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## Hazards in the use of diphencyprone

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**Key words:** diphencyprone; topical immunotherapy.

● THE topical use of diphencyprone (DCP) is frequently recommended in clinical dermatology to treat alopecia areata, the side-effects of this substance being well known among users (1, 2). We present a case of professional sensitization in a nurse who developed systemic contact dermatitis simulating a fixed drug eruption, and another of consort contact dermatitis in a housewife with acute facial eczema.

*Patient 1.* A 60-year-old nurse, with no personal or family history of allergy, who had received topical immunotherapy (DCP) for 3 years, complained of the appearance of

a 10×5 cm lesion in the right neck region. The lesion was erythematous, violet in color, and slightly scaly, and the outbreaks exhibited a weekly incidence and a development of 10 months. It was very pruriginous and simulated fixed drug eruption (Fig. 1). The patient had never before had dermatologic lesions, either on the hands or elsewhere. She presented a flare up of the lesion when going into the room where this topical therapy was being applied, with reactivation of her clinical manifestations not only in the previously affected area but also on her back, where epicutaneous tests had been applied.

*Patient 2.* A 48-year-old housewife, without previous illness, went to the emergency room for acute lesions on her face. Clinical examination revealed acute edematous erythema and itchy plaques on the cheeks and neck. She had been treated with systemic steroids in three previous episodes. The dermatitis developed 10 h after her husband underwent topical immunotherapy with DCP solution in 2% acetone, for extensive alopecia areata, for

which he was treated once a week. She could not share a bed with her husband after his use of DCP because her clinical manifestations worsened.

In both cases, patch tests with the standard contact antigen panel of the Spanish Contact Dermatitis Research Group were negative. Patch tests with DCP (dilutions 0.0001–0.1% in acetone) in the Finn Chamber, and Leukotest showed eczematous changes at 48 and 96 h.

Shah et al. (3) report the risk to medical and nursing staff. We also think (3, 4) that sensitization to DCP among pharmacists and dermatologic staff is well recognized. Notifying health-care personnel of these adverse reactions is important because the use of DCP could be questioned unless all the prophylactic measures were implemented.

Consort contact dermatitis may be due to cutaneous exposure to the allergens employed by the partner (5). Medicaments, perfumes, hair dyes, acne preparations, sunscreens, condoms and rubber diaphragms, and clothes may be allergenic



Figure 1. Clinical aspect of lesions in patient 1.

in such situations. However, connubial/consort contact dermatitis is rare and probably underreported. In the literature reviewed, we have not found another case of a patient with sensitization to the DCP used by the spouse.

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### Successful treatment of delayed pressure urticaria with montelukast

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**Key words:** delayed pressure urticaria; leukotriene inhibitors; montelukast.

• DELAYED pressure urticaria (DPU) is a type of physical urticaria, characterized by the appearance of painful skin swelling a few hours after application of mechanical

pressure. It may be accompanied by systemic signs. Most patients also suffer from chronic idiopathic urticaria (1).

Antihistamines are not very effective in the treatment of this disorder. Most patients usually

require corticosteroids for prolonged periods and frequently develop undesirable side-effects from these medications.

In this report, we present a patient with severe steroid-dependent DPU which responded to the new antileukotriene agent montelukast (Singulair®). Steroid treatment was subsequently discontinued.

A 45-year-old nurse without an atopic history was healthy until 1992. During her first pregnancy, she had developed deep, erythematous swellings of skin on pressure areas, sometimes accompanied by fever and arthralgia. She also noticed urticarial eruptions on various areas. Skin lesions were not associated with food ingestion. Extensive laboratory evaluation was unremarkable. Skin prick tests with aeroallergens and foods were negative. The standard pressure challenge test performed on her arm was strongly positive. After the diagnosis of DPU was established, she was treated with maximal doses of several antihistaminic agents, including ketotifen, with minimal or transient improvement.

Various combinations of H<sub>1</sub> and H<sub>2</sub> blockers, doxepin, sulfasalazine, tranexamic acid, and colchicine did not improve her condition. An elimination diet for 14 days was ineffective. For the next 3 years, she was treated with monthly betamethazone dipropionate injections and 10–30 mg of cetirizine daily, combined with other antihistamines. Repeated trials to discontinue steroid treatment resulted in immediate exacerbation of pressure urticaria. The patient developed side-effects of chronic steroid therapy, including weight gain, facial acne, and flushing.

#### First case of DPU to be treated with the leukotriene antagonist montelukast.

As a last resort, montelukast 10 mg a day was initiated. Within 1 week, the urticaria completely resolved. Steroid treatment was tapered off and discontinued with no recurrence of DPU for the following 20 months. Repeated pressure challenge tests were negative. A brief trial to discontinue montelukast resulted in recurrence of urticaria within 3 days. The patient is currently treated with montelukast and cetirizine, 10 mg daily, without suffering any skin lesions.

DPU is a type of physical urticaria, more common than previously thought. In addition, up to 50% of patient with chronic idiopathic urticaria have a pressure component. The pathogenesis of DPU is unknown. The late appearance and prolonged duration of lesions, the associated systemic manifestations, and the poor response to antihistamines are not consistent with the early phase of immediate-type hypersensitivity reaction. These features are more characteristic of the late-phase reaction, in which inflammatory cells and mediators play major role. Cysteinyl leukotrienes (LTC<sub>4</sub>, LTD<sub>4</sub>, and LTE<sub>4</sub>) are potent inflammatory mediators, produced by activated mast cells, basophils, and eosinophils, and in lesser amounts by monocytes, endothelial cells, and platelets. These mediators are smooth-muscle contractors and eosinophil chemoattractants. Intradermal injection of leukotrienes induces wheal and flare reactions (2). Increased levels of LTE<sub>4</sub>, the stable metabolite of LTC<sub>4</sub>, and LTB<sub>4</sub> were found in cold urticaria patients (3). However, the role of leukotrienes in the pathogenesis of DPU has not been elucidated. The effect of leukotriene inhibitors, the new class of medication for asthma treatment, in urticaria patients has not been extensively studied. Chu et al. reported improvement in eight patients with chronic urticaria treated with zafirlukast (4). Another patient responding to the same medication was described by Spector & Tan (5). Three