

REFERENCES

1. Caputo R, Grimalt R. Advances in the research on the histiocytic syndromes X and non-X. *Current Dermatol* 1995; 2: 51–54.
2. Hurwitz S. The skin and systemic disease in children. Chicago: Year Book Medical Publishers Inc., 1985.
3. Otis CN, Fischer RA, Johnson N. Histiocytosis X of the vulva: a case report and review of the literature. *Obstet Gynecol* 1990; 75: 555–558.
4. Bonifazi E. Should Langerhans cell histiocytosis always be treated? In: Gelmetti C, ed. *Pediatric dermatology: controversies and current concepts*. New York: DM Medical Publishing, 1994: 155–166.
5. Hansen RC, Bangert J. Update on childhood cutaneous histiocytosis syndromes. *Curr Dermatol* 1996; 3: 141–144.
6. Donadieu J (the French Langerhans Cell Histiocytosis Study Group). A multicenter, retrospective survey of Langerhans cell histiocytosis: 348 cases observed between 1983 and 1993. *Arch Dis Child* 1996; 75: 17–24.
7. Shian WJ, Shu G, Chu HY. Langerhans cell histiocytosis: a 10-year review. *Acta Paediatr Scand* 1994; 5: 385–390.

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Cross-allergy to Latex and Spinach

Sir,

Immediate hypersensitivity to latex proteins is frequent and affects approximately 10% of medical personnel in operating rooms and intensive care units. Cross allergy to latex proteins and food allergy is well known for avocados, bananas and kiwi fruit (1).

We report here a case of allergy to latex and spinach.

CASE REPORT

A 37-year-old man who worked in intensive care unit had had contact urticaria from latex gloves since 1995. He had no urticaria when he used vinyl gloves and was not atopic. Three re-challenge tests with latex gloves reproduced urticaria on his hands. The patient had eaten spinach twice, which produced diffuse urticaria, facial erythema and pruritis. In 1997 he experienced 2 episodes of diffuse urticaria with angioneurotic oedema during exercise (intensive swimming), 1–2 h after eating a meal including spinach. A re-challenge test with spinach alone reproduced diffuse urticaria. He sometimes ate avocados, bananas, kiwi fruit, sweet peppers and peanuts, but without reaction.

The cutaneous prick tests were compared with histamine. They were positive for latex, spinach, avocado, banana, peanut, apple and sweet pepper (Table I), but negative for kiwi fruit, melon, wheat, rice, dactyle, artemisia, phleum and birch. Total IgE antibodies were normal, at 30 kU/l. Radio-allergosorbent testing (RAST) for IgE antibody were negative for latex, avocado, spinach, banana, phleum and artemisia.

DISCUSSION

In this case we demonstrated an allergy to latex proteins (proved by contact urticaria, positive prick test and re-challenge test with latex), associated with an allergy to spinach (reproducible urticaria and positive prick test for spinach). The negativity of IgE antibodies for latex and spinach does not exclude the diagnosis of allergy, especially since total IgE antibodies were normal. IgE antibodies might be fixed in tissues and not in plasma. The patient was not atopic, had no reaction to pollens and his prick tests were negative for pollens.

Cross allergy between latex and avocados or bananas is well documented, with a common epitope (1, 2), and has been described with kiwi fruit, mango, peanut and sweet pepper (3).

Table II. *Cutaneous prick tests*

Substance	Prick test (papule (mm)/erythema (mm))
Positive control (histamine)	5/10
Negative control (–) (solvent)	0/3
Latex	5/8
Spinach	5/20
Sweet pepper	7/15
Banana	3/5
Avocado	4/10
Apple	6/15
Peanut	4/10
Wheat	0/3
Kiwi fruit	0/3
Paprika	0/3

Spinach belongs to the chenopodiaceae family. Only 2 cases of cross allergy between latex proteins and spinach have been reported previously (4). There may be a common epitope between latex and spinach.

REFERENCES

1. Lavaud F, Prévost A, Cossard C, Guérin L, Bernard J, Kochman S. Allergy to latex, avocado pear, and banana: evidence for a 30 kd antigen in immunoblotting. *J Allergy Clin Immunol* 1995; 95: 557–564.
2. Blanco C, Carillo T, Castillo R, Quiralte J, Cuevas M. Latex allergy: clinical features and cross reactivity with fruits. *Ann Allergy* 1994; 73: 309–314.
3. Maillard H, Drouet M, Sabbah A. Allergies croisées au poivron et au latex: nouvelle allergie croisée? *Allergie et immunologie* 1995; 27: 292–294.
4. Le Sellin J. Allergie croisée: latex-épinard. *Allergie et immunologie* 1994; 26: 76–78.

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Disseminated Superficial Porokeratosis Induced by Furosemide

Sir,

Porokeratosis is a heritable disorder of cornification, characterized by an extending keratotic lesion with an atrophic centre and a distinctive peripheral ridge. Histologically, the tiny keratotic spine exhibits the pathognomonic parakeratotic column, the cornoid lamella (1). Different clinical manifestations of porokeratosis are recognized (2).

The classical forms are localized. Other forms are disseminated, such as disseminated superficial porokeratosis, disseminated superficial actinic porokeratosis, porokeratosis plantaris and palmaris et disseminata.

Disseminated superficial porokeratosis is usually inherited as an autosomal dominant condition, but sporadic cases have also been observed. Porokeratosis often begins in childhood between the ages of 5 and 10 years, with widespread flat lesions on the entire integument including both sun-exposed and non-sun-exposed areas. Disseminated superficial porokeratosis has been reported in immunocompromised patients after hepatic or renal transplantation suggesting that immunosuppression may lead to a clonal development of porokeratosis (3).

Disseminated superficial actinic porokeratosis (4) is present in sun-exposed areas, especially on the arms and legs. Induction of actinic porokeratosis is also possible by artificial light exposure.

Inamoto et al. (5) reported the exacerbation of a pre-existing porokeratosis of Mibelli caused by a non-immunosuppressive drug, such as hydrochlorothiazide. Other drugs that may induce porokeratosis include corticosteroids, cytostatics and psoralens (actinic porokeratosis) (6).

In this study, the provocation of disseminated superficial porokeratosis by the hydrochlorothiazide analogon furosemide is, to our knowledge, described for the first time.

CASE REPORT

A 72-year-old male patient with a history of heart insufficiency since 1984 and coronary heart disease leading to a cardiac infarction in 1994 presented at our department with disseminated skin lesions that had occurred 8 weeks previously. He had suffered from recurrent episodes of cardiac decompensation for several years and the treatment had usually included furosemide administration. During one of these treatments with furosemide he had developed skin lesions on the lower leg. The lesions disappeared after discontinuation of furosemide. Four weeks later, due to cardiac oedema and dyspnoea he had once more been treated with furosemide, resulting in new skin lesions. Increasing the dose of furosemide due to severe cardiac decompensation had led to even more rapid development of new lesions.

Physical examination of the skin revealed disseminated, slightly atrophic maculae with a hyperkeratotic ridge on the lower legs (Fig. 1), the forearm and the capillitium, measuring up to 8 mm in diameter. Some lesions on the lower legs were haemorrhagic (Fig. 1) and showed a clinically vasculitis-like aspect.

Histopathological examination of a lesion located on the leg revealed the typical cornoid lamella (Fig. 2) with an underlying atrophic epidermis and absence of the granular layer. In the upper dermis extravasated erythrocytes responsible for the haemorrhagic clinical presentation could be identified.

Because of the correlation between the furosemide therapy and the appearance of porokeratosis, furosemide was discontinued and, as a

result, the skin lesions cleared in about 2 weeks. However, owing to a very severe cardiac decompensation during hospitalization, furosemide had to be given again, resulting in prompt recurrence of the lesions.

The porokeratosis improved again after discontinuation of furosemide treatment and the lesions disappeared. The cardiac decompensation was treated with restriction of fluid intake to approximately 750 ml/day.

DISCUSSION

Inamoto et al. (5) observed a drug eruption over pre-existing porokeratosis of Mibelli in a 72-year-old-patient, who had been suffering from porokeratosis of Mibelli for 50 years. After administering hydrochlorothiazide for 1 year because of hypertension, a drug eruption developed on the flexor aspects of the legs. Re-administration of hydrochlorothiazide resulted in new skin lesions showing the histological hallmark of porokeratosis, the cornoid lamella.

There was also a clear correlation of therapy with



Fig. 1. Porokeratosis on the lower legs, presenting as disseminated, slightly atrophic macules with a hyperkeratotic ridge and a haemorrhagic aspect.

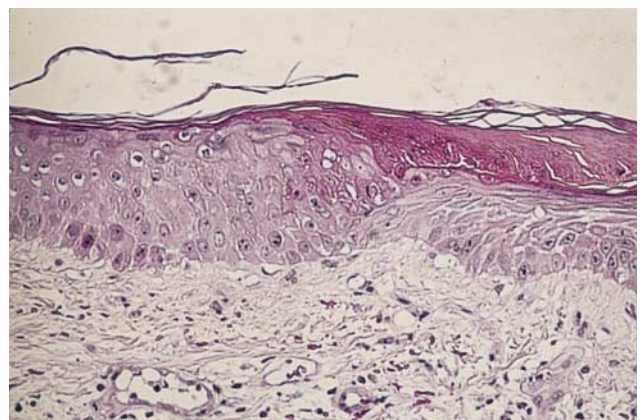


Fig. 2. Histopathological examination of a lesion located on the lower left leg. Cornoid lamella, absence of the granular layer, underlying atrophic epidermis and extravasates of erythrocytes could be identified.

furosemide and porokeratosis in the patient described here, but no history of pre-existing porokeratosis.

During furosemide therapy, photosensitivity and phototoxic reactions are rarely observed and, until now, disorders of cornification have not been known as typical side-effects of furosemide treatment. Furosemide is chemically characterized as a hydrochlorothiazide analogue.

REFERENCES

1. Wade TR, Ackerman AB. Cornoid lamellation. A histologic reaction pattern. *Am J Dermatopathol* 1980; 2: 5–15.
2. Schamroth JM, Zlotogorski A, Gilead L. Porokeratosis of Mibelli—overview and review of the literature. *Acta Derm Venerol* 1997; 77: 207–213.

3. Fields LL, White CR Jr, Maziarz RT. Rapid development of disseminated superficial porokeratosis after transplant induction therapy. *Bone Marrow Transplant* 1995; 15: 993–995.
4. Chernosky ME. Porokeratosis: report of twelve patients with multiple superficial lesions. *South Med J* 1966; 59: 289–294.
5. Inamoto N, Watanabe T, Makamura K. Porokeratosis of Mibelli: benzyhydrochlorothiazide-induced new lesions accompanied by eosinophilic spongiosis. *J Am Acad Dermatol* 1984; 11: 357–361.
6. Bruinsma W. A guide to drug eruptions. Oosthuizen: The File of Medicines, 1990; 127.

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Successful Treatment of Hailey-Hailey Disease with a Scanned Carbon Dioxide Laser

Sir,

Familial benign chronic pemphigus (Hailey-Hailey disease) is characterized by recurrent blistering lesions. Topical therapy of Hailey-Hailey disease includes application of antibiotic or antimycotic solutions and glucocorticosteroids, but these therapies do not usually induce prolonged remissions. Surgical intervention with dermabrasion or excision of the lesional sites and subsequent transplantation of grafted skin can lead to complete remission (1–3). Skin ablation can also be performed using modern CO₂ laser systems, such as the SilkTouch Flashscanner[®]. This flashscanning mechanism delivers a focused laser beam with constant velocity in a spiral pattern over a designated area. The laser energy is delivered so quickly that it never lingers at any given point in the scan longer than the thermal relaxation time of the tissue, resulting in char-free ablation and predictable, repeatable vaporization. We report here on 2 patients with Hailey-Hailey disease who were treated successfully with the SilkTouch Flashscanner[®] CO₂ laser system.

CASE REPORT

Case 1

A 38-year-old female patient with recurrent Hailey-Hailey disease of both axillas for years (Fig. 1a) was treated with the SilkTouch Flashscanner[®] CO₂ laser. After disinfection of the lesional sites and infiltration anaesthesia with Prilocain 1% (Xylonest[®] 1%) the SilkTouch Flashscanner[®] CO₂ laser vaporization was performed with 3 passes over affected axillary tissue using a 125 mm handpiece, producing a spot size of 3 mm. The power setting was 6.0 W and the scan time was 0.2 s. After every pass the char that appeared following the vaporization was removed with 0.9% NaCl solution. Finally, the treated areas were covered with Polyvidon-iodine wound gauze. Postoperatively the patient did not show any complications and reported little pain. After a follow-up of more than 1 year, the treated, scar-free healed areas revealed no clinical signs indicating the recurrence of Hailey-Hailey disease (Fig. 1b). However, recurrence of the disease was noted in the skin surrounding the treated area.

Case 2

A 31-year-old male patient with recurrent Hailey-Hailey disease of both axillas was treated similarly to case 1. Again, there were no postoperative complications and after a follow-up of 6 months the treated areas were scar-free and without clinical signs of the disease.

DISCUSSION

The conservative topical treatment of Hailey-Hailey disease is difficult and is characterized by the frequent recurrence of symptoms and lesions. Interestingly, dermabrasion or excision of lesional sites results in durable healing. An alternative to surgical intervention is the ablation with a CO₂ laser. CO₂ laser ablation is a careful, low bleeding method with less postoperative pain than dermabrasion, which is followed by rapid healing of the erosions (4, 5). Two previously published studies used CO₂ laser in defocus reported significant clinical and symptomatic improvement (5, 6). However, by using the CO₂ laser in defocus, the depth of CO₂ laser ablation cannot be controlled very effectively, resulting in a considerable risk of scarification or insufficient ablation leading to recurrence of the disease. This problem is also observed in cryosurgery or dermabrasion. The modern pulsed or scanned CO₂ laser systems deliver the laser energy so quickly that controlled vaporization of tissue in layers of 40–60 µm becomes possible (4, 7) allowing deeper ablation into the dermis compared with Er:YAG laser systems. The patients described above show that the SilkTouch Flashscanner[®] CO₂ laser system allows ablation of lesional skin with sufficient depth to induce remission of the disease without scar formation. As illustrated by case 1, treatment should extend beyond the visible skin margin (1–2 cm) to prevent recurrence.

The mechanism of durable healing of Hailey-Hailey disease lesions after vaporization of the affected areas with the scanned or pulsed (8) CO₂ laser is not clear. Theories maintain that the superficial erosions are re-epithelialized by fast proliferating adnex keratinocytes that do not express the molecular defect of Hailey-Hailey disease (2, 3). An