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An Overview on Contact Dermatitis: Simple Review

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Contact dermatitis (CD) is usually the result of cumulative exposure to sensitive irritants and accounts for 80% of all contact dermatitis cases. ICD can coexist with atopic dermatitis (AD) and allergic contact dermatitis (ACD). Patients with Alzheimer's disease and ACD may also have a lower infection threshold for ICD. Therefore, it must stand out from EA and CAD lesions. People with ICD have experienced uncontrolled tingling and burning sensations. Itching is typically manifested in patients with AD and ACD. Compared with AD and ACD, ICD lesions are usually well described. The prognosis of ICD is based on the exclusion method. Monitor patients to rule out

type 1 and type 4 hypersensitivity reactions. A negative result indicates the prognosis of ICD. Management includes identifying and avoiding irritants through the normal use of emollients. Although ICD is older, it is not uncommon in some majors, and genetics and environment play a vital role in its development.

Keywords: Contact dermatitis (ACD); irritant contact dermatitis (ICD); occupational dermatology patch test.

1. INTRODUCTION

Contact dermatitis (CD) is defined as a reactive eczematous inflammation of the skin which occurs after direct contact with a chemical but occasionally by biologic or physical agents [1].

Contact dermatitis is a frequent problem accounting for 95% of all occupational skin diseases. Categories of CD include allergic CD (ACD), photoallergic CD (PACD), irritant CD (ICD), photo irritant CD (PICD, or phototoxic CD), protein CD (PCD), the most common of which are ICD or ACD [2].

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 can be either acute type due to single exposure of a material such as chemical burns (e.g. hydrofluoric acid, hydrochloric acid, alkali) and also phototoxic ICD (require ultraviolet light A to elicit it) or could be chronic type from cumulative and repetitive exposure to an irritant substance (such as solvents, water, soap, detergents, acid, alkali, etc.) [3].

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 IHD [2].

ACD is a delayed (type 4) hypersensitivity reaction to exogenous contact antigens. ACD only occurs in sensitized patients i.e. individuals who have developed chemical-specific T cells [4]. These cells have pro-inflammatory properties and are referred toas effector T cells. Common etiological allergens for allergic contact dermatitis are nickel, balsam of Peru, chromium, neomycin, formaldehyde, thiomersal, fragrance mix, cobalt, and parthenium [5]. Poison Ivy (Toxicodendron, formerly known as Rhus), in the United States, is considered to be the most common cause of allergic contact dermatitis [6].

Symptoms of ICD may include burning, itching, stinging, soreness, and pain, particularly at the beginning of the clinical course, while pruritus is more common in allergic contact dermatitis. Patients with a history are at increased risk for developing nonspecific hand dermatitis and irritant contact dermatitis.

The diagnosis of ICD is a diagnosis of exclusion; diagnosis of CD relies on clinical presentation, thorough exposure assessment and a patch test (for ACD and PACD), which is the gold standard for the identification of contact allergens, or a skin-prick or prick-prick test (for PCD) [7].

The treatment of contact dermatitis depends on its stage. The acute phase is best treated with astringent soak and topical or systemic steroids and an antihistamine. Surgical debridement and skin grafting may be needed in very rare [8]. The chronic phase is managed by moisturizing creams for skin dryness in addition to topical steroids. Antibiotics may be needed if there is evidence of secondary infection. In all cases protection and avoidance of irritants and allergens should be implemented [9].

1.1 Etiology

ICD occurs more often than allergic contact dermatitis. The patient develop a rash when a chemical substance irritates the skin's outer layers. The rash is more painful than itchy.

Common causes of ICD include: Acids, Alkalis like drain cleaners, Body fluids, including urine and saliva, Certain plants, such as poinsettias and peppers, Hair dyes.

The chance of developing irritant contact dermatitis (ICD) increases with the duration, intensity, and concentration of the substance. ICD can also occur when the skin comes in contact with less irritating materials — like soap or even water — too often. Physical irritants like friction, abrasions, occlusion, and detergents like sodium lauryl sulfate produce synergistic effect of irritant contact dermatitis in (their combined effect greater than the sum of their separate effects) [10,11].

1.2 Pathophysiology

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 [12]. Acetone (an organic solvent), on the other hand causes disruption of the epithelial barrier by loss of lipids [13]. 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-1α, IL-1β, TNF-α, GM-CSF, IL-6, and IL-8 from the keratinocytes. 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 inflammation and NFκB 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 [14].

ICD is mainly due to the toxicity of chemicals on skin cells, which triggers inflammation by the activation of the innate immune system [15]. By contrast, ACD is due to type IV delayed-type hypersensitivity responses [16] induced by the immunogenic properties of a subset of chemicals and requires the activation of both innate and acquired immunity. The mechanisms by which chemicals cause skin irritation are poorly understood and vary from the disorganization of the lipid bilayers of cell membranes to the damage of epidermal barrier proteins such as keratins, claudins, involucrin and filaggrin [17].

Certain chemicals, such as acids, bases or detergents, trigger an intense cell necrosis, causing major disruption of the skin barrier; these chemicals are known as corrosives. Corrosive substances irreversibly damage the skin beyond repair, whereas irritant substances lead to a reversible local inflammatory reaction caused by the innate immune system of the affected tissues. Irritants have minimal and reversible effects on epidermal cells and may require repetitive applications before an ICD reaction occurs [18].

In both cases, the release of stress-associated molecular patterns (reactive oxygen species (ROS, ATP) and damage-associated molecular patterns (DAMPs; high-mobility group protein B1 (HMGB1), heat-shock proteins, IL-1α) by injured cells are sensed by receptors of the innate immune system on surrounding healthy cells [19]. The recognition of these ligands results in the release of a myriad of chemokines, derivatives of arachidonic acid metabolism and proteases within minutes or hours after contact.

Irritants may also excite nociceptors, thereby producing acute pain and neurogenic inflammation through the release of vasoactive peptides such as substance P [20]. This release induces vasodilation and the infiltration of diverse leukocytes (neutrophils, eosinophils, basophils and/or inflammatory monocytes) from the blood into the skin, which further amplify the reaction.

The resulting physiological signs of irritation include damage to the epidermis with spongiosis (characterized by intercellular oedema), microvesicle formation and/or necrosis (which can be detected by histology), and clinical manifestations such as erythema, induration (hardened skin) and oedema, which can be associated with painful and burning areas of skin [21].

1.3 Prevalence

Females, infants, elderly, and individuals with atopic tendencies are more susceptible to irritant contact dermatitis. It is reported that up to 80% of cases of occupational dermatitis are irritant contact dermatitis [22].

ICD is significantly more common in women than in men. The high frequency of hand eczema in women in comparison with men is caused by environmental factors, not genetic factors.

Occupational irritant contact dermatitis affects women almost twice as often as men, in contrast to other occupational diseases that predominantly affect men. Women are exposed more highly to cutaneous irritants from their traditionally greater role in housecleaning and the care of small children at home. In addition, women predominantly perform many occupations at high risk for irritant contact dermatitis (eg, hairdressing, nursing).

In some European studies among employees in high-risk occupations, such as hairdressing, healthcare, and metal working, the 1-year prevalence was between 20% and 30% [23]. Specifically, in Denmark, cleaners comprise the greatest number of affected workers, but culinary workers have the highest incidence. A higher proportion of prolonged sick leave is seen among those in food-related occupations compared with those in wet occupations.

The incidence rates of contact dermatitis in Germany were 4.5 cases per 10,000 workers for irritant contact dermatitis, compared with 4.1 cases per 10,000 workers for allergic contact dermatitis. The highest irritant contact dermatitis annual incidence rates were found in hairdressers (46.9 cases per 10,000 workers per year), bakers (23.5 cases per 10,000 workers per year), and pastry cooks (16.9 cases per 10,000 workers per year [24].

1.3.1 Risk factors

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 [25]. 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.

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 [26]. These mutations are predisposing factors for AD and are carried by 15–55 % of the patients with AD in European populations [27,28].

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 [29]. ICD is also common in children who may develop diaper dermatitis, perianal dermatitis, sweaty sock syndrome, woolen clothing-induced ICD, and perioral dermatitis. Moreover, it seems that ICD is seen more frequently in women than men which is likely a result of increased exposure to irritants [30].

Also there appears to be some genetic predisposition to contact dermatitis, as some people are more prone to develop allergies to chemicals, while others with the same exposure do not.But there are many systems at play that determine whether or not the patient develop the allergy or an irritant reaction. These include how well the skin acts as a barrier, how the body produces an inflammatory response, and how prone the patient are to developing allergies.While some genes have been proposed as increasing the risk, there are no definitive culprits.[31].

1.3.2 Clinical presentation

Clinically, irritant CD can occur as an acute or chronic disease. Lesions may occur anywhere but commonly appear on the hands [32]. Acute irritant CD is typically characterized by erythema, blisters, pustules, hemorrhage, crusts, scales and erosions, and also with pruritus or even pain. Skin lesions in acute irritant CD are predominantly sharply bordered in the areas of contact (distant spread does not occur) and usually asymmetric. On the other hand, chronic irritant CD is characterized by diffuse or localized lesions with typically poorly defined erythematous scaly patches and plaques, dryness of skin, lichenification and desquamation.

Irritant skin lesions commonly occur on the back of the hands and forearms (palms have greater intrinsic resistance). The disease is often asymmetric, with the dominant hand more affected. As the disease persists, lichenification and fissures develop, with possible nail damage (paronychia with nail dystrophy, pitting, oil spots, etc.). Distant reactions usually do not occur and the disease is usually limited to the areas of repeated contact [32].

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 [29].

1.4 Diagnoses

The diagnosis of ICD is made by exclusion, based on accurate, thorough medical history and careful clinical examination of the patient. It is important to obtain exposure history from work, from home and hobbies. 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. Therefore, it is important to exclude both type I and type IV allergies before making a diagnosis of ICD, especially in an occupational setting [33].

Skin scrapings of cutaneous lesions (direct microscopy) may help exclude scabies or may reveal fiberglass fibers as a cause of a patient's pruritus. To asses for scabies, superficial epidermal cells can be scraped lightly from the skin surface with a No. 15 blade. Skin scrapings can be evaluated with light microscopy on a glass slide containing mineral oil.

Patch tests with a standard tray and a special environmental allergen will verify or rule out allergic components of contact dermatitis. Also correct, pure and stable patch test material is essential for accurate patch test results and can form the basis for prevention of allergic contact dermatitis through screening [34].

If he is a worker, a visit to the workplace may be needed to identify physical irritants such as temperature, humidity or mechanical irritants and/or chemical allergens and irritants. Workplace provocation test can be carried out if the patch test is still negative and ACD is suspected [35].

1.5 Management

Compliance with avoidance is important. The key to avoidance is proper evaluation and detection of causative allergen. Wear appropriate clothing to protect against irritants at home and in a work environment [36].

High-potency topical corticosteroids, e.g., clobetasol propionate 0.05% cream, may be used to reduce the inflammation [37]. As a general rule, high-potency corticosteroids should not be used on thin skin, e.g. face, genitals, intertriginous areas, to avoid the risk of skin atrophy. Antihistamines such as hydroxyzine and cetirizine are recommended to control pruritus. Systemic steroids are advised in severe cases but should be tapered gradually to prevent recurrences. Friction should be avoided as well as the use of soaps, perfumes, and dyes. Emollients are used for hydrating the skin [38].

1.6 Prognosis

The prognosis of patients with contact dermatitis depends on the cause and lifestyle. Isolated cases usually resolve if the offending agent exposure is discontinued. Those who do not remain compliant and continue to wear jewelry with metal or are exposed to plants because of lifestyle generally tend to have a chronic course. Relapses are very common.

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 [39].

2. CONCLUSION

Although ICD is more common in certain occupations, genetics and environment play significant roles in its development. Management consists of irritant identification and avoidance with regular emollient use.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our

area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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