the eruptions from becoming aggravated by rubbing and scratching. Oral steroids are usually unnecessary for mild symptoms of atopic dermatitis.

Besides these medical treatments, improvement of the living environment (e.g., removing carpeting, keeping the temperature and humidity low to reduce perspiration), and skin care (avoiding contact with causative agents, keeping the skin clean) are important.

**Prognosis**

Atopic dermatitis tends to be chronic and recurrent. It mostly resolves spontaneously by the time the patient reaches age 10; however, the symptoms do not improve in some patients until they reach adolescence or adulthood. The incidence of adolescent and adult atopic dermatitis has been increasing in recent years.

### 3. Seborrheic dermatitis

**Synonym:** Seborrheic eczema

**Outline**

- Seborrheic dermatitis occurs on sites of skin where sebum is actively secreted. It is characterized by erythematous lesions accompanied by yellowish scales.
- This is one of the most common skin diseases, occurring in infants, adolescents and adults.
- *Pityrosporum* fungus resident in the skin is a factor in the occurrence.
- Skin care and application of topical steroids and antifungal agents are the main treatments.

**Clinical features**

There is some controversy as to whether seborrheic dermatitis in infants, adolescents and adults is the same disease, because there are minor differences in the clinical courses (Fig. 7.12). Dermatitis appears as follicular eczema on seborrheic sites or intertriginous areas in the head, face, axillary fossa, neck and external genitals. The main features of the lesions are oleaginous scales and erythematous plaques that may be slightly itchy.

In infants, yellowish crusts begin to form on the scalp, eyebrows and forehead. In infants, scaly erythematous plaques may also form 2 to 4 weeks after birth. In most cases they resolve 8 to 12 months after birth. In adolescents and adults, pityroid scales (commonly called dandruff) increase and scaly erythematous
lesions form on the eyebrows and nasolabial groove. Seborrheic dermatitis is chronic and recurrent.

Pathogenesis

Triglycerides in sebum are decomposed by microbes resident in the skin to produce free acid. The free acid reacts to cause seborrheic dermatitis. It has been reported that over-proliferation of *Pityrosporum* fungi such as *Malassezia furfur* aggravates seborrheic dermatitis.

**Differential diagnosis**

Dry seborrheic dermatitis closely resembles psoriasis vulgaris. It is also important to differentiate seborrheic dermatitis from pityriasis rosea and parapsoriasis en plaque. In infants, differentiation from atopic dermatitis is essential.

**Treatment**

Proper facial cleansing with soap and hair washing with shampoo are basic for keeping the seborrheic regions clean. Regulating daily life is also helpful. Middle class topical steroids are applied. Topical antifungal agents and antifungal shampoos are effective at resolving seborrheic dermatitis accompanied by dandruff in adolescents and older adults, which is often caused by overproliferation of *Pityrosporum* fungi.

4. **Nummular eczema (eczema nummulare)**

**Outline**

- Round, relatively large eczematous plaques are produced.
- Nummular eczema may occur at any site on the body, and it tends to progress to autosensitization dermatitis.
- Topical steroids are the first choice of treatment.

**Clinical features, Epidemiology**

Nummular eczema is frequently seen in the winter. Multiple round eczematous lesions occur, mostly on the extremities (particularly on the extensor surface of the lower extremities), trunk, hips and buttocks (Fig. 7.13).

At the periphery of the lesions, serous papules aggregate, in the center of which exudative erythema is produced with scales on the surface. Most cases are accompanied by intense itching and multiple scars from rubbing and scratching. As the lesions progress, they may produce dispersal eruptions (id dermatitis) to progress into autosensitization dermatitis.

**Pathogenesis**

Scratched insect bites may develop urticarial lichens that, when rubbed, progress to nummular eczema. Or nummular eczema may result from asthenotic eczema in the elderly, or it...
may appear as a symptom of atopic dermatitis.

**Treatment**

Topical steroids (containing ODT) are effective. In cases in which infiltration and exudation are intense, the application of topical zinc ointment sheets is also effective. Oral antihistamines are helpful in relieving the itching.

**5. Lichen simplex chronicus**

| Synonyms: Lichen Vidal, Circumscribed neurodermatitis |

Lichen simplex chronicus is chronic eczema in which round, intensely itchy lichenified plaques form on the nuchal region and extensor aspect of forearms and lower legs of middle-aged women. Pigmentation or depigmentation is present in many cases. Warty eruptions may proliferate (Fig. 7.14). When skin is repeatedly stimulated by the friction of clothing or by metal allergens and the site is rubbed and scratched for a long period of time, it leads to the occurrence of chronic eczematous lesions. Topical steroids and oral antihistamines are first-line treatments for the itching.

**6. Autosensitization dermatitis**

| Outline |

- Multiple small papules and erythematous lesions accompanied by itching occur systemically. They are caused by sudden aggravation of a localized lesion.
- This dermatitis is caused by endogenous allergic reaction (id reaction).

| Clinical features |

Reddening, swelling and acute aggravation of exudation occur in the lower extremities as primary lesions of autosensitization dermatitis (in 50% to 60% of cases). Two weeks to several weeks after acute aggravation of reddening, swelling and exudation, dispersed eruptions appear. In most cases, the eruptions (id dermatitis) are erythema, papules, serous papules, or pustules of 2 to 5 mm in diameter dispersed symmetrically on the extremities, trunk, and face. These are often accompanied by intense itching (Fig. 7.15). Systemic symptoms such as fever and fatigue may occur.

| Pathogenesis |

Autosensitization dermatitis arises from endogenous allergic reaction (id reaction). Decayed proteins, bacteria, fungal components, and toxins produced by injured tissues in a primary lesion are considered to be the antigens. These may spread through the entire body such in blood flow from the primary lesion, or they
may spread by rubbing or by an accidental dose of the causative substance (orally or intravenously). Autosensitization dermatitis is caused by sensitization against the antigens. The primary lesions can be nummular eczema, stasis dermatitis, contact dermatitis, atopic dermatitis, tinea pedis, or eczematization of a burn.

**Treatment**

Topical steroids are applied and oral antihistamines are administered, in addition to whatever treatment is given for the underlying lesion. In severe cases, oral steroids are also administered.

### 7. Stasis dermatitis

**Outline**

- Edematous erythema or eczematous plaques form on the lower thighs as a result of varicose veins or congestion in the lower extremities.
- This disease tends to affect those who work standing, the elderly, and obese women.
- It may progress to autosensitization dermatitis.
- Elastic bandages and varicose vein phlebectomy are effective in reducing congestion.

**Clinical features**

Edematous erythema occurs on the lower third of the leg, particularly at the upper ankles. The site gradually presents a dark red, scaly, eczematous plaque, pigmentation or whitish atrophie blanche (Fig. 7.16). Minor trauma may induce ulceration. Treatments for stasis dermatitis may induce allergic contact dermatitis as a complication, from the application of an antiseptic or a topical agent (lanoline, antibiotic agent, preservative). Aggregated serous papules often progress to autosensitization dermatitis.

**Epidemiology**

Stasis dermatitis is frequently found in those who work standing for long periods of time. Pregnancy may trigger stasis dermatitis as a complication of varicose veins.

**Pathogenesis**

Congestion in the cutaneous blood vessels is caused by impairment of venous outflow, which leads to bleeding from the capillary vessel loop in the dermal upper layer. Hemosiderins deposit in tissues, and the skin takes on a blackish-brown appearance. The keratinocytes are injured by further impairment of blood flow. Atrophy and scaling occur in the epidermis and there is tendency of ulceration. The skin loses its function as a barrier and becomes more reactive to extrinsic irritation, leading to eczematous lesions in many cases.
**Laboratory findings, Diagnosis**

Stasis dermatitis is easy to diagnose from the varicose veins and the characteristics and distribution of the eruption. A Doppler test and angiography are performed on the varicosity to examine the physical potential of patients for surgical treatment. A patch test is performed if allergic contact dermatitis is suspected.

**Treatment**

Topical steroids are effective in treating eczematous lesions. When there is ulceration, it is cleansed and dressed. Induced allergic contact dermatitis is carefully avoided during treatment. Intravenous circulatory impairment is treated to prevent stasis dermatitis from progressing. Pressure that is greater than that of elastic bandages and socks should not be given to the patients. They should take bed rest and keep the lower extremities elevated. Surgery such as sclerotherapy, ligation, and removal of varicose blood may be necessary for cases with severe varicosity.

### 8. Asteatotic eczema

Skin dryness (asteatosis, xerosis) occurs when sebum decreases as a result of aging or excess washing. When the horny cell layer is destroyed, the skin is vulnerable to extrinsic irritation. When asteatosis becomes inflamed and eczematous, the condition is called asteatotic eczema (Fig. 7.17). This mostly affects the lower extremities of elderly in dry seasons, especially winter. For those who have a habit of excessively washing or rubbing the body with a towel, lifestyle guidance to avoid such behavior has therapeutic effects. Use of moisturizer prevents skin dryness. Eczema is treated with topical steroids. Skin care with moisturizer is helpful afterwards (Chapter 19).

### 9. Wiskott-Aldrich syndrome

#### Outline
- The three major characteristics of this disorder are immunological deficiency (T-cell dysfunction), thrombocytopenia, and intractable eczema.
- It is hereditary (X-linked recessive).
- There are decreased levels of immunoglobulins.
- Bone marrow transplantation may be performed.

#### Clinical features

Wiskott-Aldrich syndrome is characterized by eczema or purpura that occurs in newborn babies within 6 months after birth. The eczema that occurs on the head, face, buttocks and extremities appears similar to atopic dermatitis and seborrheic dermatitis (Fig. 7.18). Purpura is caused by thrombocytopenia. Immune-deficiency-derived infections occur repeatedly as the patient grows. Infections are caused by various factors including bacteria,
viruses, fungi and protozoa. Impetigo contagious (Staphylococcal infection), pseudomonas infection, herpes simplex, varicella (herpes virus infection), and candidiasis are particularly likely to accompany this syndrome, and they tend to become aggravated and persistent. Systemic symptoms such as bloody diarrheic stool, internal organ hemorrhage, infection (e.g., tympanitis, paranasal sinusitis, pneumonia) are seen recurrently.

**Pathogenesis**

Wiskott-Aldrich syndrome is caused by abnormality of a WASP gene at Xp11.22-11.23. The function of the WASP protein is unknown; however, it is thought to be associated with the cell viability and functional activation of T cells and platelets.

**Treatment, Prognosis**

Bone marrow transplantation may be conducted as a treatment. Treatments for atopic dermatitis are given for skin lesions produced by Wiskott-Aldrich syndrome. Patients with the syndrome may not survive, because of bleeding and infection in infancy (until about age 10); nevertheless, long-term survival is possible if the patient survives this period. In long-term survival cases, autoimmune disease and malignant lymphoma may arise as complications.