1. Acne vulgaris

**Outline**

- It most frequently occurs on the face of adolescent men and women. Comedones, folliculitis, papules and pustules are produced.
- Agents and factors such as *Propionibacterium acnes*, the *Demodex folliculorum* mite, endocrine secretion and stress are associated with the occurrence.
- Lifestyle guidance and administration of sulfa drugs and antibiotics are effective.

**Clinical features**

Multiple follicular inflammatory papules occur, most commonly on the seborrheic areas of the face, precordial region, and back of men and women from the ages of about 10 to 40 (Fig. 19.5). The papules worsen at puberty. The initial eruption, called a comedo, is classified as an open comedo (the follicles are open, also called “blackheads”) or a closed comedo (small yellowish-white nodules in the skin: “white heads”). Closed comedos often progress to erythematous papules or pustules. Subjective symptoms such as itching are not usually present; however, the comedos become painful as they develop further. Acne vulgaris is characterized by intermingled eruptions of different stages.

**Pathogenesis**

The main pathogenesis involves hormonal imbalance, abnormal keratinization and bacterial infection (Fig. 19.6). Along with...

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*Fig. 19.5 Acne vulgaris.*
Multiple, follicular, inflammatory papules occur on oily areas of the face, such as the cheeks and forehead. The hair follicles are obstructed and appear as yellowish-white nodules (closed comedos).

*Fig. 19.6 Mechanism of acne vulgaris.*
these main factors, hereditary factors, the patient’s age, diet, stress and extrinsic factors such as cosmetics are complicatedly associated with the onset. Acne caused by *Demodex folliculorum* is called acne demodecica, and it occurs most frequently in women after puberty.

**Hormonal imbalance:** Androgen in the blood increases according to pubertal endocrine changes, and the function of the sebaceous glands is enhanced by adrenogenic dihydrotestosterone (DHT). Accordingly, sebum retention and bacterial proliferation readily occur.

**Follicular hyperkeratinization:** The infundibulum is obstructed by keratin as a result of poor hygiene or hereditary factors. When sebum components are decomposed by bacteria, free fatty acid is produced; stimulated by this phenomenon, the infundibulum induces keratinization. Sebum retention is accelerated by these causative factors to produce an initial comedo.

**Bacterial infection:** *Propionibacterium acnes* resident in the infundibulum break down triglycerides in the sebum, producing free fatty acids that destroy the hair follicles and lead to inflammation. The bacteria themselves also cause destruction of follicles and inflammation, especially when there is infestation by the *Demodex folliculorum* mite.

**Pathology**

Acne vulgaris is characterized by enlargement of the sebaceous glands and follicular keratinization. Cystic dilation occurs in follicles, and destruction of the follicular walls causes inflammatory reaction.

**Differential diagnosis**

Acne vulgaris must be differentiated from steroid acne, a side effect of oral or topical steroid use, and from lupus miliaris disseminatus faciei, and verruca plana. Acne-like eruptions are caused by immunosuppressants in the same mechanism as that in steroid acne; these eruptions must also be differentiated from acne vulgaris. Thorough history-taking is important.

**Treatment**

Lifestyle guidance is primary. Observance of a regular schedule of sleeping and eating, and avoidance of cosmetics (oily creams and foundation, in particular) are helpful. Washing the face and maintaining regular bowel movements are important. Topical application of retinoids (tretinoin or adapalene cream), sulfa drugs, and antibiotic ointments and oral antibiotics such as tetracyclines and roxithromycin are effective. Chemical peeling or extraction of comedos may be useful in some cases. Unless treated appropriately, acne vulgaris heals with scarring, leaving a cosmetic problem.
Rosacea is a chronic inflammatory disease that causes diffuse reddening and vascular dilation on the face of middle-aged and older men and women. Acne-like papules and pustules may be produced.

**Clinical features**

Rosacea is classified by severity into three stages. The first stage (rosacea erythematosa) and second stage (acne rosacea) occur frequently in middle-aged and older women. Progression to the third stage (rhinophyma) is more common in men. In addition to the cutaneous symptoms listed below, eye symptoms (e.g., keratitis and conjunctivitis) may occur.

1. **First stage (rosacea erythematosa):** Transient reddening appears on the tip of the nose and on the cheeks, glabella and chin. It progresses gradually to become persistent and accompanied by telangiectasia and seborrhea (Fig. 19.7). Cold and warm weather, sunlight and alcohol consumption aggravate it. Subjective symptoms such as itching, hot flashes and irritability are present.

2. **Second stage (acne rosacea):** In addition to the symptoms of the first stage, follicular papules and pustules occur. Seborrhea is intense (Fig. 19.8). The lesions spread to cover the face.

3. **Third stage (rhinophyma):** The papules aggregate and coalesce to become tumorous. The surface of the nose becomes rough and reddish purple. The skin appears orange-peel-like with open follicles (Fig. 19.9). Rosacea keratitis, conjunctivitis, and blepharitis occur as complications.

**Pathogenesis**

Involvement of sunlight, mental stress, intake of alcohol or spicy food, liver dysfunction, and infection by *Demodex folliculorum* is suspected; however, the pathogenesis is unknown.

**Treatment, Prognosis**

Rosacea progresses gradually and tends to be intractable. Spicy foods, excessive sun exposure, and stress should be avoided. Laser irradiation is performed on the telangiectasia. The treatments for acne rosacea are the same as those for acne vulgaris. Topical metronidazole, imidazoles and tretinoin may bring improvement. Steroid should never be used. Laser therapy, cryotherapy and surgical treatment are conducted for rhinophyma.

**2. Rosacea**

**Definition, Pathogenesis**

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**3. Rosacea-like dermatitis**

**Synonym:** Steroid-induced dermatitis

**Outline**

- Prolonged application of steroids to the faces induces
rosacea-like erythematous papules, diffuse flushing, and acne.

- Perioral dermatitis is also known to be caused by topical steroid therapy.
- After discontinuation of steroids, the treatments for acne vulgaris are given.

**Clinical features**

Erythema, telangiectasia, papules, pustules, diffuse flushing, and scaling occur on sites where steroids have been applied. These symptoms are accompanied by itching and burning sensation (Figs. 19.10-1 and 19.10-2). Localized rosacea-like dermatitis around the mouth is called perioral dermatitis.

**Pathogenesis**

Rosacea-like dermatitis most commonly occurs in middle-aged women. Rosacea-like dermatitis is a typical side effect of topical steroids.

**Treatment**

Steroids are immediately discontinued. After that discontinuation, rebound phenomenon occurs. Reddening and swelling aggravate, and erosion may persist for several weeks to several months. The same treatments as for acne vulgaris are performed. Topical steroids are tapered off only when rebound is severe.

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**4. Lupus miliaris disseminatus faciei (LMDF)**

**Synonym:** Acne agminata

**Outline**

- Multiple small papules of 2 mm to 5 mm in diameter the color of normal skin or redder occur on the face, particularly the lower eyelids. They are asymptomatic.
- The histology is epithelioid cell granuloma with central necrosis.
- Tetracyclines are administrated in small doses.

**Clinical features**

Lupus miliaris disseminatus faciei (LMDF) occurs in both sexes equally, most patients being in their 20s and 30s. Multiple small papules with central necrosis of 2 mm to 5 mm in diameter the color of normal skin or redder occur symmetrically on the face, especially on the lower eyelids, cheeks and sides of the nose, accompanied by pustules (Figs. 19.11-1 and 19.11-2). The disorder is asymptomatic. Small yellowish-white nodules are observed by diascopy. These heal with concave scarring one to several years after onset. The scars become indistinct in about 1 year.

**Pathogenesis**

LMDF is first thought to be a form of tuberculid, but an
association with tuberculosis has been excluded. Tuberculin reaction test is negative in most cases. The mechanism is predominantly thought to be reaction against the hair follicle tissues or their contents.

**Pathology**

A biopsy from a well established lesion shows that epithelioid granuloma with central necrosis is present.

**Differential diagnosis**

Syringoma, milium, rosacea and acne vulgaris should be differentiated from LMDF.

**Treatment**

Tetracyclines are administered in small doses. Topical steroids may be the inductive factor in some cases. After a period of months or up to 2 years, the condition resolves spontaneously.

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**5. Xerosis, Asteatosis**

Dehydration of the horny cell layer and decrease of sebum cutaneum lead to dryness and coarseness of skin, resulting in pityrioid scaling. These symptoms tend to aggravate during the winter. Xerosis is often caused by excessive washing and rubbing during bathing. It may be observed as a change in the aging process. It may also be caused by specific climates and environments. It appears as a symptom of nutritional deficiency or atopic dermatitis in some cases. It may progress to pruritus, nummular eczema or astreatic eczema (Chapter 7).

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**C. Disorders of the hair**

**1. Alopecia areata**

**Outline**

- Round, sharply margined hair loss suddenly occurs.
- Hair regrows spontaneously in several months in most cases. Cases with multiple alopecia areata may progress to alopecia totalis or alopecia universalis.
- Topical steroids and PUVA are applied.

**Clinical features**

Alopecia areata is quite common, affecting up to 1% of the population. Sharply margined hair loss occurs suddenly without prodromes or subjective symptoms (Figs. 19.12-1 and 19.12-2). Alopecia areata is usually a round or oval, single but sometimes multiple, alopecia of 2 cm to 3 cm in diameter. The alopecia patches may coalesce, progressing to complete scalp hair loss (alopecia