

CHAPTER 1

Irritant Contact Dermatitis and Allergic Contact Dermatitis

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Contact dermatitis refers to a superficial inflammation of the skin when in contact with a “noxious” substance.

Classically, there are two mechanisms by which a substance elicits contact dermatitis:

1. Irritation
2. Allergy

Irritation refers to the direct injurious action by a substance when in contact with the skin. The resultant injury causes an inflammation, appropriately called irritant contact dermatitis.

Contact allergy, on the other hand, is an action by the immune system of the body on recognising an allergen that has gained entry into the skin. The resultant inflammation is called allergic contact dermatitis.

This distinction between irritation and allergy is central to the practice of contact dermatitis.

Irritant Contact Dermatitis

Classically, irritant contact dermatitis has been polarised into two ends of a spectrum. On one end is that of an acute irritant contact dermatitis. On the other, a cumulative insult irritant contact dermatitis.

Acute irritant contact dermatitis refers to the damage caused after only one contact. Cumulative insult irritant contact dermatitis refers to the wear and tear of multiple insults on the skin, eventuating in a contact dermatitis.

This distinction is central to the understanding of irritant contact dermatitis.

Acute irritant contact dermatitis

Substances which cause acute irritant contact dermatitis are toxic enough to cause damage to the skin after just one exposure. Examples include concentrated acids, concentrated alkalis, strong oxidising and reducing agents, strong solvents, etc.

The cause of an acute irritant contact dermatitis is often obvious. In the workplace, this usually occurs as a result of an accident, or a failure to use protective equipment when handling strong irritants. Occasionally, this occurs when of a chemical is used at a wrong concentration.

The most striking clinical feature of acute irritant contact dermatitis is the sharp demarcation of the lesion (Fig. 1.1).



Fig. 1.1 Sharp demarcation of the lesion in an acute irritant contact dermatitis from an inappropriate cosmetic treatment.



Fig. 1.2 The later phase of acute irritant contact dermatitis showing erythema and crusting.



Fig. 1.3 The resolving phase of an acute irritant contact dermatitis showing scaling and residual erythema.

Also striking is the course of the acute irritant contact dermatitis over time. After contact with a strong irritant, there is a variable delay (inflammation takes time to occur), before any visible change occurs in the skin.^a The progression then goes through erythema, vesiculation, exudation, crusting, scaling, residual erythema and post-inflammatory hyperpigmentation (Figs. 1.2 and 1.3). Occasionally, there is no vesiculation, the progression then marches from erythema through scaling.

Sometimes, a very caustic substance may cause sufficient damage such that necrosis occurs. Then, instead of healing through scaling, an ulcer forms, and the healing is through scarring.

All individuals, on contact with strong irritants, will develop this acute irritant contact dermatitis. Although there is invariably a slight variation in susceptibility between individuals, by and large, it can be said that all individuals are affected by strong irritants in much the same manner. This is in contrast to cumulative insult irritant contact dermatitis, as we will see later.

Cumulative insult irritant contact dermatitis

On the other end of the spectrum of irritant contact dermatitis is cumulative insult irritant contact dermatitis. Weak irritants, e.g. water, detergent, weak solvents, oils and greases do not have sufficient toxic potential to cause damage to skin structures after one exposure. It is the cumulative wear and tear on the skin that eventually result in sufficient damage as to cause an inflammation.^b

Cumulative insult irritant contact dermatitis usually has multifactorial causes. In addition to the wear and tear by one weak irritant, e.g. cutting oil in the workplace, there is often the contributory effects of the ambient environment, friction, other weak irritants in non-occupational activities

^aIn some cases, the delay is prolonged, up to many hours. Some writers designate this type of reaction as a delayed acute irritant contact dermatitis. An example of this is skin damage from kneeling on wet cement.

^bThe initial wear and tear on the skin is on the outermost layer of the skin, the stratum corneum. The initial damage to the stratum corneum is not yet a contact dermatitis. Some authors refer to this as an irritant reaction. This stage of irritant reaction is rapidly amenable to treatment using moisturizers. There is a threshold, after which contact dermatitis occurs, when the stratum corneum is finally broken through.

such as hobbies and activities in the home. Indeed, a cumulative effect of multiple irritants, appropriately reflected in the term cumulative insult irritant contact dermatitis.

Unlike acute irritant contact dermatitis by strong irritants, where there is little variability in susceptibility between people, certain categories of people are more predisposed to cumulative insult irritant contact dermatitis. Atopic dermatitis patients particularly, are more prone to cumulative insult irritant contact dermatitis.^c

The clinical picture of cumulative insult irritant contact dermatitis is that of a chronic dermatitis. This chronic dermatitis is polymorphic, ranging from a dry, slightly scaly erythematous dermatitis on the palms of housewives (Fig. 1.4) with repeated exposure to water and detergent, to discoid lesions



Fig. 1.4 Cumulative insult irritant contact dermatitis on the hands of a housewife.

^cThere is an interesting view that there is really no such entity as cumulative insult irritant contact dermatitis. The opinion is that all these patients have endogenous eczema, and the eczema is unmasked by multifactorial irritation.

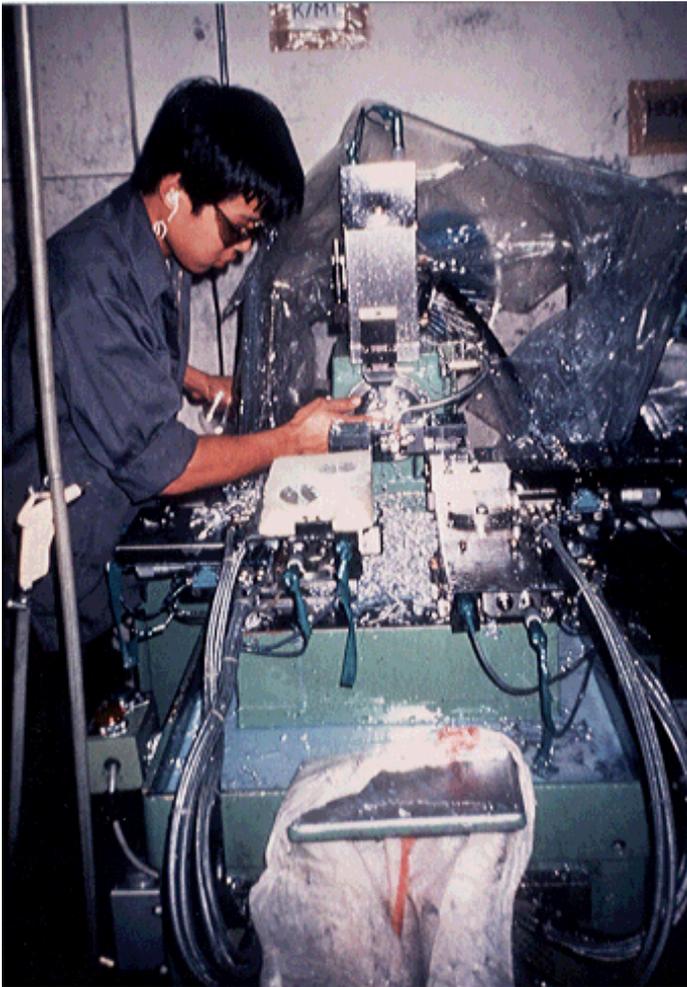


Fig. 1.5 A machinist exposed repeatedly to coolant oils.

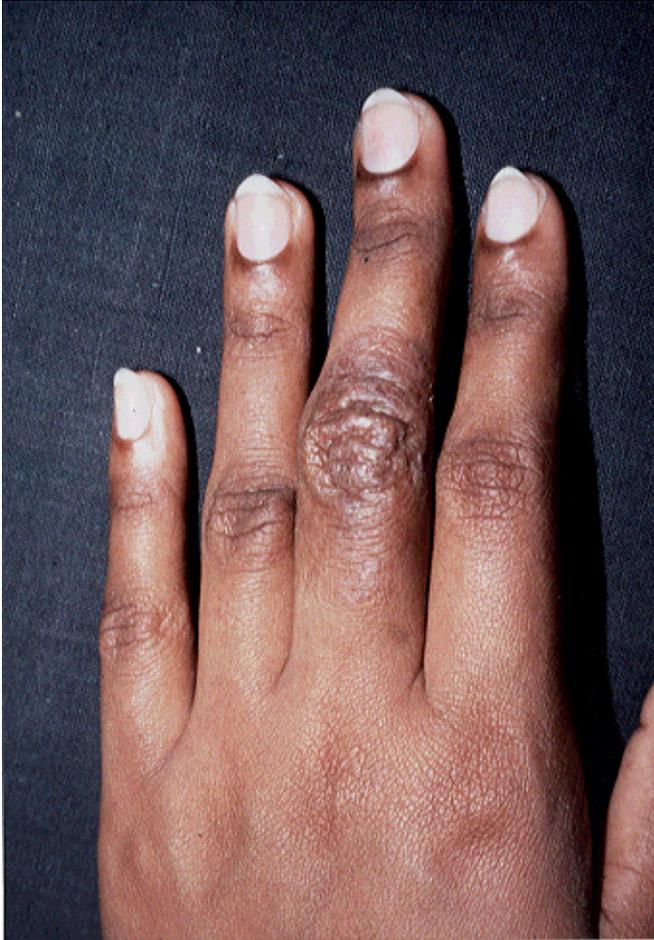


Fig. 1.6 Discoid type of cumulative insult irritant contact dermatitis due to repeated exposure to coolant oils.

on the knuckles of machinists exposed repeatedly to cutting oil (Figs. 1.5 and 1.6), to periungual and finger web eczema seen in workers exposed to solvents in the printing industry.

Allergic Contact Dermatitis

Allergic contact dermatitis, in contrast to irritant contact dermatitis, is not due to a direct injury but as a result of an immunological reaction. There is

no direct injury to the skin by the substance. It is the immune system, mediated by sensitised T-lymphocytes, that elicits a Type IV delayed hypersensitivity reaction in the skin on recognition of an allergen that has entered into the skin.

Only people who have been exposed to the substance, and have acquired an allergy (i.e. sensitised) will develop an allergic contact dermatitis. Allergic contact dermatitis, like all other forms of allergy, is specific to a substance.

There are therefore two phases in allergic contact dermatitis:

1. Sensitisation phase

This refers to the process whereby a person acquires allergy to a substance. In allergic contact dermatitis, sensitisation is usually via cutaneous contact. The latent period for sensitisation to develop is very variable. It is dependent on the sensitisation index of a substance,^d the concentration of the substance, the degree of exposure and the integrity of skin exposed.^e The minimum time needed for sensitisation to take place is about four days. Sensitisation, on the other hand, may only develop after many years of exposure.

There is no evidence of a genetic predisposition to sensitisation. Atopic dermatitis patients are neither more nor less predisposed to sensitisation.

2. Elicitation phase

This refers to the development of allergic contact dermatitis when an already sensitised person comes into contact with the specific allergen.

After contact with the allergen, there is a latent period before the allergic contact dermatitis is manifested. The reaction peaks at 48 to 72 hours.

Allergic contact dermatitis is usually eczematous. (There are some, a minority, of allergic contact reactions that are non-eczematous. This will be covered later.) An allergic contact dermatitis can be very acute, presenting

^dThe sensitisation potential of a chemical is often referred to as its sensitisation index. SADBE (squaric acid dibutyl ester) has a sensitisation index of close to 100%, making it a good agent for topical immunotherapy of severe alopecia areata.

^eDamaged skin predisposes to sensitisation. An example is stasis eczema. Patients with stasis eczema are prone to acquire allergy to their topical medicament.



Fig. 1.7 Acute allergic contact dermatitis: vesiculobullous.

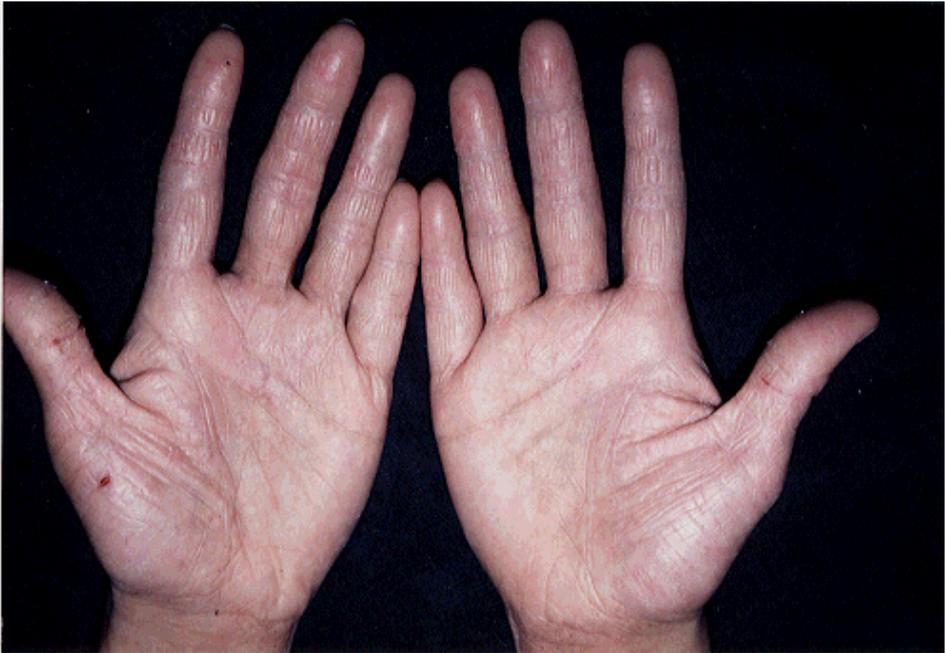


Fig. 1.8 Chronic allergic contact dermatitis showing dry, scaly erythematous plaques.

as an acute vesiculobullous eczema if the exposure is to a high concentration of a strong allergen (Fig. 1.7). Repeated contact to a low concentration of a weak allergen, on the other hand, produces a chronic lichenified fissuring eczema (Fig. 1.8).

The clinical picture of allergic contact dermatitis is often so similar to irritant contact dermatitis that it is difficult to differentiate the two. The investigative procedure to differentiate these two is the patch test, which will be covered later.

It goes without saying that allergic contact dermatitis occurs at the site of the eliciting contact. The margins may, like an acute irritant contact dermatitis, be sharp. More often though, in contrast to an acute irritant contact dermatitis, there is contiguous spread beyond the site of contact. Less frequently, there is a non-contiguous spread, and only very rarely is there a generalised spread of the allergic reaction (Fig. 1.9 and 1.10). Some allergens appear to have a propensity to cause a secondary spread of allergic contact dermatitis, e.g. proflavine and Vitamin E.



Fig. 1.9 Allergic contact dermatitis to proflavine.



Fig. 1.10 Secondary spread of allergic contact dermatitis to proflavine.