CASE REPORT

Airborne contact dermatitis caused by epoxy resin: case report

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INTRODUCTION

Airborne contact dermatitis (ACD) is a skin injury characterized by acute or chronic dermatoses caused by allergic or irritative substances in gaseous, liquid or solid states. When such substances are released into the air, they generate different kinds of lesions when exposed to dermal tissue. There is no reliable information in regards to an incidence of this type of dermatitis, due to an underestimated frequency. ACD presents different types of lesions: allergies produced by different types of woods, insecticides, plants, plastics; irritant lesions by fiberglass or charcoal, ethylene oxide, mustard gas; photo-urticaria induced by chlorpromazine; phototoxicity caused by psoralsens; contact urticaria provoked by latex and epoxy resins; photosensitive lesions produced by thioureas or pesticides; purple skin disease by epoxy resins; paresthesias induced by pyrethroid; acneiform lesions caused by epoxy resins and chlorinated hydrocarbons; pustulosis subcorneal injuries provoked by trichlorethylene; erythema multiforme caused by tropical timber; lichen plaques by color developers; fixed erythema lesions by tropical wood; dyspigmentation caused by fragrances; exfoliative dermatitis by trichlorethylene; telangiectasia’s produced by fluorochemical products. The overall prognosis is noteworthy and the prevention at work using specific creams and protective clothing for the activity is the only etiopathogenic treatment. In connection with symptomatic treatment, using topical steroids is recommended. Pruritus should be treated with systemic antihistamines while antibiotics should only be used in case of bacterial infection.
Airborne contact dermatitis

An individual’s occupation. ACD is produced when a sensitizing substance spreads through the air and is then deposited predominantly on the face as well as the back of hands and arms; it can also adhere to the lower extremities, particularly on females. Clinical evolution is characterized by an itching or burning sensation, and erythematous lesions are often accompanied by signs of scratching; injuries vary depending on the etiopathogenic agent. The differential diagnosis should be established with psoriasis, seborrheic dermatitis, dermatophytosis and atopic dermatitis; therefore, different mechanisms can coexist, thus complicating proper diagnosis. However, associating the clinical progression with a positive patch test on D4 for BADGE, could confirm the diagnosis of ACD induced by epoxy resins. Treatment for ACD depends on which etiologic agent conditions the dermatoses, that said, the use of protective creams and protective clothing at work is recommended. Usually ACD responds appropriately with the use of oral antihistamines and topical corticosteroids, as did in our case. We conclude there is an undeniable risk to develop such condition in different professions, especially in areas where workers are exposed to different sensitizing agents. Consequently a worker with compatible skin lesions, you should think getting tested for a possible ACD diagnosis.

REFERENCES


CASE REPORT

A 21-year-old male presented with a 1-week history of recurrent pruritic rash on his face. No medical history of interest was provided. Skin reactions appeared when he entered the clean room laboratory, of which walls are painted with a substance that contains bisphenol A epoxy resin (BADGE); despite the area being provided with a continuous airflow system. Upon physical examination, acneiform, erythematous, popular, lichenified lesions were detected on the exposed areas of the face (Figure 1). The patient presented no other general symptoms. The diagnosis was made using patch test, which was positive for BADGE on D4 (according to International Contact Dermatitis Research Group guidelines). The patient was diagnosed with ACD caused by BADGE. The patient was then treated with oral antihistamines (levocetirizine 5 mg/24h orally) and topical steroids (clobetasone 0.05%/day). Skin lesions cleared within 2 days after the initiation of treatment.

DISCUSSION

The case presented shows how it is possible to identify ACD, which demonstrates a direct correlation with

Figure 1. Inflammatory acne lesions located on the exposed parts of the patient’s face.