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Review

Management of contact dermatitis

Sultan T. Al-Otaibi *, Hatem Ali M. Alqahtani

Department of Family and Community Medicine, University of Dammam, Dammam, Saudi Arabia

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Abstract

Skin disorders compromise more than 35% of all occupationally related disorders. Most of these are contact dermatitis as a result from contact with a chemical substance. Contact dermatitis can be either irritant or allergic type. Each type has a different mechanism while the clinical presentation is the same. Management of contact dermatitis must include both medical treatment and workplace modifications as appropriate to reduce exposure to the causative agents. Physicians should be aware of this preventable medical condition.

Keywords: Dermatitis; Contact; Management

Contents

* Corresponding author at: University of Dammam, PO Box 2208, Al-Khobar 31952, Saudi Arabia. Tel.: +966 13 8948964; fax: +966 13 8645612.
E-mail address: otaibist@hotmail.com (S.T. Al-Otaibi).
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1. Introduction

Skin is the most commonly injured organ in industry today, whereas skin disorders compromise more than 35% of all occupationally related disorders (Diepgen and Kanverva, 2006). Contact dermatitis is the most common occupational disease in many countries. A challenge is that contact dermatitis is under reported as work related illness. Health care worker should be aware of this occupational illness. It will also require appropriate diagnosis and management.

Contact dermatitis (CD) is defined as a reactive eczematous inflammation of the skin which occurs after the direct contact with a chemical but occasionally by biologic or physical agents (Holness, 2014; Chew and Maibach, 2003). Contact dermatitis can be either due to irritation from direct exposure to a substance, irritant contact dermatitis (ICD) or as a result of exposure to allergic substance, allergic contact dermatitis (ACD) (Chew and Maibach, 2003; McFadden, 2014). ICD is the most common form of occupational skin disease which accounts for nearly 80% of CD (McFadden, 2014; Cahill et al., 2004; Lau et al., 2011).

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 irritant substance (such as solvents, water, soap, detergents, acid, alkali, etc.).

ACD includes contact urticaria which is type I hypersensitivity as an immediate but transient localized swelling and redness that occurs on the skin after direct contact with an offending substance such as latex, food (beans, egg, fish), antibiotics (penicillin, neomycin), ingredients of cosmetics and medicaments such as Balsam of Peru and Benzoic acid. ACD also includes contact dermatitis which is type IV hypersensitivity (dermatitis begins within 24–48 h after contact) e.g. chrome, nickel, epoxy resin, rubber additives, etc. Sometimes ACD could be photoallergic that requires UV light after exposure to allergen. Atopic skin remains the single most important risk factor in an occupational setting (Holness, 2011; Holness et al., 2013; Diepgen, 2006; Keegel et al., 2009; Ibler et al., 2012; Luk et al., 2011; Lysdal et al., 2012).

2. Mechanism

The mechanism of contact dermatitis depends on its type (Cahill et al., 2004; Keegel et al., 2009).

ICD is characterized by skin damage which could be mild to severe depending on the causative agent as a result of direct, local, toxic effect on the cellular elements of the skin. This leads to removal of the lipid film, denaturation of keratin of the skin, release of lysosomal enzymes and inflammatory response.

Contact urticaria occurs through either allergic (immunologic) or non-allergic (non-immunologic) mechanism. Allergic contact urticaria is mediated by an IgE mechanism leading to a cascade of events causing inflammation of the skin. In non-immunologic contact urticaria a direct effect on the blood vessel wall occurs with release of vasoactive substances leading to hives (McFadden, 2014).

Allergic contact dermatitis arises from a cell mediated delayed hypersensitivity reaction. Sensitization is initiated after an agent or hapten combines with skin protein to form a complete antigen. This antigen is processed by epidermal Langerhans cells, then T lymphocytes interact with Langerhans' cell processed antigen. Later on T lymphocytes release lymphokines which serve as mediators of inflammation (Holness, 2014).

3. Clinical presentation

It is impossible to differentiate between ICD and ACD clinically (Chew and Maibach, 2003).

However, acute ICD is manifested by red, swollen, itchy, painful and ulcerated skin. Hydrofluoric acid burns are associated with hypocalcemia, and hypomagnesemia. While chronic ICD is characterized by eczematous skin eruption, erythema, dryness, cracking and fissuring of the skin. Secondary infection may supervene. It mainly involves the back of the hands including the fingers and the finger webs and subsequent involvement of the palm (Ibler et al., 2012; Luk et al., 2011; Lysdal et al., 2012).

Contact urticaria appears as hives occurring within a few minutes up to an hour of skin exposure to the offending agent. Allergic contact dermatitis is characterized by redness, itching and scaling of the skin at the site of the contact, but very frequently involvement of the eyelids