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Short Communication

Allergic contact dermatitis from 6α-methylprednisolone aceponate and budesonide

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Case Report

A 26-year-old non-atopic nurse had a histologically confirmed chronic lichenified eczema of both hands. Worsening was related to the use of topical corticosteroids, which she had frequently changed due to lack of responsiveness.

Patch tests with the GIRDCA standard series and with a rubber series were negative, while patch tests with the numerous topical corticosteroids used by the patient showed positive reactions only to Advantan® cream (+D2/++D3) and Bidien® cream (+D2/++D3), tested as is. Further patch tests with a corticosteroids series and the constituents of the 2 creams, kindly supplied by the manufacturers, were carried out (Table 1). No further positive reactions to corticosteroids were observed, reading at D7.

Discussion

6α-methylprednisolone aceponate (MPA) is a non-halogenated diester of 6α-methylprednisolone. It has a propionate group at the C17 and an acetate group at the C21; these ester groups increase the lipophilicity of the molecule and its penetration into the skin (1). The metabolite of MPA, methylprednisolone 17-propionate (MPP), is the active principle in the skin; it is quickly inactivated by conjugation with glucuronic acid, which explains the low systemic activity of MPA (1, 2). MPA has been classified as a potent glucocorticosteroid with reduced atrophogenic potential and good local tolerability; MPA is therefore used in Italy and in many European countries in the treatment of various forms of eczema (2, 3).

Table 1. Patch test results

Corticosteroid series	D2	D3
hydrocortisone 25% pet.	−	−
prednisolone 5% pet.	−	−
betamethasone-17-valerate 1% pet.	−	−
clobetasol-17-propionate 0.5% pet.	−	−
tixocortol pivalate 1% pet.	−	−
budesonide 0.1% pet.	−	++
hydrocortisone-17-butyrate 1% eth.	−	−
Constituents of the creams		
methylprednisolone aceponate 1% eth.	+	++
budesonide 1% pet.	−	++
excipients of the creams	−	−

In spite of its wide use, to our knowledge, only 1 previous case of allergic contact dermatitis has been described, thus suggesting a low allergenic potential of MPA (4) in contrast to the well-documented allergenicity of other corticosteroids (5). In the case described by Balato et al. (4), true cross reaction was observed with hydrocortisone-17-butyrate (H-17-B). Both MPP and H-17-B belong to the D group of the Coopman et al. (6) classification, based on different substitution on the D-ring or in the C20–C21 position of the side chain of the steroid molecule.

In our case, sensitization to MPA was associated with sensitization to budesonide. We could not find reactions to any other corticosteroids, using either the commonly advised markers of corticosteroid sensitivity (tixocortol pivalate for group A, budesonide for groups B and D, H-17-B for group D) (7), or other standardized corticosteroid allergens (Table 1). In accordance with the Coopman et al. classification (6), the different chemical structures of budesonide (group B) and MPA and its metabolite, methyl prednisolone 17-propionate (group D), would suggest probable independent concomitant multiple sensitization in our patient. Lepoittevin et al. (8), on the basis of statistical analysis and conformational study of major corticosteroids, confirmed clinical observations of cross-reactions between budesonide and the corticosteroid ester group D. Therefore, in our patient, a cross-reaction may be hypothesized as well. The patient's clinical history of prolonged use of both MPA and budesonide, together with many other topical corticosteroids, does not allow us to resolve the question.

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