



Irritant Contact Dermatitis

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Abstract

Contact dermatitis accounts for 95% of occupational skin disorders. Irritant contact dermatitis (ICD) is often caused by cumulative exposure to weak irritants, accounting for 80% of all cases of contact dermatitis. ICD can co-exist with atopic dermatitis (AD) and allergic contact dermatitis (ACD). Patients with AD and ACD may have a lower inflammatory threshold for developing ICD. Therefore, it needs to be distinguished from lesions of AD and ACD. ICD Patients report stinging and burning in excess of pruritus. Pruritus is classically reported by patients with AD and ACD. ICD lesions are typically well-demarcated unlike AD and ACD. ICD is diagnosed by exclusion. Patients undergo testing to rule out type I and type IV hypersensitivity. Negative results suggest a diagnosis of ICD. Management consists of irritant identification and avoidance with regular emollient use. Although ICD is more common in certain occupations, genetics and environment play significant roles in its development.

Keywords Irritant contact dermatitis (ICD) · Allergic contact dermatitis (ACD) · Contact dermatitis · Patch testing · Occupational skin disease

Introduction

Contact dermatitis is a frequent problem accounting for 95% of all occupational skin diseases. It is an acute or chronic inflammation of the skin caused by contact with chemical or physical agents. It can be categorized as either irritant contact dermatitis (ICD) or allergic contact dermatitis (ACD). ICD accounts for 80% of all cases of contact dermatitis, and is most often caused by cumulative exposure to weak irritants such as soap and water. ICD is characterized by a direct injury of the skin epidermal cells which triggers the innate immune system causing an inflammatory cutaneous response to various external stimuli. ACD, on the other hand is characterized by a Type IV delayed hypersensitivity (immune) reaction to an allergen. It can be difficult to differentiate between the two types of dermatoses based upon clinical features alone.

ICD is a complex reaction modulated by both intrinsic (genetic) and extrinsic (environmental) factors, both of which are important in the pathogenesis of ICD especially of hand dermatitis. Age, sex, body region, and the presence of atopy influence the susceptibility to ICD. As well, the nature of the irritant, amount of exposure, concentration, duration, repetition, and the presence of overlying environmental and mechanical factors should be considered in the evaluation of ICD as it is not evident whether endogenous or exogenous factors make a stronger contribution to the development of ICD.

ICD occurs in both occupational and non-occupational settings. It is more common in specific occupational groups who do wet work in low-humidity conditions. Frequent hand washing and glove use, such as in health care workers are associated with the development of hand dermatitis.

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Clinical Features

The most common type of ICD encountered in a physician office is chronic ICD caused by repetitive exposure to a weak or marginal irritant over years. It classically presents with a dry, dull, red, scaly rash, and lichenified lesions. It is associated with a poor prognosis [1]. AD and ACD are other common causes of a similar rash, and must be differentiated from ICD. Sometimes they may co-exist in the same patient. ICD

typically affects young adults in an occupational setting [2]. AD commonly presents in children, whereas ACD more commonly presents in adults. Patients with AD usually also have allergic rhinitis and/or asthma, and a positive family history of atopy. The rash of AD is typically not well-demarcated from uninvolved skin. The rash of ICD is well-demarcated, typically confined to the area of contact with the irritant. In ACD, initially, the rash is well-demarcated; however, it may spread past the site of contact with allergen. Whereas AD is frequently complicated by impetigo (*Staphylococcus aureus* infection) and eczema herpeticum (HSV infection), these are uncommon in both ICD and ACD. Likewise, bacterial colonization is more common in patients with AD as compared to patients with ACD and ICD. Whereas both AD and ACD are characterized by a prominent itch, burning is more prominent than itch in patients with ICD.

Pathogenesis

ICD is caused by the direct toxic effect of an irritant on epidermal keratinocytes which results in skin barrier disruption and triggers the innate immune system. An irritant can be directly toxic to epidermal keratinocytes, as is the case with sodium lauryl sulfate, an irritant found in detergents [3]. Acetone (an organic solvent), on the other hand causes disruption of the epithelial barrier by loss of lipids [4]. This disrupts the epithelial barrier allowing increased permeability of irritants and even allergens. Chronic epithelial injury, usually upon repetitive exposure to a weak irritant, triggers the innate immune response with release of several proinflammatory cytokines including IL-1 α , IL-1 β , TNF- α , GM-CSF, IL-6, and IL-8 from the keratinocytes [5]. In turn, these cytokines activate Langerhan cells, dermal dendritic cells, and endothelial cells. Irritants can also be recognized as “danger signals” by TLRs and Nod-like receptors which activate the inflammasome and NF κ B pathways. These cells then release chemokines which results in the recruitment of neutrophils, lymphocytes, macrophages, and mast cells to the epidermis which causes further inflammation (Fig. 1). ICD, ACD, and AD often mimic each other, and may co-exist in the same patient. All three disorders are characterized by chronic inflammation.

Differences Between ICD and ACD Both ICD and ACD are caused by repeated contact with low molecular weight hapten. ACD is a delayed (type IV) hypersensitivity reaction to a hapten (acquired immune response) or non-protein contact allergens whereas ICD does not involve antigen/allergen-specific T cells. Therefore, ACD only occurs in susceptible individuals who have been sensitized. ICD, on the other hand does not require sensitization, and may be observed with initial exposure. The identification of hapten specific T cells in

ACD patients (in skin, patch test sites or blood-ELISPOT) helps to distinguish ACD from ICD [6]. Although susceptibility varies among individuals, given sufficient exposure to an irritant, anyone can develop ICD. Patients with ICD are more susceptible to the development of contact sensitization to allergens [7].

Differences Between ICD and AD Both AD and ICD are characterized by epithelial barrier disruption. The barrier defect of AD is due to loss of lipids, loss of terminal differentiation proteins, down regulation of barrier genes, and other proteins that comprise tight junctions [7–9]. The epithelial barrier is disrupted in affected and normal skin of AD patients resulting in increased permeability of antigens and irritants. Patients with AD have a heightened type I immune response (IgE mediated) to protein antigens which occurs in genetically predisposed individuals [10, 11]. In AD, the activated Langerhans cells (antigen presenting cells) produce T_H2 cytokines, whereas when ICD is induced experimentally the keratinocytes produce T_H1 cytokines [12, 13].

Predisposing Factors

The Host

Age is not consistently correlated with ICD, however, elderly patients have dry skin due to lower lipid content, and their skin does not heal quickly after injury resulting in a disrupted epithelial barrier. These are the main causes of asteatotic and perineal ICD in the elderly population [1]. ICD is also common in children who may develop diaper dermatitis, perianal dermatitis, sweaty sock syndrome, woolen clothing-induced ICD, and perioral dermatitis.

ICD is seen more frequently in women than men which is likely a result of increased exposure to irritants [14].

The face, dorsal aspect of hands, and the finger webs are more prone to chemical irritants than the palms, soles, and the back. This is most likely due to the fact that the skin is thinner in these areas, and therefore more susceptible to irritants [15, 16].

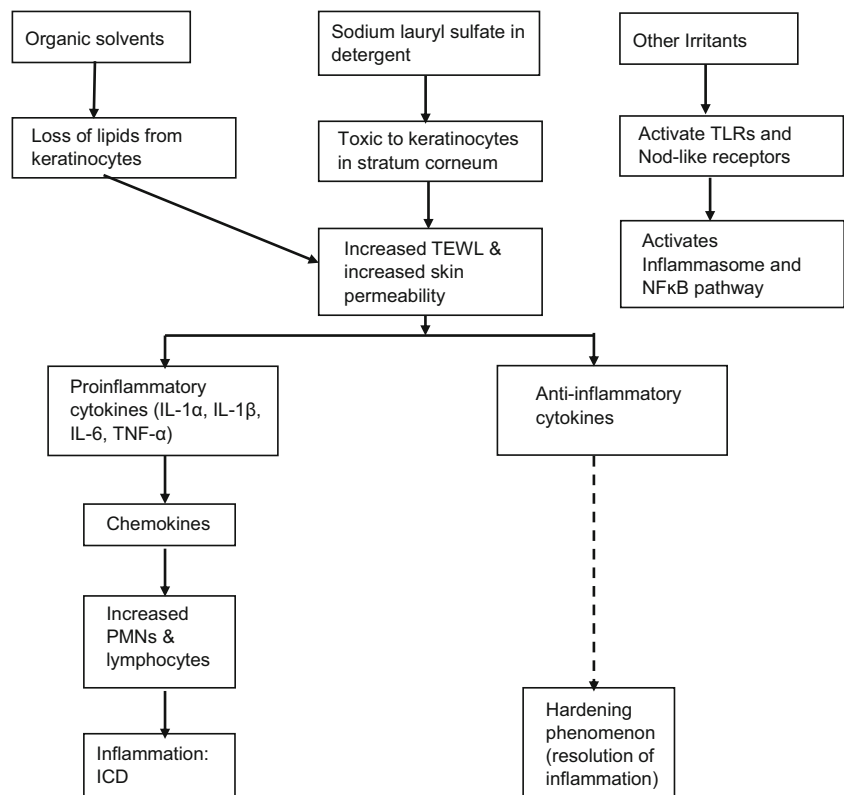
Atopic patients may have a lower inflammatory threshold for irritants, therefore promoting the development of ICD in patients with AD [17]. The frequency of irritant reactions is also increased in patients with a greater number of positive patch test reactions [18]. Twin studies indicate that genetic factors other than atopy may influence susceptibility to ICD [19].

The Environment

Ambient conditions such as temperature, airflow, humidity, and occlusion affect the skin's response to irritants [20]. Cold temperatures and low humidity increase transepidermal water loss [21]. On the other hand, increased humidity can

Fig. 1 Mechanisms of ICD: skin barrier disruption, epidermal cellular changes, and cytokine release. Chemicals such as organic solvents and detergents can cause barrier disruption leading to TEWL and increase skin permeability. The release of cytokines that cause inflammation results in ICD. TEWL, transepidermal water loss. ICD, irritant contact dermatitis.

TEWL: Transepidermal water loss
ICD: Irritant contact dermatitis



disrupt the skin barrier. Both result in an increased susceptibility to irritants [22]. Certain occupations increase the risk of ICD due to repeated contact with water, detergents, organic compounds, and other chemicals. The following occupations are high risk for the development of ICD: medical personnel, hairdressers, metalworkers, food worker, and construction and cement workers [2, 23].

The Irritant

Irritants are physical and chemical agents which can cause cellular damage when in contact with the skin for prolonged periods of time or at high concentrations. Common irritants include animal products, cosmetics, degreasing agents, detergents, dusts, foods, friction, low humidity, metalworking fluids, solvents, tear gases, topical medications, and water and wet work (see Table 1).

Water and wet work

The criteria for wet work are loosely defined, and include: (1) hands regularly in a wet environment for >2 h per day, (2) frequent hand washing (>20 times/day), (3) use of hand disinfectants 20 times in a working day, and (4) use of protective gloves for >2 h per day or change of gloves >20 times/day

[24]. It has been shown that unprotected wet work for >2 h per day is a risk factor for the development of ICD in hairdressers. In 1996, Ramsin et al. found that use of occlusive gloves for >6 h/day for 14 days had a negative effect on the skin barrier; however, in 2009, Wetzky et al. could not show the same negative effect (4 h/day for 7 days), warranting more studies on the effect of occlusion of rubber gloves. There is a wide variation between individual inflammatory responses to irritants. Therefore, not everyone doing wet work goes on to develop ICD, and simply a history of wet work does not rule out other causes of dermatitis.

Chemical Irritants

Detergents, surfactants, disinfectants, and antiseptics are common causes of occupational dermatitis—both ICD (42%) and ACD (26.3%) [25]. Shampoos contain the irritating chemical sodium lauryl sulfate and related detergents. Benzalkonium chloride, a surfactant and irritant [26] is widely used in cosmetics, skin disinfectants, medicated shampoos, and ophthalmic preparations. Solvent based mascaras are more likely to cause ICD [27]. Acids and alkalis cause a severe acute ICD with ulceration or trauma-like burns, usually in an occupational setting.

Table 1 Common causes of ICD based on region of involvement

Region involved	Clinical features	Common causes	Differential diagnosis	Management
Diffuse or generalized	Diffuse eczema	-Rough material: wool, burlap -Occlusive materials: polyester, nylon	-Textile ACD -Systemic contact dermatitis -AD	-Use untreated, finish-free cotton fibers -Avoid dry cleaning
Eyelids	Stinging and burning of the eyes and eyelids	-Propylene glycol -Sunscreen -Soap emulsifiers in eye formulations	-ACD (marked eyelid edema usual with poison ivy and hair dye allergy) -Seborrheic dermatitis -AD	-Tolerance may develop in some cases -Some react to emulsifiers in water-based mascara and may tolerate an anhydrous waterproof or a cake mascara
Face and other exposed areas (airborne ICD)	Eczema on face and other exposed areas	-Compositae plant family (most common) -Others include woods, plastics, rubbers, glues, natural resins, insecticides and pesticides -Pomades applied to the scalp especially in patients of color (pomade acne) -Violin players (fiddlers' neck)	-Occupational airborne ACD	-Use of personal protective equipment
Forehead	Acneiform lesions of the forehead, sometimes face and scalp		-Acne vulgaris	
Neck	Localized lichenification of left side of neck below angle of jaw		-ACD: run-off pattern of dermatitis due to cosmetic allergy or nail polish allergy -ACD: due to fragrance	Patch testing helpful (not indicated for dyspareunia)
Vulvitis	Itching without rash	-Feminine hygiene products -ICD due to the propellant		
Buttocks	Follicular type dermatitis and wet blisters	-Prolonged contact with wet bathing suits (bikini bottom)	Baboon syndrome (systemic contact dermatitis—erythema of buttocks, inner thighs, and axillae) ACD due to nail hardeners (may contain formaldehyde-releasing agents)	
Nails	Koilonychia (spoon shaped nails) Traumatic onycholysis Nail discoloration Leukonychia	-Organic solvents (e.g., thinners) in cabinet makers -Motor oils in mechanics -Permanent wave solutions in hairdressers -Weed killers and insecticides -Traumatic causes: poultry pluckers, rifleman, joggers, and tennis, soccer and karate players -Detergents, solvents, wet-work [housewife's eczema or dishpan hands, medical and dental workers, food preparation and service workers (corn, pineapple juice), janitors, and housekeepers] -Trapping of soap and detergents under a ring often initiates hand ICD -65% of pts. with AD who work in the hospital develop hand ICD		
Hands	Localized dermatitis without vesicles in the webs of fingers extending onto the dorsal and ventral surfaces (apron pattern), dorsum of hands, palms, and ball of thumb. ICD due to wet work is often associated with paronychia		-ACD (vesicles usual, favors fingertips, nail folds, and dorsum of the hands) -AD (dorsal hands and fingers along with the volar wrist, popliteal, and antecubital areas) -Dyshidrosis (discrete vesicles on sides of fingers, palms, and soles) - Psoriasis (examine nails, elbows, knees, and scalp)	Use cold compresses and cold water for washing, UV radiation, and training programs
Diaper area	Erythema over the external genitalia and buttocks, usually sparing the creases. May progress to vesicles and bullae	-Prolonged contact with urine or feces or both -Contact with residual antiseptics, soaps, and detergents in the diapers; and by friction	-Candida infection -Psoriasis early manifestation -Seborrheic dermatitis -ACD	Disposable diapers are better than cloth diapers in prevention
Thighs	Clearly defined dermatitis on the posterior aspect of the thighs and buttocks	Toilet seat dermatitis due to lacquer or paint covering the seat, or strong detergents used to clean the seat		

Table 1 (continued)

Region involved	Clinical features	Common causes	Differential diagnosis	Management
Perineal	-Starts as erythema with mild pruritus which progresses to erosions and vesicles -Tendency for superinfection with <i>Staphylococcus aureus</i> , candida and tinea	Usually affects elderly patients with incontinence due to frequent liquid or soft stools and undigested food particles in feces	-ACD	Coat area with zinc oxide ointment
Feet	Lesions affect the interdigital spaces (sweaty sock syndrome)	Excessive sweating	-ACD due to shoes: typically spares the interdigital spaces -Tinea pedis -AD	-Wear non-occlusive footwear, all cotton hose -Permanganate foot baths and dusting powder
Axillae		Shaving and use of depilatories	-ACD -AD	
Perioral dermatitis and contact cheilitis		Frequent licking, chewing gum, contact of cheeks with foods such as spinach, carrot, and citrus fruits	-AD -ACD	

Physical Irritants

Common physical irritants include metals tools, wood, and fiberglass [28]; plant parts such as thorns, spines, sharp-edged leaves [29]; and wool, paper, dust, and soil [30].

Wool

Rough-textured and woolen clothing and occlusive footwear frequently cause dermatitis in children. The latter is called sweaty sock dermatitis, and is due to excessive sweating, wearing socks containing synthetic fibers or sneakers, rubbers, or rubber-soled shoes for prolonged periods of time. This causes an eruption of the toes and inter-web areas. These areas can readily become eczematized and infected. Paronychial infections and dystrophic nail changes in neglected cases is not uncommon [31].

Paper/Dust/Soil

Coal, rock, stone dust, chemical dusts, cement dust, and sawdust (from teak, mahogany, and rosewood) produce dry, lichenified dermatitis, frequently with a follicular pattern. Sawdust dermatitis usually affects the face, penis, and scrotum of carpenters and woodworkers.

Diagnosis

Contact dermatitis is suspected from the clinical presentation and possible exposure to a contact allergen. ICD is typically a diagnosis of exclusion and may be over-diagnosed for example, if a patient’s occupation involves frequent wet work or wet-dry cycles. This is especially important since up to 40% of all occupations involve excessive contact with irritants. Accordingly, persons in these occupations will most likely fulfill criteria for wet work, and ICD if they develop dermatitis [32]. Therefore, it is important to exclude both type I and type IV allergies before making a diagnosis of ICD, especially in an occupational setting.

History

The history is aimed at identifying potential allergens and irritants, and excluding other potential diagnoses (Table 2). Patients with ICD typically have onset of symptoms within minutes to hours of exposure to an irritant. The rash is limited to areas in contact with the irritant. Classically, pain, burning, stinging, or discomfort exceeds itching. The latter is more prominent in ACD and AD.

It is important to ask about daily activities, including occupation and hobbies: any obvious exposure to chrome, epoxy,

Table 2 History

Onset: acute or chronic
Typically pain, burning, stinging, or discomfort exceeds itching
Suspected triggers or exposures
Facial dermatitis: hair/facial and nail cosmetics, dyes, toiletries, and makeup applicators
Daily activities including occupation and hobbies
Work conditions: wet work, use of occlusive gloves & cleansers, mechanical trauma, and oils
List of topical and oral medications including complementary medicines
Recent antibiotic use
Recent surgery
History of allergic rhinitis, asthma
History of psoriasis or other chronic skin disorders
Known history of contact allergies
Family history of atopy or psoriasis

acrylics, gloves, clothing, first aid creams, preservatives, plants, and other chemicals, can point to contact dermatitis. Hobbies, for example, gardening involves the risk of airborne contact dermatitis in response to being near plants. Rubber dermatitis may be present in patients who wear facemasks or goggles during scuba diving or swimming. Ask about the environment at work (temperature, humidity, and exposure to dusts). Ask about use of protective gloves or gear. Inquire about wet work, use of occlusive gloves, and cleansers at work. Improvement of symptoms away from work may suggest an occupational dermatitis. Ask about whether other workers are similarly affected which may suggest an ICD. Common irritants at work include: wet work, glove use, mechanical traumas, and oils [32].

For patients presenting with facial dermatitis, it is important to inquire about use of hair dye; cosmetics/toiletries of the face, hair, and nails (nail polish, nail varnish, acrylic nails); and cosmetic applicators and tools such as rubber sponges, eyelash curlers, and adhesives used for false eyelashes. Infants and children may acquire cosmetic dermatitis by contact with cosmetics worn by the care giver.

It is important to get a list of topical and oral medications including complementary therapies. Inquire about recent antibiotic use. Predisposing factors for ICD include AD, atopic respiratory disease, or other inflammatory skin disease; therefore, it is important to inquire about these, and any known contact allergies. Reactions to suture and wound dressings may be irritant rather than allergic.

Ask about a family history of atopy, psoriasis, and other chronic skin conditions. Family history of atopy is important because AD may present for the first time during adulthood.

Lastly, it is important to get a thorough history before and after patch testing to formulate the correct diagnosis, and treat and counsel a patient.

Physical Exam

The distribution of the dermatitis (pattern of involvement) is often the single most important clue to the diagnosis of contact dermatitis. The site of eruption is usually in the area of contact with the irritant. Since prior sensitization is not required, ICD can present after a single episode of exposure to a strong irritant, e.g., strong acids or alkalis or repeated exposure to weak irritants. The thinner eyelid and genital skin are more susceptible to ACD and ICD. Allergens in lotions and creams which are applied all over sometimes produce reaction in intertriginous areas, where the chemicals tend to concentrate.

Patterns of Dermatitis

Hand

ICD commonly presents as a localized dermatitis without vesicles in the webs of fingers; it extends onto the dorsal and ventral surfaces (apron pattern), dorsum of hands, palms, and ball of thumb. On the other hand, ACD often has vesicles and favors the fingertips, nail folds, and dorsum of the hands and less commonly involves the palms. ICD often precedes ACD, which will cause a progression of the distribution of rash [33]. Involvement of the dorsal hands and fingers along with the volar wrist suggests AD as a contributing causative factor [34].

Face and eyelid

Marked edema of the eyelids is often a feature of poison ivy or hair dye dermatitis. Stinging and burning of the eyes and lids on application of the cosmetic suggest ICD. Usually, these symptoms are transient and not associated with physical signs. Some common irritants include propylene glycol, sunscreen, and soap emulsifiers in eye-area formulations.

Diaper Area

The dermatitis of ICD is present over the external genitalia and buttocks, usually sparing the creases. The eruption may spread to include the lower abdomen, and even the skin of the feet coming into contact with urine. Mild cases present with slight erythema confined to the diaper area. It can progress to a scalded appearance with edema and vesicles. Eroded bullae may become eczematous, and pyodermic lesions may appear. Herpetiform ulcers are not uncommon. It is produced by prolonged contact with urine or feces or both; by residual antiseptics, soaps, and detergents in the diapers; and by friction. Toilet seat dermatitis may be produced by strong detergents used to cleanse the seat. In addition, lacquer or paint covering the seat may cause a clearly defined pattern of dermatitis on the posterior aspect of the thighs and buttocks.

Feet

The rash in ICD typically affects the interdigital spaces, and is due to excessive sweating. It may be mistaken for tinea pedis, thus scraping and cultures for tinea may be helpful. In contrast, ACD to shoes usually spares the interdigital areas. It is easy to mistake it for AD especially if hyperhidrosis also causes dermatitis of the flexural areas thus patch testing may be indicated.

Perioral Area

Contact cheilitis and perioral dermatitis in children affect the lips and adjacent areas, which are commonly irritated due to a habit of licking, chewing gum, contact of cheeks with foods such as spinach, carrot, and citrus fruits.

Perineal Area

Incontinence dermatitis is typical for older individuals and is mostly related to urinary and/or fecal incontinence [35]. It initially presents with a mild erythema, sometimes pruritic which becomes complicated by the development of small erosions and vesicles. These have a tendency for superinfection with *Staphylococcus aureus*, candida, and tinea. In severe cases, pressure ulcers develop.

Morphology

ICD may be subdivided into acute, sub-acute and chronic ICD depending on how fast the dermatitis develops following exposure. Whereas, acute ICD often develops following contact with a strong acid or alkali and mimics a chemical burn with a scalded appearance of the epidermis, chronic ICD is characterized by lichenification and fissuring. See Table 3. Furthermore, there are distinct morphologic forms of ICD elicited by specific irritants (see Table 4).

Sensory Irritation

Status cosmeticus manifests as itching, stinging, or burning sensation of the face after application of any cosmetic. There may be minimal physical findings [36] and the differential diagnosis includes pityriasis folliculorum. Common causative agents are benzoic acid, bronopol, cinnamic acid compounds, Dowicel 200, formaldehyde, lactic acid, nonionic emulsifiers, propylene glycol, quaternary ammonium compounds, sodium lauryl sulfate, sorbic acid, and urea. Management includes using cosmetics without these agents.

The use of anti-irritant cosmetic compounds such as carboxyl compounds, hydroxyl compounds (polysorbate 20, *Aloe vera* gel), and imidazole compounds (Germall-115-

imidazolidinyl urea allantoin, imidazoline amphoteric surfactants – used in no tears shampoos) needs investigation.

Laboratory Tests

Role of Patch Testing

Patch testing is the gold standard for diagnosing ACD, and its proper performance and interpretation require experience. Due to the clinical similarity of ACD and ICD, patch testing may provide helpful information. Commercially available individual patch test allergens in a dilute, non-irritating concentration are applied to the upper back for 48 h. After the patch tests are removed, the sites of the patch tests are evaluated at least twice, usually after removal at 48 h and again at 72–96 h or beyond. Some irritant reactions on patch testing appear within the first 48 h then disappear (decrecendo effect) by 96 h. Allergic reactions tend to increase (crescendo effect). Thus, the second patch test read is important to help determine if the reaction is an irritant or an allergic one because an irritant patch test is considered a negative test and has no clinical significance. Results at both readings are graded according to intensity of reaction at the patch test site on a scale of 0 to 3⁺. Relevance of positive reactions to present or past episodes of dermatitis must be determined by correlating the patch test results with chemicals, products, and processes encountered in the environment.

Patch tests should not be applied if the patient's dermatitis is active or involves the back. A screening patch test series is typically applied and supplemented by other patch test chemicals based on the patient's history and occupation. Consider skin and/or ImmunoCAP testing to latex and foods in patients who wear rubber gloves and handle food frequently.

Measurement of transepidermal water loss (TEWL) is a non-invasive method of assessing the skin irritant response but may not be readily available to the clinician.

Skin biopsy is of limited value in diagnosing contact dermatitis since the findings depend on the stage of the process and nature of contactant. Furthermore, most types of eczema show similar histopathological changes and cannot be distinguished with certainty. The histology of ICD is very different from ACD when biopsies are taken during the first day or two following exposure. This is expected because in ICD, the epidermal damage is caused directly by the toxic agent, whereas in ACD the damage is due to the host's immune reactions. Within a few hours of exposure to a strong irritant, dermo-epidermal separation begins. By 24 h, epidermal necrosis sets in, often with sub-epidermal blister formation [37]. Lymphocytes are relatively rare. In contrast, the bulk of the inflammatory infiltrate is made up of neutrophils which appear within 6–8 h of exposure. Different irritants can produce distinct histologic findings. Unfortunately, these differences

Table 3 Subtypes of ICD

Subtype	Description
Acute ICD	Bright erythema, vesiculation, weeping, mimics a chemical burn or sunburn. Develops within minutes to hours of exposure to a strong chemical, such as acid or alkali
Subacute ICD or delayed acute ICD	Presents similarly to acute ICD but develops 8–24 h after contact. Typical irritants include: anthralin, benzalkonium chloride, tretinoin and tetra ethylene glycol diacrylate, dithranol, epichlorhydrin, podophyllin, and propane sulphone
Chronic ICD	Erythema, scaling and fissuring. May be due to physical agents, repeated micro trauma, or exposure to weak irritants over years
Sensory irritation	Stinging, burning, tightness, itching, or painful sensations after contact with cosmetics, usually in middle-aged White and Asian women

between early ICD and ACD are rarely of clinical benefit because biopsies within the first few days of onset are usually unavailable. The histologic findings of chronic low-grade ICD may be identical to those of ACD; however, the presence of eosinophils may suggest ACD, whereas the absence of exocytosis of lymphocytes may suggest ICD. Mast cells are increased in AD only. There are no eosinophils or mast cells in patient with ICD. Neutrophils, on the other hand are usually observed in the epidermis of patients with ICD, and not in patients with AD or ACD.

Furthermore, hyperplasia and hyperkeratosis are observed in ICD, whereas, parakeratosis is evident in ICD and ACD, and orthokeratosis is typical of AD. The granular layer is normal in ACD and ICD, whereas it is reduced or absent in patients with AD [38].

Fungal, bacterial, and viral smears and cultures; potassium hydroxide examination for fungi; and microscopic examination for glass fibers can also be helpful in the evaluation of a patient with suspected ICD.

Differential Diagnosis

Contact dermatitis is normally differentiated from other types of dermatitis on the basis of clinical findings, knowledge of

exposure to potential allergens or irritants, and diagnostic patch testing. See Table 5.

Management

The first step is to accurately diagnose ICD; however, there are no universally accepted tests for diagnosing ICD. It is a diagnosis of exclusion. The next step is to identify the irritant. For example, cosmetics and fragrances can cause either ACD or ICD. Some common irritants are listed in Table 1. A positive patch test with current relevance makes ACD more likely than ICD although both can occur at the same time. Any allergen at high enough concentration can produce an ICD.

Avoidance of Irritant(s)

The definitive treatment of contact dermatitis is the identification and avoidance of the underlying cause. Patients with ICD should be counseled on how best to avoid irritants both at home and in the workplace. After an irritant has been identified, measures should be taken (e.g., the use of personal protective equipment in the workplace) to reduce the risk of future exposure. Use cold compresses and cold water for

Table 4 Distinct morphological forms of ICD

Morphology	Reported causes
Erythema, vesiculation and weeping, bullae (acute ICD)	Potent irritant: laboratory or industrial chemical
Erythema, scaling fissuring (chronic ICD)	Cumulative exposure to a weak irritant such as water and soap.
Ulceration	Strong acids and alkalis
Folliculitis	Oils and greases
Miliaria	Aluminum chloride
Hyperpigmentation	Heavy metals
Hypopigmentation (contact vitiligo)	Phenolic detergents
Acneiform lesions of the forehead (pomade acne)	Pomades—hair straighteners applied to the scalp, usually used by black patients
Petechial and purpuric eruptions	Woolen garments, textile finishes, fiberglass, pressure
Pustular dermatitis	Metals, tar, oil, chlorinated agents, naphthalene

Table 5 Differential diagnosis

Condition	Location	Subtype of ICD it may mimic	Differentiating features
ACD	Area in contact with allergen	Any (face, eyelid, perioral, cheilitis, hand, foot, perianal, generalized)	Prior sensitization is required. Itching is more prominent than burning or stinging (latter more common in ICD). Lesions usually well-demarcated. Vesicles
AD	Dorsal hands, fingers and volar wrist, antecubital fossa, popliteal fossa, posterior neck, behind ears	Any (face, eyelid, perioral, cheilitis, hand, foot, generalized)	History of allergic rhinitis, asthma, food allergy, family history of atopy. Usually presents in childhood but may be seen in adults also
Psoriasis	Hands, knees, ankles, nail involvement, +/- systemic involvement	Hand dermatitis	Nail involvement does not rule out ICD, ACD, or AD
Infection: impetigo, superficial fungal infections, herpes simplex, varicella zoster, cellulitis	Impetigo is commonly superimposed on AD lesions, less often with ICD and ACD	-Hand dermatitis -Diaper dermatitis -Foot dermatitis	
Asteatotic eczema			
Dyshidrotic eczema		Hand dermatitis	
Factitious eczema			
Nummular eczema			
Photo allergic dermatitis			
Phototoxicity			
Seborrheic dermatitis		-Diaper dermatitis -Scalp dermatitis -Eyelid dermatitis -Facial dermatitis	
Sunburn		Acute ICD	
Stasis dermatitis with autoeczematization		Leg dermatitis	
Cutaneous T cell lymphoma		Thigh eczema due to matchstick	
Lupus erythematosus		Facial rash	

washing. UV radiation can be beneficial, and training programs at the workplace can be effective.

Treat Inflammation

The regular use of emollients enhances barrier function of the skin and is an important part of the management of contact dermatitis. Emollients or occlusive dressings may improve barrier in dry, lichenified skin. Moisturizers are believed to increase hydration or prevent TEWL, thereby maintaining skin barrier function and reducing the risk of ICD [39]. Traditional petrolatum based emollients are accessible and inexpensive, and they have been shown to be as effective as an emollient containing skin-related lipids. Restore skin barrier function by using less irritating substances, such as soap substitutes when washing.

ICD due to saliva and food juices often does not respond to creams and ointments. Adherent, protective pastes such as plain Lassar's paste give better results. For infants Burrow's solution 5 mL+anhydrous lanolin 10 g, Talc USP 10 g, Zinc oxide ointment up to 6 g (Fisher Contact Dermatitis, 5th

edition) can be used. To relieve pruritus, a lotion of camphor, menthol, and hydrocortisone (Sarnol HC) is soothing, drying, and antipruritic. Pramoxine, a topical anesthetic in a lotion base (Prax) can also relieve pruritus. Topical corticosteroid use in ICD is controversial and may be effective for the treatment of the inflammation in contact dermatitis if the underlying irritant is avoided.

Prevention

Gloves and barrier creams

The use of vinyl gloves with cotton liners to avoid the accumulation of moisture that often occurs during activities involving exposure to household or other irritants or foods (e.g., peeling or chopping fruits or vegetables) may be helpful [40]. In the workplace, verify that gloves are safe to use around machinery before recommending their use. Barrier creams are generally a last resort and are probably best used in workers with no dermatitis.

Prognosis

Patients with severe disease have poorer prognosis despite improvements in general working conditions, better availability of diagnostic patch testing, improved understanding of cutaneous biology, and treatment with topical and systemic steroids. A history of chronic dermatitis, delay of adequate treatment, a history of AD, and poor understanding by the worker of his or her disease are associated with a worse prognosis. AD is an important factor in susceptibility to persistent post-occupational dermatitis.

Discussion Section

Contact dermatitis accounts for 95% of all occupational skin diseases with ICD accounting for a vast majority of cases. Although more common in specific occupational groups, it can be found in both occupational and non-occupational settings. The allergist should be able to evaluate and manage these cases when it presents in their office. The clinical features of ICD and ACD overlap but certain characteristics suggest to the physician on how to proceed in the evaluation (patch testing), management (improving barrier function and avoidance of precipitation factors), and use of medications (topical corticosteroids).

Summary

ICD is a complex reaction modulated by both intrinsic (genetic) and extrinsic (environmental) factors, both of which are important in the pathogenesis of ICD especially of hand dermatitis. The host (patient) and the irritants (chemical, amount of exposure, concentration, duration, repetition) should be considered in the diagnosis and management of ICD.

Although AD, ACD, and ICD can co-exist, differentiation if possible and proper treatment can lead to earlier resolution of the rash. Patients with AD usually also have allergic rhinitis and/or asthma, and a positive family history of atopy. The rash of ICD is well-demarcated, typically confined to the area of contact with the irritant. The rash of ACD may spread past the site of contact with allergen.

ICD is caused by the direct toxic effect of an irritant on epidermal keratinocytes which results in skin barrier disruption and triggers the innate immune. ACD is a delayed (type IV) hypersensitivity reaction to a hapten (acquired immune response) or non-protein contact allergens. Important predisposing factors in the development of ICD include both the host and the environment. Also, certain occupations increase the risk of ICD due to repeated contact with water, detergents, organic compounds, and other chemicals. This includes medical personnel, hairdressers, metalworkers, food worker, and construction and cement workers.

Water and wet work predisposes to the development of ICD especially on the hands. It has been shown that unprotected wet work for >2 h per day is a risk factor for the development of ICD in hairdressers. Detergents, disinfectants, and antiseptics are common causes of occupational dermatitis. Solvent, oxidizing agents, acids, and alkalis can cause a severe acute ICD with ulceration or trauma-like burns, usually in an occupational setting.

Aside from chemical irritants, physical irritants such as metals tools, wood, fiberglass, plant parts (such as thorns, spines, sharp-edged leaves), wool, paper, dust, and soil can cause ICD. Contact dermatitis is suspected from the history, clinical presentation, and possible exposure to a contact allergen. ICD is typically a diagnosis of exclusion, thus patch testing may be needed to rule out ACD. Fungal, bacterial, and viral smears and cultures; potassium hydroxide examination for fungi and microscopic exam for glass fibers can also be helpful in the evaluation of a patient with suspected ICD. Other tests such as measurement of TEWL and skin biopsy are usually of limited value.

The management of ICD includes accurate diagnosis and identification of the irritant if possible. The definitive treatment is the avoidance of the underlying cause. The use of personal protective equipment in the workplace may help reduce the risk of future exposure. The regular use of emollients enhances barrier function of the skin and is an important part of the management of CD. Gloves, barrier creams, and ointments may improve ICD. Topical corticosteroid use in ICD may be effective for the treatment of the inflammation if the underlying irritant is avoided. However, patients with severe disease have poorer prognosis despite improvements in general working conditions, better availability of diagnostic patch testing, improved understanding of cutaneous biology, and treatment with topical and systemic steroids.

Compliance with Ethical Standards

Conflict of Interest Sonia Bains is currently a Speaker for Pfizer 090117. Luz Fonacier received research and educational grants from Winthrop University Hospital, Genentech, Baxter, Pfizer, and Regeneron. She is also a Treasurer of the American College of Allergy, Asthma & Immunology (2015–2018) and a member of the American Board of Allergy and Immunology (ABAI) (2016–present). Pembroke Nash declares no conflict of interest.

Ethical Approval This article does not contain any studies with human participants or animals performed by any of the authors.

References

1. Seyfarth F, Schliemann S, Antonov D, Elsner P (2011) Dry skin, barrier function, and irritant contact dermatitis in the elderly. *Clin Dermatol* 29(1):31–36

2. Diepgen TL, Coenraads PJ (1999) The epidemiology of occupational contact dermatitis. *Int Arch Occup Environ Health* 72(8): 496–506
3. Fartasch M, Schnetz E, Diepgen TL (1998) Characterization of detergent-induced barrier alterations – effect of barrier cream on irritation. *J Invest Dermatol Symp Proc* 3(2):121–127
4. Yang L, Mao-Qiang M, Taljebini M, Elias PM, Feingold KR (1995) Topical stratum corneum lipids accelerate barrier repair after tape stripping, solvent treatment and some but not all types of detergent treatment. *Br J Dermatol* 133(5):679–685
5. Smith HR, Basketter DA, McFadden JP (2002) Irritant dermatitis, irritancy and its role in allergic contact dermatitis. *Clin Exp Dermatol* 27(2):138–146
6. Nosbaum A, Vocanson M, Rozieres A, Hennino A, Nicolas JF (2009) Allergic and irritant contact dermatitis. *Eur J Dermatol: EJD* 19(4):325–332
7. Gittler JK, Krueger JG, Guttman-Yassky E (2013) Atopic dermatitis results in intrinsic barrier and immune abnormalities: implications for contact dermatitis. *J Allergy Clin Immunol* 131(2):300–313
8. Suarez-Farinas M, Tintle SJ, Shemer A, et al. (2011) Nonlesional atopic dermatitis skin is characterized by broad terminal differentiation defects and variable immune abnormalities. *The Journal of allergy and clinical immunology* 127(4):954–964.e951–954
9. Palmer CN, Irvine AD, Terron-Kwiatkowski A et al (2006) Common loss-of-function variants of the epidermal barrier protein filaggrin are a major predisposing factor for atopic dermatitis. *Nat Genet* 38(4):441–446
10. Proksch E, Brasch J (1997) Influence of epidermal permeability barrier disruption and Langerhans' cell density on allergic contact dermatitis. *Acta Derm Venereol* 77(2):102–104
11. Shimada S, Caughman SW, Sharrow SO, Stephany D, Katz SI (1987) Enhanced antigen-presenting capacity of cultured Langerhans' cells is associated with markedly increased expression of Ia antigen. *J Immunol (Baltimore, Md : 1950)* 139(8):2551–2555
12. Onoue A, Kabashima K, Kobayashi M, Mori T, Tokura Y (2009) Induction of eosinophil- and Th2-attracting epidermal chemokines and cutaneous late-phase reaction in tape-stripped skin. *Exp Dermatol* 18(12):1036–1043
13. Mori T, Kabashima K, Yoshiki R, Sugita K, Shiraiishi N, Onoue A, Kuroda E, Kobayashi M, Yamashita U, Tokura Y (2008) Cutaneous hypersensitivities to haptens are controlled by IFN-gamma-upregulated keratinocyte Th1 chemokines and IFN-gamma-downregulated Langerhans cell Th2 chemokines. *J Invest Dermatol* 128(7):1719–1727
14. Thyssen JP, Skare L, Lundgren L, Menné T, Johansen JD, Maibach HI, Lidén C (2010) Sensitivity and specificity of the nickel spot (dimethylglyoxime) test. *Contact Dermatitis* 62(5):279–288
15. Clark SC, Zirwas MJ (2009) Management of occupational dermatitis. *Dermatol Clin* 27(3):365–383 vii–viii
16. Rougier A, Dupuis D, Lotte C, Roguet R, Wester RC, Maibach HI (1986) Regional variation in percutaneous absorption in man: measurement by the stripping method. *Arch Dermatol Res* 278(6):465–469
17. Scharschmidt TC, Man MQ, Hatano Y, et al. (2009) Filaggrin deficiency confers a paracellular barrier abnormality that reduces inflammatory thresholds to irritants and haptens. *J Allergy Clin Immunol*;124(3):496–506, 506.e491–496
18. Klas PA, Corey G, Storrs FJ, Chan SC, Hanifin JM (1996) Allergic and irritant patch test reactions and atopic disease. *Contact Dermatitis* 34(2):121–124
19. Lerbaek A, Kyvik KO, Mortensen J, Bryld LE, Menne T, Agner T (2007) Heritability of hand eczema is not explained by comorbidity with atopic dermatitis. *J Invest Dermatol* 127(7):1632–1640
20. Zhai H, Maibach HI (2001) Skin occlusion and irritant and allergic contact dermatitis: an overview. *Contact Dermatitis* 44(4):201–206
21. Uter W, Gefeller O, Schwanzitz HJ (1998) An epidemiological study of the influence of season (cold and dry air) on the occurrence of irritant skin changes of the hands. *Br J Dermatol* 138(2):266–272
22. Fluhr JW, Akengin A, Bornkessel A, Fuchs S, Praessler J, Norgauer J, Grieshaber R, Kleesz P, Elsner P (2005) Additive impairment of the barrier function by mechanical irritation, occlusion and sodium lauryl sulphate in vivo. *Br J Dermatol* 153(1):125–131
23. Bock M, Schmidt A, Bruckner T, Diepgen TL (2003) Occupational skin disease in the construction industry. *Br J Dermatol* 149(6): 1165–1171
24. Menne T, Johansen JD, Sommerlund M, Veien NK (2011) Hand eczema guidelines based on the Danish guidelines for the diagnosis and treatment of hand eczema. *Contact Dermatitis* 65(1):3–12
25. Lodde B, Paul M, Roguedas-Contios AM, Eniafe-Eveillard MO, Misery L, Dewitte JD (2012) Occupational dermatitis in workers exposed to detergents, disinfectants, and antiseptics. *Skinmed* 10(3):144–150
26. Oiso N, Fukai K, Ishii M (2005) Irritant contact dermatitis from benzalkonium chloride in shampoo. *Contact Dermatitis* 52(1):54
27. Loden M, Wessman C (2002) Mascaras may cause irritant contact dermatitis. *Int J Cosmet Sci* 24(5):281–285
28. Bordel-Gomez MT, Miranda-Romero A (2008) Fibreglass dermatitis: a report of 2 cases. *Contact Dermatitis* 59(2):120–122
29. Modi GM, Doherty CB, Katta R, Orengo IF (2009) Irritant contact dermatitis from plants. *Dermatitis: contact, atopic, occupational, drug* 20(2):63–78
30. Morris-Jones R, Robertson SJ, Ross JS, White IR, McFadden JP, Rycroft RJ (2002) Dermatitis caused by physical irritants. *Br J Dermatol* 147(2):270–275
31. Gibson WB (1963) Sweaty sock dermatitis. *Clin Pediatr* 2:175–177
32. Friis UF, Menne T, Schwensen JF, Flyvholm MA, Bonde JP, Johansen JD (2014) Occupational irritant contact dermatitis diagnosed by analysis of contact irritants and allergens in the work environment. *Contact Dermatitis* 71(6):364–370
33. Fonacier LS, Dreskin SC, Leung DY (2010) Allergic skin diseases. *J Allergy Clin Immunol* 125(2 Suppl 2):S138–S149
34. Simpson EL, Thompson MM, Hanifin JM (2006) Prevalence and morphology of hand eczema in patients with atopic dermatitis. *Dermatitis: contact, atopic, occupational, drug* 17(3):123–127
35. Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, Talley NJ (1999) Prevalence of combined fecal and urinary incontinence: a community-based study. *J Am Geriatr Soc* 47(7):837–841
36. Fisher AA (1980) Cosmetic dermatitis. Part II. Reactions to some commonly used preservatives. *Cutis* 26(2):136–137 141–132, 147–138
37. Medenica M, Rostenberg A Jr (1971) A comparative light and electron microscopic study of primary irritant contact dermatitis and allergic contact dermatitis. *J Invest Dermatol* 56(4):259–271
38. Suarez-Farinas M, Gittler JK, Shemer A, Cardinale I, Krueger JG, Guttman-Yassky E (2013) Residual genomic signature of atopic dermatitis despite clinical resolution with narrow-band UVB. *J Allergy Clin Immunol* 131(2):577–579
39. Chew AL, Maibach HI (2003) Occupational issues of irritant contact dermatitis. *Int Arch Occup Environ Health* 76(5):339–346
40. Brancaccio RR, Alvarez MS (2004) Contact allergy to food. *Dermatol Ther* 17(4):302–313