Chapter 13
Physiochemical Injury and Photosensitive Diseases

An important role of the skin is to protect the body from extrinsic stimuli such as sunlight, heat and cold. Melanin pigment and intercellular bridges in the epidermis prevent DNA from being damaged by sunlight and ultraviolet rays. Perspiration and blood capillaries work to maintain the body temperature. The horny cell layer and intercellular bridges protect the body from mechanical shock. Nonetheless, skin barrier functions can be destroyed when extrinsic stimuli exceed a certain level, resulting in injury such as electrical burn, chemical burn, frostbite, radiation damage and solar dermatitis. This chapter introduces skin disorders and photosensitive diseases with physiochemical causes.

Physiochemical injury

1. Burns (Fig. 13.1)

Outline
- Burns are injuries to cutaneous tissues caused by high temperature. The damage is divided by depth into first, second, and third degree.
- Burn size is measured by “the rule of nines” or “the rule of fives.”
- The basic treatment is cooling. Systemic intensive care or escharotomy may be necessary for severe cases.
- The first infusion, if necessary, is lactated Ringer’s solution, whose amount is decided according to the extent of burn (e.g., by the Baxter method).

Clinical features, Classification
Burn severity is evaluated by depth, size and patient’s condition.

Classification of burn by depth (Table 13.1)
The depth of a burn depends on the temperature of the heat source and the contact time. Burns are classified into first-degree, second-degree and third-degree. However, the severity is difficult to determine immediately after a burn. A burn may deepen with time after the burn incident.

<table>
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<tr>
<th>Diagnosis</th>
<th>Synonym</th>
<th>Clinical Findings</th>
<th>Treatment</th>
<th>Aftereffects</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree burn</td>
<td>Epidermal burn</td>
<td>Painful erythema, edema</td>
<td>Application of ointment</td>
<td>Scarring (-)</td>
</tr>
<tr>
<td>Second-degree burn</td>
<td>Superficial dermal burn (SDB)</td>
<td>Painful blisters with clear fluid and red blister bottoms</td>
<td>Application of ointment</td>
<td>Heals in about 2 weeks, scarring (-)</td>
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<tr>
<td></td>
<td>Deep dermal burn (DDB)</td>
<td>Hypalagesic blisters with white bottoms</td>
<td>Débridement, skin graft</td>
<td>Heals in 3-4 weeks, scarring (+)</td>
</tr>
<tr>
<td>Third-degree burn</td>
<td>Deep burn (DB)</td>
<td>Grayish-white or brown carbonized skin</td>
<td>Débridement, skin graft</td>
<td>Scarring (+)</td>
</tr>
</tbody>
</table>

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Fig. 13.1 Burn.
Second-degree burn. Mix of superficial dermal burn (SDB) and deep dermal burn (DDB), caused by hot water.
First-degree burn (epidermal burn): Painful erythema and edema occur, healing in 3 to 4 days without scarring.

Second-degree burn (dermal burn): Intense burning sensation is present. Erythema is followed within several hours by tense blistering (Fig. 13.2).

Second-degree burn is subclassified into superficial dermal burn (SDB) and deep dermal burn (DDB). SDB presents as painful blisters whose bottoms are rose pink; there is mild damage to the dermis, which heals without scarring in about 2 weeks. DDB affects the deep dermal layer and heals with scarring in 3 to 4 weeks. The bottom of a DDB blister is white and has reduced sensation. DDB often progresses to third-degree burn.

Third-degree burn (deep burn): All cutaneous layers and sometimes even deeper areas are damaged (Fig. 13.3). The skin appears gray-white. Blistering is not present, or the skin is carbonized to appear brownish. It becomes necrotic: Eschar forms and autodestruction occurs. Proliferation of the epidermis around the burn is the only spontaneous recovery; skin graft may be necessary.

A needle is sometimes used to examine the severity. The needle is stuck gently into the skin of the affected area; if it is painful, the diagnosis is second-degree burn; otherwise, the diagnosis is third-degree burn. If hairs come out when pulled gently, the diagnosis is DDB or third-degree burn.

2 Estimating the burn extent

“The rule of nines” is generally adopted to determine the extent of burns in adults; “the rule of fives” or the Lund and Browder chart is used for children (Fig. 13.4).

3 Evaluation of burn severity

Systemic intensive care is conducted in cases of second-degree burn on 10% or more of the body in children and on 15% or more in adults. Cases with 15% or greater burn index score (Fig. 13.4) are treated as severe burn.

Pathogenesis

Burns are caused by high temperature. They are extremely common in infants and children under age 10. However, low-temperature burns caused by prolonged use of electric pads and air heaters have been increasing among patients with cerebrovascular disease or diabetes (Fig. 13.5). In severe burns, histamines and cytokines are secreted from damaged tissue, leading to an increase in systemic vascular permeability. Accordingly, leakage of plasma proteins and extracellular fluid loss occur, resulting in burn shock.

Renal disorder, pulmonary edema, disseminated intravascular coagulation syndrome (DIC), and multiple organ failure may occur. Respiratory failure may also occur in patients with tracheal edema from inhaling hot air from a fire. Extensive burns are prone to infection (sepsis), and they tend to induce peptic ulcer (Curling’s ulcer) within a week after the burn incident. After epithelization and hypertrophic scar formation or a prolonged course of burn, squamous cell carcinoma may be caused.
**Fig. 13.3 Burns.**
a, b: Second-degree burns caused by hot water. Marked blistering is present. c: The epidermis has exfoliated with the stripping of clothes that were wet with scalding water. Third-degree burn is also present.

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**Fig. 13.4 Calculation of burn index score.**

\[
\text{burn index} = \frac{1}{2} \times (\text{area of second-degree burn}) + (\text{area of third-degree burn})
\]

* Add 5% if breast or two legs are burnt.

<table>
<thead>
<tr>
<th>Area</th>
<th>Age (years)</th>
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<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>A=1/2 of head</td>
<td>1 1/2%</td>
</tr>
<tr>
<td>B=1/2 of one thigh</td>
<td>2 3/4%</td>
</tr>
<tr>
<td>C=1/2 of one lower leg</td>
<td>2 1/2</td>
</tr>
</tbody>
</table>
In cases with hypertrophic scarring of the surface of joints, contracture may occur.

**Treatment**

1. **Local treatment**

   The primary treatment for burns is cooling with running water for at least 30 minutes to relieve pain, inflammation and edema.

   In second- and third-degree burns, it is important to prevent infection. Blister puncture may be performed. Appropriate antibiotic and incandescent ointments are chosen according to the skin condition. Affected sites of DDB and third-degree burns are removed (débridement), and a skin graft may be performed. The burn depth can be more clearly determined about 2 weeks after the time of the burn, which often makes it possible to determine whether the affected site should be treated for conservative preservation or whether surgical treatment is necessary. In the case of extensive burns, mesh skin grafts or fresh homografts are applied. In the event that severe edema disturbs the blood flow in the extremities, escharotomy is necessary to prevent necrosis.

2. **Systemic treatment**

   Airway management and infusion are primarily performed on patients with severe burns. The Baxter method is a widely applied infusion therapy *(Table 13.2)*. Infusion fluid is controlled by monitoring urine output, central venous pressure, and serum sodium and potassium concentration. Antibiotics or other drugs are applied systematically under observation if there are signs of sepsis, peptic ulcer, cardiac failure, pulmonary edema, or renal dysfunction.

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**2. Chilblain (perniosis), Frostbite**

**Outline**

- Chilblains and frostbite are cutaneous disorders caused by exposure to the cold.
- Edema-like and erythema multiforme-like eruptions are caused by local vascular constriction.
- Avoidance of the cold is the basic treatment. It is important not to warm the affected sites rapidly.

**1) Chilblain, Perniosis**

**Clinical features**

Chilblain (perniosis) occurs most commonly on the hands, fingers, feet, heels, auriculae and cheeks of schoolchildren *(Fig. 13.6)*. Chilblains are localized, usually tender, inflammatory, erythematous, often itchy lesions that may blister or ulcerate. Abnormal hypersensitive reaction is thought to be the cause; however, it is not clear why some people have this reaction while others do not.

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**Table 13.2 Parkland (Baxter) formula.**

Lactated Ringer’s solution [4cc x %TBSA (total body surface area) x weight (kg)] is given for the first 24 hours after the time of burn. Half the amount is given in the first 8 hours. The rest is given in the subsequent 16 hours.

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*Fig. 13.5 Third-degree low-temperature burn.*

a: This burn was caused by adhering a heated pad to the skin for a long time during sleep. Although the burn seems small and superficial, it is deep and third degree. b: Third-degree burn caused by a hot-water bottle during sleep. The patient has diabetes and diminished sensation in the peripheral nerves.
Chilblains are caused by exposure to low temperatures. Small arteries and veins become congested by repetitive exposure to cold, and the congestion causes inflammation. The condition occurs more often in early winter and early spring than in mid-winter, and is seen even in regions with warm temperatures. In addition to low temperature, moistness from perspiration and heredity factors are closely associated with chilblain occurrence.

Treatment

Exposure to the cold should be avoided. The affected sites are warmed and dried. Massaging is helpful. Vitamin E preparations, topical steroids, and orally administered peripheral circulatory dilators may be given.

2) Frostbite

Clinical features

Frostbite is acute freezing of tissues from exposure to extreme cold. Even just a few seconds of exposure may be sufficient to cause it. The fingers, ears and nose are most easily affected. It tends to occur in those who are not accustomed to the cold, and in the elderly. A few severe cases of frostbite in winter mountain climbers and in drunken persons, and from occupational accidents, have been reported. The skin becomes white to purplish-red, and reduced sensory perception is accompanied by hypoesthesia. As it progresses, blistering, necrotic ulceration, and mummification occur. The depth classification for burns is used to determine the severity of frostbite (Table 13.1).

Pathogenesis

Inadequate blood flow and thrombus formation (circulatory disorder) is caused when skin is exposed to the cold, leading to intercellular dehydration, destruction of cellular membranes (from tissue freezing), and blood vessel constriction. Frostbite most frequently occurs at or below −12°C. The length of exposure and the wind speed are factors in the occurrence and severity of frostbite. When the entire body surface is exposed to the cold for a long period, lethargy may set in and freezing death may result.

Treatment

The affected sites are warmed gradually as an emergency treatment. Rapid warming and strong friction should be avoided. The sites are warmed with 40°C water for 20 minutes and kept clean and protected. Surgical removal and care to prevent infection and necrosis are necessary. Intravenous vasodilators are useful in cases where the frostbite is related to circulatory disorder.
3. Chemical burn

In chemical burn, cutaneous tissues are damaged by acidic, alkaline or other escharotic chemicals (Fig. 13.7). Acid induces coagulative necrosis. Crusts appear, and their color depends on the causative acid (brown for sulfuric acid, yellow for hydrochloric and nitric acids, and white for hydrofluoric acid). The affected sites should be flushed with running water immediately. Neutralizing agents are not applied. The treatments afterwards are the same as those for burns.

4. Electric burn

In electric burn, cutaneous tissues are damaged by the passage of electrical current (Fig. 13.8). These burns, called “flash burns,” are caused on sites that come into contact with an electrical current. They result in ulceration and necrosis. Flash burns spread to become electric burns with dendritic reddening and ulceration. Lesions are caused by metal contained in electrodes that melts and fuses to the skin. The treatments are basically the same as those for burns.

5. Radiodermatitis

Synonyms: Radiation dermatitis, Radiation-induced dermatitis

Outline

- In radiodermatitis, cutaneous lesions are caused by radiation. The condition is divided into acute radiodermatitis, in which lesions occur immediately after exposure, and chronic radiodermatitis, in which lesions occur later.
- The treatments are the same as those for burns.
- Actinic keratosis and squamous cell carcinoma (radiation cancer) may develop. Surgical removal of the affected site is also chosen as a treatment.

Clinical features

1. Acute radiodermatitis

Acute radiodermatitis is caused by a single large exposure of radiation. The symptoms vary depending on the amount of irradiation, the site and the patient’s age. With a comparatively small amount irradiation (up to 5 Gy of gamma rays), erythema occurs several minutes after irradiation and disappears in 2 or 3 days, followed by edematous erythema, pigmentation, atrophy and telangiectasia. Blistering and erosion are caused by radiation doses between 5 Gy and 10 Gy (Fig. 13.9), and intractable ulceration and burn symptoms are caused by irradiation greater than 10 Gy.

2. Chronic radiodermatitis

Chronic radiodermatitis is commonly caused by a small...
amount of fractionated radiation. It often occurs on the site where a malignant tumor has been produced and radiation therapy has been repeatedly performed. It is also seen in the hands of medical professionals who deal with radiation (Fig. 13.10). There are four stages of chronic radiodermatitis: atrophy (atrophy, pigmentation, alopecia, and telangiectasia, occurring 6 months after the irradiation incident), keratinization (proliferation of horny cells), ulceration (intractable), and canceration (squamous cell carcinoma or basal cell carcinoma, occurring 15 to 20 years after irradiation).

Pathogenesis
Radiodermatitis is caused by exposure to X-rays, radioactive materials, or corpuscular radiation. All kinds of radiation cause cutaneous lesions of different severity. Radiation injures intercellular genes by DNA degeneration and DNA synthetase inhibition, which impairs cellular functions.

Treatment
The treatments for acute radiodermatitis are the same as those for burns. For chronic radiodermatitis, the affected site is protected by ointment application and bandaging. Extrinsic stimulation should be avoided. Ulcers and tumors are removed and the site is repaired with tissue that has good blood circulation, such as by pedicle flap procedure.

6. Pressure ulcer
Synonyms: Decubitus ulcer, Pressure sore, Bedsore

Clinical features
Pressure ulcers mostly occur in the sacral division, ischial tuberosity, and ankles (Fig. 13.11). Erythema, edema and induration are produced in areas subjected to constant pressure, and ulceration develops. Ulcers may be as deep as the bone, or spread to joints, rectum or vagina. The periphery of the ulcer is erosive and the lesion is often larger inside than it appears from the outside. The bottom of the ulcer is moist and covered by necrotic tissue and accumulated pus. Secondary infection such as by anaerobic fungi may result in sepsis.

Treatments and skin care are chosen according to the state of the affected site; therefore, stage classification is important (Table 13.3).

Pathogenesis
Circulation disorder caused by persistent pressure leads to necrosis of skin and subcutaneous tissues. It most commonly occurs in bedridden elderly and patients with spiral cord injury who are not able to change position by themselves. Thin persons and those who have underlying diseases such as under-nutrition and diabetes are also prone to pressure ulcer.
The primary treatment for pressure ulcer is quick removal or reduction of pressure to alleviate the impairment of blood flow. The affected site is locally cleansed, incarnative and antibiotic ointments are applied, and dressings are applied for protection. In the chronic stages, the affected site is cleaned with water and the necrotic tissue is removed. Disinfectant tends not to be used unless there is apparent infection. It is most essential not to worsen the condition.

Synonyms: Multiple neurotic gangrene, Hysteric gangrene

Clinical features

Erythema, erosion, gangrene and ulcer occur suddenly, mostly at sites within reach of the hands (extremities, chest and face). Right-handed patients tend to have dermatitis artefacta on the left side of the body. Eruptions may present different appearances according to the cause (e.g., fingernail scratch, knife injury, misuse of drug).

Pathogenesis, Diagnosis

In dermatitis artefacta there are factitious lesions. Patients with mental stress, hysteria, depression, mental disability or schizophrenia injure themselves in the skin, nails or mucous membranes. Most patients deny that the injury is self-inflicted. The specific types include trichotillomania (Chapter 18) and onychotillomania. Akatsuki disease (Fig. 13.12) and navel stone (Fig. 13.13) resemble dermatitis artefacta; however, they are

Table 13.3 Stages of pressure ulcer.

<table>
<thead>
<tr>
<th>Stage I</th>
<th>Stage II</th>
<th>Stage III</th>
<th>Stage IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>In lightly pigmented skin, lesion appears as a defined area of persistent redness. In darker skin tones, the lesion may appear with persistent red, blue, or purple hues. Lesions occur within the epidermis.</td>
<td>Partial-thickness skin loss involving epidermis, dermis, or both. The ulcer is superficial and presents clinically as an abrasion, blister or shallow crater. The ulcers spread to the dermis and subcutaneous fat.</td>
<td>Full-thickness skin loss involving damage to, or necrosis (death) of, subcutaneous tissue that may extend down to, but not through, underlying fascia (the sheet of fibrous material overlying muscles). The ulcer presents as a deep crater with or without undermining of adjacent tissue. The muscles are involved.</td>
<td>Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (tendons, joint capsule). Adjacent tissue undermining sinus tracts (an abnormal cavity in the tissue) may also be associated with this stage. The ulcers spread to the skeletal tissue. The bones and joints are damaged.</td>
</tr>
<tr>
<td>Reddening persists for more than 30 minutes after pressure is removed. Damage is within the epidermis.</td>
<td>Partial-thickness skin loss involving epidermis, dermis or both. The ulcer is superficial and may appear as an abrasion blister or shallow crater.</td>
<td>Full-thickness skin loss involving damage to, or necrosis of, subcutaneous tissue, which may extend down to but not through underlying fascia. The ulcer may appear as a deep crater with or without undermining of adjacent tissue.</td>
<td>Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon or joint capsule).</td>
</tr>
<tr>
<td>Non-bleachable erythema of intact skin, the heralding lesion of skin ulceration.</td>
<td>Partial-thickness skin loss involving epidermis, dermis or both (e.g., abrasion, blister, or shallow crater).</td>
<td>Full-thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia (deep crater with or without undermining). The ulcer presents clinically as a deep crater with or without undermining adjacent tissue.</td>
<td>Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon or joint capsule).</td>
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</tbody>
</table>

Clinical images are available in hardcopy only.

Fig. 13.12 Akatsuki disease in a woman in her twenties.

There is marked deposition of keratin. This patient hardly ever washed her nipples for fear that she would contract a skin disease.

Clinical images are available in hardcopy only.

Fig. 13.13 Navel stone.

This is so-called bellybutton lint. The patient consulted a doctor on what seemed to be a black tumor in the navel. When it was pulled out, there was a grimey mass of keratin. This patient had believed the superstition that the navel must not be washed.
Cutaneous symptoms should be treated appropriately. If necessary, treatment for mental imbalance may be necessary with cooperation from a psychiatrist.

Photosensitive diseases

1. Solar dermatitis, Sunburn

Erythema and blisters are produced by prolonged exposure to sunlight (mainly UVB). Pathologically, sunburn cells (apoptotic epidermal cells), epidermal spongiosis, edema in the dermal blood vessels, inflammatory cellular infiltration, necrosis and subcutaneous blistering are present. Erythema occurs several hours after photoradiation on the exposed site, and it gradually becomes edematous (Fig. 13.14). Solar dermatitis is most severe 12 to 24 hours after irradiation, after which it gradually resolves. Exfoliation and pigmentation occur in several days. Pigmentation is left after healing in some cases. Application of sunscreen is helpful for prevention. Cold compresses and steroid ointments are effective. When blistering is present, the same treatments as those for burns are applied.

2. Photosensitive dermatoses

Outline

- Photosensitive dermatoses are cutaneous diseases that are caused or aggravated by sunlight exposure.
- Both extrinsic factors (e.g., drugs) and intrinsic factors (e.g., inherited diseases, metabolic disorders) may be involved.
- They are caused by direct action of drugs (phototoxic dermatitis) or by immunological mechanism (photoallergic dermatitis).
- Xeroderma pigmentosum is a hereditary photosensitive dermatosis that is induced by intrinsic factors.

Pathogenesis

The two main causative factors of photosensitive dermatoses

MEMO

Suntan

The skin is darkened by exposure to the sun. Oxidation of melanin occurs in the epidermis (primary tanning), and there is enhanced production of melanin (secondary tanning).