There are many disorders of the sebaceous glands. Acne is the most relevant condition in the field of dermatology that is tested in medical examinations. Acne vulgaris is also considered 1 of the most common skin diseases. The clinical picture of these conditions as well their treatment is covered in this article.

Overview of Sebaceous Glands

Sebaceous glands (Lat. *glandula sebacea*) are microscopic exocrine glands on the skin that secrete sebum. They are found in almost all areas of the body except the palms and sole of the feet. Rich in sebaceous glands are the head, face, back and the upper body. The produced tallow, also referred to as sebum, consists of triglycerides, fatty acids, and wax esters. Androgen hormones stimulate the production of sebum from these glands.

Nevus Sebaceous Glands (Nevus Sebaceous)

The nevus sebaceous gland is also called organoid nevus and consists of sebaceous glands, ectopic glands, and malformed hair follicle residues.
Epidemiology

Nevus sebaceous occurs at an early age but is usually inherited. They have no sexual or racial predilection.

Clinical features of nevus sebaceous

It presents as a flat, white-yellowish, waxy tumor without hair and most frequently occurs in the hair area where it may develop alopecia. Sebaceous glands nevus occurs mostly solitary on the face and neck. In young children, maternal hormones may lead to an increase in growth while in the pubertal age the hormones lead to a verrucous appearance.

In old age, the nevus may evolve into a benign or malignant adnexal tumor. The most common one is trichoblastoma.

The therapy of nevus sebaceous

Ablative techniques i.e. cryotherapy is a proven treatment method. Here, low temperatures are usually used locally, to destroy pathological tissue.

Medical therapy i.e. photodynamic therapy uses topical aminolevulinic acid.

Surgical therapy i.e. excision of a nevus sebaceous can be performed for cosmetic reasons or even to avoid complications such as the development of tumors thus the risk of malignancy must be assessed. Adolescent tumors are almost always excised as most have a verrucous appearance that may suggest malignancy.

Sebostasis

Sebostasis means a reduction in the activity of sebaceous glands. The result is dry skin (xerosis cutis) and brittle hair.

The etiology of sebostasis

Sebostasis is age-related and has an occurrence of about 80% in older people. Mechanical or chemical degreasing such as frequent washing or very dry hair can also cause sebostasis.
Clinic of sebostasis

The clinical symptoms show a scaly, rough skin and the patient often suffers from itching and an unpleasant tightness of the skin.

Dry skin often leads to barrier disorder. Therefore, secondary inflammation and superinfection by bacteria may result. Consequently, the skin cracks and forms an eczema craquele, also named drying eczema or asteatotic eczema.

Typical symptoms of sebostasis are brittle and scruffy hair.

Treatment of sebostasis

Consistent anti-sebostatic cleaning serving as prophylactic treatment is recommended. It is necessary to maintain the skin regularly with fatty externa, special fatty acids, and urea.

The patient should shower, if possible, only briefly under warm, but not hot water. Alcoholic lotions must be avoided, as they cause the skin to dry out.

Seborrhea

Seborrhea means, ‘tallow flow’ and refers to a strongly increased sebum production, which leads to oily skin and greasy lank hair. Seborrhea is often a predisposition for acne, rosacea, and seborrhoeic eczema.

Etiology of seborrhea

In 80% of cases, seborrhea occurs at adolescence. This suggests that hormonal changes play a decisive role. An increase in androgen production and a higher density of androgen receptors are responsible for sebum production. Thus, hormonal medicines, such as anabolic steroids or contraceptives, can affect the sebaceous glands.

Systemic diseases such as Parkinson’s disease, acromegaly or polycystic ovarian
syndrome go hand in hand with seborrhea.

**Clinical presentation of seborrhea**

A greasy shine of the face, scalp and hair are the consequence of increased sebum production. Partially large pores are visible.

An increase in lipid secretion favors the conditions for *Propionibacterium acnes* and yeast fungi (e.g., *Malassezia furfur*) to propagate.

**Treatment of seborrhea**

A prophylactic anti-seborrheic skincare is recommended. For cleaning and care, disinfectants (e.g., benzoyl peroxide) and soap-free synthetic detergents, as well as low-fat externa (lotions and gels) can be used.

Topical and systemic retinoids are anti-inflammatory and against abnormal keratinization. In severe cases of acne the use of oral retinoids, such as Isoretinoid, are proven to be highly effective.

**Note:** Retinoids are teratogenic and contraindicated in pregnancy. Women can use systemic anti-androgens against seborrhea. Topical application of estradiol for men can lead to systemic side effects.

**Acne vulgaris**

Acne is a common chronic inflammatory disease of the pilosebaceous unit (sebaceous glands and hair follicles). Acne is characterized, depending on the shape and characteristics, by comedones, pustules or papules. Particularly affected are the areas rich in sebaceous glands such as the face, scalp, as well as back and chest.

Vulgaris means common. Thus, acne vulgaris refers to the most common form of acne to differentiate it from other forms of acne. Frequently, acne vulgaris occurs over the age of 12.

**Note:** 65% of young people suffer from acne!
The etiology of acne vulgaris

In most cases, acne vulgaris appears with the beginning of puberty. By the end of the 3rd decade of life, skin disease usually ends by itself. Boys and girls are affected to the same extent. Typical for girls is a deterioration of the condition before and during menstruation.

**Note:** For young people, the disease can be, psychologically, very stressful. Acne often leads to a feeling of distortion. The typical comedones (“blackheads”) are the result of a faulty tallow drain in the closure of the follicle excretory ducts.

Androgens enlarge sebaceous glands and increase their activity. This may, as already described above, lead to seborrhea.

The increase of sebum production and thus, an increase in bacteria such as *P. acnes*, results in a bacterial superinfection. The bacteria split the fat into free fatty acids, which induce the secretion of pro-inflammatory mediators and release an inflammatory response.

Nicotine abuse leads to phospholipase-induced inflammation.

**Clinical presentation of acne vulgaris**

The clinical picture of acne vulgaris shows in particular comedones, papules, and pustules and in severe forms of acne, the formation of scars.

**Acne vulgaris can be divided into 3 stages**

1. **Non-flammable stage**

Here we find *acne comedonica*, which appears centrofacial and is characterized by
open (blackhead) and closed (whitehead) comedones.

2. **Flammable stage**

The clinical picture of **acne papulopustulosa** includes pustules and papules. Often affected is the face, back, and upper torso. **Acne nodosa** forms up to 1 cm large knots that decline very slowly. The next stage is either acne conglobata or a transition to the defect stage. **Acne conglobata** is the most severe form of inflammatory acne and appears as deep, inflammatory nodes in the face, chest, and back. This form is especially common in young men.

3. **Defect stage**

Acne comedonica and papulopustulosa usually heal without scarring. In the defect stage, scars, cysts and abscessed fistula gears are typically noted.

### Treatment of acne vulgaris

To prevent exacerbation of chronic acne and scar formation a stage-related treatment of acne vulgaris is necessary. The following forms of therapy are to be considered:

- **Topical therapy**: Used in cases of papulopustulosa or acne comedonica; benzoyl peroxide (BPO) 5–10% can be used as a washing suspension to clean the skin.

  Topical retinoids, such as adapalene and Isotretinoin are effective. However, retinoids work well with teratogens. Therefore, special clarification for women within childbearing age is observed. Oral contraception – 1 month before the treatment and up to 1 month after treatment – plus additional contraceptive methods (condoms) are necessary. Clindamycin alone or in combination with benzoyl peroxide, as well as topical antibiotics, can be used.

- **Systemic therapy**: Used against pustulosa or acne conglobata. Systemic treatment methods such as tetracyclines, hormones (contraceptives with anti-androgenic effect) and retinoids are considered.

- **Peelings etc.**: E.g., peelings with fruit acids can have supportive effects. Also, treatment with dermabrasion or hyaluronic acid injection can reduce acne scars.

### Special forms of acne

- Acne neonatorum: is already present at birth or occurs in the 1st weeks of life. The sebaceous glands are stimulated by maternal androgens.
- Acne infantum: is typical in the 3rd–6th month of life by the influence of testosterone.
- Acne exocrine: is a surface ulceration caused by constant manipulation and excoriation
- Acne tarda: is acne, which occurs after puberty through androgen effects. Hormonal disorders must be excluded.
- Acne venenata: might develop through contact with tar, oils, halogens (chlorine) or cosmetics

**Acne aestivalis** (mallorca acne): arise in a warm, humid climate in combination with the application of oily sun creams
Rosacea is a chronic inflammatory, non-infectious disease of the sebaceous glands and connective tissues. It has a relapsing course in the central facial area. In contrast to acne, rosacea forms no comedones.

Etiology of rosacea

So far the etiology of this disease is largely unexplained. But there are certain known factors which seem to deteriorate rosacea. Ultra-violet (UV) exposure, hot drinks, alcohol, and spicy food, as well as hormonal influences or temperature changes, can affect disease activity.

Clinical presentation of rosacea

- Stage 1: Rosacea is characterized by a flat redness on the face – the flush. In addition, telangiectasia and persistent erythema are present.
- Stage 2: Facial pustules and papules, partially exceed the forehead-hairline.
- Stage 3: The rhinophyma is a tuberous hypertrophy of sebaceous glands in the nose.

Diagnosis of rosacea

For the diagnosis of rosacea, knowing the typical clinical picture is crucial. Ophthalmorosacea occurs in up to 50% of cases and should, therefore, be excluded.

Treatment of rosacea

Particularly relevant for the treatment of rosacea is the avoidance of aggravating factors.

Stage 1 is treated with 0.75% metronidazole-containing externa. Stage 2 combines the therapy of the 1st stage with a systemic antibiosis (doxycycline) or retinoids (low dose) over the course of several weeks. Rhinophyma can be treated using operational or laser surgical resection.
Perioral dermatitis

Perioral dermatitis is an inflammatory skin disease that presents with facial rash and bumps around the mouth. It appears in perioral and periocular regions due to excessive skin care. This dermatosis mostly affects young women who represent 90% of the cases but it can also affect older women. In some cases, the lesions may develop around the ears, eyes, or nose.

Etiology of perioral dermatitis

The etiology of this disease is still unclear. Factors that can trigger perioral dermatitis are excessive cosmetic care, the use of external corticosteroids, fluoridated toothpaste, soaps, and being exposed to stressful situations and hormonal disturbances.

The result of excessive care could lead to swelling of the horny layer. This disrupts the skin barrier and creates room for superinfections. Topical glucocorticoids often reinforce this effect.

Clinical presentation of perioral dermatitis

Small bound follicular papules and pustules appear around the mouth and eyes. Burning and the feeling of skin tension are also typical symptoms.

Perioral dermatitis therapy

The treatment of perioral dermatitis is “zero-therapy.” This means that the patient should avoid all external causes of the condition. Initially, it usually progresses to a deteriorated condition in the 1st 2 weeks. For patients with the habit of using cosmetics, zero therapy is often very difficult to achieve. The patient should be informed about probable deterioration.

Oral or topical antibiotics may be considered for very severe cases with superinfection.

References


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