

Allergic contact dermatitis from formaldehyde mimicking impetigo and initiating rosacea

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Two cases are presented in which other diseases (impetigo and rosacea) had initially been suspected until the

final diagnosis of allergic contact dermatitis caused by formaldehyde was established.

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Case Reports

Case 1

A 21-year-old woman, without a family or personal history of atopy or skin disease, had worked as hair-dresser for 3 years when bullous lesions appeared on both cheeks. She had suffered from the facial dermatitis

for 3 months when she was first seen at the General Dermatology Department; previously, herpes simplex was suspected. Impetigo contagiosa was diagnosed, and a topical corticosteroid/antibacterial (Kenacutan®; Bristol-Myers Squibb, Solna, Sweden) was given. Bacterial cultures were negative. The treatment was effective for a week, but, when one new bullae reappeared on dry, erythematous skin, another topical corticosteroid was administered. Twenty days after the first visit, highly pruritic, eczematous lesions with crusts appeared on the face (Fig. 1), and, at this point, even lupus erythematosus was suspected. The patient was prescribed Betnovate® with chionoform (GlaxoSmithKline, Solna, Sweden). One month later, she had not improved. Hence, she was referred to us and seen 3 days after referral. It was then pointed out by the patient that all skin lesions had started since she had been using a new facial day cream, which had been emptied until the visit. Despite the use of topical corticosteroids, pustules were noted on the forehead, cheeks, and eyelids.

Patch testing with the Swedish baseline series, a hairdresser series and a cosmetic series (Chemotechnique Diagnostics, Vellinge, Sweden) and various work materials, such as shampoos, was performed according to ICDRG guidelines, with Finn Chambers® (diameter, 8 mm; Epitest, Tuusula, Finland) on Scanpor® tape. The dose for the preparations in aq. was 15 µl (1). At the first patch test reading on day (D) 3, there were no reactions. On D6, 1 day before the intended second reading, the patient consulted the general dermatologist because of severe exacerbation of her face lesions (Fig. 2), and was prescribed systemic and topical corticosteroids and antibiotics along with ultraviolet (UV) B treatment. The diagnosis at this point was nummular microbial eczema. A punch biopsy for microscopic examination was taken 1 week later. Owing to a high dose of prednisolone, the reading on D7 was not performed.

Despite this treatment, the patient's dermatitis always became worse when she worked. Three months later, she was again seen by us. At this point, we even suspected an artefact, as the patient claimed that her skin lesions always appeared as soon as she entered the hair salon. A new patch test session was started with the same series as before. Positive test results are shown in Table 1. As the patient reacted to formaldehyde, a chemical analysis with the chromotropic acid method for the presence of formaldehyde in the two shampoos that she had been tested with, and was currently using, was performed. Relevant amounts of formaldehyde were detected in both shampoos used at work and at home (2). The biopsy showed rosacea-like histology with spongiosis. The patient's dermatitis cleared after she avoided



Fig. 1. Allergic contact dermatitis caused by formaldehyde, with dryness and crusting.



Fig. 2. Allergic contact dermatitis caused by formaldehyde, imitating rosacea. The clinical picture had characteristics of both eczema and rosacea, with macules, pustules, crusts, and oozing.

formaldehyde-containing skin care products and after treatment with systemic tetracyclines. Two months later, facial papulopustules reappeared. When her current shampoo and new facial cream were checked, they both contained formaldehyde, in the cream at a concentration equivalent to >40 µg/ml, and in the shampoo at a concentration equivalent to 40 µg/ml. DMDM hydantoin was labelled on the shampoo, but the patient had failed to identify this as a formaldehyde releaser. The cream was unlabelled. Other cosmetics were negative for formaldehyde. The patient remained clear at follow-up 3 months later.

Case 2

A 15-year-old girl without personal atopy or prior skin disease suddenly developed pruritic papular dermatitis

Table 1. Positive patch test reactions

	Day 3	Day 7
Case 1		
Formaldehyde 1.0% aq.	+	?+
Gold sodium thiosulfate 2.0% pet.	++	++
Shampoo A 5% aq.	?+	–
Shampoo B 5% aq.	?+	–
Shampoo B 0.5% aq.	?	–
Case 2		
Formaldehyde 1.0% aq.	+	?+
Quaternium 15	+	?+

?+, erythema (doubtful reaction).

involving the face, body, and hands. The dermatologist suspected nummular dermatitis, and prescribed UVB treatment combined with Betnovate® (GlaxoSmithKline) cream for the body, Emovate® (GlaxoSmithKline) cream for the face, and Ficortril® (Pfizer, Sollentuna, Sweden) cream for the eyelids. Despite this treatment, an exacerbation of the dermatitis was seen in the groins and axillae, with crusts, and impetigo was suspected. Internal antibiotics were prescribed, and the patient was sent for a patch test. While waiting, she had a recurrence, and systemic corticosteroids were given. At presentation, she had suffered from skin lesions for 7 months. She was tested with the Swedish baseline series, and contact allergy to formaldehyde was detected (Table 1). A shampoo that she used was analysed for the presence of formaldehyde, and a concentration of $>40\text{ }\mu\text{g/ml}$ was found. Because she also had perioral dermatitis, she was given tetracyclines, and the dermatitis eventually cleared. The skin lesions on the body and hands vanished when she stopped using skin care products containing formaldehyde or formaldehyde-releasing preservatives.

Discussion

These cases show that allergic contact dermatitis may present in an unusual clinical pattern. In case 1, this had characteristics of both eczema and rosacea (Figs. 1 and 2); the results from patch testing and the biopsy were the clues to the diagnosis: allergic contact dermatitis caused by formaldehyde imitating rosacea. In the second case, we found a shampoo that contained sufficient amounts of formaldehyde to cause skin lesions mimicking nummular dermatitis and impetigo in a patient with contact allergy to formaldehyde.

Formaldehyde is a well-known contact sensitizer, and should be tested at 2.0% aq., as done in the Swedish baseline series since 2014, but the 2 patients were tested before this. All finished products containing formaldehyde or formaldehyde releasers must be labelled with the warning 'contains formaldehyde' when the concentration exceeds 0.05% (3–5). However, as we can see from these 2 cases, contact allergy to formaldehyde is difficult and sometimes impossible to suspect from the patient's history. Allergic contact dermatitis caused by formaldehyde is often chronic, as it is difficult to avoid formaldehyde-containing cosmetic products (4). Mesteach and Goossens published an article describing 2 cases of nail damage mimicking psoriasis caused by nail hardeners containing formaldehyde (6).

Our 2 cases show that it is crucial to give the patients a list of formaldehyde releasers and their INCI names according to legislation, and to explain the importance of scrutinizing labels. However, formaldehyde may be found in cosmetic products even when unlabelled (7). In conclusion, contact allergy to formaldehyde is common, but the clinical picture may be misleading, delaying proper diagnosis and treatment. Contact allergy should be suspected in patients with chronic and treatment-resistant, and recurrent dermatitis.

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