

zole, and between isoconazole and tioconazole (3, 11, 14). Cross-sensitivity has rarely been reported between clotrimazole and other structures, bifonazole and other structures, or ketoconazole (also a phenylethyl imidazole) and other structures (3).

The case reported here is unique in that the patient showed positive reactions to croconazole and itraconazole, besides clotrimazole, and had apparently used only 1 azole. Croconazole, belonging to the phenmethyl imidazoles like clotrimazole, was first marketed in Japan and Korea in 1986 and was introduced to the European market only recently. There are a number of publications on hypersensitivity to it (8, 9, 21) and cross-sensitivity between croconazole, isoconazole, bifonazole, and clotrimazole was reported in 1988 (8). Croconazole seems to owe its position as the only strong sensitizer among the azole derivatives to its methylene group (7).

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Contact dermatitis due to diethyl sebacate

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Key words: allergic contact dermatitis; diethyl sebacate; patch testing technique; emulsifiers; topical medications. © Munksgaard, 1999.

A 28-year-old woman was seen in June 1998 with pruritic erythema on both feet. She had started using an antimycotic over-the-counter preparation, 0.2% pyrrolnitrin and 0.4% clotrimazole ointment (Pyroace W[®], Fujisawa Co. Ltd. Osaka, Japan) about 2 weeks before, and a few days later developed erythema. She then changed to New Eurax G[®] (OTC) (Fujisawa), but the eruption did not improve.

We performed patch tests with Pyroace W[®] ointment (as is) and New Eurax G (as is) initially. Positive reaction was seen to Pyroace W[®] ointment (as is). Secondly, the patient was patch tested with 1% and 10% pet. of pyrrolnitrin, clotrimazole, and 6 other constituents (crotamiton, white beeswax, isopropyl myristate, polyoxyethylene lauryl ether, and diethyl sebacate). Only diethyl sebacate showed a positive response on D2 at 10% pet. and on

Table 1. Patch test results

		D2	D3
Pyroace W [®] ointment		++	++
New Eurax G [®]		—	—
diethyl sebacate	30% eth.	++	++
	10% eth.	++	++
	1% eth.	—	+
	0.1% eth.	—	—
ethanol		—	—
diethyl sebacate	10% pet.	++	++
	1% pet.	?+	+
	0.1% pet.	?+	—
petrolatum		—	—

D3 at 1% and 10% pet. Further, we did patch tests with diethyl sebacate 0.1, 1, and 10% pet. and 0.1, 1, 10, and 30% eth. Diethyl sebacate 10% pet., 10%, and 30% eth. were positive at D2, and 1%, 10% pet., 1, 10, and 30% eth. were positive at D3 (Table 1). Patch tests with 0.1, 1, and 10% pet. and 0.1, 1, 10, and 30% eth. of diethyl sebacate in 6 normal subjects showed no erythema or papular response at D2 or D3.

Discussion

Although emulsifiers are rare sensitizers (1), stearyl alcohol (2) stearic acid, and glyceryl stearate (3) are known to cause allergic contact dermatitis. Diethyl sebacate, an emulsifier, is widely used in cosmetics (4, 5) and surfactants (6) in Japan. In the USA, it is also used as a synthetic flavor in food (7). Only 5 cases of contact dermatitis due to diethyl sebacate in topical medicaments have been reported (8–11). Pyroace W[®] contains 15% diethyl sebacate.

The former 3 cases (10, 11) showed positive patch tests to high concentrations of diethyl sebacate, 20–30% eth.

In our cases, patch tests with lower concentrations (1 and 10%) of diethyl sebacate were positive, in both petrolatum and ethanol. Patch tests are usually performed with 0.2–2.7 % pet. of common emulsifiers (1). Our results suggest that 1 and 10% pet., and 1 and 10% eth., are appropriate for patch tests with diethyl sebacate.

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Contact dermatitis from flavourings in chewing gum

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Key words: allergic contact cheilitis; allergic contact dermatitis; flavourings; chewing gum; product labelling; eugenol. © Munksgaard, 1999.

Allergic contact cheilitis, including circumoral rashes, has been reported from lipsticks, sunscreens, foods, toothpastes and mouthwashes (1). Allergic contact dermatitis has been attributed to flavourings in such products (2–4), including chewing gum.

Case Report

A 10-year-old girl presented with a 6-month history of an intermittent circumoral rash. Her lips and oral mucosa were unaffected. There was no history of atopy. Examination revealed a well-circumscribed light brown perioral

rash, suggestive of post-inflammatory hyperpigmentation, prompting a diagnosis of lip licking. Later, perioral eczema was noted. The patient was patch tested to the European standard series and a facial series (Table 1).

She was further patch tested to a bakers series (Table 2). In the meantime, she was advised to avoid highly flavoured foods and drinks, including chewing gum and toothpaste.

She was able to re-introduce her original toothpaste, Signal[®], without adverse affect; however, chewing her favourite gum caused flare of her rash. The manufacturers of the toothpaste confirmed that neither eugenol