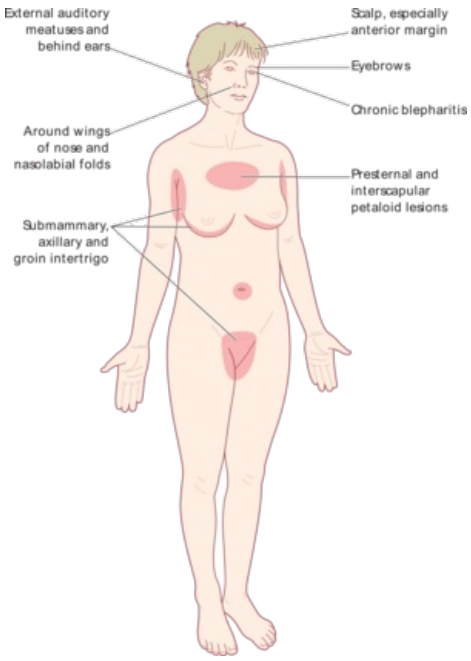
Seborrheic dermatitis

Seborrheic dermatitis (SD) is a **common papulosquamous skin disease** of unknown etiology with a subacute to chronic course. It mainly affects the so-called seborrhoeic localizations, i.e. skin areas rich in sebaceous glands, namely the facial area (nasolabial folds, eyebrows, eyelids, forehead), hairy parts of the head, upper part of the trunk and intertriginous areas. It is characterized by scaling in greasy yellowish scales and redness of varying intensity. It affects in various forms newborns and infants, but also adults around the 4th decade, mostly male. The prevalence in the adult population is estimated at 3-5%. In the treatment of SD, mainly topical antifungal agents and corticosteroids are used.[1][2]



Typical localizations of seborrheic dermatitis in adults

Etiopathogenesis

The etiopathogenesis is currently not fully elucidated. Predisposing factors are considered to be:

- increased activity of sebaceous glands with overproduction of sebum - so-called seborrhea or seborrhoeic status;
- hormonal influences (especially androgens);
- proliferation of lipophilic yeasts of the genus Malassezia (or Pityrosporum sp.), which form the normal skin microflora;[3][4][5]
- hyperhidrosis;
- external application of oil preparations, systemic application of corticosteroids or immunosuppressants;
- climatic factors (worsening of manifestations in the winter months, on the contrary, difficulties subside in the summer, in the mountains and by the sea);
- endogenous influences: heredity (familial occurrence observed, but the influence of heredity was not confirmed)[6], obesity, Cushing's disease, diabetes mellitus, hypovitaminosis B, Parkinson's disease, stroke, menopause, emotional stress and reduced cellular immunity;
- HIV positivity (frequent occurrence of severe forms of SD).

Clinical forms

Dermatitis seborrhoica infantum (SD of infants)

More than 2/3 of infants aged 3 weeks to 3 months are affected by seborrheic dermatitis. Manifestations therefore appear earlier than in atopic dermatitis.

Typical manifestations:

1. small yellow-brown scales on a pale pink base in the brush (especially frontal and parietal);
2. cracked, yellow-gray or yellow-brown, tightly adhering gooey scales in the area of the large fontanelle - "cradle cap".[1]

From the brush, it can spread to the eyebrows and the middle part of the face in the form of sharply defined light red deposits with peeling fine, oily yellow scales. More severe forms also spread to the intertriginous areas in the form of infiltrated non-wetting erythematosquamous foci. Complications include association with yeast infection.[7][8][1] The course is more severe in pasty-fed infants.

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

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

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

Tinea amiantacea (asbestos)

Tinea amiantacea (asbestina) is a form of SD in school children. It manifests itself in the form of limited, non-inflammatory, scaly deposits in the scalp - the scales overlap in a roof-like manner and resemble asbestos or dermatomycosis in appearance and color.[9][10][1]

Erythema paranasale

Erythema paranasale is a form of SD in adolescents. It manifests as sharply demarcated erythema with seborrheic scales in the nasolabial and nasofacial folds. Very resistant to conventional treatment. Long-term or repeated treatment with local corticosteroids leads to perioral dermatitis.[1]

Seborrheic dermatitis capitilii

Dermatitis seborrhoica capilitii is a form of SD that often appears in teenagers and adults.[1]

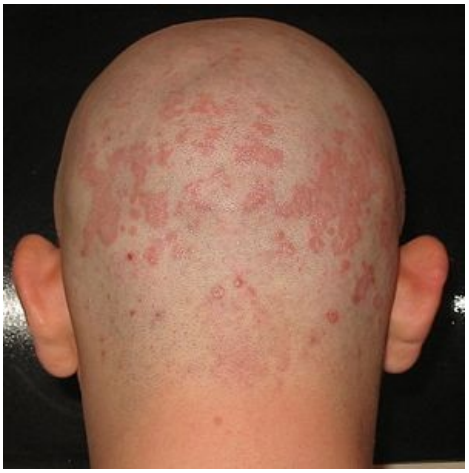
Pityriasis simplex capilitis

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

Dermatitis seborrhoica adultorum (Adult SD)

In its basic form, it affects seborrheic areas. Here, sharply demarcated, yellowish-pink, flaking oily scales form deposits. In the box it is layered into a larger deposit.

Several characteristic forms are distinguished: *retroauricularis, mediothoracica, intertriginosa* etc. In terms of differential diagnosis, it is necessary to differentiate: psoriasis, pityriasis rosea, mycosis fungoides and others.[6]



Acute form of seborrheic dermatitis

Treatment

Treatment tends to be lengthy due to the chronically relapsing course. In the first 2 weeks of treatment, '*combined preparations of corticoids with antifungals* can be used, followed by long-term monotherapy with local antifungals.[1] Locally applied azole antifungals containing flutrimazole, etoconazole, econazole can be used.[6]

SD in the chest of infants from 6 months' - salicylic oil (*Rp. Ac. salicylici 3.0; Ol. olivarum ad 100.0*). It is recommended 2 times a week to soak the deposits with scales at least 3 hours before bathing to soften the scales and then comb with a fine brush and wash with shampoo. There is a risk of percutaneous resorption of toxic amounts of salicylic acid in infants and small children.[1]

SD of younger infants - mechanical removal of scales by 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 hair with a warm wet cloth[11][2]; creams and lotions with urea can also be applied (even daily).[1]

Mild forms of *SD in the hair of 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 begins only after 6-8 weeks of use. More serious lesions in the hair area - shampoos with antiseborrheic ingredients + intermittent treatment with local corticosteroids (solutions, gels, lotions - often combined with salicylic acid).[1]

Intertriginous areas in infants - antifungal pastes, soft zinc pastes. **Outside the intertriginous area** - a magistraliter of soft pastes or creams with corticoids and antibacterial substances (chloroxin - endiaron) or tar additives (tinctura carbonis detergent, ichtamol).[1]

Links

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