

Urticaria to methylprednisolone sodium hemisuccinate

J. M. Borja*, P. A. Galindo, F. Feo, E. Gomez

Key words: corticosteroids; drug allergy; hypersensitivity; methylprednisolone; methylprednisolone sodium hemisuccinate.

● CORTICOSTEROIDS (CS) are widely used in the treatment of allergic reactions. The prevalence of adverse reactions due to CS hypersensitivity is about 0.3% in systemic administration. Urticaria/angioedema, bronchospasm, and anaphylaxis have been reported, especially with hydrocortisone and methylprednisolone. Some authors (2–5) have reported positive skin tests with those CS and others such as paramethasone, prednisolone, and dexamethasone, suggesting an IgE-mediated mechanism.

Cross-reactivity among systemic CS is variable, and classification into groups by

No cross-reactivity with methylprednisolone.

this criterion is not possible. In contrast, topical CS have been classified into four groups by chemical structure and cross-reactivity.

A 26-year-old woman, diagnosed as having allergic rhinoconjunctivitis and mild and intermittent asthma, received an intramuscular dose of methylprednisolone sodium hemisuccinate (s.h.) due to bronchospasm secondary to airway infection. After 10 min, she developed facial pruritus, angioedema of the lips and face, and hives. She was treated with hydrocortisone sodium phosphate (s.p.), and the clinical picture resolved in 1 h. We carried out the following study after receiving her informed consent. Skin prick tests with methylprednisolone s.h. (40 mg/ml), prednisolone s.h. (25 mg/ml), hydrocortisone s.p. (25 mg/ml), dexametha-

sone s.p. (4 mg/ml), and deflazacort (22 mg/ml) were negative. Intradermal tests were positive (7×5 mm) with methylprednisolone s.h. (4×10⁻³ mg/ml) and negative with prednisolone s.h., hydrocortisone s.p., dexamethasone s.p., and deflazacort. Seven atopic and nonatopic controls were negative. Single-blind, placebo-controlled challenge tests (SBPCCT) performed with prednisolone s.h., hydrocortisone s.p., dexamethasone s.p., and deflazacort were negative. SBPCCT with methylprednisolone s.h. were performed with intramuscular doses of 1, 5, and 10 mg separated by 1 h. By 30 min after the last dose, the patient developed nasal blockage, rhinorrhea, dry cough, and macular exanthema on her neck and abdomen. She received epinephrine and dexchlorpheniramine. Challenge tests with excipients of methylprednisolone s.h. were negative. To investigate the possible high specificity of her sensitization, as related to the succinate group, we performed an oral challenge test with methylprednisolone, with negative result.

Severe adverse reactions to systemic CS have been reported more frequently with the parenteral route. Some cases (1) of bronchospasm have occurred with hydrocortisone s.h. in asthmatic patients with ASA intolerance. Hydrocortisone and methylprednisolone (2–5) are the more frequently implicated CS in adverse reactions with a probably IgE-mediated mechanism. CS sodic succinate has produced more severe reactions than CS s.p. or acetate. This fact suggests that the succinate group has a certain immunogenic role. Doezeema (2) reported a severe respiratory reaction after intravenous administration of methylprednisolone sodium succinate. The skin tests performed were positive. Methylprednisolone acetate was negative in the skin test. Fernández et al. (4) reported a case of bronchospasm induced by methylprednisolone s.h. Skin tests were positive with methylprednisolone

s.h. and negative with hydrocortisone s.p. The patient tolerated this steroid and various CS s.p. Asakawa et al. (5) reported a case of bronchospasm worsened by hydrocortisone s.h. and methylprednisolone s.h. Skin tests performed with both CS were positive. In this case, skin and challenge tests with betamethasone s.p. were negative.

In our case, the clinical picture and positive skin test suggest an IgE-mediated hypersensitivity to methylprednisolone s.h. The remarkable specificity of the patient's sensitization was demonstrated after the tolerance of methylprednisolone. Moreover, the immunogenic role of the succinate group was evidenced, as in previous reports. Cross-reactivity is variable among systemic CS, and skin and challenge tests are necessary in patients with adverse reactions.

*c/ Olivo no. 37. 13.170. Miguelturra Ciudad Real Spain
Tel. 34-26-242371
Fax: 34-26-225154

Accepted for publication 22 March 2001
Allergy 2001; 56:791
Copyright © Munksgaard 2001
ISSN 0105-4538

References

1. PARTRIDGE MR, GIBSON GJ. Adverse bronchial reaction to intravenous hydrocortisone in two aspirin sensitive asthmatic patients. *BMJ* 1978;1:1521–1522.
2. DOEZEMA D. Anaphylaxis following administration of intravenous methylprednisolone sodium succinate. *Am J Emerg Med* 1987;5:4–44.
3. ESCRIBANO-RODRIGUEZ MM, GONZÁLEZ-POL J, MUÑOZ-BELLIDO FJ, DE LA CALLE-TORAL A, VELÁZQUEZ-AMOR E, CONDE-HERNÁNDEZ J. Immediate reaction to methylprednisolone with tolerance of other corticosteroids. *Allergy* 1997;52:677–678.
4. FERNÁNDEZ S, REAÑO M, VIVES R, et al. 6-Methylprednisolone-induced bronchospasm. *Allergy* 1997;52:780–782.
5. ASAKAWA T, ARAKI T, IMAI I, TSUTSUMI Y, KAWAKAMI F. Skin tests in steroid allergy. *Allergy* 1999;55:645–646.