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 pityroïd 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 asteatotic 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

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Fig. 19.11-2 Lupus miliaris disseminatus faciei.

Fig. 19.12-1 Alopecia areata.

Sharply demarcated hair loss occurs. In active alopecia areata, the hairs around the lesion easily come out.
Besides occurring in the scalp, alopecia areata may occur in the eyebrows, beard areas and the extremities; cases in which hair on the whole body is affected are called alopecia universalis, which is intractable. In nails, desquamation, coarseness, cloudiness and slight depression occur.

**Pathogenesis**

Hair matrix cells are impaired temporarily for unknown reason. Theories include nutritional failure, heredity and mental stress; however, the pathogenesis is unknown. Some cases are accompanied by autoimmune thyroid deficiency and atopic dermatitis. Autoimmune involvement is suspected.

**Pathology**

In the lesion, there is infiltration of CD4+ T cells and the appearance of Langerhans cells in the hair follicles at the anagen stage. Expression of MHC class II in hair bulb epitheliocytes, and deposition of C3, IgG and IgM in the hair follicular basement membrane are observed. There is possible involvement of autoimmunity. The affected hair follicles form abnormal atrophic hair that falls out.

**Diagnosis**

Alopecia areata is easily diagnosed by the clinical features. The hairs around the lesion easily fall out at the early stages of the lesion. The hairs are characteristically thin and atrophic at the end of the hair root, giving them the appearance of exclamation marks (“exclamation-point hair”). The hairs stop falling out and newly grown hairs are seen during the healing period.

**Differential diagnosis**

Trichotillomania and traumatic alopecia are distinguished from alopecia areata. Trichotillomania, which produces short, breakable, hard hair in the lesion, occurs most commonly in children; however, there is no diseased hair in trichotillomania, and the hair around the lesion does not come out easily. In traumatic alopecia, the lesion is not round, and it is caused by extrinsic factors such as scarring. Fibrosis and pigmentation are also found. Alopecia areata also should be distinguished from systemic lupus erythematosus (SLE) and alopecia caused by syphilis.

**Treatment**

Alopecia areata resolves spontaneously in several months, although in some cases it may be intractable or recurrent. It is important to address the patient’s distress about hair loss. Sedatives may be used if necessary. Steroids, immunosuppressants and hair-growth lotions are topically applied. In severe cases, PUVA therapy, steroid injection, cryotherapy, and application of squaric acid dibutylester (SADBE) are performed. Steroids and immunosuppressants are administered orally in alopecia totalis or universalis.
**2. Androgenetic alopecia**

**Synonyms:** Male-pattern baldness, Alopecia prematura

**Clinical features**

Androgenetic alopecia also called male pattern baldness, is hair loss in adolescent and adult men. Androgenetic alopecia is a very common disorder, affecting at least 50% of men by the age of 50. The hairline recedes to form an M shape (with vellus hair at the frontal region of the head) or an O shape (with vellus hair on the top of the head). These patterns may appear separately or simultaneously. The diameter of the vellus hair is smaller than that of normal hair. The density (hairs per unit area), also decreases. It progresses to complete hair loss.

**Pathogenesis**

Patients usually have a familial history of baldness. Elevated sensitivity of hair follicles to androgen (dihydrotestosterone, in particular) begins at some point. The anagen period is shortened, hairs at telogen decrease in number, hair follicles contract, and vellus transformation occurs. The thin, sparse vellus hair produced in androgenetic alopecia becomes less densely distributed, eventually progressing to alopecia.

**Treatment**

Topical minoxidil and anti-androgenetic drugs such as 5α-reductase inhibitor fenasteride are effective in some cases. Stimulating the affected site, stimulating the local circulation of the scalp by massaging, and using hair growth lotions containing female hormones are helpful.

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**3. Congenital alopecia**

Congenital atrichia, alopecia and oligotrichia are observed in various conditions, including those listed below.

① **Atrichia congenita**

It is autosomal recessive. Hair may be present at birth; however, it falls out between several months after birth and puberty, until no hair remains on the body. Involvement of the hairless (hr) gene has been identified as a cause in some cases of certain subtypes.

② **Hypotrichosis congenita**

Normal hair is present at birth; however, alopecia gradually leads to thin, sparse hair (**Fig. 19.14**).

③ **Arrichia and alopecia associated with hereditary syndrome**

Arrichia and congenital alopecia are associated with congenital ectodermal defect (aplasia cutis congenita (**Fig. 19.15**)), dermatothlasia, Werner’s syndrome, poikiloderma congenital and Netherton syndrome. Odontogenesis imperfecta, abnormal nail plates, palmoplantar keratosis and anhidrosis often occur as complications.
4. Alopecia pityrodes

Pityriasis capitis (“dandruff”) occurs in combination with alopecia most frequently in men after puberty. Fine, dispersed, grayish-white scaling occurs constantly on the scalp. The hair is thin and the natural gloss is not present. Itching and reddening of the scalp often occur. The treatments are the same as for seborrheic dermatitis.

5. Trichotillomania

Patients with trichotillomania, who tend to be in their late childhood, have an uncontrollable compulsion to pull out their own hair. The patients may deny this hair-pulling behavior. Vaguely circumscribed, irregular-shaped, incomplete alopecia is present. Both short and broken remaining hairs and newly produced hairs are observed in the same alopecia, which is within reach of the hand, often on the frontal and temporal region of the head on the right side. The patient’s psychological background, personality and domestic environment may trigger trichotillomania; cooperation with a psychiatrist is necessary for treatment.

6. Scarring alopecia

As a result of scarring caused by injury, burn, or discoid lupus erythematosus. The hair follicles are irreversibly destroyed, leading to alopecia. Surgical treatment is necessary.

D. Disorders of nails

a. Color change of nail plates

1. Melanonychia

Melanonychia may be caused by increased number of nail matrix melanocytes (e.g., from nevocellular nevus, inflammation, melanocytes activated by pressure), fungal infections, malignant melanoma, Behçet’s disease, subungual hemorrhage, Addison’s disease or drugs (e.g., 5-FU, bleomycin, hydroxyurea). When the skin of the nail fold region is also affected, it is called Hutchinson’s sign and has a high likelihood of indicating a malignant melanoma (Fig. 19.16).

Approximately 20% of ethnic Japanese and up to 96% of ethnic Africans have pigmented streaks.

2. Yellow nail

It is caused by nutritional deficiency or infection of nails, or by aurantiasis cutis, or jaundice. When yellowing of the nails occurs...