Irritant Contact Dermatitis: Diagnosis and Risk Factors

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6.1 Introduction

Irritant contact dermatitis (ICD) occurs after exposure of the skin to irritants from outside. Irritating agents or factors induce a disruption of the skin barrier and lead to an inflammatory reaction mainly mediated by the innate immunity. Any agent that causes damage to the skin is an irritant. The damage to the skin is determined by the chemical, physical or mechanical properties of the agent but also by the extent and duration of exposure. There are two variants: acute ICD and chronic ICD. Acute ICD is mostly due to one toxic event, usually caused by an accidental, short contact with a strong irritant. The chronic variant of ICD develops as a result of prolonged or repeated exposure to primary irritants and depends on the duration and intensity of exposure to the potentially responsible agent(s) [1]. Malten described in 1981 chronic ICD as a result of a sequence of a variety of skin irritating events, each being not strong enough to induce overt dermatitis, but when events taking place before the skin could recover from the previous event, the effect

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becomes clinically discernable [2]. Example of such skin damage incidents assigns its exposure to detergents, shampoos, abrasives, solvents and physical factors such as dry air, moisture and occlusion (by wearing gloves) but also water. At the moment that eczema has developed, even a minimal skin irritation, like a trivial exposure to water and soaps in normal personal care, can cause or maintain eczema.

6.2 Clinical Features

Clinical signs are dependent on the location and duration of exposure to the irritant as well as its chemical structure. Moreover, the clinical signs may vary with the susceptibility of the exposed individual. The morphology of the skin lesions depends greatly on the stage of the dermatitis at which the patient is first examined. In general, skin changes appear quite sharply defined and are located at places where the skin is exposed to aggressive substances. Acute ICD presents with erythema, oedema, infiltration and erosions. Subsequently, scaling and pustules may arise. In case of exposure to strong irritants, the skin changes may include blisters up to necrosis. Chronic ICD develops after repetitive exposure to a variety of damaging factors, such as water, soaps and detergents. Dry skin and mild erythema are often the first clinical signs. Proceeding the exposure leads to an obvious chronic ICD.

In the early phases of ICD, the web spaces, the dorsal surfaces of the fingers and backs of the hands are often affected, since these surfaces are more sensitive to irritant influences than the palm (Fig. 6.1). Subsequently the palmar surfaces are involved. Although vesicles usually do not occur in ICD, the clinical picture may be identical to that of allergic contact dermatitis (ACD).

6.3 Main Groups of Irritants

ICD is induced by direct contact of the skin with liquids, pastes and solids, including contact between aerosols, gases and vapors and the skin. Exposures to irritants that give rise to hand eczema are listed in Table 6.1.



Fig. 6.1 Irritant contact dermatitis in the web spaces, the dorsal surfaces of the fingers and back of the hand

Chemical irritants	Physical irritants	Other
Acids (also from fruit)	Mechanical	Water
Alkaline substances	Friction	Climate: cold
Cement/lime	Pressure	Environmental condition: low
Cooling lubricants	Heat	relative humidity
Oil products, including cutting oils	Dusts	
Organic solvents (benzene, acetone)	Occlusion (gloves)	
Detergents	Mineral and glass	
	fibers	
	Sand	
	UV radiation	
	Ionizing radiation	
	Wool	

Table 6.1 Main groups of irritants

Wet work is an important stressor for the skin and plays a prominent role in the majority of ICD cases. Activities during which workers spend a considerable portion of their working time in a wet work environment or wear liquid-tight gloves or wash their hands frequently or intensively count as wet work. Although gloves protect the skin from contact with allergens and irritants, the occlusion of the skin caused by the glove itself is a risk factor for hand eczema [3, 4]. The liquid-tight effect of protective gloves prevents the dissipation of perspiration to the outside; subsequently, the skin swells up as the time the gloves are worn increases, which reduces the barrier effect. Fartasch et al. investigated the differences between water exposure and occlusion by gloves in an experimental setting on forearm skin [3]. They demonstrated that short occlusion seems to harm the skin less than water exposure for the same time. However, their experiments were performed on forearm skin which indicates that the results may have been different when performed on the hands, in which occlusion of gloves may cause more perspiration due to abundant eccrine sweat glands on the palmar surfaces.

When the skin is pre-damaged by irritants or liquid-tight gloves, it becomes easier for irritants, potentially allergenic substances or infectious agents to penetrate [5]. In the case of combinations of irritative conditions, the damage to the skin is more than the separate effects, e.g., the harmful effect of soap is increased if it is followed by the use of liquid-tight gloves. Fartasch et al. demonstrated that previous occlusion and water exposure were capable of inducing higher susceptibility to sodium lauryl sulfate (SLS) irritation [3].

6.4 Risk Factors and Skin Barrier Dysfunction

The cause of hand eczema is often multifactorial. In addition to exogenous risk factors, there are endogenous risk factors that influence the development of ICD. A current or previous history of atopic dermatitis (AD) increases the risk for ICD [6, 7]. However, among individuals with atopic dermatitis who are exposed to irritants, it is difficult to distinguish between atopic hand eczema and hand eczema as a manifestation of ICD. Patients with AD have an impaired skin barrier, also in uninvolved skin, which was demonstrated by a higher penetration of SLS [8]. However, individuals without a history of atopic dermatitis may also have an increased susceptibility to irritants. Tupker et al. [9] investigated the susceptibility of the skin to various irritants, among other SLS, in individuals with a history of AD, individuals with a dry skin, and in individuals with clinically normal skin. In those with a previous history of AD, the transepidermal water loss values were both preexposure and throughout the entire period of exposure, higher than in the other groups. Though also individuals with clinically dry skin, without a history of AD, appeared to be more susceptible to irritants than those with normal skin, there was no difference noted in the preexposure barrier function.

The uppermost layer of the skin, the stratum corneum, acts as a barrier that prevents the entry of external irritants, microbes and allergens and controls the transcutaneous movement of water. An impaired skin barrier function in AD can partly be explained by a reduction or absence of the protein filaggrin in the skin. The filaggrin gene (FLG) encodes the protein profilaggrine, a major component of the keratohyalin granules in the stratum granulosum of the epidermis. During the later stages of epidermal differentiation, profilaggrine is dephosphorylated and cleaved to form filaggrin monomers, which contribute to the cornified cell envelope [10]. The filaggrin monomers are further proteolyzed, contributing to the natural moisturizing factor of the stratum corneum, and playing a central role in the hydration of the stratum corneum. Loss-of-function mutations in the FLG result in either a reduction or complete absence of epidermal filaggrin and its degradation products [11]. These mutations are predisposing factors for AD and are carried by 15-55 % of the patients with AD in European populations [12-14]. However, epidermal filaggrin and its degradation products are influenced not only by the filaggrin genotype but also by inflammation and exogenous stressors [11]. Filaggrin deficiency is observed in patients with AD regardless of filaggrin mutation status [11].

De Jongh et al. [7] demonstrated an increased risk for the development of ICD in individuals with *FLG* mutations. However, this association appeared to be dependent on the presence of a history of AD. In one study, a small, but significant association between ICD and *FLG* mutations persisted after adjustment for the history AD [11]. Both a history of AD and *FLG* mutations contribute to the development of ICD. More research into the skin barrier function in patients with AD and patients with *FLG* mutations is warranted to investigate the role of the different predisposing stimuli in the development of ICD.

6.5 How to Make the Diagnosis

The morphology and distribution of eczema are of limited help in making the diagnosis, and specific tests for ICD are not available. Allergic contact dermatitis and contact urticaria/protein contact dermatitis should be excluded as contributory causes, since combinations of irritant and allergic cases are common. Therefore, diagnostic patch testing should be performed in all patients with hand eczema with duration of more than 3 months and/or relapse, to identify the role of contact allergens [1]. Hand eczema patients reporting immediate skin reactions may have protein contact dermatitis. This is a distinct form of allergic or irritant hand eczema in which IgE-mediated mechanisms or nonimmunological mechanisms give rise to clinical manifestations characterized by an initial urticarial phase followed by eczema [1]. Triggers are natural rubber latex, food allergens or certain animal proteins. Skin prick testing or serum radioallergosorbent (RAST) testing should be performed to assess these reactions. However, nonimmunological types also exist [1]. See also Chap. 8 on protein contact dermatitis.

A history of hand eczema or AD provides important information on risk factors to develop ICD. Determination of allergen-specific IgE levels can help to establish the atopy status, though is not routinely recommended.

The diagnosis of ICD is based on a documented exposure to an irritant that is quantitatively likely to cause contact dermatitis [1]. A careful history about occupational and nonoccupational exposure to irritants is necessary. Occupational information should be obtained about accidents, new products or defective machinery. Environmental conditions such as changes in season, temperature and humidity and the influence on the contact dermatitis should be obtained. Questioning about the conditions of exposure is crucial to find the offending agents. Information about preceding or concomitant exposure is important if more than one product is involved. Detailed information on chemicals, products and materials should be traced. In case there are suspected materials or products from patient's work environment, material data safety sheets and lists of ingredients should be examined carefully for information about the product, the ingredients, concentrations, etc. [15].

It is important to estimate the duration of the exposures to irritants at the workplace, at home and during leisure activities. In addition, it is important to have insight into the frequency of exposure, whether it is a single exposure or a repeated exposure. Working procedures should be reviewed in order to quantify exposure to irritants. A well-defined exposure to irritants likely to cause ICD is wet work. TRGS 401 is the only existing guideline to regulate exposure to wet work [5]. Criteria for wet work include wet hands or wearing of liquid-proof gloves for 2 h or more per day or more than 20 handwashes daily. This limit of 2 h should be included in the quantification of exposure to irritants to make the diagnosis of ICD. The only correct method to measure wet work seems to be observation since questionnaires appeared not reliable in a study [16]. In Table 6.2, a work-up for a consultation of a patient with ICD is presented. Actually, the work-up is suitable for hand eczema in general. However, some parts such as the extensive, detailed history regarding exposure are important if ICD is suspected.

6.6 Management of Irritant Contact Dermatitis

The first and most important measure in ICD is to eliminate or reduce exposure to irritants and wet work. Healthcare workers should use alcohol-based skin disinfectants instead of water and soap. However, healthcare workers often experience the disinfectants as burning because of the damaged skin, though such a damaged skin

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History of hand eczema	Duration, primary site and type of skin changes, work related
Endogenous risk factors	History of atopic dermatitis, dry skin
Exposure	Frequency, duration, combination of irritative conditions
	Cleaning activities, detergents
	Handwashing, usage of soap, alcohol-based skin disinfectants
	Physical irritants (Table 6.1), cold air, low relative humidity
	Leisure activities (e.g., garden work)
	Types of gloves, plastic or rubber (e.g., natural rubber latex, nitrile, PVC)
	Chemicals, products, materials: concentration, dose, frequency, materials safety data sheets
	Wet work environment
Absenteeism	Duration, frequency, influence on hand eczema
Skin care	Usage of emollients and frequency
Impact on quality of life	E.g., hand eczema specific: QOLHEQ [17]
Examination of the skin	Anatomical distribution of skin changes on the hands, assessment of severity
	Skin changes on other sites of the body (e.g., feet)
	Other skin diseases (e.g., psoriasis, mycosis)
Assessment of severity	E.g., Physician Global Assessment (PGA), photographic guide or hand eczema severity index (HECSI) [18, 19]
Diagnostics	Patch testing (to exclude contact allergy)
	Prick testing or sIgE (to exclude protein contact dermatitis, if suspected)
Treatment, advice, educat	
Reduction of exposure	Reduce exposure to irritants, wet work, allergens
Protective measures	Usage of gloves adjusted to the irritants and type of work
	Wet work environment: fluid-tight gloves with cotton lining or inner gloves underneath fluid-tight gloves when gloves are worn for longer than 10 min
Gloves: good chemical	Latex: biologic materials and water-based materials
resistance to various	Nitrile: solvents, oils, greases, selected acids and bases
materials [4]	Vinyl: acids, bases, oils, greases, peroxides and amines
	Polychloroprene: acids, bases, alcohols, fuels, peroxides, hydrocarbons, oils, greases and phenols
	Polyvinyl alcohol: aromatic and chlorinated, solvents, ketones, esters, methacrylate (expensive)
	Butyl: ketones, aldehydes, esters (expensive)
	Multilayer laminates gloves (laminated glove of ethylene-
	vinylalcohol-polyethylene 4H®): almost all substances (poor fit)
Handwashing	Reduce frequency
	Wash only if visibly dirty: lukewarm water with fragrance-free soap without well-known sensitizers such as MCI/MI, dry thoroughly, alcohol-based disinfectants instead of water and soap for hand hygiene
Hand hygiene (e.g.	Alcohol-based skin disinfectants (instead of water and soap), dry
healthcare workers)	thoroughly before putting on gloves

 Table 6.2
 Work-up consultation of a patient with hand eczema in general, with specific focus on exposure to irritants and recommendation on various gloves

Daily skin care	Emollients several times per day	
Medical treatment	Topical steroids, calcineurin inhibitors (efficacy limited), (tar), UV therapy, systemic therapy (only recommended in severe hand eczema)	
Education	Nature of ICD, role of triggering factors like scratching, contact with water and soap, climate Use of topical steroids, guidance on the potency, duration and reducing steroid use, instruction on application of emollients/skin care	
Support	Strengthening of self-efficacy and self-management	

Tab	le 6.2	(continued)
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condition should always be avoided. Gloves protect the skin from contact with irritants. Different gloves protect for various exposures making it important to find out the right glove. A recommendation on various gloves [4] is made in Table 6.2. However, the occlusion of the skin caused by the glove itself is a risk factor for hand eczema. As the skin of the hands will sweat after about 10 min in liquid-tight gloves, it is recommended to wear it no more than 10 min continuously. When gloves are worn for longer than 10 min, cotton linings or inner gloves underneath occlusive gloves are recommended. See also Chap. 24 on workers' protection.

Acute hand eczema should be treated quickly and consequently to avoid the development of chronic hand eczema [1]. Often the patient has a certain work routine or routine in daily life in which one is accustomed over the years. However, without a thoroughly adjustment of skin damaging behavior, a medical treatment is unsuccessful. Table 6.2 provides detailed information on the treatment of hand eczema. Education is necessary on the nature of ICD, the role of triggering factors like scratching, contact with water and soap, skin protection and daily skin care. Nurses can have an important role in the education, instruction and guidance of patients with hand eczema. Most emollients improve the hydration state of normal skin/stratum corneum and are effective for treatment of contact dermatitis [1]. Besides treatment with emollients, the first line local treatment is a topical corticosteroid, in which once-daily application seems sufficient.

6.7 Prognosis and Personal Prevention

Hand eczema is often a long-lasting disease with a poor prognosis. Meding et al. reported the negative effect of the extensiveness of symptoms on the prognosis. Petersen et al. recently reported a 7-year follow-up study in which they evaluated the clinical course of patients with hand eczema [20]. Patients with a greater risk of a poor outcome were characterized by frequent eruptions, severe hand eczema and more widespread eczema [20]. A poorer prognosis of hand eczema is also associated with longer delay before medical attention [21]. Early accurate medical intervention is recommended to improve the prognosis.

The aim of primary prevention is to decrease the incidence of ICD by limiting exposure to its risk factors. Previous or current AD is a significant endogenous risk factor for development of ICD, counselling about avoiding wet and soiled occupations should be given to adolescents with AD. Clear risk occupations (hairdressers, healthcare workers, construction staff, cleaning staff, metal workers) should be discouraged. Nevertheless, if patients insist on these jobs, personal protective measures should be started immediately.

In patients with ICD, secondary prevention strategies are indicated. The objective of secondary prevention is to spot early skin changes in order to rapidly implement corrective measures [1]. The first and most important measure is to eliminate or reduce risk factors. Exposure to irritants and work-related skin burden should be avoided. Wearing gloves is often the first option to protect the skin from irritants. However, gloves may contribute to persistent damage by occlusion. In Table 6.2, a recommendation is made on the treatment and advises which are important in secondary prevention.

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