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Leucoderma after Chinese sofa dermatitis

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Dimethylfumarate is a potent contact sensitizer used for its antimicrobial and mould-prevention effects (1). An epidemic of sofa dermatitis affecting many hundreds of patients occurred because of sachets containing dimethylfumarate, which was used to inhibit mould in furniture while it was being stored or transported (2–5). We describe a case of allergic contact dermatitis caused by dimethylfumarate present in a leather sofa. The condition was followed by persistent depigmentation after the resolution of eczematous lesions.

Case Report

A 49-year-old man was referred with a widespread pruritic erythematous dermatitis lasting for 2 weeks. It had started on the thighs, and then extended to the back, buttocks and arms, at skin sites in contact with a sofa purchased 6 months previously (Fig. 1). A diagnosis of allergic contact dermatitis resulting from a leather sofa was made. The dermatitis healed with oral prednisone and after avoidance of contact with the sofa, but the patient developed hypopigmented areas on the pre-existing dermatitis 4 weeks after the resolution of the lesions (Fig. 2). A biopsy of the depigmented skin showed an absence of melanocytes. We performed patch tests with the baseline Spanish contact dermatitis research group series, textile dyes and footwear series (Chemotechnique Diagnostics, Vellinge, Sweden). A non-relevant positive result for wool alcohols (D2+/D4++) was observed. Patch testing with dimethylfumarate 0.01% pet. was positive (D2++/D4++) (Fig. 3). No depigmentation at

the patch test site was observed. The depigmented area is still present after 2 years of follow-up. There was no personal or family history of vitiligo or other autoimmune diseases.



Fig. 1. Extensive erythema on the back.



Fig. 2. Leucoderma secondary to contact dermatitis from exposure to dimethylfumarate.

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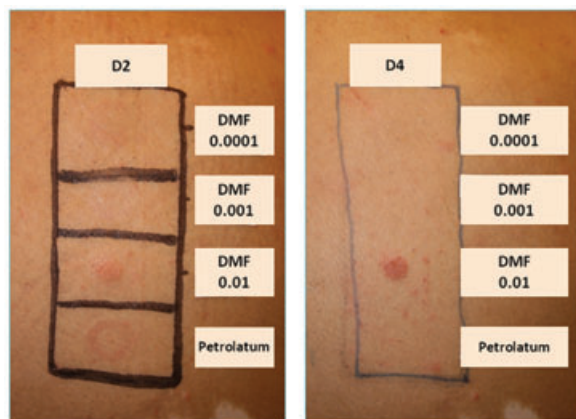


Fig. 3. Patch tests reactions at D2 and D4 to dimethylfumarate.

Discussion

Contact leucoderma is characterized by the loss of pigment on exposure to certain chemicals that produce specific and selective melanocytopenia. It can occur after a preceding allergic dermatitis or because of toxic depigmenting effects (6). The onset of dermatitis in our patient was 6 months after the sofa was purchased; therefore, we believe that it resulted from allergic contact dermatitis caused by dimethylfumarate. The exact mechanism underlying this kind of pigmentary change remains to be elucidated. It could be a chemical Koebner phenomenon in vitiligo (the chemical acts as

a triggering factor in initiating autoimmune responses against melanocytes) or result from the cytotoxic or cytostatic effects on the melanocytes (7). Contact eczema and subsequent depigmentation has been previously reported in different settings, either occupationally, or following private use of sensitizing agents such as phenol and catechol derivatives or sulfhydryl compounds (5, 8). In our case, the lack of a history of vitiligo makes this possibility less likely. Although vitiligo could not be excluded, the repeated exposure to dimethylfumarate for 6 months, the localization of the acquired depigmented patches to the area of contact dermatitis and the temporal association are more suggestive of leucoderma (6). Post-inflammatory hypopigmentation has more subtle, ill-defined macules; complete depigmentation does not occur, and the hypomelanosis immediately follows resolution of the primary process. We regard this as a case of contact leucoderma after dimethylfumarate dermatitis. This might result from a melanocytotoxic effect that is independent of its sensitizing potential. It seems that genetic susceptibility and the nature of the chemical were important contributory factors in determining the severity of the lesions. This is the first reported case of contact leucoderma caused by dimethylfumarate in a Chinese sofa. Dimethylfumarate should be added to the list of substances capable of inducing depigmentation. Fortunately, in May 2009, the European Commission banned products containing dimethylfumarate from being placed on the market (European Directive 2009/251/EC) (9).

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