and duration of skin exposure to PPD are important factors in the elicitation of contact dermatitis. Our current data show that patients with a history of using temporary black henna tattoos are often highly sensitized to PPD after exposure to such high concentrations and require only very low concentrations of PPD for elicitation of an allergic response. As the current practice of using PPD 1% pet. often results in unacceptably strong blistering reactions, we propose patch testing with PPD 0.01% pet., which can elicit a sufficiently positive reaction in a highly sensitized individual. If this is negative at the 1st reading, the concentration of PPD can then be stepped up to 0.1%, or even 1% to ensure that an allergic contact dermatitis from PPD is not missed.

References

Case Report
A 40-year-old man was referred for patch testing. He reported an 8-month history of a rash that began as blisters on his feet and then spread to involve his hands. He had been treated first with antibiotics and then with prednisolone. Further flares occurred, which required treatment with topical corticosteroids and narrow band UVB light, which improved his condition.

He reported wearing leather boatshoes (made in China) intermittently over a 3-month period and had noticed dye leaking from the shoes onto his skin. Patch testing showed strong reactions to chromate (+++) and cobalt (+). The patient was diagnosed with allergic contact dermatitis (ACD) from chromate and advised to avoid chrome-tanned leather shoes.

He was reviewed 3 months later and reported that his foot dermatitis was much improved, but that he had developed dermatitis on his left anterior thigh, at the site of contact with his leather wallet. He was subsequently reviewed 3 months later, and reported that, after changing to a plastic wallet, the dermatitis on his thigh had completely resolved for 2 months, though it had recently flared again in the same localized area on his thigh, despite no known leather exposure.

Discussion
Chromate is a common allergen, and leather products are an important source of chromate exposure (1). A recent study by Moed et al. (2) investigating the phenomenon of local skin memory and flare-up reactions, reported that, after clinical recovery from an ACD reaction, CD4+ CCR10+ memory T cells apparently persist locally. This may explain recurrent symptoms in a previously affected site, as in this case. Another possible cause for the flare of the rash may be oral ingestion of chromate, which has been previously reported (3, 4).

References

Localized recurrence of chromate dermatitis

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Key words: allergic contact dermatitis, chromate, memory T cells, oral provocation.
greenhouses. A few years later, she had had to discontinue wearing rubber gloves and boots, because of a burning sensation and eczema at contact sites.

Examination showed psoriatic plaques over the patient’s trunk and limbs. There was confluent erythema, hyperkeratosis with scaling and fissuring on the palms of the hands and lichenification on the dorsa. The distal forearms were also involved, with parakeratotic papules scattered along the borders of the dermatitis. The patient underwent a standardized diagnostic procedure for farmers’ occupational diseases (1).

Biopsy from the involved forearm skin showed granulocytic infiltration of dermal papillae; there were foci of parakeratosis and spongiosis in the epidermis, the granular layer being preserved. This picture thus comprised features of both psoriasis and dermatitis. Prick and intracutaneous tests with environmental and occupational allergens were all negative.

Patch tests included European standard series (Chemotechnique, Malmö, Sweden), rubber series (Jaworski, Katowice, Poland) and pesticide series (Institute of Agricultural Medicine, Lublin, Poland). A positive reaction was recorded to thiuram mix 1% pet. on D3, D4 and D7. The test reaction corresponded with a + + score – there was pronounced erythema and infiltration, though pustules were present instead of typical vesicles.

Within 2 weeks, a parakeratotic plaque of psoriasis had developed on the positive patch test site. This Köbner (isomorphic) phenomenon was consistent with the above-mentioned parakeratotic patches at the borders of the eczema.

Besides rubber gloves and boots, seed protectants were identified as a major source of thiuram in the patient’s work environment. She was regularly treating vegetable seeds with such protectants, which according to the products’ labels consisted of up to 32% thiuram.

Discussion

Köbnerizing occupational contact dermatitis from thiuram has previously been described only once – in a nurse allergic to rubber gloves (2). Nurses’ gloves were also the cause of recurrent erythroderma in a thiuram-allergic patient with psoriasis (3).

A pustular patch-test reaction to thiuram has previously been seen in a rubber factory worker with occupational pustulosis palmaris; in that case, however, there was no pre-existing psoriasis and no Köbnerization (4). Allergy to thiuram is relatively frequent among farmers – it was found in 7% cases of disabling occupational dermatitis (5).

The present case, besides the rarity of the clinical picture, clearly shows that seed protectants and seeds treated with those chemicals are relevant sources of thiuram in agriculture and horticulture.

References


Key words: allergic contact dermatitis; benzyl alcohol; diclofenac; medicaments; polyethylene glycol monomethyl ether 350 (PEGMME 350); Solaraze® gel.

Case Reports

Case no. 1

A 65-year-old woman with disseminated superficial actinic porokeratosis developed contact allergy to stearyl alcohol in Efudix cream®. Thereafter, Solaraze® gel (Shire Pharmaceuticals, Basingstoke, UK) was prescribed, but she began to develop further contact dermatitis within a few weeks of starting to apply it. Patch tests were performed with the ingredients of Solaraze® gel, provided by the manufacturers, at the following concentrations: diclofenac 1% aq., benzyl alcohol 5% pet., polyethylene glycol monomethyl ether 350 (PEGMME 350) 1% and 5% aq., sodium hyaluronate 0.5%, 1% and 5% aq. and Solaraze® gel 5% and 10% pet. Positive reactions were seen to diclofenac 1% at D2 (+) and D4 (+++); Solaraze® gel 5% pet. at D4 (+) and Solaraze® gel 10% pet. at D2 (+) and D4 (+).

Case no. 2

Solaraze® gel was prescribed for a 60-year-old woman who had actinic keratoses on the right shin. After a single application, vesicular eczema was noted at the application site. Patch tests with the aforementioned ingredients demonstrated positive reactions to PEGMME 350 1% aq. at D4 (+), PEGMME 350 5% aq. at D4 (+++), Solaraze® gel 5% pet. at D4 (+), Solaraze® gel 10% pet. at D4 (++) and Solaraze® gel (as is) at D4 (++++).

Case no. 3

An 80-year-old man used Solaraze® gel intermittently for 6 months to treat actinic keratoses on the face and hands. He developed a pruritic, eczematous eruption at the sites of application. Patch tests showed positive reactions to benzyl alcohol 5% pet. at D2 (+) and D4 (++) and Solaraze® gel (as is) at D2 (+) and D4 (+).

15 control subjects showed no reaction on patch testing with Solaraze® gel and all its constituents.

Contact dermatitis from 3 different allergens in Solaraze® gel

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