CASE REPORT

Contact dermatitis: A case study

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Abstract: Chronic exposure to the contact material can cause cutaneous xerosis, thickening, and scaling, or pruritic, erythematous, indurated, scaly plaques. Allergic contact dermatitis is an inflammatory cutaneous response that follows immunological sensitization to the contact substance. Over 4000 contact allergens have been identified, and the reaction is a type IV reaction mediated by T cells (delayed hypersensitivity response). Since prevention is the key to treating this illness, it should be of the highest significance to identify occupational risk factors, reduce sources of antigen, and provide adequate protective equipment. This study discusses the risk factors and outcomes of allergen exposure in the workplace by presenting the example of a 28-year-old male student who acquired pruritic eczematous lesions on the lower limbs.

Introduction

The inflammatory cutaneous response known as allergic contact dermatitis (ACD) is typified by pathological alterations in the epidermis [1]. This cutaneous condition is mostly caused by a type IV reaction (delayed-type hypersensitivity response) mediated by T cells. It takes immunological sensitivity to the touch substance for allergic contact dermatitis to develop [2-4]. So far, almost 4000 contact allergens have been identified. Contact allergens with low molecular weight are usually responsible for delayed-type allergic reactions. According to studies, metals like nickel, preservatives like parabens, perfumes, adhesives like epoxy resins, and topical medicines are some of the allergens that cause reactions the most often [5, 6]. It is yet unknown how common and severe ACD is in the general population. Information is usually extrapolated from research about skin problems associated with the workplace. Nevertheless, research indicates that in affluent nations, cutaneous symptoms account for up to 30.0% of all occupational disorders, with irritant and contact dermatitis accounting for more than 90.0% of cases of occupational skin problems [7]. This case report represents practical observation with relevant important aspects of contact dermatitis.

Observations: The instance of a 28-year-old male student who smokes and has a personal history of dyslipidemia and arterial hypertension. The patient came to our clinic complaining of itchy, lower limb eczema sores that started around two years ago. The observation disclosed a few months of experience working as a chemical technician in an aluminum production plant, resulting in extended exposure to workplace dangers including acetone, epoxy resins, and various acids (nitric, hydrofluoric, and hydrochloric acid). The individual has had yearly physical examinations, along with consultations in ophthalmology and lung health. The condition lesions are shown in **Figure 1**.

After receiving systemic medication that included topical corticosteroids, antihistamines, and 16 mg/day of methylprednisolone, the patient reported a reduction in his symptoms. He quits taking systemic

methylprednisolone after two months so that she may do Patch testing. The allergy tests indicated multiple allergies for several substances such as nickel, epoxy resin, palladium chloride, lanolin wool alcohols and bisphenol A glycerolate dimethacrylate. A diagnosis of allergic contact dermatitis was established after taking into account the results of both the allergy Patch testing and the histological analysis after six months. It is crucial to identify and ascertain the existence of occupational risk factors (long working hours, heavy instruments) to establish the occupational genesis of this illness.

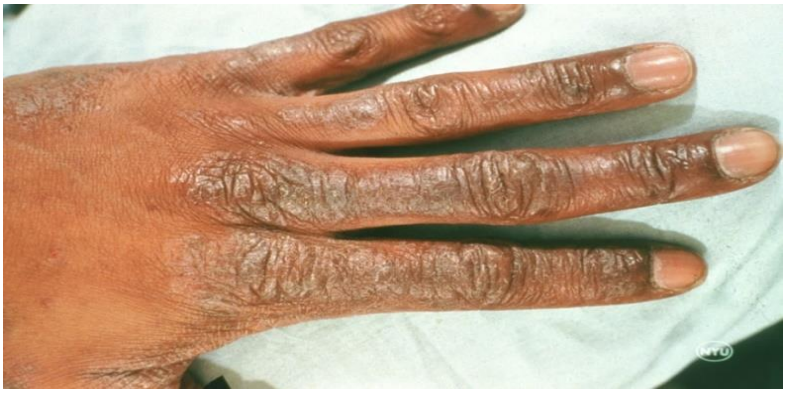

Figure 1: Lesions of the hands of the patient

Discussion

T cells operate as a mediator for a delayed-type hypersensitivity reaction that leads to ACD. This response happens when a dangerous chemical comes into touch with the skin; it affects those who have already become sensitive to that specific substance in the past. It is mostly an occupational illness, and managing it can be difficult due to resistance [8]. Allergens often need only trace amounts to cause an allergic contact response. Thus, a novel contact allergen will activate effector cells specific to the antigen, which will in turn activate and release pro-inflammatory cytokines such as tumor necrosis factor-α and interleukin-1. In most cases, the inflammatory response occurs 48-72 hours following exposure and presents clinically as contact dermatitis [9, 10]. An itchy, red, indurated, scaly plaque is one of the clinically observed acute lesions of ACD. Vesiculae and bullae may be seen in extreme situations. In contrast, long-term exposure to allergens causes chronic illness that manifests as thickening, scaling, and xerosis of the skin. Later on, cracks and lichenification may form. Although broad distribution happens frequently, lesions in ACD are typically limited to the skin's exposed parts. As opposed to irritant contact dermatitis (ICD), cutaneous manifestations in allergic contact dermatitis have imprecise edges and a tendency to scatter, these features becoming useful in differentiating the two similar diseases. Both the clinical signs and the patient history are important in the diagnosis of ACD. However, the identification of the relevant contact allergens is done by the use of an epicutaneous patch test. But most of the time, a histological examination is equally crucial to the procedure, exposing several suggestive findings such as dilated arteries in the stratum papillare and upper stratum reticulare, perivascular lymphocytic infiltrates, eosinophils, and epidermal spongiosis.

Conclusion: The main way that prevention is achieved is by teamwork and policies designed to reduce contamination, particularly in the workplace. As a result, the occupational physician plays a crucial role in diagnosing symptoms brought on by exposure to workplace dangers. Thus, it should be of the highest significance to identify the risk factors in the workplace, reduce antigen sources, and provide suitable protective equipment.

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