

hydrocortisone-17-butyrate 1%, and budesonide 0.1%, all in pet) of the standard series. In addition to this, the patient was patch tested with our additional corticosteroid series to confirm the ACD diagnosis. After 96 hours, only a positive reaction to hydrocortisone-17-butyrate 1% in pet in this additional series was observed.

Avoiding the use of this corticosteroid cream, the patient had no relapse of the disease in the following 4 months.

Allergic contact dermatitis to topical corticosteroids is a common problem. It was first described in 1959 and observed in up to 6% of patients undergoing patch tests often presenting eczematous reactions.^{1,2} The authors wanted to report a peculiar case of eczematous patches with few vesicles, papules, and pustules mimicking a papular-pustular rosacea. As reported by Jappe et al,^{3,4} ACD due to cosmetics/medicaments can develop on the face, triggering or resembling rosaceiform dermatitis. It can be caused mainly by nickel sulfate, fragrance mix, and thimerosal. Moreover, the presence of pustules was also described in a case of contact dermatitis by corticosteroid by Broesby-Olsen et al.⁵

In conclusion, ACD from topical corticosteroids mimicking a rosacea is a rare manifestation, not hitherto reported. This case emphasizes also the importance of a detailed anamnesis and performing patch test for the formulation of the correct diagnosis.

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Contact Allergy to a Permanent Marker

To the Editor:

Allergic contact dermatitis to permanent markers used in patch tests has been rarely reported. We describe a 32-year-old woman who presented with skin lesions on both feet. Patch testing was performed with standard series of GEIDAC (Spanish Contact Dermatitis and Cutaneous Allergy Research Group) and shoes series (Chemotechnique Diagnostics). After 96 hours, a positive reaction was obtained for potassium dichromate (++), colophony (+++), fragrance mix I (++), and fragrance mix II (+++). At the second reading (96 hours), she showed a vesicular reaction close the patch tests sites marked with a permanent black marker (Impega). Epicutaneous tests were carried out with the ink “as is,” abitol, abietic acid, and fragrance series. Positive results was seen to the ink of the marking pen Impega (++), abietic acid (+++), geraniol (++), oakmoss absolute (+), rose oil absolute (++), and geranium oil Bourbon (++). The manufacturers were asked about the presence of colophony in the permanent black marker, but they did not answer. This marker pen has been used for many years in patients undergoing patch tests in our hospital without adverse reactions.

Colophony and related compounds are the most frequent allergens described as responsible for allergic contact dermatitis to permanent markers. McKenna and Burrows¹ described 2 cases of positivity to colophony associated with an Artline 70 marker pen, which presumably contained this substance. A patient with vulvar eczema showed positivity to the colophony contained in sanitary towels and who also had a positive reaction to the black marker used in the patch tests.² Recently, Romaguera et al³ described a case of allergic eczema caused by a red Edding 3000 marker pen containing colophony. In other different medical uses, abitol was seen to be the allergen responsible in a patient on whom a blue Edding 3000 marker pen was used to delimit an area marked for liposuction; in this patient, positivity was observed for blue, red, and black pens (Edding 3000) due to the abitol they contained, but colophony was negative.⁴ The use of different types of colophony may complicate the study of some patients because a standard series contains only colophony. Moreover, it has been shown that products containing modified colophony cause stronger reactions than the unmodified type.⁴ Finally, a male patient showed active

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sensitivity to a colophony after accidental contact with a Pentel marker.⁵ It has been described that colophony and derivatives have been used as adhesive in several inks (ceramic, marking, and printing) and support to make sharp lines.

The origin of the sensitivity to colophony in our patient was her shoes; she was also sensitive to the black ink of the Impega marker pen used and to abietic acid. An adhesive strip (Steri-Strip) was used in the corners of the patch tests in a second round of epicutaneous tests.

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Blue Contact Urticaria

To the Editor:

Intraoperative lymphatic mapping has become common practice for breast cancer, melanoma, and other malignancies.¹ Blue dyes (BDs) are commonly used for sentinel node biopsy. Patent blue (alphazurine 2G) is a rosaniline dye of the triphenylmethane type.² Currently, patent BD is the first-line agent to sentinel node mapping. The reported incidence of allergic reactions to these dyes is 1% to 2%, ranging from mild local reactions to anaphylactic shock.³ Here, we report a patient with

breast cancer who developed blue urticaria a few minutes after intradermal injection of patent blue for sentinel node biopsy.

We report a 43-year-old woman with a medical history of obesity, hypertension, and depression. Her treatment included alprazolam, escitalopram, furosemide, and losartan. She did not have any prior personal or family history of asthma, allergies, or atopic dermatitis. She was evaluated to have adenocarcinoma on the left breast on April 2010, and she was scheduled for radical mastectomy accompanied by sentinel node biopsy. When surgical process commenced, she was anesthetized, and no complications were observed. Then, intradermal patent blue was injected to establish sentinel node. Injection was performed close to the malignant parenchyma. In a few minutes, her left breast started to become diffusely bluish, and immediately, blue hives were easily recognized. It started surrounding site of injection, but it was disseminated in approximately less than 20 minutes. Blue hives were bilateral and mostly symmetric. Arms, chest, breasts, abdomen, pelvis, and thighs were involved (Fig. 1). It is important to note that she remained hemodynamically stable. She maintained an oxygen saturation of 96%, and neither bronchospasm nor hypotension was observed. Immediate intravenous therapy consisted of 60 mg of methylprednisolone and 1 vial of dextrochlorpheniramine. The surgical process was stopped, and monitoring of blood pressure, oxygen saturation, and heart rate was continued. Surprisingly, blue hives progressively disappeared in approximately 2 hours, and no recurrences have been noted to date. She did not experience other complications. Subsequently, she was referred to our institute's allergy department. Both intradermal and prick tests were positive for patent blue and methylene blue. Hence, allergic hypersensitivity to these agents was confirmed, and she was evaluated to have contact urticaria due to patent BD.

Blue hives have rarely been described.⁴ However, its pathogenesis seems to be widely accepted. The immediate systemic absorption of the dye may cause blue hives not only limited to the zone of injection. As in our patient, urticaria is the consequence of the contact between the circulating blue agent and serum protein complexes.¹ It may be enhanced by possible accidental intravascular injection. Vasoactive mediators such as histamine, leukotrienes, and prostaglandins are released. This increased vascular permeability allows fluid and protein to leak into the superficial dermis, causing urticarial edema.⁴ Finally, the complexes blue-serum proteins reach the skin through the enhanced permeability, and blue hives are the result.⁵

It is difficult to identify the cause of the previous exposure to the agent because BDs are found in large amounts in medical devices, lotions, detergents, paper, and agriculture products and they are even being used as food coloring. In addition, previous antigen sensitization may be cutaneously, orally, or through inhalation. Thus, the likelihood of previous sensitization is high. Clinically, some authors suggested 4 stages for immune contact urticaria.⁶ According to them, our patient presented with stage 2, in which a generalized urticaria with no extracutaneous involvement occurred.

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