Treatment of Protein Contact Dermatitis with Topical Tacrolimus

Pedro Mercader, Jesús de la Cuadra-Oyanguren, Mercedes Rodríguez-Serna, Gerard Pitarch-Bort and Jose Miguel Fortea-Baixauli

Dermatology Service, Consorcio Hospital General Universitario de Valencia Av/tres Cruces S/N, Valencia 46014, Spain.
E-mail: pmercader@aedv.es
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Sir,

Since 1952 it has been known that eczema can be induced by contact with food (1). This was called protein contact dermatitis (PCD) by Hjorth & Roed-Petersen (2). These authors described several food handlers with eczema and negative epicutaneous tests, but with positive scratch tests and specific IgE to the food they handled. PCD appears as a subacute or chronic dermatitis located on hands and forearms. It cannot be clinically distinguished from a classical contact dermatitis. It occurs in food handlers, who not only are exposed to the antigen, but also suffer from alterations of the epidermal barrier, making the contact with proteins easier (3). Treatment of these patients is the same as for patients with eczema. This paper presents two food handlers with PCD who had a good response to tacrolimus 0.1% cream.

CASE REPORT

Case 1. A 35-year-old woman with previous intolerance to imitation jewellery, had an 11-month history of itchy dermatitis on the back of the hands and volar aspects of the forearms. The symptoms began after 3 months work of cleaning chickens in a bird slaughterhouse. She improved on sick leave, but had a relapse on the day she returned to work. Epicutaneous tests done with the Spanish standard series were positive for nickel sulphate...
2.5% petrolatum (++) and for cliquinol 5% petrolatum (++; unknown relevance). Prick tests using chicken viscera were positive with gizzard (a modified muscular pouch behind the stomach in the alimentary canal of birds, that has a thick lining and often contains ingested grit that aids in the breakdown of seeds before digestion), heart and liver. She was diagnosed as having PCD. She had been on 2 months sick leave, and despite treatment with clobetasol propionate 0.05% cream, she was not cured (Fig. 1a). Tacrolimus 0.1% ointment was administered and she showed a significant improvement within 7 days (Fig. 1b).

Case 2. A 24-year-old woman who had a history of psoriasis and was 4 months pregnant, had worked for 2 years in a frozen fish manufacture plant. A few months after starting work she developed itchy erythematous lesions on her hands and forearms. These injuries improved with sick leave and holidays, but 48 h after going back to work the injuries became worse.

On epicutaneous testing we found sensitivity to para-phenylenediamine 1% petrolatum (++) and thimerosal 0.1% petrolatum (++), both of unknown relevance. Prick test with the fish handled by the patient (halibut) was positive. She also had positive specific IgE for hake (class 2) and tuna (class 3), which confirmed the PCD diagnosis.

She had been on sick leave for 2 months and received clobetasol propionate 0.05% without significant improvement of her lesions. We replaced her treatment with tacrolimus 0.1% ointment twice a day; after 48 h there was an almost complete cure.

DISCUSSION

In both patients, the dermatitis symptoms located on the hands of a food handler suggested PCD, which was confirmed by the prick tests. Although the pathogenic mechanisms are not clear, it is believed to be produced by the action of the specific IgE in Langerhans' cells and other antigen-presenting cells producing the eczema symptoms (3). The efficacy shown by tacrolimus in these patients would partly support this hypothesis, because it is known that tacrolimus decreases the expression of the high affinity IgE receptor of Langerhans' cells and epidermic dendritic inflammatory cells (4) and also inhibits the stimulatory function of the Langerhans' cells (5).

The response in both patients cannot be attributed only to a lack of exposure to the antigen, because when seen in our clinic both patients had spent several days off work and had clobetasol propionate cream treatment, and despite this both still presented with eczema. The clinical response to tacrolimus 0.1% was significant in both patients, which suggests that this could be the initial treatment for these patients.

REFERENCES