstrates a large number of staphylococci out-side the cells or in the neutrophils. An inflammatory infiltrate of neutrophils and lymphocytes surrounding the dilated capillaries is seen in the dermal papillary layer.

Therapy The causes conducive to the origin of ostial folliculitis are removed. Some of the pustules are opened and the pus removed, after which the foci of affection are painted twice a day with 1–2 per cent alcohol solution of aniline dyes (l % Sol. Gentianvioleti, seu Methyleni coerulei, seu Virides uitens) in 70 per cent ethyl alcohol or with an aqueous solution of potassium (dark-cherry coloured). The hair in the area of the lesions is cut, but not shaved, and for preventive purposes the surrounding skin is wiped with 2 per cent salicylic or boric acid or with a solution of camphor and alcohol (2.5 ml of camphor alcohol and 45 ml of 40 per cent rectified spirit).

Folliculitis

Definition: Hair follicle infection or irritation. The most common forms are caused by invasive staphylococci, but other bacteria, viruses, and fungi may also be responsible. Yet other forms (eosinophilic folliculitis in HIV/AIDS) are noninfectious. Mechanical irritation is also a factor, such prolonged sitting (*truck driver folliculitis*) or tight clothes (*blue jean folliculitis*); exposure to cutting oils is another factor.

Superficial Folliculitis. Synonyms: Bockhart impetigo.

Clinical features: Tiny pustules with erythematous border localized in superficial aspect (infundibulum) of follicle.

Localization: In children, usually scalp; in adults, trunk, buttocks, thighs, beard area.

Histopathology: The process begins with the formation of an infiltrate around the follicle. Neutrophils and lymphocytes are found in the infiltrate. Later, the follicle melts and dies and is replaced by connective tissue.

Therapy: The lesions are painted with Castellani's paint, 1–2 per cent alcohol solution of methylene blue or brilliant green. The healthy skin areas close to the pustules are wiped with 2 per cent salicylic or camphor spirit to prevent dissemination. Topical antiseptics or antibiotics (fusidic acid or erythromycin) can be used. If lack of response, systemic antibiotics (penicillinase-resistant penicillines or first-generation cephalosporin for 7–10 days). Baths and showers are forbidden for some time.

Furuncle

Definition: Furuncle is one of the common forms of pyoderma. It is acute staphylococcal inflammation of the hair follicle and the surrounding connective tissue.

Epidemiology: All age groups affected. The causative agent of furuncle is Staphylococcus aureus and, less frequently, Staphylococcus albus. Particular attention should be paid to occupational and household factors of this kind which may facilitate the development of furuncles in very many people. The important endogenic factors are emaciation of the organism, metabolic diseases (diabetes, obesity), gastro-intestinal diseases, anaemia, hypovitaminosis, diseases of the nervous and endocrine systems, alcoholism, regular overcooling or overheating, etc., which lead to a decrease in the body's general immunobiological reactivity. Furuncles occur more frequently in the spring and autumn. The incidence is lower among children than among adults and higher among males than among females.

Pathogenesis: A furuncle may form on previously healthy skin or may be a complication of an already existing superficial or deep staphyloderma. Besides the virulence and pathogenicity of the strain of the causative agent, predisposing exogenic and endogenic factors play an important role'in the development of furuncle and furunculosis. Among the exogenic factors are mechanical injury inflicted to the skin by particles of dust, coal or metal which create the site of entry for the infection.

Clinical features: A hard elevated, bright-red infiltrate first forms around the hair follicle. The infiltrate is not sharply circumscribed and is attended with a pricking sensation or mild pain. The infiltrate acquires gradually the form of a firm tumour which grows along the periphery and becomes more painful; there is swelling of the surrounding tissues (the swelling in the region of the cheeks, eyelids, and lips may be sharply pronounced). The second stage sets in on the third or fourth day: the furuncle grows to 1–3 cm in diameter and a necrotic core with a pustule on its surface forms in the center. The furuncle takes the shape of a conic tumour with smooth, lustry blue skin.The pain is very severe in this period, body temperature may rise to 37–38 °C, and symptoms of

toxicosis may develop (general indisposition, malaise, headache, etc.). The top of the pustule opens spontaneously or is opened artificially and pus, sometimes with an admixture of blood, is discharged from the furuncle after which a' yellowish-green necrotic 'plug' (necrotic core), comes out. After removal or rejection of the core, swelling, infiltration, and pain subside; and the remaining crater of the furuncle is filled with granulations which are replaced by a scar in two to three days. The scar is bluish-red at first, then gradually turns white and is sometimes hardly visible. The developmental cycle of a furuncle commonly lasts eight to ten days. In a subclinical course of the process, a painful infiltrate forms but there is no suppuration or necrosis. A small furuncle is distinguished from the lesion formed in folliculitis by the small central necrotic core. In weak patients emaciated by other diseases or in inadequate treatment, the furuncle may develop into an abscess (phlegmonous furuncle). Furuncles may form on any area of the skin, with the exception of the skin on the soles and palms devoid of hair follicles. Solitary furuncles occur most frequently on the back of the head, the forearm, small of the back, abdomen, buttocks, and lower limbs. A furuncle of the external acoustic meatus is marked by severe pain, while a furuncle of the upper lip is a dangerous disease because thrombosis of the lymphatics and veins with the formation of septic phlebitis of the cerebral vessels and general sepsis may occur. Acute inflammation of lymphatic vessels and lymphadenitis may develop when furuncles of the neck, chest, and thighs form close to the lymph nodes. Metastasis to the liver, kidneys, and other internal organs may occur. All these complications make furuncles a very grave disease in some cases. Complications may be promoted by attempts to squeeze out the furuncle, cuts from shaving, inadequate local treatment; localization of a furuncle on the face, in the nasolabial triangle, and on the skin and mucous membranes of the nose is also conducive to the development of complications.

Localization: Neck, face, axillae, groin, upper back.

Caution: There is a risk of sepsis in immunosuppressed patients.

Furunculosis is a condition in which there is multiple (though not always) and recurrent eruption of furuncles. Furunculosis may be localized (on a circumscribed skin area) or diffuse, disseminated. According to the course, furunculosis may be acute (lasting several weeks to one or two months and marked by the appearance of very many furuncles) or chronic (a small number of furuncles appearing at short intervals or uninterruptedly for months).

Differential diagnosis: The diagnosis of characteristic cases is easy. The condition has to be differentiated from anthrax, hydradenitis, granuloma. Anthrax sets in with the formation of a papulovesicular lesion which acquires a brownish-black scab, there are also marked infiltration of the dermis and hypoderm, sharp pain, and severe disturbance of the general condition. In hydradenitis, purulent inilamnia, inflammation of the apocrine glands (in the axillae, inguinal folds, nipples: usually here is no central necrotic core. The medical history (contact with animals), the absence of severe pain, no

pyonecrotic core, and the detection of fungi in the pathological material on microscopy are important in making the diagnosis. In some cases, furunculosis has to be differentiated from erythema.

Therapy: Treatment of furuncle depends to a great measure type and spread of the pathological process. In a case with a solitary furuncle and no complications, for instance, only external therapy is prescribed (particularly when the patient applies for medical advice early). In recurrent and complicated furuncles, in furuncles of hazardous localization, and in furunculosis, especially in the chronic and disseminated forms, external therapy is supplemented by general measures which act on the microbial flora, stimulate the defense reactive forces of the body, and contribute to the removal of intercurrent diseases revealed during examination of the patient.

Antibiotics are used extensively. Penicillin is given intramuscularly in a dose of 50 000–100 000 U every three or four hours to a total dose of 1 000 000–3 000 000 U in acute forms and 5 000 000–10 000 000 U and more in chronic forms. Out-patients are treated with bicillins (benzathine penicillin) which are long-acting penicillin preparation. The former is injected into the muscle once a day in a dose of 600 000 U, and the latter once in three or four days in a dose of 1 200 000–1 500 000 U (3 000 000 to 8 000 000 U are administered in the course of treatment).

Growing-resistance of the cocal flora, staphylococci in particular, to penicillin and its derivatives is now noted. In view of this, in the treatment of furunculosis more and more importance is attached to broad spectrum agents, which have an antimicrobial effect, namely macrolids – erythromycin and oleandomycin, and their combinations with tetracycline – oletetrin, signiamycin, and tetraolean.

Semisynthetic penicillins are now used more and more extensively in the management, of some pyodermas, chronic furunculosis in particular. These are methicillin (intramuscular injection of 1.0 g every four to six hours), oxacillin (taken in tablets or capsules of 0.25–0,5 g every-four; to six hours for five days or injected intramuscularly in a dose, of 0.25–0.5 g two or four times a day). Combination of antibiotics with oral antihistamine agents is advisable.

Sulphonarhides (sulphathiazole, sulphadimidine, sulphadimethoxine, sulphamethoxypyridazine) and other antimicrobial agents by a general action, are used. Nitrofuran derivatives: furazolidone, furazolin, furadonin (nitrofurantoin) and furagin have been lately prescribed in staphylodermas resistant to antibiotics and sulphonamides. They are given orally in tablets of 0.1 g two or four times a day after a meal for five-seven-ten days (the single dose of furazolin for infants under 12 months of age is 0.01–0.015 g, for children of 1 to 2 years of age 0.02 g, for children of 2 to 5 years of age 0.03–0.04 g and for children of 5 to 14 years of age 0.05 g given three or four times a day 15 to 20 minutes after, a meal).

Treatment of obesity, diabetes, intestinal atony, diseases of the internal organs, anaemia, etc. is a very important component in the complex management of patients suffering from chronic furunculosis. The diet of such

patients should contain food that is easily assimilated and no piquant and spicy dishes are given. Alcoholic beverages are not allowed. Vitamins A, C and the B complex as well as preparations of iron and phosphorus (phytoferrolactol, one tablet given three times a day for 15 to 20 days) are recommended.

The skin around the furuncle is disinfected with a solution of salicylic alcohol, camphor spirit or vodka. The hair is cut (but not shaved!) in the area of the furuncle and in the area immediately surrounding it (to prevent the development of folliculitis and new furuncles); this is done from the center to the periphery. The hair is then removed from the furuncle with sterile for ceps, pure ichthammol (possessing bactericidal, keratoplasty, local anaesthetic, and anti-inflammatory effects) is applied and covered with a thin layer of sterile cotton.

Such treatment of a solitary furuncle that has not opened sometimes prevents the further development of the pathological process. After the furuncle is opened, a dressing with a hypertonic saline solution may be applied and the periphery of the ulcer painted with pure ichthammol. A mercury plaster is sometimes applied to the furuncle; after the furuncle opens the ulcer is treated with ointment dressings: 5 per cent camphorichthammol, Vishnevsky's (3 parts tar, 3 parts xeroph-rm, 94 parts castor oil), 2 per cent ammoniated mercury, 10 per cent ichthammol, 1–2 per cent yellow mercuric oxide, 5 per cent chlortetracycline or erythromycin, dibiomycin ointment. Dry heat (heater, sollux, Minin's reflector) or exposure to the effect of UHF electromagnetic field is advisable. Moist heat (wet compress) and water procedures are not allowed during the disease. Surgery is recommended when the furuncle develops into an abscess, as well as intensive antibiotic therapy combined with immunotherapy (hyperimmune gamma globulin, hyperimmune antistaphylococcal plasma, staphylococcus toxoid).

Carbuncle

A carbuncle is diffuse pyonecrotic inflammation of the deep layers of the dermis and hypoderm with involvement of several neighbouring hair follicles into the process. Unlike a furuncle, the pyonecrotic infiltrate in a carbuncle spreads over a larger area and penetrates into the deeper layers of the dermis and hypoderm. The lesion is called a carbuncle (L. carbo charcoal) because the large necrotic areas formed during the pyonecrotic inflammation are dark and resemble charcoal.

The back of the head, the back, and the loins are the favoured localization.

The causative agent is Staphylococcus aureus and less frequently other staphylococcal species.

Prognosis: Deep necrosis of the-lower-parts of the dermis and hypoderm is revealed. The necrosis spreads gradually to the periphery. These foci are seen in a thick infiltrate of neutrophils. The prognosis depends on the patient's general condition.

Clinical picture and course: A few individual hard nodules are found in the skin at first, which coalesce into a single infiltrate. Carbuncles usually occur as solitary lesions. Their development is'attended with high fever, excruciating

pain of a tearing, pulling character, a chill, and indisposition. A carbuncle may take a malignant course in old age, in emaciated patients suffering from severe diabetes, and in neuro-psychic overstrain. This infiltrate grows, sometimes to the size of a child's palm. *1st stage:* a few individual hard nodules are found in the skin at first they coalesce into a single infiltrate which grows sometimes to the size of a child palm. It surface becomes semispherical, the skin is tense and cyanotic and in its center there is a localized tenderness (it takes about 8–12 days). After a few pustules are formed in the area of the infiltration, the top of them is opened and they appears as sieve. Pus and green necrotic masses with an admixture of blood are discharged from these openings, larger areas in the center of the carbuncle undergoes necrosis. Ulcer is formed and sometimes it may reach the muscles. *2nd stage (stage of suppuration and necrosis*): last for 14 to 28 days after that the ulcer in filled with granulating tissues and a deep scar is formed and it fuses with the underlying tissues. After surgical removal of the carbuncle a large scar is formed.

Differential diagnosis: diagnostic and differential diagnosis of carbuncles always includes general measures and does not differ in principle from the treatment of furuncles.

Treatment: The treatment of carbuncles always includes general measures and does not differ in principle from the treatment of furuncles. Antibiotics are given together with sulphonamides in severe cases. Radiotherapy produces a favourable effect at the onset of the disease. In rapid development of the carbuncle, a wide and deep cross-like incision is indicated with excision of the necrotic areas; this is carried out by a surgeon as a rule; antibiotic therapy is applied at the same time (500 000 U of streptomycin is often injected twice a day simultaneously with injections of penicillin in a daily dose of 1 000 000 U or injections of its analogues). The skin around the carbuncle is disinfected with 2 per cent camphor spirit or salicylic acid twice a day without fail and all scratches and excoriations are painted with Castollani's paint or alcohol solution of iodine.

Hydradenitis

Hidradenitis (Gk. hidros sweat, aden gland) is purulent inflammation of the apocrine sweat glands in the axillae (usually unilateral) or inguinal area frequently around the nipples and in the region of the large pudendal lips, scrotum, and anus.

Epidemiology: The most common causative agent is Staphylococcus aureus, which enters the efferent duct of the apocrine gland through the orifice of the hair follicle.

Pathogenesis: Geneval weakening of the organism, increased sweating and sweat of alkaline reaction in the axillae, inguinal folds and anus (especially in individuals with faulty hygienic habits), macerations, microtraumas, cuts during shaving, scratches on the skin consequent upon pruritic dermatoses in individuals with nervous and endocrine (diabetes, gonadal dysfunction) disorders, and diminished local resistance are predisposing factors. The sweat

apocrine glands develop only in the period of puberty (earlier in girls than in boys). There are more of them in females than in males. By old age the activity of these glands is extinguished and hidradenitis therefore does not develop in the old. The disease is encountered more often among females than among males.

Clinical picture and course: At the onset of the disease, solitary small hard mound-like nodes are palpated in the thickness of the dermis or hypoderm. The patient experiences mild itching or pain at this time. The nodes grow rapidly in size, adhere to the skin, become pear-shaped and protrude like nipples. The isolated nodes coalesce, soften, and fluctuation appears after which they open spontaneously. Hard disk like infiltrate resembling a phlegmon forms sometimes, in which case pain is felt not only during movements but at rest too, and disables the patient. Maturation of the lesion is attended as a rule with indisposition, moderately elevated temperature, and marked painfulness. After the nodes open, the sensation of stretching and pain subside and the ulcers heal in a few days (resolution of the infiltrate takes longer). Recurrences are frequent, however, and lend the process a protracted course. Axillary hidradenitis is usually unilateral, though bilateral lesions are also encountered. The average duration of hidradenitis is 10 to 15 days, but a protracted recurrent course is observed quite often (particularly in obese individuals, in patients with hyperhidrosis, diabetes, and in persons who pay little attention to skin.

Diagnosis: The diagnosis is made easily from the peculiar localization of the process and the typical clinical picture. The absence of a necrotic core distinguishes hidradenitis from furuncles. Tuberculosis colliquative is characterized by a more protracted course, involvement of the lymph nodes at the very onset of the process, no pain, the development of extensive ulcerative surfaces and many fistules, and healing with the formation of bridge-like scars.

Treatment: To prevent the further development of the lesions in the early stages, it is recommended to apply ultrasonics, UHF current, ultraviolet irradiation, pure ichthammol ('cakes'). Surgery is resorted to when agminated abscesses form. Injection of 0.5–1.0 procaine hydrochloride solution (8–10 ml) with penicillin (300 000–500 000 U) around the lesions is advisable in marked infiltration and pain. Such blockades are made every other day, treatment consisting of four or five procedures. Vaccine therapy is a rational measure in persistent and recurrent hidradenitis. In other respects hidradenitis is treated along the same principles as furuncles. Prevention consists in proper hygienic habits (frequent washing of the body with soap and sponge) and disinfection of the axillae with salicylic alcohol or borocamphor spirit.

Sycosis:

Definition: Staphylogenic sycosis (vulgaris) is a chronic recurrent pyoderma encountered predominantly among males. Areas of ostial folliculitis and folliculitis form usually on the scalp, in the region of the moustache and beard, and less frequently on the inner surface of the wings of the nose, on the eyebrows, in the axillae, on the eyelid margins, and on the pubis.

Pathogenesis: Despite the fact that the aetiology of non-parasitic sycosis is known since long ago (staphylococcal flora), the pathogenesis, the mechanism of a persistent, often recurring form in particular, is still not clear. A. I. Pospelov believed that the persistent character of sycosis was caused by disturbed innervation of the sebaceous hair apparatus as a result of which-the composition of the secretions changed and acquired properties favourable for the multiplication and development of pyogenic cocci. Importance is attached today to diminished immunobiological resistance of the body and to the presence in the hair follicles of other foci of localized infection, besides the staphylococcal infection, which sensitize the body (the state of infectious allergy). Moreover, endocrine disorders are also held responsible to a great measure. Among the exogenic factors conducive to the development of sycosis are cutting of the skin in shaving, damage inflicted to it by coal and metal dust, and maceration of the skin on the upper lip in persistent rhinitis.

Clinical features: This condition is characterized by small superficial follicular pustules, some of them ruptures to discharge beads of pus and the rest is dried up to form a crust. Folliculitis develops rapidly involving more follicles, and the infection becomes chronic and the skin looks congested, swollen, and infiltrated. Usually pain is absent, itching and burning sensations are the only symptoms.

Sycosis vulgaris is usually a persistent condition (remaining-for years) which exacerbates now and again and has a depressing effect on the patient's mental condition, especially if it is localized on the face. In some cases there are no subjective disorders, in others the lesions are attended with a .sensation of burning mild itching or pricking.

Histology: A pustule filled with neutrophils, similar to the pustule in ostial folliculitis, forms in the ostial epithelium of the hair follicle. The infiltration around the follicles penetrates the entire upper part of the dermis and consists mainly of lymphocytes, plasma cells, and histiocytes, a small number of polymorphonuclear leucocytes, and occasional giant cells. Sometimes the sebaceous glands are destroyed.

Differential diagnosis: Differential diagnosis is made with infiltrative-suppurative trichophytosis (parasitic sycosis), which is characterized by a more acute, formation of thick and deeper-seated nodules, a tendency to disappear without any treatment; laboratory examination reveals elements of a fungus (of the ectothrix group) in the hairs on the periphery of the foci. If eczematization of sycosis occurs (extension of hyperaemia beyond the, boundaries of the main focus and the appearance of exudative papules), the clinical picture may resemble that of eczema complicated by pyoderma.

Therapy: The management of sycosis usually takes a very long time and calls for patience on the part of both the physician. All identified exogenic irritating factors should be removed (treatment of focal infection, improvement of sanitary and hygienic conditions of work at enterprises with the removal .of

the causes of injury to and soiling of the skin on the face, etc.). A general effect must be exerted on the patient's organism when deviations in its activity are revealed; this may be accomplished (by prescribing hydrotherapy, sedatives, treatment :at health resorts, a change in the surroundings (in functional disorders of the nervous system), autohaemotherapy, autovaccines (as non-specific and specific-stimulation therapy), medication with preparations of iron, arsenic, sex hormones (e.g. methyl testosterone in diminished sexual function), and vitamin B_{12} (in anaemia). Broad spectrum antibiotics (gentamicin, erythromycin) are prescribed. External therapy includes disinfectant lotions (applied during exacerbation), e.g. 1 : 1000 ethoxydiaminoacridine lactate solution, 1 : 3000 potassium permanganate solution, 2 per cent boric acid solution. In the period of abatement daily painting with 2 per cent solutions of aniline dyes (methylene blue, gentian violet, brilliant green, etc.) is advisable or the prescription of ointment containing boric acid and tar. Ultraviolet irradiation (erythema doses) is prescribed in marked infiltration in the foci of affection.

Pemphigus epidermicus neonatarum

Definition: The disease arises frequently on the first days of life usually on the 7^{th} to 10^{th} days after birth. Small bullae, of a pea or cherry size formed on the previously clear or slightly erythematic skin within a few hours.

Clinical features: The bulla has thin tense top and clear serous yellowish contents. After their development the child is restless, with fever, then the content of bulla turns to a cloudy purulent in character. Then the bulla grows in size and spread all over the body, their top ruptures and a bright red, moist itching erosive surfaces are exposed with the reminisce of epidermis or on the periphery. The discharge from the erosions changes to form a serous-purulent crust. The favorable locations are naval, abdomen, chest, back, buttocks, and limbs.

Ritter's disease (Staphylococcal Scalded Skin Syndrome)

Definition: Widespread superficial skin loss caused by exfoliation.

Clinical features: Most patients are newborns or small infants. Rapid onset (sometimes with prodrome) of diffuse erythema and fever. After 12 hours, Nikolski phenomenon positive – stratum corneum can be pushed over underlying layers. Problems with temperature and fluid control because of widespread skin loss. The disease is like pemfigus epidermicus neonatarum, it develop in the first week of the infant life. A bright edematous inflammatory erythema appears first in the mouth and then descends rapidly to the folds of the neck to the naval, gentiles, and anus. A large spherical dense bulla formed against background which soon burst and causes weeping eroded surfaces. The wildest injury the swollen and lose epidermis is detached in same places of the epidermis around the erosion are pulled with forceps they separate from the underlying layer far beyond the healthy skin (positive Nikolsky sign).

Diagnostic approach: The organism usually cannot be cultured from the skin, but often from pharynx or other sites. Biopsy with frozen section.

Differential diagnosis: In SSSS, the skin biopsy shows a very superficial epidermal split, whereas in toxic epidermal necrolysis, there is full-thickness epidermal necrosis.

Therapy: Topical antiseptics or fusidic acid. Place on bed covered with nonadherent sheeting. Attention to fluid replacement, electrolytes, temperature control. Systemic antibiotics (penicillinase-resistant penicillins or first-generation cephalosporins; as soon as possible, culture and sensitivity-directed choice of agents). Search for staphylococcal carrier among parents or especially nursing personnel in case of nursery epidemics. Systemic corticosteroids are not effective in SSSS and should be avoided.

Acne

Etiology: Acne, an inflammatory disorder of the sebaceous glands, is exceedingly common among teen-agers and frequently continues into adulthood. The fact that innumerable remedies are advertised to the medical profession and the public indicates that none is satisfactory. Certain drugs, notably the iodides and bromides, but also systemic corticosteroids, can produce acneiform eruptions. Acneiform eruptions may be caused or aggravated by externally applied fluorinated hydrocarbons, cutting oils, pomades, and other greasy materials.

Clinical features: Acne manifests most commonly in areas of the body that have larger, more numerous sebaceous glands, such as the face, back, chest, and shoulders, upper arms. Acne lesions can be separated into inflammatory and noninflammatory lesions. Inflammatory lesions can appear as pink papules, pustules, or cysts (see Inflammatory lesions). Noninflammatory lesions are closed or open comedones and contain a thick, white material mostly composed of keratin (see comedonal lesions). Most patients with acne present with a combination of different acne lesions at varying states of formation (see comedonal and inflammatory lesions). Inflammatory papules, pustules, and cysts often resolve with postinflammatory hyperpigmentation (areas of discoloration) that can last for several weeks to months. Cysts and nodules can result in long-term scarring.

Treatment: The therapy of noninflammatory lesions differs from that of inflammatory lesions. Comedones (blackheads) and closed comedones (whiteheads) are noninflammatory lesions. They usually cause little distress. These lesions can be removed mechanically with comedone extractors and a fine needle or pointed blade to open closed comedones. Such maneuvers are temporary; new comedones form promptly. Many dermatologists believe without proof that mechanically removing comedones prevents the formation of inflammatory lesions. Topical antibiotics combine proven effectiveness with cosmetic elegance. The currently available preparations are tetracycline (Topicycline), erythromycin, clindamycin, and meclocycline subsalicylate. Topical tetracycline, erythromycin lotions, and topical clindamycin have hydroalcoholic vehicles that most patients tolerate well. For patients with dry or irritable skin, I favor topical meclocycline cream or the erythromycin ointment. Of the nonantibiotic antimicrobials, benzoyl peroxide deserves mention. It's a potent antimicrobial

available in a variety of creams, gels, and liquids. Sulfur, resorcinol, and salicylic acid are the classical time-tested topical agents.

Streptodermas

There are many schemes for classifying streptococci. *Streptococcus* pyogenes (group A, β-hemolytic streptococci) account for 90 % of infections. *Streptococcus viridans* (α-hemolytic streptococci) and *Streptococcus pneumoniae* are other important members of the group.

Impetigo

Definition: Superficial skin infection.

Epidemiology: Most patients are children. Infections usually in late summer and fall; more common under poor hygienic conditions.

Pathogenesis: In Europe most impetigo is caused by group A streptococci (*Streptococcus pyogenes*), as well as by mixed infections with *Staphylococcus aureus*. It is impossible to distinguish between staphylococcal and streptococcal impetigo on clinical examination. Furthermore, many infections are mixed.

Clinical features: Crusts that develop from tiny blisters and superficial pustules. Usually on face or hands.

Complications: Glomerulonephritis is very common; rheumatic fever almost unheard of.

*Diagnostic approach***:** Culture usually reveals mixed infection. Antistreptolysin (ASL) and antistreptodornase-B (ADB) titers elevated. Check urine status at start of therapy and after 6 weeks.

Therapy: Topical therapy with disinfectants or fusidic acid ointment is satisfactory for mild cases. Crusts should be removed with disinfectant soaps. Systemic antibiotics, usually penicillin, may speed healing and will reduce spread to contacts. Avoid contact with other children, as well as shared wash clothes and towels.

Bullous Impetigo

Epidemiology: In Germany most impetigo is caused by streptococci; in the USA, most caused by staphylococci. Bullous lesions suggest staphylococcal origin, but lack of blister is not diagnostically helpful.

Pathogenesis: Staphylococci in phage group II produce a toxin, exfoliatin, coded by the phage virus, which is capable of splitting the epidermis in the stratum granulosum (acting on desmoglein 3). This action produces large superficial blisters or more diffuse superficial skin loss.

Clinical features: Most patients are neonates (neonatal pustulosis) infants, or small children. Suddenly appearance of small blisters that rapidly enlarge; little associated erythema. Soon form yellow crusts.

Diagnostic approach: Bacterial culture; see if siblings have similar lesions.

Therapy: Topical antiseptics or fusidic acid. Systemic antibiotics (penicillinase-resistant penicillins or first-generation cephalosporins) may slightly speed course of healing.

Prognosis: Rapid healing with therapy; less than 5% mortality.

Ecthyma

Definition: Ulcerative infection usually caused by group A streptococci.

Epidemiology: Patients often show immunosuppression, inadequate nutrition, poor hygiene (homeless, drug abusers). Also common in tourists following visits to the tropics.

Clinical features: Punched-out ulcers, usually on legs, presumably at sites of minor trauma. Typically 0.5–3.0 cm with peripheral erythema. Healing is slow and with scarring.

Diagnostic approach: Culture and sensitivity.

Therapy: Address predisposing factors; compression therapy may be needed. Topical disinfectants or fusidic acid ointment; in difficult cases, mupirocin ointment. Culture-directed systemic antibiotics.

Erysipelas

Definition: Acute superficial cellulitis involving dermal lymphatics; caused by group A streptococci.

Pathogenesis: There is usually a portal for entry. On the face, it is often herpes simplex; on the legs, interdigital tinea with maceration. The streptococci come from nasal or perineal carriage, or from respiratory tract infections.

Clinical features: Bright red, sharply demarcated, rapidly spreading erythematous patch. On the face, usually symmetrical involving the cheeks. On the legs, unilateral with associated swelling. Fever, chills, malaise.

Complications: Recurrent infections lead to lymphatic damage and then lymphedema. Facial: swollen lip or lid edema; leg: *elephantiasis nostra*. Glomerulonephritis. In immunosuppressed patients, there is a risk of sepsis, necrotizing fasciitis, or shock if treatment is not prompt.

Diagnostic approach: Lesion very difficult to culture; can attempt aspirates from edge. Elevated white blood cell count, sed rate and C-reactive protein; ASL and ADB titers raised.

Therapy: High-dose penicillin i.v.; raise limb; cool compresses. Later attempt to address portal of entry; consider compression, prophylactic antibiotics.

Therapy of Streptococcal infections. Principles: Culture and sensitivity for serious manifestations. Antibiotic of choice is penicillin G or amoxicilline; in case of penicillin allergy, rely on local sensitivity guidelines. If mixed infection with staphylococci is suspected, then penicillinase-resistant penicillin, perhaps combined with ampicillin. Duration of therapy is for at least 10 days. *Mild infections (impetigo, scarlet fever, mild erysipelas):* procaine penicillin (penicillin G) 600,000 IU i.m. 1–2 times daily. Penicillin G 250 mg p. o. 4–6 times daily. If mixed staphylococcal infection is suspected, dicloxacillin 500–1000mg p. o. in 8 h. *Penicillin allergy:* erythromycin 500 mg p. o. q. i. d. or clindamycin 150–300 mg p. o. t.i.d.

Viral dermatoses

Dermatoses of virus etiology form a rather large and frequently encountered group of skin diseases. It includes herpes, warts and condyloma acuminatum, molluscum contagiosum.

Herpes Simplex Virus Infections

Definition: Diseases of skin and mucous membranes caused by infections with herpes simplex virus type 1 (HSV-1) or type 2 (HSV-2).

Etiology: Herpes simplex infections are caused by the herpes hominis virus, which has two distinct strains, HSV 1 and HSV 2. Genital herpes is usually caused by type 2 virus, while the type 1 strain is responsible for most facial infections.

Pathogenesis: Initial infection: HSV enters via small defects in skin or mucosa and starts to replicate locally; then spreads via axons to sensory ganglia where further replication occurs; through centrifugal spread via other nerves, affects wider areas. After resolution of the primary infection, the virus remains latent in the sensory ganglia. Recurrent infection: reactivation of virus by various stimuli (UV light, trauma, emotional stress, menstruation) as well as local or systemic immunosuppression leads to seeding of the virus into area served by the sensory ganglia and thus to local recurrences. The trigeminal ganglia are most commonly involved in orofacial HSV infections, while, the sacral nerve root ganglia (S2-S5) are involved in genital HSV infection.

Epidemiology: Almost everyone suffers from HSV-1 infection; the first infection is silent in 90 %, non-specific in 9 %, and clinically manifest in only 1 %. Type 1 primary herpes simplex infection usually occurs in children as an inflammation of the oral mucosa and gums, it sometimes occurs in adults as well. HSV-2 appears after start of sexual activity and affects 25–50 % of population. Primary genital herpes is mostly a disease seen in adults, since it's usually acquired through sexual intercourse. Following the primary infection, the virus establishes residence in a nerve root ganglion and may periodically travel down the nerve to the skin to produce the recurrent disease. Both viruses can be shed when patient is asymptomatic, easing transmission.

Clinical features: Common findings: Incubation period 6–8 days. Both HSV types can cause oral and genital infections; their clinical presentations are identical. In the genital area, the recurrence rate for HSV-2 infections is 10× greater than for HSV-1, while with orofacial infections, HSV-1 has a significantly higher recurrence rate.

Orofacial HSV infections:

Initial infection occurs as a *herpetic gingivostomatitis* usually in infants and children 5–6 years with extensive vesicular lesions, erosions and hemorrhagic crusts on lips and oral mucosa; difficulty feeding, foul smelling breath, systemic signs and symptoms: fever, anorexia and listlessness. Adults may also

develop acute gingivostomatitis, but it is less severe and is associated more often with a posterior *pharyngitis and tonsillitis* with vesicles, ulcerative lesions with grayish exudates on the tonsils and the posterior pharynx. Tender regional lymphadenopathy, perioral skin involvement due to contamination with infected saliva. The incubation period is 3–6 days, acute herpetic gingivostomatitis lasts 5–7 days, and the symptoms subside in 2 weeks. Viral shedding from the saliva may continue for 3 weeks or more.

Recurrences: small grouped blisters on erythematous base, rapidly become pustules and then eroded; often painful with dysesthesias and neuralgias. Common sites: lips (herpes labialis), chin, cheeks (herpes facialis), nose (herpes nasalis), periorbital region.

Viral shedding is in first 24 hours (maximum) of the acute illness and may last 5 days.

The following clinical forms are distinguished: 1) mild (abortive) variety with rapid resolution of the few lesions that had erupted; 2) edematous form accompanied with bright hyperemia and marked swelling; 3) severe form – herpes simplex ulcerosa; 4) zosteriform –herpes simplex zosteriformis; 5) frequently recurring form localized on the lips (vermilion border), buttocks, external genitals.

Eczema herpeticatum (Kaposi's eczema): patients with atopic dermatitis can develop extensive orofacial HSV infections which disseminate, especially favoring areas of active dermatitis. Neck is most common site. The eruptions occur suddenly with severe toxicosis, sharp elevation of body temperature (up to 39–40 ºC), confused consciousness and enlargement of the liver and lymph nodes. Pneumonia, meningeal phenomena and encephalitis, otitis, keratoconjunctivitis sometimes with corneal ulceration, and gastrointestinal disorders may develop.

Periungual HSV infection: Herpetic whitlow most often affects doctors, dentists, and health personal; sharp reduction since more extensive use of gloves because of HIV. Periungual erythema, pain, and then vesicles, regional lymphadenopathy. Differential diagnosis includes candidiasis and pyoderma. No incision and drainage.

Genital HSV infections: In 80 % of patients: HSV-2. *Initial infection:* Disseminated, rapidly eroded vesicles leading to small painful superficial ulcers, pain, itching, dysuria, vaginal and urethral discharge, and tender lymphadenopathy. Cervix involved in 80 % of women. Systemic signs and symptoms: malaise, fever, headache, myalgia. Incubation period of primary genital herpes is 3–7 days, healing after 2–3weeks. *Recurrences:* prodromal tenderness, pain, and burning at the site of eruption that may last from 2 hours to 2 days, grouped blisters or pustules on erythematous base, erosions and crust, in some patients sacral neuralgia occurs. Differential diagnosis includes all genital ulcers. *Uncommon sites:* buttocks or upper thigh; anal or rectal involvement more painful with paresthesias, retention of urine or stool, impotence.

Herpes gladiatorum: Wrestling or other close contact sports (rugby: scrum pox) are ideal for transfer of HSV between team members, usually HSV-1 spread when beard is rubbed on upper trunk, face, arms or neck of opponent. Widespread lesions in areas of body contact.

Neonatal HSV infections: HSV-2 (and increasingly HSV-1) in birth canal with direct transfer to newborn and potential for HSV sepsis. Genital HSV recurrences in women are asymptomatic in 70 % of cases, making diagnosis most difficult. Course of HSV in newborns tends to be severe because of incomplete immune response. Sepsis, encephalitis; 30 % have no skin findings. If mother has genital herpes, cesarean section and antiviral therapy for newborn.

Postherpetic erythema multiforme: Over 95 % of patients with recurrent erythema multiforme have recurrent HSV as trigger. This hypersensitivity reaction, severe enough to overshadow may be the herpetic infection completely and responds dramatically to systemic corticosteroids.

Diagnostic approach: Clinical findings usually so typical that laboratory investigations not needed. *Most rapid approach:* Tzanck smear searching for multinucleated giant cells. *Other possibilities:* Identification of virus: immunofluorescent staining of smear with monoclonal antibodies, PCR, electron microscopy, culture. Serology (ELISA): most useful for epidemiological studies.

Differential diagnosis: Deciding between HSV and early zoster can be difficult, but zoster should be unilateral and not recurrent. HSV also develops more rapidly following immunosuppression than does VZV. The yellow crusting stage of herpes simplex is sometimes mistaken for bacterial infection, bacterial superinfection of herpes appears to be rare.

Therapy: Systemic treatment with acyclovir, valaciclovir, or famciclovir. The adult dose acyclovir for treating either primary or recurrent herpes simplex is one 200 mg capsule every three-four hours while awake, for a total five capsules in each 24-hour period. For primary herpes simplex a 10-day course is recommended, for recurring – 5-day course. It is critical to initiate antiviral therapy early. Prophylaxis for recurrences: If patient has more than six recurrences yearly, consider acyclovir 400 mg p. o. b.i.d. or valaciclovir 1000 mg p. o. daily. Use for1 year; then vacation to check for improvement. Same regimen can be employed for recurrent erythema multiforme. Drying measures: Zinc oxide lotion, calamine lotion. HSV vaccines was effective in preventing HSV-1 genital disease and infection. Neonatal HSV: Specific hyperimmune globulin and i.v. acyclovir.

Herpes Zoster (shingles, zoster)

Definition: Segmental (dermatomal) acute painful skin disease caused by reactivation of varicella-zoster virus (VZV), that is the cause of varicella (chickenpox).

Epidemiology: 10–20 % of seropositive adults develop clinically apparent zoster. Peak age 50–70. Risk factors are: HIV and iatrogenic immunosuppression, primary VZV infection in utero or in early infancy (low immune response), anti-TNF therapy, leukemia or other malignancies.

Pathogenesis: Following the initial varicella infection, VZV persists life-long in the sensory ganglia of the spinal cord and cranial nerves. When reactivated, it follows the associated nerves into the skin; thus both the peripheral nerve and the skin of its dermatome involved.

Clinical features: Preeruptive phase (preherpetic neuralgia)*:* dysesthesias or pain in distribution of the affected nerve without visible skin changes; may last up to 7 days, typically burning or lancinating pain, may be also accompanied by malaise, myalgia, headache, photophobia, and, uncommonly, fever. The neuralgia of shingles may precede the skin eruption by a week or more. Acute eruptive phase: eruption of grouped vesicles and then pustules on an erythematous base appear unilaterally and stopping abruptly at the midline of the limit of involved dermatome; clear vesicles become cloudy and purulent, occasionally the rash is hemorrhagic or necrotic; also lasts about 7 days then covered by crust and involute. Always respects the midline, and only few lesions are outside the involved dermatome and its two immediate neighbors. The eruptions are characteristically asymmetric and unilateral. More widespread disease suggests immunosuppression. The following clinical varieties are distinguished: 1) generalized herpes zoster (herpes zoster generalisatus, disseminates marked by bilateral and disseminated lesions; 2) herpes zoster haemorrhagicus, in which the clear contents of the vesicles turn purulent and then, when the process penetrates deeper into the dermis, becomes hemorrhagic; 3) herpes zoster gangrenous, a severe form, in which the floor of the vesicles undergoes necrosis and scars form in their place; 4) mild (abortive) form; 5) bullous form characterized by the appearance of both vesicles and bullae.

The disease is accompanied by regional lymphadenopathy, severe pain; scarring is possible. Post herpetic neuralgia (chronic phase) – persistent pain or hyperesthesia lasting >30 days after acute infection or crusted lesions.

Complications: Ocular involvement: When 1st branch of trigeminal nerve (opthalmic nerve) is involved, 50 % have ocular involvement, including keratitis, scleritis, episcleritis, corneal erosions, conjunctivitis, iridocyclitis, retinitis, choroiditis, secondary glaucoma, optic neuritis, optic atrophy, impairment of muscles (double vision), facial paralysis. Vesicles on the tip of the nose (Hutchinson sign) indicate nasociliary nerve involvement and greater likelihood of eye involvement. Always consult the ophthalmologist.

Otic involvement: Involvement of inner ear when 8th cranial nerve is affected, leading to reduced hearing, vertigo, and zoster lesions of tympanic membrane and outer ear canal. *Ramsay Hunt syndrome*: involvement of both 7th and 8th cranial nerves, leading to facial paralysis, hearing loss, vertigo, and zoster lesions of tympanic membrane and outer ear canal. Affected sacral ganglia can lead to retention of urine or stool. *Generalized zoster*: Usually in immuno-suppressed patients, resembles varicella but starts in a dermatome before disseminating. The clinical picture is more synchronous and uniform than in varicella. *Uncommon variants:* Zoster pneumonitis, encephalitis, nephritis, cystitis,

and cholecystitis. The disease may also be complicated by meningitis (herpes zoster meningitidem) and encephalitis. *Zoster in pregnancy:* No serious problems.

Diagnostic approach: Clinical features, Tzanck smear, if questions exist, then direct fluorescent antibody (DFA) testing or polymerase chain reaction (PCR) of vesicular fluid or a corneal lesion. With trigeminal nerve involvement or any signs and symptoms of eye involvement, always get ophthalmologic consultation.

Differential diagnosis: HSV infection; in such a case, the immuno-fluorescent examination of a smear can readily separate VZV from HSV.

Therapy: Acute zoster: Drying measures (zinc oxide lotion, calamine lotion) or wet dressing with 5 % aluminum acetate (Burrow solution) applied for 30–60 minutes 4–6 times daily. Antiviral therapy ideally should be started within 48 hours of presence of vesicles during 7 days (Acyclovir 4 g/d, Valacyclovir 3 g/d, Famcyclovir 1,5 g/d). Analgesia with non-steroidal anti-inflammatory drugs (ibuprofen 600–2 400 mg daily or acetaminophen 1.5–4 g daily). Postherpetic neuralgia: Pain therapy is associated with a wide variety of side effects. Inexperienced physicians should work closely with a special pain clinic or physician.

Prevention: live attenuated VZV vaccine has been effective in preventing from varicella zoster infection. Administration of varicella-zoster immune globulin is recommended for immunocompromised patients. The patients with herpes zoster should be considered as contagious as if they had varicella. They are potentially contagious to infants and small children who have never had varicella, as well as to any adult whose immune system has been altered by illness and /or drugs. In particular, patients must scrupulously avoid anyone with malignancies or long-term corticosteroids or immunosuppressant's (kidney transplant, systemic LE, and like).

Warts

The primary clinical manifestations of HPV infection include common warts, genital warts, flat warts and deep palmoplantar warts.

Definition: Wart is benign proliferation of the skin and mucosa caused by the human papillomavirus (HPV).

Etiology: More than 100 types of HPV have been identified currently. HPV is a double-stranded DNA virus. Some types of HPV (6, 11, 16, 18, 31, 35, 39, 40, 43, 45, 51–56, 58) are associated with malignant transformation.

Pathophysiology: HPV infects the epithelium, viral replication occurs in differentiated epithelial cells in the upper level of epidermis.

Epidemiology: The warts of any HPV type may infect skin and mucosa at any anatomic site. Warts are transmitted by direct or indirect contact. Predisposing factors include disruption of the normal epithelial barrier. Warts affect 7–12 % of population with peak at 12–16 years school-aged children till 10–20 %. Genital warts prevalence is greatest in 17–33 aged persons, with peak

incidence 20–24 y.o. Risk factors of transmission of warts: trauma or maceration of skin, wet work involving of hands, hyperhidrosis of feet, swimming pools, nail biting, butchers and slaughterhouse workers. Smoking, oral contraceptives, multiple sex partners, and early coital age are risk factors for genital warts. HP virus is spread by direct contact as well as indirect. Autoinoculation is possible, which cause local spread of rash. Incubation period is 1–6 month, however latent period up to 3 or more years is possible.

Prognosis: 65 % of warts disappear spontaneously within 2 years without scarring. Recurrence rates of genital warts exceed 50 % after 1 year.

Clinical picture: **common warts (verruca vulgaris)** – hyperkeratotic papules with rough surface 0.1–1 cm in diameter on any part of body, most commonly on the hands and knees. **Filiform warts** – long thin growths around the lips, eyelids, nares. **Deep palmoplantar warts (myrmecia)** – begin as small papules and progress to deep, painful, sharply defined round lesion with rough surface, surrounded by a smooth collar of calloused skin. *Localization* – plantar and palmar surface with tend to be subungual and periungual. The periungual warts are very resistant to therapy with tendency to recurrence. The plaque of closely grouped myrmecia is named mosaic warts, which are seen as tightly compressed individual warts. **Flat warts (verruca plana, juvenile warts)** – smooth flat slightly elevated flesh-colored papules 1–5 mm, usually multiple, from few to hundreds, may become confluent. Localization – anywhere, face, hands and shins are more common. Flat warts can appear after traumatization of skin (Koebner phenomenon), spontaneous regression is possible, usually after previous inflammation. **Genital warts (condyloma accuminata)** affect penis, vulva, vagina, cervix, perineum and perianal area. Single or multiple pearly, filiform, fungating, cauliflower or plaque-like popular eruptions may be seen on affected area. Lesions have smooth, verrucous or lobulated surface, color vary from that of skin to red and brown. Pruritus and discharge may be complaints.

Differential diagnosis. Extremely large and resistant to treatment warts may be verrucous carcinoma. Other diseases for differential DD: lichen planus, callosities, seborrheic keratosis, molluscum contagiosum, tuberculosis verrucosa cutis. DD of genital warts: condyloma lata (secondary syphilis), herpes simplex, nevi, neoplasia.

Diagnostic approaches: minute black dots (thrombosed capillaries) become seen after clearing of warts. Skin biopsy and histological examination is necessary rarely. Laboratory study: immunohistochemical detection of HPV structural proteins, viral DNA identification using Southern blot hybridization to identify HPV type in tissue, polymerase chain reaction to amplify viral DNA for testing; HPV may be detected in younger lesions. In cases of genital warts, the patients have to be checked for other STD.

Treatment: Home application 1–2 t/d for several weeks of topical salicylic acid (5–40 % creams, paints, gels, karaya gum, impregnated plasters, collodion or

sodium carboxycellulose tape) in combination with lactic acid in wart varnishes is a first line therapy. Another home topical agent Podophyllin (0,5 % purified solution 2 t/d for 3 consecutive days, repeated weekly, not exceed 4 weeks.

Topical agents for physician's office. Cantharidin (extract of the blister beetle) 0.7 % solution applied sparingly with the wooden end of cotton-tipped applicator, repeat at 3–4 week intervals if needed. Dibutyl squaric acid/diphencyclopropenon is applied in light-shielded accessible location to achieve initial sensitization, then applied on warts 1–2 weeks. Another topical agents. Aminolevulenic acid is used in combination with UV-light as a photosensitizer to treat verruca plana. Imiquimod (5 % cream) is an immune response modifier used to treat genital warts. Cidofovir (antiviral agent) may be used in various concentration for topical treatment of recalcitrant warts. Podophyllotoxin 0.5 % solution (purified substances of podophyllin) is used to treat of genital warts. Topical 5-fluorouracil may be effective in treating warts used under occlusion daily for up to 1 month. Topical tretinoin (retinoid acid) has been successful in treating flat warts. Systemic treatment with retinoids (synthetic vitamin A analogs) may help with extensive hyperkeratotic warts in immunocompromised patients. Alternative therapy: adhesiotherapy (applying duct tape to the wart daily), hypnosis, hyperthermia (immersing the involved surface in hot water 45 ºC for 30–45 minutes, 2–3 times per week), propolis for common or plantar warts (500 mg/day until warts resolve or till 3 month). *Surgical care.* Cryosurgery with liquid nitrogen (-196 ºC) is considered the most effective method. Laser is considered the most expensive method, used to treat resistant or large warts (carbon dioxide laser, Nd:YAG laser). Electrodessication and curettage may be effective, but risk of scarring and pain is higher and HPV can be isolated from the plume.

Prevention. Vaccine is used to decrease the risk for HPV-induced cancer and precancerous lesions.

Molluscum contagiosum

Etiology. Molluscum contagiosum virus is unclassified member from family Poxviridae which I-IV types have been identified. It is considered more than 96 % of infections are caused by I type; II type causes most infections in HIV-positive patients. Molluscum contagiosum is a large DNA virus with high molecular weight, slightly smaller than the smallest bacteria and visible in microscope.

Pathogenesis. Molluscum contagiosum virus replicates in cytoplasm of epithelial cells and infects only the epidermis. The viral particles occur in basal cells and have been never found until spindle and granular layers of epidermis. Viral proliferation produces lobulated epidermal growths compressing epidermal papillae, while interlobular fibrous septa produces pear-shaped clumps with apex upwards.

The center of lesion is ultimately destroyed with forming of large hyaline bodies (i.e., molluscum bodies, Henderson-Paterson bodies), containing masses of virus material. These bodies are appeared as a white depression at the center of papule.

Epidemiology: Infection follows contact with infected persons or contaminated objects, virus may be inoculated along a minor skin trauma and may spread by autoinoculation. Incubation period is usually 2–7 weeks, but it is known that some cases accompanied by latent period till 6 months. Molluscum contagiosum is most common in children who become infected through direct skin-to-skin contact and indirect (bath towels, sponges, gymnasium equipment). Spontaneous resolution of rash occurs by 18 months in healthy persons, but the rash can persist till 5 years. Patients with HIV, atopic dermatitis, or those receiving prednisone, methotrexate, or other immunosuppressive medication may have more extensive and resistant molluscum contagiosum.

Clinical presentation: Skin lesions are consisting of single or multiple, spherical, dome-shaped, pink, waxy papules 2–5 mm (rarely 1–2 cm – giant molluscum) in diameter. Lesions have umbilicated center containing caseous whitish plug. Some lesions become confluent to form a plaque (agminate form). Typical localization: face, arms, chest, trunk, legs. The lesions usually asymptomatic, but some elements rarely may be tender or pruritic. Some elements become inflamed leading to suppuration crusting and eventual resolution. Molluscum contagiosum may rarely occur on the mucosa of lips, tongue, buccal area. Atopic patients may develop multiple hundreds lesions. Molluscum contagiosum in adult most commonly is a sexually transmitted disease with few lesions, which are limited to the perineum, genitalia, lower abdomen, buttocks. Immunocompromised or HIV-infected children and adults demonstrate widespread, persistent and atypical molluscum contagiosum with resistance to therapy. The following clinical variants are distinguished: 1) molluscum contagiosum milliare with multiple tiny lesions; 2) molluscum contagiosum pediculatum marked by pedunculated lesions; 3) molluscum giganteum with giant lesions formed after coalescence of smaller ones.

Differential diagnosis: Cutaneous cryptococcosis, histoplasmosis, aspergillosis, epidermal cysts, sebaceous hyperplasia, verruca vulgaris, lichen planus, flat warts, basal cell carcinoma, milia.

Workup: Histologic examination of biopsy: acanthosis, intracytoplasmic inclusion bodies (molluscum bodies). Microscopy of pasty core of lesion, crushing between two microscope slides and staining it to reveal molluscum bodies. Polymerase chain reaction is used to detect molluscum contagiosum virus in skin lesions.

Treatment: Cryotherapy, laser removing, curettage, expression of the central core with tweezers, rupture it with needle or toothpick, electrodesiccation, radiowaving removing, shave removal, duct tape occlusion of individual lesions. Topical using of tretinoin (cream 0.1 % or gel 0.025 %),

podophyllotoxin (cream 0.5 %), dilute povidone iodine and cantharidin can be self-administered with instructions of applications. Bichloracetic acid, trichloroacetic acid, salicylic acid (17 % collodion), lactic acid, glycolic acid, potassium hydrochloride (10 % aqueous solution), silver nitrate is applied in the hospital by the doctor. Subcutaneous intralesional interferon-alfa and topical cidofovir may be useful in immunodepressed patients.

Parasitosis

Scabies

Definition: Human scabies is an intensely pruritic skin infestation caused by the host-specific mite *Sarcoptes scabiei var. hominis.*

Epidemiology: Worldwide distribution. In some areas, such as certain Caribbean islands, it is endemic with virtually everyone infested. Approximately 300 million cases of scabies are reported worldwide each year. In the past, it typically appeared in cycles (*seven year itch*), but this is no longer the case. In recent years, epidemics in homes for the elderly have become a problem.

Pathogenesis: Sarcoptes scabiei hominis is an obligate human parasite. The infestation occurs by close personal (skin-to-skin) contact including sexual contact or, less commonly, by contact with infested fomites (e.g. clothing and towels). *Sarcoptes scabiei* female mites burrow into human epidermis just below the stratum corneum laying eggs that hatch and develop into adults in 2 weeks. The life cycle of *Sarcoptes scabiei* is 4–6 weeks. The mites and mite products (faeces, eggs and dead parasites) generate an immediate or delayed (type IV) hypersensitivity reaction with scabies symptoms typically starting 3–6 weeks after primary infestation and 1–3 days after re-infestation. The first infestation remains asymptomatic for a period of weeks, until an immune response develops and pruritus results. Upon re-infestation, the symptoms appear in a matter of days.

Clinical features: Specific manifestations include intense itch and disseminated inflammatory papules. *Signs and Symptoms:* erythematous papules disseminated on the periumbilical area, waist, genitalia, breasts, buttocks, axillary folds, fingers (including interdigital spaces), wrists and extensor aspects of the limbs; nodules on the penis and scrotum,; pustules on the palms and soles of infants; nocturnal itching, generalized, severe itching; other lesions: vesicles (usually at the start of a burrow), weals; rash present for 4 to 8 weeks has suddenly become worse; rash is present in several members of the same family. Burrows are a pathognomonic sign and represent the intraepidermal tunnel (sometimes erythematous, irregular lines with a terminal swelling) created by the moving female mite. Non-specific manifestations which may also occur are skin excoriation, secondary eczematization and impetiginization. *Intense pruritus*: Few skin diseases itch as much as scabies; usually worst at night. *Dermatitis*: Immune reaction (type IV) to mites leads to both pruritus and diffuse exanthem. Typical sites are thighs, buttocks, trunk. *Variations: Pyoderma:* Pruritus leads

to excoriations and erosions which become secondarily infected. In some areas, there is a vicious cycle of scabies – impetigo, glomerulonephritis.

Scabies incognita: Patients with meticulous personal hygiene (*scabies of the cleanly*) or those using topical corticosteroids may completely mask the findings of scabies, complaining only of pruritus.

Nodular scabies: Nodules occur in 7–10 % of patients with scabies, particularly young children, favoring the groin, axillae, and genitalia. Occasionally seen in adults once again genitalia most common. There is a lymphocytic infiltrate on biopsy. Remain for months after elimination of all mites; blamed on "antigen persistence".

Crusted scabies (Norwegian scabies): Massive scabies infestation with crusted hyperkeratotic, psoriasiform lesions, as well as subungual lesions. It occurs in patients with severe immune deficiency due to disease (e.g. AIDS, HTLV1-infection, malignancy and leprosy) or therapy (e.g. immunosuppressant drugs and biologicals), sometimes in Down syndrome, neurological disease causing reduced sensation, immobility with reduced ability to scratch or in genetically susceptible.

Patients may present with thick, crusted lesions on the hands and feet, nail abnormalities, generalized erythematous scaling eruptions, and scalp involvement. Because patient`s pruritus is mild or absent in this form, scabies is often misdiagnosed, leading to large nosocomial outbreaks. In crusted scabies, thousands of mites may be present, and the risk of transfer is greater.

Animal scabies: There are over 40 different forms of animal scabies, including those involving cats, dogs, and birds. These mites cannot reproduce on humans. They tend to bite at sites of contact (hands, arms, face if sleeping with pet), cause pruritus without any incubation period, and then die.

Complications: Acarophobia: fear of persistent infection following cure; major psychological problem.

Diagnostic approach: Scabies may be diagnosed with a history of pruritus, rash in the typical distribution, and history of itching in close contacts. The diagnosis is confirmed by a positive microscopic examination of skin scrapings which identifies mites, larvae, eggs. A negative microscopic result does not exclude scabies. Dermoscopic examination can identify skin burrows, mites. In sexually active patients, STI screening, including HIV test is recommended.

Differential diagnosis: All pruritic diseases; the issue is always to include scabies in the differential diagnosis (eczema, atopic dermatitis, allergic contact dermatitis).

Therapy: Scabies treatment includes administration of a scabicidal agent, appropriate antimicrobial agent as well if a secondary infection has developed. Permethrin 5 % cream has the best safety record and the least reports of resistance. Lindane lotion is the worldwide standard, but not as effective as permethrin. Topical treatment should be applied to all skin regions including scalp, groin, navel, external genitalia, finger and toe web spaces and the skin

beneath the ends of the nails at night and left in place for 8–12 h. The treatment must be repeated after 7–14 days. Benzyl benzoate lotion 10–25 % applied once daily at night on 2 consecutive days with re-application at 7 days. After applying treatment, patients should change into clean clothing. All the patient's close personal contacts should be treated simultaneously to avoid re-infestation. For resistant cases, epidemics or crusted scabies, ivermectin 150–400 μg/kg per oral administered on days 1 and 14 is highly effective. Pruritus may be partially alleviated with an oral antihistamine.

A warm bath prior to application will increase the efficacy of treatment; remember that with eroded areas, absorption is increased. Clothing, bedding, towels and other items should be washed in hot cycle of washing machine, dry-cleaned, or sealed and stored in plastic bag for 1 week. Patients should be informed that rash and pruritus of scabies may persist for up to 2 weeks after treatment. Post-treatment itch should be treated with repeated application of emollients. Oral antihistamines and mild topical corticosteroids may also be useful.

Prevention: The risk of scabies can be reduced by limiting the number of sexual partners and observing strict personal hygiene when living in crowded spaces (e.g. no sharing of underwear clothing, bedding and towels and avoidance of skin-to-skin contact). Transmission is not prevented by condom use.

Pediculosis

Definition: Pediculosis is infestation with the human head-and-body louse *Pediculus humanus*. It is highly contagious diseases which spread by direct head-to-head contact.

Etiology: Lice (*Pediculus spp*.) are blood-sucking, wingless, ectoparasitic insects that can infest the head, body and pubic region.

Epidemiology: Louse infestation remains a major problem throughout the world, making the diagnosis and treatment of louse infestation a common task in general medical practice. All socioeconomic groups can be affected.

Pathogenesis: Lice feed on human blood after piercing the skin and injecting saliva, which may cause pruritus due to an allergic reaction.

Disease transmission: Lice transmit a number of important diseases: Epidemic typhus (*Rickettsia prowazekii*). Relapsing fever (*Borrelia recurrentis*). Trench fever (*Bartonella quintana*).

Pediculosis Capitis

Definition: Infestation with the head lice, *Pediculus humanus capitis*.

Epidemiology: Pediculosis has a worldwide distribution and is endemic in both developing and developed countries. The prevalence of pediculosis capitis is usually higher in girls and women. Head lice are mainly spread by direct contact with an infected person. Head-to-head contact can be common among children during play at school, home, and elsewhere (e.g., sports activities, playgrounds, camp, and slumber parties). Infestation with head lice (*Pediculus humanus capitis*) is most common among preschool- and elementary school-age

children and their household members and caretakers. Pediculosis capitis results in significant psychological stress in children and adults and missed schooldays in children. Fomites, such as clothing, headgear, hats, combs, hairbrushes, hair barrettes, may occasionally play a role in the spread of head lice.

Pathogenesis: Lice live on the scalp and suck blood there. They firmly attach their eggs (nits) to the hair shaft just at the skin surface.

Clinical features: Head lice infestations can be asymptomatic, particularly with a first infestation or when an infestation is light. Itching ("pruritus") is the most common symptom of head lice infestation and is caused by an allergic reaction to louse bites. It may take 4–6 weeks for itching to appear the first time a person has head lice. Other symptoms may include the following: a tickling feeling or a sensation of something moving in the hair; irritability and sleeplessness. The hairs may become matted from repeated scratching (*plica polonica*). Scalp itch leads to scratching, secondary infection and cervical lymphadenopathy.

Diagnostic approach: The diagnosis is confirmed by identifying the living louse or nymph on the scalp or on a black sheet of paper after careful fine-toothed combing of wet hair following conditioner application. The empty egg cases ('nits') are easily seen on the hair shaft and are hard to dislodge.

Misdiagnosis of head lice infestation is common. The diagnosis of head lice infestation is best made by finding a live nymph or adult louse on the scalp or hair of a person. Because adult and nymph lice are very small, move quickly, and avoid light, they may be difficult to find. Visualization can be improved by the use of a bright light, a magnifying lens, and combing the hair with a "lice comb" (fine-toothed comb) and examining the comb teeth If crawling lice are not seen, finding nits attached firmly within ¼ inch of the base of hair shafts suggests, but does not confirm, the person is infested. Nits frequently are seen on hair behind the ears and near the back of the neck. Nits that are attached more than ¼ inch from the base of the hair shaft are almost always non-viable (hatched or dead). Head lice and nits can be visible with the naked eye, although use of a magnifying lens may be necessary to find crawling lice or to identify a developing nymph inside a viable nit. Nits are often confused with other particles found in hair such as dandruff, hair spray droplets, and dirt particles. If no nymphs or adults are seen, and the only nits found are more than ¼ inch from the scalp, then the infestation is probably old and no longer active – and does not need to be treated.

Differential diagnosis: True nit infestation must be distinguished from hair casts (pseudonits). Hair casts look similar to nits, but form an encompassing cylinder whereas the nits are attached at an angle. Many scalp conditions can cause pruritus follow as: seborrheic dermatitis, atopic dermatitis. Other problems to be considered in the differential diagnoses of head louse infestation include the following: dandruff, fibers, scabs, desquamated cells, dirt, dried hairspray/gel, dermatophyte infection, piedra, hair shaft abnormalities (e.g.

monilethrix, trichorrhexis nodosa), delusions. *Piedra* is much less common and consist of clumps of bacteria or fungi.

Therapy: Treatment of pediculosis has 2 aspects: medication and environmental control measures. Increasing emphasis is being placed on understanding the life cycle of lice in order to provide effective treatment. Treatment for head lice is recommended for the affected individual and any infected household/school contacts. Eradication in school populations is difficult because of poor adherence and treatment resistance. The goal of therapy is to eliminate lice and eggs. Chemical pediculicides are the mainstay of pharmacotherapy. With most medications, treatment should be repeated in 7–10 days (the time needed for the eggs to hatch) because nits are less effectively killed than adults. *Eyelash/brow infestation* can be treated effectively with petrolatum ointment (e.g. vaseline), applying twice daily for at least a fortnight. *Topical treatment* with permethrin lotion 5 % or permethrin shampoo, benzyl alcohol lotion 5 %, ivermectin lotion 0.5 %; carbaryl or, less often, malathion in lotion or aqueous formulations may be effective and should be applied twice at an interval of 7–10 days. Resistance of head lice to pediculicides has become a problem. Check for local resistance pattern. All agents should be applied twice, 7–14 days apart. Recommendations vary regarding length of application, including overnight use, but all appear effective when used for 10–30 minutes and rinsed. Malathion 0.5 % lotion is most effective, but there is resistance in France and the UK. Pyrethrins and the synthetic permethrin have fair action and a reasonable resistance profile. Lindane shampoo 1 % (gamma benzene hexachloride) is still widely used, but relatively ineffective for this indication and with marked resistance. The nits are always a problem; many schools have rules banning children returning as long as nits are present. Wet-combing' (physical removal of live lice by regular combing of conditioned wet hair – 'bug busting') can suffice but may be less effective than pharmacological treatments. Best solutions following treatment are soaking with vinegar and water (50 : 50) and using a fine-toothed nit comb. Treatment resistance and recurrence can be problematic. Ivermectin (400 mcg per kg) is an oral antiparasitic that has demonstrated effectiveness for the treatment of resistance pediculosis.

High-temperature washing of clothing and bedding is required. Machine wash and dry clothing, bed linens, and other items that the infested person wore or used during the 2 days before treatment using the hot water (50 °C) laundry cycle and the high heat drying cycle. Clothing and items that are not washable can be dry-cleaned or sealed in a plastic bag and stored for 2 weeks. Soak combs and brushes in hot water (at least 50 °C) for 5–10 minutes.

Prevention: Avoid head-to-head (hair-to-hair) contact during play and other activities at home, school, and elsewhere (sports activities, playground, slumber parties, camp). Do not share clothing such as hats, scarves, coats, sports uniforms, hair ribbons, or barrettes. Do not share combs, brushes, or towels. Disinfest combs and brushes used by an infected person by soaking them in hot water (at

least 50 °C) for 5–10 minutes. Do not lie on beds, couches, pillows, carpets, or stuffed animals that have recently been in contact with an infected person.

To prevent head lice re-infestation, all household members and contacts of a patient should be examined and treated at the same time if infested. Prevention of re-infestation with body lice can be accomplished by ensuring that infested clothing, bedding, and towels were appropriately laundered with hot water (at least 60 °C) and then dried in a dryer using a hot setting to destroy the lice, improving the individual's hygiene, regularly laundering clothing, and changing to clean clothing at least weekly.

Pediculosis Corporis

Definition: Infestation with *Pediculus humanus corporis*.

Epidemiology: Pediculosis corporis is primarily a disease of the unwashed. It is common in homeless people and during wars (trench fever) and other disasters. These are similar to head lice but live on clothing, particularly in seams, and feed on the skin. Poor hygiene and overcrowded conditions predispose. Risk factors for body lice infestation are included close, crowded living situations (e.g. crowded buses and trains, prison camps) and infrequent washing and/or changing of clothing. *Pediculus humanus corporis* is spread through direct contact with a person who has body lice or through contact with articles such as clothing, beds, bed linens, or towels that have been in contact with an infected person. Body lice can transmit disease. Epidemics of typhus and louse-borne relapsing fever have been caused by body lice.

Pathogenesis: The lice feed on the body, but live in the clothing and tend to lay their eggs along the seams.

Clinical features: Intense itching ("pruritus") and rash caused by an allergic reaction to louse bites are common symptoms of body lice infestation. As with other lice infestations, intense itching leads to scratching which can cause sores and secondary bacterial infection of the skin. In longstanding infestation, the skin may become lichenified and hyperpigmented, particularly on the trunk.

Diagnostic approach: Body lice infestation is diagnosed by finding eggs and crawling lice in the seams of clothing. Although body lice and nits can be large enough to be seen with the naked eye, a magnifying lens may be necessary to find crawling lice or eggs.

Differential diagnosis: All pruritic dermatoses, especially scabies. Other problems to be considered in the differential diagnoses of body louse infestation include the following: folliculitis, insect bites, scabies, acne, delusions of parasitosis, xerosis with excoriations, impetigo, postinflammatory hyperpigmentation.

Therapy: The mainstay of treatment for body lice is disinfection of clothing and bedding (boiling, hot ironing, fumigation) and regular bathing. Attempt to change living conditions. A pediculicides are usually not needed. In mass epidemics, usually insecticidal dusting powders employed. In the case of body lice, infested clothing and towels need to be washed in hot water and with a hot dryer. The infested individual should be counseled on proper hygiene, changing

clothing at least once a week, and proper laundering of clothing. Treatment options are as for head lice. For heavy infestation, oral ivermectin may be indicated.

Prevention: Bathe regularly and change into properly laundered clothes at least once a week; launder infested clothing at least once a week. Machine wash and dry infested clothing and bedding using the hot water (at least 50 °C) laundry cycle and the high heat drying cycle. Clothing and items that are not washable can be dry-cleaned or sealed in a plastic bag and stored for 2 weeks. Do not share clothing, beds, bedding, and towels used by an infected person. Fumigation or dusting with chemical insecticides sometimes is necessary to control and prevent the spread of body lice for certain diseases (epidemic typhus).

Pediculosis Pubis

Definition: Pediculosis pubis (sin. crab louse) is an infectious disease caused by the infestation with the parasite *Phthirus pubis*.

Epidemiology: Pubic lice infestation is found worldwide and occurs in all races and ethnic groups and in all levels of society. The infection is transmitted by sexual contacts, close body contact or, less commonly, by contact with objects (e.g. clothing, towels) and it are most common in adults. *Phthirus pubis* infests the terminal hairs of the pubic and perianal areas. The parasite is not adapted for crawling but can be found of the hairs of the legs, forearms, chest or face. Pubic lice do not transmit disease; however, secondary bacterial infection can occur from scratching of the skin. Finding pubic lice should prompt an evaluation for other sexually transmitted infections.

Clinical features: Patients' main complaint is of itch in the pubic area. Patients usually identify moving lice on their pubic hairs (*crabs*). Nits are usually on pubic hair, but occasionally elsewhere (axillary or body hairs; eyelashes, eyebrows). The feeding sites turn into distinctive blue-gray hemorrhagic macules (*maculae ceruleae* or *taches bluetres*). Small blood stains can be observed on the underwear.

Diagnostic approach: Diagnosis is usually based on the typical clinical findings**.** There is identification of lice or nits.

Differential diagnosis: Other problems to be considered in the differential diagnoses of pubic louse infestation include the following: dermatophyte infection, folliculitis, delusions of parasitosis, contact dermatitis.

Therapy: Patients should be advised to avoid close body contact until they and their partner(s) have completed treatment and follow-up. Pubic hair may need to be shaved. Sexual and other close contacts should also be treated. The topical treatment is applied to all suspected infested regions: genital and anal areas, thighs, trunk, axillae, moustache and beard areas. Permethrin cream 1 % or shampoo or lindane lotion 1 % or shampoo applied for 10minutes; repeat in 1 week. Lotions are likely to be more effective than shampoos, and should be applied to all body hair including the beard and moustache if necessary. Ivermectin 250 mkg/kg orally is also effective in a series of Pediculus pubis cases, at dosage 400 mkg/kg orally effective for resistant cases. The nits must be removed from the hair.

Clothing, bedding, towels and other items should be machine washed (at 50 °C or higher) or dry-cleaned or sealed and stored in a plastic bag for 3 days. When starting the treatment, patients should wear clean underwear and clothing.

Prevention: Patients with Pediculosis pubis should not share their clothes, bedding and personal hygiene products. Transmission by sitting on toilet seats is not possible. The disease is not prevented by condom use.

Demodicosis

Definition: Human demodicosis is a skin disease of the pilosebaceous units associated with human *Demodex* mites that involves predominantly the face and head.

Etiology: Its causative agents are *Demodex* mites which belong to the family Demodicidae of the class *Arachnida* in the order *Acarina*. The mite *Demodex* spp., lives around hair follicles or in the secretory ducts of sebaceous glands connected to the hair follicles of humans. The incidence of demodicosis steadily increases with the individual's age. Only two specious of *Demodex* (*D. follicullorum* and *D. brevis*) have been identified in humans. They have been implicated in at least three facial conditions: pityriasis follicullorum, rosacea-like demodicosis and so called *demodicosis* gravis.

Pathogenesis: Demodex folliculorum is a saprophytic mite of the human pilosebaceous unit. The preferred sites are facial skin, forehead, cheeks, eyelashes and external ear channels. Infestation with them may frequently be free of symptoms. However, suppurative or granulomatous reactions and inflammation may occur in acute and chronic demodicosis in humans.

Classification: There are forms of the demodicosis follow as: erythemato-squamous, erythematous-telangiectatic, papular, papulopustular, nodulocystic or conglobate demodicosis, infiltrative-productive, ocular demodicosis, inducing chronic blepharitis, chalazia or, less commonly, keratoconjunctivitis; and auricular demodicosis causing external otitis or myringitis.

Clinical features: Two clinical variants, primary and secondary, can be observed. *Primary demodicosis* can be defined according to following diagnostic criteria: absence of pre-existing or concurrent inflammatory dermatoses, such as acne, rosacea or perioral dermatitis; abnormal increase in mite colonization, which should be identified from the active lesions at the time of examination; and remission of the disease only after adequate treatment with topical or systemic acaricides/arachnicides, but not with antibiotics possessing anti-inflammatory effects. *Primary human demodicosis* is clinically characterized by late onset, usually after age 40 years and especially in the elderly population; facial involvement, typically affecting periorificial areas (perioral, periorbital or periauricular); usually asymmetric distribution, grouped in an irregular shape with satellite lesions within one affected area; being follicle bound; and being asymptomatic or mildly pruritic. Skin lesions associated with an abnormal increase of *Demodex* mites in patients with other known skin or

systemic diseases can be classified as *secondary demodicosis*. It occurs most commonly in significantly immunosuppressed patients, such as those with leukaemia and HIV infection, those being treated with immunosuppressants including topical glucocorticoids or topical calcineurin inhibitors as well. *Secondary demodicosis* can occur early in life and show a more diffuse facial distribution or truncal involvement with more extensive inflammation.

Diagnostic approach: The diagnosis is confirmed by a positive microscopic examination of skin scrapings which identifies mites, eggs.

Differential diagnosis: Other problems to be considered in the differential diagnoses of demodicosis include the following: rosacea, acne vulgaris, systemic lupus erythematosus, photosensitivity disorders, seborrhoeic dermatitis, meibomian gland dysfunction, Phthiriasis (pubic lice) of the lids, cyanoacrylate glued false eyelashes and other eye diseases.

Therapy: Mild disease may respond to topical antimicrobials, such as metronidazole or azelaic acid. Topical use of acaricides, such as permethrin 5 %, benzyl benzoate 10–25 %, topical metronidazole 0.75–2 %, crotamiton 10 %, lindane 1 % or malathion 0.5 %, were effective for the treatment of demodicosis. Topical ivermectin may be beneficial in some cases, supporting a contributory role of *Demodex* in pathogenesis. Tetracycline or erythromycin for 3–6 months is usually effective in inflammatory pustular disease resistant to topical therapy. Relapse may require intermittent or chronic antibiotic use. Erythema and telangiectasiae do not usually respond well to antibiotics but vascular laser therapy may be effective. Topical vasoconstrictors, such as the α_2-adrenoceptor agonist brimonidine, may be of benefit in some cases where erythema and telangiectasiae predominate. Systemic isotretinoin may be helpful in severe resistant disease and rhinophyma may require laser therapy or surgery.

Fungal diseases

Nomenclature: dermatophytes. Molds. Yeast. Subcutaneous mycoses. Systemic or deep mycoses.

Classification:

1. *Keratomycosis*: multicolored pitiasis.

2. *Dermatophytes*: Tinea cruris. Tinea pedis. Rubrophytosis. Trichophytosis (anthrophilic, zooantrophilic). Microsporosis (antrophilic, zooantrophilic). Favus.

3.*Candidasis*: Superifical. Visceral. Generalized. Chronic.

4. *Deep mycosis.*

5. *Pseudomycosis*

Keratomycosis

Pityriasis versicolor (pityriasis furfuracea)

Definition: A common superficial yeast infection. Well-defined scaly plaques appear on the torso, which appear white on tanned skin and light brown on white skin.

Pathogenesis: Pathogen *Malassezia furfur,* which is a pathogenic form of commensal cutaneous yeast *Pityrosporon ovale and Pityrosporon orbiculare.* This is a conditionally pathogenic microorganism that lives in the stratum corneum of the epidermis and in the hair follicles in people over 15 years old. The mechanisms of pigment changes are unclear; darkening is the result of hyperkeratosis, but lightening may reflect the effect of an umbrella, as well as a direct effect on melanocytes.

Clinical features: typical places: Upper half of the body, shoulders, neck, abdomen, axilla, groin, thighs, genitals. Occasionally - a face. The scalp is the most common reservoir of Pityrosporon. In fair-skinned people, lesions are usually hyperpigmented, with 1–3 cm brown oval spots, often merging. With easy cleaning, they are very scaly; a good diagnostic clue, since few other disorders release so many scales. On tanned skin – light brown, on tanned – white, blacks – dark brown. All shades of brown; yellowish white. Dimensions and shape. Round or oval spots of various sizes. Over time, the foci increase, sometimes merge, forming drawings similar to a geographical map.

Diagnostic approach: KOH examination reveals hyphae and spores (spaghetti and meatballs). Another approach is stripping with adhesive tape, staining with methylene blue for 5 seconds, washing and examination under a microscope. Culture is impossible.

Differential diagnosis: *Hypopigmented lesions:* vitiligo, alpha-pitiriasis, lichen white, post-inflammatory hypopigmentation.

Hyperpigmented lesions: epelids, lentigines, coffee-a-light macula.

Erythematous lesions: Tinea corporis, pityriasis rosea, secondary syphilis, teardrop-shaped psoriasis, seborrheic dermatitis.

Therapy: The standard approach is to use shampoo with imidazole (ketoconazole) or shampoo with selenium. The scalp and the entire surface of the body in the groin should be washed and then washed after a few minutes. Initially, treatment is daily for 7–10 days. Always relate to the scalp; it is the main reservoir for yeast. Relapses are common; patients can simply use a 1–2-hour treatment shampoo weekly to infinity. In persistent cases, a short course of itraconazole or fluconazole can be used; some recommend 1 tablet per week for prevention.

Dermatophytes

Dermatophytes are a group of filamentous fungi that infect keratinous keratinocytes (the stratum corneum of the epidermis, nails and hair). They can be subdivided into: *anthropophilic*: found in humans. *Zoophilic:* found in animals. *Geophilic:* found in soil.

Clinical picture: determined by the nature of the dermatophyte, the tissue into which it enters, and the degree of reaction of the host. Dermatophyte infections commonly called shingles; an anatomical site such as shingles with inflammation of the scalp. A clinical infection usually begins at the site of vaccination and spreads peripherally; while ring lesions with an active border.

In non-medical jargon, the diagnosis is often "ringworm." The most common cause of fungal infections in Europe is *Trichophyton rubrum*. Zoophilic and geophilic infections always cause a more intense immune response and, therefore, seem more aggressive. An immune response to dermatophytic infections can also cause disease in remote places where there are no mushrooms. The most typical picture is dyshidrotic dermatitis.

Diagnostic Approach: Sampling: Sanitize the area first to reduce contamination.

Skin: with a scalpel or with the edge of a glass slide, remove the upper layer of the epidermis, the scales are placed in the center of the glass slide and cover with a coverslip.

Nails: the material is obtained with a scalpel (blade No. 15) or a small curette (1 mm): with distal-lateral subungual onychomycosis, scraping is done from the nail bed or from the inner surface of the nail plate; with white superficial onychomycosis, scraping is done from the outer surface of the nail plate; with proximal subungual onychomycosis, they make scraping from the inner surface of the nail plate.

Hair: use tweezers or a needle holder to remove broken hair, place on a glass slide and cover with a coverslip.

Treatment with potassium hydroxide. The resulting material is treated with 5–20 % potassium hydroxide. For this, a drop of the solution is placed at the edge of the coverslip: the solution penetrates under the glass under the action of capillary forces. Then the drug is slightly heated until bubbles form using a match or a lighter. Excess alkali is removed with filter paper. The preparation thus enlightened is examined under a microscope by lowering or removing the condenser. Study. Mycelium of dermatophytes looks under a microscope as a plexus of thin tubes (hyphae), inside of which septa (septa) are visible.

Inspection under the *Wood lamp.* Carried out in a darkened room. With hair damage Microsporum spp. there is a green glow.

Culture: many standard culture media are available; usually two cultures are prepared, one on a carrier containing cycloheximide (for dermatophytes) and one without (yeast and mold). Hair can be placed directly on culture media; fragments on the underside of the nail should be used. A light wood study is useful for Microsporon and Trichophyton schoenleinii species; A light inspection of a negative tree does not exclude a fungal infection. Hyphae or spores are grown on Saburo's medium (glucose + peptone + agar-agar).

Therapy: standard therapy includes both local and systemic drugs (Griseofulvin, Ketoconazole, Itraconazole, Terbinafine)

Tinea capitis

Definition: infection of the scalp with dermatophytes involving the hair shaft. Sources of infection – a person (patient or carrier), animals, household items contaminated with spores of fungi. Infection with fungal spores occurs through cuticle defects in the hair and epidermis.

Pathogenesis: various clinical forms develop based on the reaction of the host and the nature of the hair lesion. 90 % of cases of dermatophytosis of the scalp are caused by *Trichophyton tonsuran*s, the rest are *Microsporum canis, Microsporum gypseum, Trichophyton mentagrophytes, Trichophyton rubrum.* Previously, the most common pathogen was *Microsporum audouinii* (very contagious).

Clinical features: Trichomycosis pathogens are divided into two groups: *ectotrix fungi*, which affect the outer root vagina of the hair, and *endotrix fungi*, which affect the brain and cortex of the hair shaft. Trichomycosis caused by ectotrix fungi. The infection is concentrated in the external root follicul. Fungus hyphae penetrate the hair cuticle. With the formation of art rockonidia, the cuticle is destroyed. Ectotrix fungi include *Microsporum spp. (Microsporum audouinii and Microsporum canis).*

Trichomycosis caused by endotric fungi themselves. The infection is concentrated in the brain and cortex of the hair, where arthroconidia form. The hair cuticle is not damaged. Endotrix fungi include *Trichophyton spp.* (Trichophyton tonsurans is more common in North America, *Trichophyton violaceum* in Europe, Asia and Africa). *"Black-dot" dermatophytosis of the scalp*. This trichomycosis resembles seborrheic dermatitis. Black dots represent stumps of broken hair. *Kerion*. The foci do not have clear boundaries, there is a diffuse lesion. Trichomycosis, which occurs with inflammation of the hair follicles, suppuration and the formation of deep painful nodes. Painful nodes or plaques, soft to the touch. From the openings of the hair follicles, like honey from a honeycomb, pus is secreted. Affected hair does not break, but loosens and falls out. They can be easily and painlessly removed with tweezers. After healing, cicatricial alopecia occurs. Synonym: kerion Celsus.

Favus. Trichomycosis, in which air bubbles are found in the destroyed substance of the hair along with arthroconidia. Scooters are thick, yellow crusts welded to the skin, from which the remaining hair sticks out. Scooters consist of destroyed keratinocytes, dried exudate and hyphae of the fungus. The unpleasant "mouse" smell is characteristic. Skin atrophy, scarring, cicatricial alopecia. In Western Europe and North America, the disease is rare; and in the Middle East and South Africa it is widespread.

Synonym: scab.

Differential diagnosis. Flaky area of baldness (gray spot) Seborrheic dermatitis, psoriasis, diffuse neurodermatitis, limited neurodermatitis, alopecia areata. "Black-dot" dermatophytosis Seborrheic dermatitis, psoriasis, seboro-psoriasis, diffuse neurodermatitis, limited neurodermatitis, discoid lupus erythematosus, alopecia areata. Kerion Phlegmon, boil, carbuncle. Favus Impetigo, ectima, Norwegian scabies.

Additional research. Examination under a Wood lamp. Indicated for all patients with a flaky lesion on the scalp or alopecia of unknown origin.

Microsporum canis and Microsporum audouinii give a bright green glow, Trichophyton tonsurans does not glow.

Microscopy of a potassium hydroxide-treated preparation. The sample should contain hair roots and epidermis flakes. Material is collected using a toothbrush and tweezers. Hyphae and arthrospores are found in scales. Trichomycosis caused by ectotric mushrooms themselves: hyphae and arthrospores in the hair cuticle. Trichomycosis caused by endotrix fungi: arthrospores in the hair substance. *Favus*: loosely arranged chains of arthrospores and air bubbles in the hair substance.

Sowing on media forfungus. A dry toothbrush vigorously rub the affected area, and then firmly press it with bristles to the nutrient medium. To collect material, you can use a wet cotton swab. Dermatophyte colonies grow in 10–14 days.

Therapy: local therapy is ineffective; systemic antifungal drugs for 1–2 months until the culture becomes negative. Only griseofulvin is officially approved for children, but everyone else is safe and effective. In the case of kerion with a secondary infection, add systemic antibiotics based on culture and sensitivity.

Tinea barbae

Definition: Dermatophytic infection in the beard area of men. Reminds dermatophytosis of the scalp caused by endotrix fungi.

Synonyms: tinea barbae, infiltrative suppurative trichophytosis of the face, parasitic sycosis.

Pathogenesis: similar to kerion; trichophyton verrucosum or trichophyton mentagrophytes.

Clinical features: Patients are usually farmers with close contact with animals. Red inflamed papules or pustules around the mouths of the hair follicles, purulent discharge, crusts form on the skin. When papules merge, a plaque is formed, covered with pustules. Affected hair is loose and easily removed with tweezers. Instead of folliculitis, flaky round reddish plaques can be observed, within which the hair is broken off at the skin level. As with dermatophytosis of the scalp, kerion is found – a soft painful node with purulent discharge. Color. red.

Differential diagnosis: carbuncle, acne vulgaris, rosacea, ingrown hairs, staph infection with multiple boils (sycosis barbae); it usually hurts.

Therapy: local therapy is ineffective; systemic antifungal drugs for 1–2 months.

Tinea Pedis

Definition: Dermatophytic infection of the feet and toes.

Synonym: athlete's foot, epidermomycosis of the feet, rubrophytosis of the feet, epidermophytosis of the feet.

Epidemiology: the most common fungal infection; 30–50 % of adults were affected.

Pathogenesis: The most common agents are *Trichophyton rubrum*, *Trichophyton mentagrophytes and Epidermophyton floccosum*. Infections caused by poor hygiene, excessive sweating, occlusal shoes; possibly due to impaired peripheral circulation. Pools, public showers and saunas are likely sources of infection.

Clinical features: the picture is highly dependent on the causative dermatophyte. *Hyperkeratotic type:* also known as moccasin type; diffuse small scales, rarely symptomatic, often overlooked or mistaken for palmoplastic keratoderma. First noticed with the involvement of the nail. Commonly called Trichophyton rubrum. *Chronic interdigital type:* usually includes the space between the more lateral fingers. Favorite localization is the gap between the little finger and the fourth finger. In addition to the interdigital spaces, adjacent areas of the foot may be affected. The macerated epidermis is white and fissured. It can extend to the sole, but rarely to the upper part of the foot. Commonly called Trichophyton mentagrophytes var. interdigitale. *Dyshidrotic type*: repeated bouts of itchy vesicles and pustules, identical dyshidrotic dermatitis. The same principle as the fungal reaction, but organisms (usually Trichophyton mentagrophytes var. interdigitale). Sole, medial surface of the foot, interdigital spaces.

Complications: gram-negative cobwebs, the site of penetration of erysipelas, a predisposing factor for cellulite post-coronary bypass grafting.

Differential diagnosis: Candidiasis infection. May also cause maceration, but rarely. Gram-negative web infection: usually associated with tinea pedis, look for both. Juvenile plantar dermatosis: bilateral, symmetrical, associated with atopy. Dyshidrotic dermatitis. Palmoplastic pustulosis. Allergic contact dermatitis. Erythrasma; impetigo; small point keratolysis; interdigital candidiasis; interdigital infection bullous impetigo.

Therapy: Primary prevention In public baths, pools and even at home, when taking a shower, slippers should be worn. Topical antifungal drugs; in severe cases, systemic antifungal drugs for 1–3 months. The treatment of concomitant onychomycosis and the continued prophylactic use of local drugs are important to reduce the recurrence rate. In case of macerated forms, keep the area dry, wear absorbent socks, use sandals in the summer. Shoes should be disinfected with antifungal sprays to reduce the likelihood of reinfection. Tinea pedis is very common and very difficult to eradicate. May be seen as a parasite that is just too well adapted to the host. The older the patient, the less likely the cure.

Tinea Manuum

Definition: Dermatophytic infection of the palms.

Pathogenesis: Pathogens include *Trichophyton rubrum*, *Trichophyton mentagrophytes* var. *interdigitale*, less commonly *Trichophyton violaceum* and *Trichophyton erinacei*. Either mainly vaccination (treatment of hedgehogs or infected farm animals), or secondary to tinea pedis.

Clinical features: most often dry hyperkeratotic form; then always check legs; dermatophytosis of the hands is chronic and often combined with dermatophytosis of the feet. Often only one arm is affected (the right-handed one has a right). A peculiar, but not very rare, option is “a disease of one arm, two legs”. If caused by a bestial fungus, is more localized and inflamed.

Differential diagnosis: Erythema and peeling on the hands Diffuse neurodermatitis, limited neurodermatitis, simple and allergic contact dermatitis, psoriasis, Devergey disease, squamous cell carcinoma in situ, cladosporiosis.

Therapy and prevention: The course is chronic. Relapses occur until concomitant onychomycosis or dermatophytosis of the feet is eliminated. Cracks and erosion serve as a gateway for bacterial infections. Topical antifungal medications are usually inadequate; systemic therapy is required.

Tinea Inguinalis

Synonym: sometimes incorrectly called tinea cruris (but cruris refers to the lower leg in Latin); marginal eczema.

Definition: Inguinal dermatophytosis - for an acute or chronic disease with damage to the skin of the thighs, pubic and inguinal areas.

Pathogenesis: The most common pathogens are *Epidermophyton floccosum* and *Trichophyton rubrum; Trichophyton mentagrophytes, Epidermophyton floccosum*. Often associated with tinea pedis or onychomycosis. More common in men.

Clinical features: Inguinal dermatophytosis is usually combined with dermatophytosis of the feet and onychomycosis of the feet. Elements of a rash. Large flaky plaques with clear borders. Erythema. Healing starts from the center. Along the edges of the plaques are papules and pustules. Treated foci: there is no desquamation, black patients have post-inflammatory hyperpigmentation. In patients with allergic diseases, due to constant combing of the skin, limited neurodermatitis develops. It can spread to the buttocks. The scrotum and penis are rarely affected.

Differential diagnosis: candidiasis (more common in women, satellite lesions), erythrasma (Wood's mild positive reaction), reverse psoriasis (look for psoriasis elsewhere), contact dermatitis, Haley-Haley disease. Erythema and peeling in the inguinal region of Erythrasma, psoriasis of skin folds, candidiasis of skin folds, pityriasis versicolor, histiocytosis X. Diaper rash is a superficial irritation that occurs when skin folds touch each other, like in the groin, under chest or abdominal folds. This is most often observed in obesity. Occlusion and moisture retention leads to maceration, which may predispose to any of the above diseases. We see this as a description, not as a disease.

Therapy and prevention: Topical antifungal medications are usually sufficient. Drying measures (absorbent powders, cementitious solutions, dry the area after washing, then apply the medicine). Check for the presence of associated onychomycosis or tinea pedis, which will require systemic antifungal medications. To prevent relapse, it is necessary to cure concomitant foot dermatophytosis and onychomycosis. In public baths, pools and at home, taking a shower (especially if other family members are sick), slippers should be used. Treatment of the feet and inguinal region with benzoyl peroxyde or with powder with antifungal agents.

Onychomycosis

Definition: Infection of the nail fold and nail plate with dermatophytes (Trichophyton rubrum, Trichophyton mentagrophytes, Epidermophyton floccosum), as well as mold (Hendersonula toruloidea, Scopulariopsis brevicaulis) and yeast (Candida albicans, other types of Candida). Applies only to dermatophytic nail infection. Covered under diseases of the nails.

Pathogenesis: Primary onychomycosis. Healthy nails are affected. The risk of onychomycosis increases with circulatory disorders (in the elderly, with heart failure, obliterating atherosclerosis), after fractures of the leg bones, with impaired innervation (damage to the brachial plexus, spinal injury). Secondary onychomycosis Affected nails are affected (injured, affected by psoriasis, etc.). Dermatophytosis of toenails usually occurs against the background of dermatophytosis of the feet; dermatophytosis of nails on the hands - against the background of dermatophytosis of the hands, body, scalp. The big toe and little toe suffer more often than others due to constant friction on the shoes.

Etiology: Dermatophytes. Anthropophilic: *Trichophyton rubrum, Trichophyton mentagrophytes* var. *interdigitale, Trichophyton violaceum, Trichophyton schoenleinii, Epidermophyton floccosum*. Zoophilic: *Trichophyton verrucosum* (usually affects the fingernails). *Yeast. Candida albicans, Candida tropicalis, Candida parapsilosis. Mold fungi*. More than 40 types of pathogens of onychomycosis are known. *Scopulariopsis brevicaulis* (a widespread inhabitant of the soil) is the most common causative agent of onychomycosis among yeast and mold fungi. It affects both healthy and diseased nails. Other pathogens are *Aspergillus spp., Alterna ria spp., Acremonium spp., Fusarium spp., Scytalidium dimidiatum* (outdated – *Hendersonula toruloidea*), *Scytalidium hyalinum*.

Onychomycosis caused by molds usually occurs on the legs. The clinical picture does not differ from dermatophytosis of nails, which causes diagnostic errors and treatment failures.

Dermatophytosis of nails. Men get sick more often. Candidiasis of nails and nail ridges. Women get sick more often.

Dermatophytes. Anthropophilic fungi: contact-household (through household items) and contact (especially among family members) ways of infection. Arthrospores remain viable up to 5 years.

Yeast. *Candida spp.* – representatives of human microflora. They cause infection with weakened immunity and in violation of the normal composition of microflora.

Mold fungi. They are found everywhere in the environment. Not transmitted from person to person.

Proximal subungual onychomycosis. In the past, it was quite rare; now - mainly in HIV-infected people. Toenails usually suffer. Damage to the fingernails arises as a complication of chronic paronychia.

Candidiasis of nails and nail ridges Frequent contact of hands with water. Right-handed people usually suffer from the right hand, left-handed people – the left; the nails of the thumb and middle finger are most often affected.

Toenails rarely suffer. Unlike other onychomycosis, candidiasis of nails and nail ridges is characterized by pain and pain when pressed. HIV-infected children are especially susceptible to the disease. Candidiasis of nails and nail ridges is often accompanied by candidiasis of the mucous membranes.

Chronic generalized granulomatous candidiasis In a child, candidiasis of the mucous membranes and candidiasis of paronychia may be the first manifestation of autoimmune polyglandular syndrome of type I (hereinafter, hypoparathyroidism, primary adrenal insufficiency and other disorders are added). In adults, chronic generalized granulomatous candidiasis is often combined with thymoma, myasthenia, myositis, aplastic anemia, neutropenia, hypogammaglobulinemia.

Distal-lateral subungual onychomycosis. A white spot with clear boundaries, adjacent to the free or lateral edge of the nail plate. The lesion is localized on the inner surface of the nail plate and on the nail bed. Over time, the stain becomes yellow, brown or black. The nail loses its transparency, thickens, rises (due to subungual hyperkeratosis) or separates from the nail bed, crackes and crumbles. This process takes months and years (for HIV-infected patients it is much less, only a few weeks). The clearly defined whitish stripes extending from the free edge of the nail in the proximal direction are filled with horn masses and air. According to the clinical picture, onychomycosis caused by dermatophytes are indistinguishable from onychomycosis caused by mold fungi.

Toenails suffer much more often than on toenails. Favorite localization - thumbs and little toes. With damage to the nails on the hands, only one arm usually suffers. With a combined lesion of the nails on the hands and feet, a typical localization of the lesion is one hand, both feet.

Proximal subungual onychomycosis. A white spot that appears from under the posterior nail roll. The spot gradually fills the hole and moves in the distal direction, capturing almost the entire inner surface of the nail plate. After a course of treatment with antifungal agents, spot growth stops. Toenails usually suffer. Candidiasis of nails and nail ridges. The disease begins with candida paronychia. Nail rollers painful, swollen, hyperemic.

Differential diagnosis Paronychia Candida paronychia, herpetic panaritium, diffuse neurodermatitis, allergic contact dermatitis, lichen planus. Distal-lateral subungual onychomycosis Nail psoriasis (oil slick symptoms and thimble for onychomycosis are uncharacteristic), diffuse neurodermatitis, Reiter's syndrome (blennorrhagic keratoderma), pseudomonadal onychia (pathogen Pseudo-monas aeruginosa, dark green toenail color), onychogryphosis, "nail-ticks", congenital onycho-dystrophy. White superficial onychomycosis. Injury of the nail.

Therapy: Topical antifungal agents usually inadequate; systemic therapy required.

Yeasts

Definition: Skin candidiasis is a superficial infection gravitating to moist areas of the skin. Sweating, diabetes mellitus, and immunity disorders are predisposed to the disease.

Synonyms: candidosis cutis, candidamycosis, yeast mycosis, moniliasis.

Pathogenesis: Almost all pathogenic human yeast belongs to the genus *Candida*. The main species *are Candida albicans* types 1 and 2 (formerly known as *Candida stellatoidea*). *Candida albicans* is part of the normal flora of the mouth, gastrointestinal tract, and vagina; it is present in limited quantities, growing and decreasing. Usually it is not on the skin or in the respiratory tract. Infections of the skin and mucous membranes are caused by candidal mycelia; systemic candidiasis usually caused by blastospores (budding yeast). The interaction of the host organism with *Candida albicans* is very complex and poorly understood; experts disagree on the importance of yeast in feces, for example. Some believe that gastrointestinal candidal colonization or a quantitative increase in yeast may be associated with diseases such as atopic dermatitis or urticaria. Treatment with nonabsorbable oral antiseptic agents such as nystatin sometimes gives an improvement, but we are still skeptical. Patients with very specific immune defects tend to have extremely persistent infections (mucocutaneous candidiasis).

Epidemiology: *Candida albicans* causes disease in young, elderly, or immunocompromised people; Exceptions are vulvovaginitis and balanitis, which affect patients with intact immunity. Over 90 % of infections are caused by *Candida albicans*, but other species cannot be ignored; often difficult to identify and usually more resistant to therapy. Candidiasis infections are often a sign of immunosuppression (diabetes, HIV/AIDS, prolonged antibiotic therapy, hematological malignancies). Patients with recurrent or refractory disease should be evaluated for the presence of such risk factors.

Clinical features: Candidiasis is an endogenous infection. *Candida albicans* is a representative of the normal microflora of the oropharynx and gastrointestinal tract. In healthy people, it does not live on the skin. Possible infection with candidi dose balanitis from a sexual partner.

Risk groups People who have constantly wet hands: housewives, young mothers, medical staff, bartenders, flower sellers, etc.

Risk factors. Diabetes mellitus and other endocrine diseases, obesity, sweating, hot climate, maceration of the skin, impaired immunity, treatment with corticosteroids, exhaustion.

Classification: Candidiasis of moist skin. It affects the skin folds and other closed areas of the skin – those where there is a warm moist microclimate necessary for the reproduction of the pathogen. *Intertriginous candidiasis:* Candidiasis of skin folds: axillary hollows, inguinal folds, intergluteal fold, folds under the mammary glands. There is redness, itching, weeping in the armpits, inguinal and intergluteal folds, under the mammary glands. Interdigital candidiasis: interdigital spaces of the feet and hands. There is itching, burning, erosion of the interdigital spaces of the feet or hands; frequent contact of hands with water. *Genital candidiasis:* Balanitis, balanoposthitis – irritation of the glans penis, pain, burning, discharge from the cavity of the foreskin. Vulvite – pain, burning, itching, rapid and painful urination. *"Dressing" candidiasis.* It occurs under

occlusive and plaster dressings or on the back in bedridden patients. Itching, redness, soaking under an occlusive or plaster dressing, in bedridden patients – on the skin of the back. *Candidiasis folliculitis.* Localization – the same as in the "dressing" candidiasis. Diaper dermatitis. Perineal dermatitis in infants. It can be both primary and secondary. *Candidiasis of nails and nail ridges.* Paronychia. Onychia. *Chronic generalized granulomatous candidiasis*. Continuous or recurrent infection of the skin, nails and mucous membranes caused by *Candida albicans.* It occurs in patients with congenital disorders of cellular immunity and endocrine diseases (hypothyroidism, hypo-parathyroidism, primary adrenal insufficiency, diabetes mellitus). It begins in the first 3 years of life. Usually, the oral cavity is first affected, then the scalp, trunk, hands, feet, fingertips and nails.

Differential diagnosis. Candidiasis of skin folds diaper rash, psoriasis of skin folds, erythema. Interdigital candidiasis Scabies. Balanoposthitis.Psoriasis, pruritic dermatitis. Diaper dermatitis .Diffuse neurodermatitis, psoriasis, simple contact dermatitis, seborrheic dermatitis. Candidiasis of nails and nail ridges Dermatophytosis of nails, staphylococcal paronychia, herpetic panaritium. Candidiasis folliculitis Staphylococcal folliculitis; pseudomonas folliculitis; folliculitis caused by fungi of the genus Pityrosporum; common acne.

Therapy: polyene antifungal drugs, such as nystatin, amphotericin B and natamycin, are old drugs, but are still effective; they form complexes with ergosterol in the plasma membrane and thus inhibit growth. Nystatin is not absorbed and thus often used to treat oral and intestinal infections. Imidazoles, both local and systemic, are also very effective. They are available as lozenges for diseases of the oral cavity and gastrointestinal tract, as well as for vaginal use. It is imperative to correct predisposing factors, from occlusion and maceration to weight loss, control of diabetes, or avoidance of long-term antibiotic usage.

Oral Candidiasis

Clinical features: various clinical forms that vary greatly in appearance:

Acute pseudomembranous candidiasis (thrush): the classic form known to everyone as a mother; thick, curd-like plaques that can be easily scraped off, exposing erythematous base. Most common in children. Common places include the mucous membrane of the cheeks, tongue, palate. *Acute atrophic candidiasis*: often painful, flat erythematous areas; usually includes the tongue and is secondary to the long-term use of antibiotics.

Chronic hyperplastic candidiasis: white plaques that cannot be removed; with prolonged treatment with antifungal agents, the plaques gradually resolve. *Chronic atrophic candidiasis:* most often affects dentures; atrophic dark erythematous region, limited to the area under the prosthesis; often confused with an allergic or irritating reaction. Objectively smooth red foci of epithelial atrophy.

Angular cheilitis: painful rags in the corner of the mouth; predisposing factors: salivation (infants and the elderly), eating disorders (malnutrition and forced vomiting), poorly fitting prostheses. Often co-infection with *Candida albicans* and bacteria. *Median rhomboid glossitis*: erythematous rhomboid patch

without papillae the middle line of the dorsal surface of the tongue when moving from middle to rear end; I thought for a long time to present the embryological defect of the merger,but now another form of candidiasis is being considered.

Differential diagnosis. Lichen planus, scleroatrophic lichen. Stomatitis, glossitis, pharyngitis. Thrush: hairy leukoplakia of the mouth, genital warts, geographical tongue, hairy black tongue, lichen planus, damage to the mucosa by the teeth.

Therapy: Nystatin or Imidazole for 5–10 days; easier to use than the direct use of drugs. In the case of angular stomatitis, protective imidazole pastes are useful. In resistant cases, consider 10 days of nystatin to reduce the burden on the intestines. Correct the predisposing factors.

Intertriginous Candidal Infections

Clinical features: any wet internode can be infected; examples include submammary, inguinal, perianal and sometimes axillary diseases, under the mammary glands, perineum. Lesions usually macerate with fissures and satellite lesions, often pustules, which open with the formation of erosion. Growth and fusion of erosion lead to the appearance of clearly defined eroded foci with scalloped edges. On the periphery of the foci - small pustules (daughter rashes). In addition, candida vulvovaginitis and balanitis are common. Most diaper dermatitis is annoying, but then quickly colonized by *Candida albicans*; satellite damage is a good key to this. The problem is worse when topical corticosteroids are used. Granuloma gluteale infantum refers to the reactive red-brown inflammatory nodes that develop in this setting.

Differential diagnosis: Candidiasis of skin folds diaper rash, psoriasis of skin folds, erythema.Interdigital candidiasis Scabies. Balanoposthitis.Psoriasis, pruritic dermatitis. Diaper dermatitis.Diffuse neurodermatitis, psoriasis, simple contact dermatitis, seborrheic dermatitis. Candidiasis of nails and nail ridges Dermatophytosis of nails, staphylococcal paronychia, herpes panaritium. Candidiasis folliculitis Staphylococcal folliculitis; pseudomonas folliculitis; folliculitis caused by fungi of the genus Pityrosporum; common acne.

Therapy and prevention: Skin folds must be kept clean and dry. Since this is not always possible, it is recommended to treat them daily with benzoyl peroxide and powder with miconazole usually enough drying. Dye solutions are effective and economical, but dirty. In Europe, solutions of methylrosaniline chloride and eosin still in use. Topical nystatin or imidazole products are also widely used. Oral nystatin is also safe. With vulvovaginitis, vaginal suppositories are usually severely effective; If not, or with a recurring disease, oral administration of imidazoles (usually fluconazole) is recommended.

Chronic mucocutaneous candidiasis

Definition: a heterogeneous group of congenital immune defects with severe and persistent candidal infections.

Clinical features: Classification of chronic mucocutaneous candidiasis (CMC) is indicated in patients with persistent skin, mucous and nail infections, usually with Candida albicans. Some forms are associated with endocrinological disorders.

The diagnostic approach. Persistence, age of occurrence and related symptoms usually lead to diagnosis; Accurate immunological assessment and, in some cases, genetic assessment.

Therapy: fluconazole has become standard therapy; usually 200–800 mg daily. Higher doses are required for the detection of Torulopsis glabrata. In persistent cases Itraconazole (200–400 mg per day) is promising.

Subcutaneous mycosis

Subcutaneous mycoses are not a biological family, but a group of clinical problems caused by direct inoculation of the body into the skin and subcutaneous tissue. They are more common in tropical and subtropical regions, presumably due to the increased growth of fungi in the soil and on plants and the increased likelihood of injuries (walking barefoot, less protective clothing).

Sporotrichosis

Definition: Sporotrichosis usually develops as a result of skin damage. An ulcerated node appears at the site of the pathogen introduction, then lymphangitis and regional lymphadenitis join. With weakened immunity, the infection spreads through the hematogenous pathway from the primary focus (skin or lung) and disseminated sporotrichosis occurs.

Synonyms: sporotrichosis, Schenck's disease.

E*pidemiology:* although Sporothrix schenckii is found worldwide, sporotrichosis is mainly observed in North and Central America; a famous epidemic among South African miners.

Pathogenesis: the fungus is vaccinated against injuries, usually from a tree or plants; classic pink thorn injury.

Clinical features: Localized sporotrichosis. After inoculation, violent papules and plaques develop. Then, solid subcutaneous nodules spread along the lymphatic drainage pathway. In the United States, diseases that spread in this way (nocardiosis, atypical mycobacterial infections, plaques, and many others) are called sporotrichoids. Superficial ulcerative sporotrichosis: in this form, many spores are inoculated by abrasion. The classic setting is a gardener or farmer carrying bales of sphagnum moss that are rubbed on his stomach. Systemic sporotrichosis: rare; includes lungs, muscles, bones, and even the central nervous system.

Localization. Most often - sporotrichous chancre on the back of the hand or on the finger in combination with lymphangitis of the forearm. Cutaneous sporotrichosis: in children – face, in adults – hands. Disseminated sporotrichosis: generalized rash, with the exception of the palms and soles.

Classification: Sporotrichous chancre. It is observed in 40 % of cases. At the site of injury after a few weeks, a papule, pustule or nodule appears that fuses with surrounding tissues and ulcerates. The surrounding skin turns pink or purple. A sporotrichous chancre is formed - a painless ulcer on a dense base,

with uneven saped edges. Enlargement and inflammation of the regional lymph nodes. *Lymphatic (sporotrichoid) sporotrichosis*. It is observed in 60 % of cases. It occurs due to the spread of infection from the primary focus (sporotrichous chancre) through the lymphatic vessels. Lymphangiitis: a dense thick cord with multiple nodes along the draining lymphatic vessel. *Cutaneous sporotrichosis.* Crusty ulcers; plaques with a warty surface; foci similar to ecthyma and gangrenous pyoderma; papules and plaques on an infiltrated base. *Disseminated sporotrichosis.* The infection spreads through the hematogenous route, affects the skin, joints, eyes, and meninges. Generalized rash: nodes and ulcers, covered with crusts.

Differential diagnosis. Sporotrichous chancre.Lupus; infections caused by atypical mycobacteria; tularemia; felinosis; primary syphilis.Diagnostic approach: sometimes with a biopsy you can see yeast in the form of cigars with periodic acid-Schiff (Pas) staining; Sporothrix schenckii is dimorphic; culture in 25 °C shows mold, and at 37 °C yeast.

Therapy: Itraconazole 400–600 mg daily for 6–8 weeks. With systemic involvement, amphotericin B is also possible. To accelerate healing, localized lesions can be treated with cryotherapy or hyperthermia.

Mycetoma

Definition. Chronic, slowly progressing local infection that affects the skin, subcutaneous tissue, fascia, muscles and bones. Most often, the feet and hands suffer. Swelling and an increase in the limb in volume, tumor-like growths, the formation of many fistulas with abundant purulent discharge are characteristic. In the exudate are drusen – yellow, white, red, brown or black grains containing pathogens.

Synonym: Madura Foote (from the Indian state of Madura), botryomycosis. Pathogens are bacteria.

Etiology and classification of *Pseudomycetoma:* The most common pathogen: *Staphylococcus aureus.* Other pathogens: *Staphylococcus epidermidis, Pseudomonas aeraginosa, Escherichia coli, Bacteroides spp., Proteus spp., Streptococcus spp. Actinomycetoma*. Pathogens - actinomycetes - bacteria of the order Actinomycetales (previously this order was attributed to the kingdom of fungi). *Actinomyces spp:* in addition to mycetomas, they cause cervical-maxillofacial, thoracic, abdominal actinomycosis. *Nocardia spp*: In addition to mycetomas, they cause superficial skin infections, lymphangiitis and disseminated nocardiosis. *Actinomadura spp., Streptomyces spp*. *Eumitsetoma*. Pathogens are real fungi. The most common pathogens: *Pseudallescheria boydii, Madurella grisea, Madurella mycetomatis.*

Pathogenesis: the list of pathogens is very long. All of them cause the disease in the same way - they are inoculated into the skin through trauma, and then multiply in the subcutaneous tissue, spreading to the fascia, muscles and bones. Two types: *Eumycetoma:* caused by fungi in the genera Aspergillus,

Exophiala, Madurella, Pseudallescheria and others. *Actinomycetoma*: Caused by bacteria in the genera Actinomadura, Actinomyces, Nocardia and Streptomyces.

Clinical features: Initial detection is swelling of the soft tissues, usually involving the foot. The process develops with the involvement of deeper structures, as well as with the formation of abscesses and draining sinuses with the release of colored granules (colonies of organisms). The primary focus is a papule or node at the site of introduction of the pathogen. The limb gradually swells and grows in size. The node ulcerates, pus containing drusen is secreted from it. Druze is an encapsulated colony of the pathogen of various sizes (they can be less than 1 mm or more than 5 mm in diameter). Around the fistulous opening, tumor-like growths form. The infection spreads to the underlying tissues – fascia, muscles, bones. The old mycetoma consists of scars and purulent fistulas. Limb deformity. Palpation. Usually painless. When pressed from the fistula, pus is secreted. Location The old foci are ring-shaped, since healing is taking place in the center. *Localization.* The defeat is one-sided. Shins, feet, hands. Occasionally – the trunk, forearms, head, hips, buttocks.

Diagnostic approach: granules have different colors (white, yellow, black) that give clues to organisms; microscopic examination and culture necessary to confirm the diagnosis.

Differential diagnosis: Chromomycosis with verrucous exophytic lesions, subcutaneous inflamed node Chromomycosis, North American blastomycosis, pyoderma, foreign body granuloma, dermatophytosis, leishmaniasis, gangrenous pyoderma.

Therapy: cultural antibiotic therapy or antifungal therapy, usually with amphotericin B. Bacterial forms are relatively sensitive to therapy; eumycetomas is often so persistent that amputation is the most sensible approach.

Systemic Mycoses

Pathogenesis: The systemicmycoses are caused by dimorphic fungi that live as a yeast or a mold, depending on environmental conditions. They often cause asymptomatic infections in healthy individuals, but are frequently more aggressive in weakened individuals. Risk factors include HIV/AIDS, cancer chemotherapy, solid organ transplantation, and long-term intensive care treatment. Depending on what organisms are locally common, a variety of prophylactic regimens are employed in high-risk patients.

Epidemiology: In Europe the two most common infections are cryptococcosis and aspergillosis. To avoid diagnostic errors, it is important to know where the various deep fungal infections are common.

Diagnostic approach: In all causes, histologic examination of skin lesions can reveal the causative organisms. Final confirmation is made via culture. Infectious diseases texts should be consulted for more advanced diagnostic techniques including serological studies.

Therapy: Systemic therapy is always required.

Cryptococcosis

Definition. This is a disseminated fungal infection that begins with lung damage. From there, the pathogen hematogenously enters the meninges, and in some patients into the skin and mucous membranes.

Synonyms: cryptococcosis, European blastomycosis, Busse-Bushke disease, torulosis.

Epidemiology: found worldwide; first described in Berlin, Germany.

*Etiology.*The causative agent is the yeast Cryptococcus neoformans, serotypes A, B, C, D. The tissues are represented by round yeast cells with a diameter of 3.5–7 microns, enclosed in a mucous capsule of various thicknesses. Propagated by budding; the daughter cell is connected to the maternal narrow jumper.

Pathogenesis: Cryptococcus neoformans is thought to spread from bird droppings. Primary infection through the lungs; Distribution is observed only in immunocompromised individuals.

Clinical features: skin fading is rare; they reflect the spread and indicate a critically ill patient. In HIV/AIDS, disseminated papules are very similar to molluscum contagiosum. Cryptococcal meningitis is the most dangerous complication. Diseases of the kidneys and bones are also common, but less catastrophic.

*Differential diagnosis*Pyoderma, molluscum contagiosum (rashes on the face of HIV-infected people), blastomycosis, histoplasmosis, phlegmon of a different etiology.

Therapy: The previous therapy basics are amphotericin B and flucytosine. Fluconazole is effective for meningitis; Voriconazole also shows promise.

Blastomycosis

Definition. This is a deep mycosis that begins with lung damage. Hematogenous dissemination of infection leads to damage to the skin and other organs.

Synonyms: blastomycosis Gilchrist, Gilchrist blastomycosis, Gilchrist blastomycosis.

Epidemiology: Blastomyces dermatitidis is a soil fungus found primarily in the central United States. The fungus exhibits dimorphism. In tissues, it is represented by yeast cells with a diameter of 10 μm with a thick (1 μm) wall. Propagated by budding; the daughter cell is connected to the maternal wide jumper.

Pathogenesis: The primary infection is pulmonary and is usually asymptomatic. Later, the body can cause pulmonary cavitation or spread, most often affecting the bones or prostate, as well as the skin. The causes of reactivation are poorly understood; blastomycosis is not a problem with HIV/AIDS.

Clinical features: Primary pulmonary infection. Erythema nodosum, polymorphic exudative erythema.

Disseminated infection. An inflamed node that grows in size and ulcerates. The subcutaneous node over which many small pustules form. In the future – plaques with a warty surface, covered with crusts, with clear winding borders.

If you lift the crust, pus comes out from under it. Peripheral growth in one direction leads to the fact that the lesion resembles a half or three quarters of the moon. Healing starts from the center; an atrophic scar resembling a geographical map is formed.The form. It is bizarre. *Localization*: Usually symmetrical damage to the body, less often – the face, hands, forearms. Half of the patients have multiple lesions. With contact infection, the focus is localized at the site of introduction of the pathogen. *Mucous.* In 25 % of patients, the mucous membranes of the mouth and nose are affected, in half of them the focus extends to the adjacent skin. The defeat of the larynx. *Lungs.* Infiltrates, caverns, multiple small foci. *Bones.* Affected in half of the patients. Osteomyelitis of the thoracic and lumbar vertebrae, sacrum, pelvic bones, skull, ribs, long tubular bones. It may be accompanied by the formation of fistulas and large subcutaneous abscesses. Purulent arthritis. Lymph nodes. Increase in regional limphatic nodes.

Differential diagnosis. Plaque with a warty surface Squamous cell carcinoma of the skin, gangrenous pyoderma, fungoid mycosis (tumor stage), ecthyma, warty tuberculosis of the skin, actinomycosis, nocardiosis, mycetoma, tertiary syphilis (gumma), donovanosis, leprosy, bromoderma.

Therapy: Itraconazole is usually the treatment choice; in severe cases, amphotericin B can be used.

Histoplasmosis

Definition. This is a deep mycosis, which begins with lung damage. Disseminated histoplasmosis occurs infrequently, while the pathogen hematogenously enters the mucous membranes, skin, liver, spleen and bone marrow.

Synonyms: histoplasmosis, Darling's disease, reticuloendothelial cytomycosis.

Epidemiology: Histoplasma capsulatum var. capsulatum is also most common in the central United States, but is also found in South America, Asia, Australia, and Africa. Histoplasma capsulatum var. Duboisia causes African histoplasmosis.

Pathogenesis: primary infection in the lungs. Reactivation occurs after many years, causing chronic lung disease. An aggressive course can occur in children or with HIV/AIDS with widespread involvement.

Classification: Pulmonary histoplasmosis. *Acute pulmonary histoplasmosis* (often asymptomatic). Elements of the rash are similar to erythema nodosum, a polymorphic of exudative erythema. *Chronic cavernous pulmonary histoplasmosis.* Other forms of pulmonary histoplasmosis. *Disseminated histoplasmosis*. Elements of the rash –red papules and nodes, keratinizing or necrotic. Hyperemic spots; folliculitis, sometimes pustules or acne eruptions; vegetative plaques; panniculitis; erythroderma. When the adrenal gland is affected, diffuse hyperpigmentation due to adrenal insufficiency. *Acute disseminated histoplasmosis. Chronic disseminated histoplasmosis*.

Clinical features: skin lesions rarely with disseminated disease; less than 10 % have papules or nodules after hematogenous culture. Oral infiltrates and

ulcers are more common. The main systemic problems are hepatosplenomegaly, bone marrow infiltrates with pancytopenia, endocarditis and meningitis. With African histoplasmosis, skin nodules and abscesses occur; they can be deleted.

Differential diagnosis: Disseminated histoplasmosis, miliary tuberculosis, leishmaniasis, coccidioidosis, cryptococcosis, lymphomas.

Therapy: Itraconazole or ketoconazole is used for non-life-threatening cases. In immunocompromised patients, initial monitoring should be performed with amphotericin B. In the case of HIV/AIDS, when infection is controlled, lifelong prophylaxis is provided with itraconazole.

Antifungal Agents

Griseofulvin

Mechanism of action: Griseofulvin is incorporated into newly synthesized keratin, so it must be taken for a long time – up to 18 months in the case of toe nails. Its half-life is around 24 hours; micronized forms are better absorbed and distributed. It interferes with microtubule formation.

Indications: Dermatophyte infections; griseofulvin is not effective against yeasts and molds. Organism should be cultured before starting therapy.

Contraindications: Liver disease, porphyria, LE, pregnancy.

Drug interactions: Griseofulvin is metabolized in the liver and interacts with many agents including: Impairs action of coumarin. Reduces effectiveness of oral contraceptives. Cross-reactions with penicillin possible.

Dosage: Griseofulvin is still the only agent approved for tinea capitis in children; it has been replaced in most of its other uses by the more effective imidazoles. Children 10 mg/kg daily; adults 500–100 mg p. o. daily. If no response, dose can be doubled after 2weeks.

Side effects: Hepatic toxicity, gastrointestinal bleeding, leukopenia, granulocytopenia. Check CBC before therapy and after 2–3weeks. Exanthems, urticaria, photosensitivity. May trigger acute intermittent porphyria or systemic lupus erythematosus.

Itraconazole

Mechanism of action: Inhibits cytochrome P450-dependent synthesis of ergosterol, a key component of fungal cell walls.

Indications: Effective against dermatophytes, molds, and many yeasts. Excellent against *Candida albicans* and *Candida krusei*; moderately effective against other *Candida* species. Cutaneous mycoses, including onychomycoses. Mycoses in HIV/AIDS. Mucocutaneous and systemic candidiasis. Recurrent vaginal candidiasis. Aspergillosis. Soft tissue mycotic infections.

Contraindications: Pregnancy; contraception until 4weeks after end of therapy.

Drug interactions: Inhibits cytochrome P450; many interactions; enhances coumarin, oral hypoglycemic agents, theophylline, and phenytoin.

Dosage: Cutaneous mycoses: 100–200 mg p. o. daily for 2–4weeks. *Onychomycosis:* Interval therapy; 200 mg p. o. b.i.d. for 7days; repeat in weeks 4 and 7. *Vaginal candidiasis:* 200 mg p. o. twice in one day. Longer-term, higher-dose therapy in HIV, soft tissue, and systemic mycoses.

Side effects: Only common effect is nausea.

Fluconazole

Mechanism of action: Inhibits cytochrome P450-dependent synthesis of ergosterol, a key component of fungal cell walls.

Indications: Effective against dermatophytes and yeasts; not molds. Effectiveness reduced against *Trichophyton mentagrophytes*, *Candida glabrata*, and *Candida guilliermondii*. Useful for candidiasis in almost all settings from acute vaginal to HIV/AIDS to chronic mucocutaneous candidiasis. Dermatophyte infections, including onychomycoses.

Contraindications: Severe liver disease, pregnancy and nursing; contraception untill 7days after completing therapy.

Drug interactions: Inhibits cytochrome P450; many interactions. Enhances coumarin, midazolam, oral hypoglycemic agents, phenytoin, tacrolimus, theophylline.

Dosage: Vaginal candidiasis: Single dose of 150 mg. *Systemic candidiasis:* 200–800 mg p. o. daily (depending on organism) in adults; can be used in children if no suitable alternative: 3–6 mg/kg p. o. daily. *Dermatophytes:* Adults: 50 mg p. o. daily. Children: 1–2 mg/kg p. o. daily; higher dose for zoophilic fungi. *Onychomycosis:* 150 mg weekly in single dose, for 3–6months (fingernails) or 6–12 months (toenails).

Side effects: Seizures, leukopenia, thrombocytopenia. *Hepatic injury* – monitor liver enzymes. Toxic epidermal necrolysis**;** be very cautious about continuing in patients developing an exanthema.

Terbinafine

Mechanism of action: Inhibits sterol biosynthesis by blocking squalene peroxidase causing accumulation of squalene and cell death.

Indications: Primarily dermatophytes.

Contraindications: Renal or hepatic disease.

Dosage: Cutaneous disease 250 mg daily p. o. for 2–4 weeks; onychomycosis 250 mg daily p. o. for 6–12 weeks or interval therapy.

Side effects: No common serious side effects; elevated liver enzymes, disturbed taste; rare toxic epidermal necrolysis.

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