Seborrhoeic dermatitis

Seborrheic dermatitis (SD) is a common **papulosquamous skin disease of** unknown etiology with a subacute to chronic course. It mainly affects the so-called seborrheic localizations, ie areas of the skin rich in the presence of sebaceous glands, specifically the facial area (nasolabial furrows, eyebrows, eyelids, forehead), hairy parts of the head, upper torso and intertriginous areas. It is characterized by peeling in greasy yellowish scales and redness of varying intensity. It affects various forms of newborns and infants, but also adults around the 4th decade, mostly male. Occurrence in the adult population is estimated at 3-5%. Topical antifungals and corticosteroids are mainly used in the treatment of SD.[1][2]

Etiopathogenesis

The etiopathogenesis is currently not fully elucidated. The following are considered predisposing factors:

- increased activity of sebaceous glands with overproduction of sebum - so-called seborrhea or status seborrhoicus;
- hormonal effects (especially the action of androgens);
- multiplication of lipophilic yeasts of the genus *Malassezia* (or *Pityrosporum sp.*), which form a common cutaneous microflora;[3][4][5]
- hyperhidrosis;
- external application of oil preparations, systemic application of corticosteroids or immunosuppressants;
- climatic factors (worsening of symptoms in the winter months, on the contrary, the remission of difficulties in the summer, in the mountains and at sea);
- endogenous effects: heredity (familial occurrence observed but the effect of heredity has not been confirmed)[6], obesity, Cushing's disease, diabetes mellitus, hypovitaminosis, Parkinson's disease, stroke, menopause, emotional stress and decreased cellular immunity;
- HIV positivity (frequent occurrence of severe forms of SD).[2]

Clinical forms

Dermatitis seborrhoica infantum (SD infants)

More than 2/3 of infants aged 3 weeks to 3 months are affected by seborrheic dermatitis. Thus, the symptoms start earlier than in atopic dermatitis.

Typical manifestations:

1. small yellow-brown scales on the pale pink base in the forehead (especially frontally and parietally);
2. cracked yellow-gray or yellow-brown firmly adhering deposit of greasy scales in the area of the large fountain - "cradle cap".[1]

It can spread from the forehead to the eyebrows and the middle part of the face in the form of sharply demarcated light red deposits with peeling fine oily yellow scales. Heavier forms also spread to intertriginous areas in the form of infiltrated non-wetting erythematosquamous deposits. Complications include the association yeast infection.[7][8][1] The more serious course is usually in pasty infants on artificial nutrition.[1]

Pathogenesis: increased activity of the sebaceous glands due to temporary high endogenous production of androgens by the adrenal cortex; altered ratios of unsaturated fatty acids; temporary changes in δ-6-desaturase enzyme function (conversion of linolenic and arachidonic acid to long chains of unsaturated fatty acids); immunological abnormalities, complement activation and colonization by lipophilic yeast organisms of the Malassezia type (Pityrosporum ovale). In infants with seborrheic dermatitis, Candida albicans is detected in the faeces and on the skin.[6]

In immunodeficient children, it may progress to erythrodermia (*erythrodermia desquamativa Leiner*) with marked lamellar peeling of the skin of the whole body, fever, diarrhea and vomiting with dehydration and metabolic acidosis. [6][2]

In childhood, the clinical manifestations of SD subsequently disappear or are only very mild, similar to adolescents.[1]

Tinea amiantacea (asbestina)

Tinea amiantacea (asbestina) is a form of SD in school children. It manifests itself in the form of bounded non-inflammatory, peeling deposits in the forehead - the scales overlap in roofs and resemble asbestos or dermatomycosis in appearance and color. [9][10][1]

Erythema paranasale

Erythema paranasale is a form of SD in adolescents. It is manifested by a sharply demarcated erythema with seborrheic scales in the nasolabial and nasophacial grooves. Very resistant to normal treatment. Long-term or repeated treatment with topical corticosteroids leads to perioral dermatitis. [1]

Dermatitis seborrhoica capitilii

náhled| Akutní forma seboroické dermatitidy *Dermatitis seborrhoica capilitii* is a form of SD, that often occurs in adolescents and adults. [1]

Pityriasis simplex capiliti

According to some sources, *Pityriasis simplex capiliti* or common dandruff is one of the most common forms of SD. However, according to other sources, it is a separate disease.[1]

Dermatitis seborrhoica adultorum (SD dospělých)

In its basic form, it affects seborrheic sites. The deposits here are sharply demarcated, yellowish pink, peeling greasy scales. The layer is layered in a larger deposit.

There are several characteristic forms: retroauricular, mediothoracica, intertriginosa, etc. Differential diagnostics must be distinguished: psoriasis, pityriasis rosea, mycosis fungoides and others.[6]

Treatment

Treatment is usually lengthy due to a chronically recurrent course. In the first 2 weeks of treatment, combined corticosteroids with antifungals can be used , followed by long-term monotherapy with topical antifungals.[1] Topically applied azole antifungals containing flutrimazole, etoconazole, econazole may be used.[6]

SD in infants from 6 months – salicylic oil (*Rp. Ac. salicylici 3,0; Ol. olivarum ad 100,0*). It is recommended to soak the bearings with scales twice a week at least 3 hours before bathing to soften the scales and then comb them with a soft brush and wash with shampoo. Infants and young children are at risk of percutaneous resorption of toxic amounts of salicylic acid.[1]

SD of younger infants – mechanical removal of scales by a gentle massage with fingers or a soft brush while washing the head with baby shampoo; or one hour before washing the head, apply mineral oil to the hair and wrap the brush with a warm wet cloth[11][2]; ; urea creams and lotions can also be applied (even daily).[1]

Mild forms of **SD in children and adolescents** – over-the-counter shampoos with antiseborrheic ingredients (tars, salicylic acid, organic sulfur compounds, antifungals, antimicrobials, zinc pyrithione, octopirox and others). The effect does not start until 6-8 weeks of use. More serious focal lesions of the hair area - shampoos with antiseborrheic additives + interval treatment with local corticosteroids (solutions, gels, lotions - often combined with salicylic acid).[1]

Intertriginous areas in infants - antifungals in pastes, soft zinc pastes. **Outside the intertriginous area** – a masterpiece of soft pastes or creams with corticoids and antibacterial substances (cloroxin - endiaron) or tar additives (tinctura carbonis detergens, ichtamol).[1]

References

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