

a high cockroach-allergen concentration in the family room, independently of race and income, may also induce an increased risk of bronchial inflammation through nonallergenic or allergenic mechanisms (6), it should be noted that the indoor environment may be appreciably different between Dresden and Marseille (France).

*Unité de Médecine Infantile
CHU Timone-Enfants
13385 Marseille Cedex 5
France
Tel. 33.(0)4.91.38.67.39
Fax: 33.(0)4.91.38.67.26
E-mail: jdubus@mail.ap-hm.fr

Accepted for publication 8 January 2001
Allergy 2001; 56:351–352
Copyright © Munksgaard 2001
ISSN 0105-4538

References

- HIRSCH T, STAPPENBECK C, NEUMEISTER V, et al. Exposure and allergic sensitisation to cockroach in East Germany. *Clin Exp Allergy* 2000;**30**:529–537.
- CALL RS, SMITH TF, MORRIS E, CHAPMAN MD, PLATTS-MILLS TAE. Risk factors for asthma in inner city children. *J Pediatr* 1992;**121**:862–866.
- KANG B, JOHNSON J, VERE-THORNER C. Atopic profile of inner-city asthma with a comparative analysis on the cockroach-sensitive and ragweed-sensitive subgroups. *J Allergy Clin Immunol* 1993;**92**:802–811.
- ROSENSTREICH DL, EGGLESTON P, KATTAN M, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 1997;**336**:1356–1363.
- DONFACK J, TSALENKO A, HOKI DM, et al. HLA-DRB1*01 alleles are associated with sensitisation to cockroach allergens. *J Allergy Clin Immunol* 2000;**105**:960–966.
- GOLD DR, BURGE HA, CAREY V, MILTON DK, PLATTS-MILLS T, WEISS ST. Predictors of repeated wheeze in the first year of life: the relative roles of cockroach, birth weight, acute lower respiratory illness, and maternal smoking. *Am J Respir Crit Care Med* 1999;**160**:227–236.

Urticaria to hydrocortisone

L. Rasanen, K. Tarvainen*, S. Makinen-Kiljunen

Key words: hydrocortisone; immunospot; specific IgE; urticaria.

● TOPICAL corticosteroids are well-known contact sensitizers. Immediate allergic or allergic-like reactions to systemic corticosteroids also occur but less frequently (1). We have seen two atopic patients who developed urticaria from a hydrocortisone injection or infusion.

Patient 1 (21 years of age) had had atopic flexural eczema in childhood and hand eczema for the last 2 years.

She had used various topical corticosteroid preparations without any noticeable side-effects. After being stung by a wasp or bee, the patient took three 50-mg tablets of hydrocortisone. Within 30 min, she developed rhinorrhea, shortness of breath, and fatigue, and had to go to a local medical center.

Possible IgE-mediated allergy to injected drug.

There she received 50 mg Solu-Cortef® IV (hydrocortisone succinate, benzyl alcohol as a preservative), after which her symptoms worsened. She was then treated with epinephrine IV, which relieved her condition. The patient was transferred to the nearest central hospital, and there she again received 100 mg Solu-Cortef IV. Within 2 min, she started to cough, wheeze, and develop urticaria all over the skin. IV epinephrine was then administered, with a favorable response.

Patient 2 (46 years of age) had a history of childhood atopic eczema and had suffered from seronegative rheumatoid arthritis, bronchial asthma, hypothyrosis, and epilepsy for years. Various drugs were suspected to have caused immediate allergic or pseudoallergic reactions, including sulfonamides, penicillins, lorazepam, clindamycin, ASA, and parenteral corticosteroids. The patient received 100 mg Solu-Cortef IV several hours after an arthrodesis operation. Within 15 min, she developed urticaria over her trunk. Atarax® was given *per os*,

Table 1. Results of skin tests

Subject	Prick/ ID	Hydrocortisone	Prednisolone	Methyl- prednisolone	Betamethasone (Betapred®)
1	Prick	5×4/7×5 ¹ (10 mg/ml) ²	3/6×4 50 mg/ml	4×3/6×4 (50 mg/ml)	0/6×4 (4 mg/ml)
1	ID	n.d.	7×5/4 ³ (0.1 mg/ml)	7×6/4 (0.1 mg/ml)	0/4 (4 mg/ml)
2	Prick	n.d.	0/6 (25 mg/ml)	n.d.	n.d.
2	ID	n.d.	20 ps, generalized itching (2.5 mg/ml)	n.d.	n.d.

¹ Diameter(s) of corticosteroid-induced wheal/diameter(s) of positive control wheal on prick test in millimeters.

² Corticosteroid concentration in parenthesis.

³ Diameter(s) of corticosteroid-induced wheal/diameter(s) of negative control wheal on intradermal test.

ID: intradermal test; HSA: human serum albumin; n.d.: not done; ps: pseudopodia.

and the urticaria started to subside. Two years later, after a shoulder region operation, the patient received 100 mg Solu-Cortef IM and again developed widespread urticaria within 15 min. She received oral Phenergan[®], after which her urticaria resolved in hours. The patient had also experienced periarticular itching and erythema after receiving intra-articular methylprednisolone injections (Solomet Depot[®]). Due to her rheumatic arthritis, the patient had been on oral prednisolone for years (daily doses 5–30 mg) without any allergic side-effects.

On skin tests, patient 1 was positive to hydrocortisone, prednisolone, and methylprednisolone, while betamethasone and preservatives (benzyl alcohol 9 mg/ml, sodium metabisulfite 1 mg/ml) remained negative. Patient 2 had a positive intradermal test reaction to prednisolone (Table 1). When 11 control persons were skin-tested with the same corticosteroids and preservatives, no positive reactions were seen. Both of our patients had elevated IgE values (286 and 448 kU/l, respectively). Immunospot was performed as described before (2), and patient 2 had IgE antibodies to hydrocortisone and methylprednisolone conjugated with human serum albumin (HSA), while patient 1 was negative. Neither of the patients exhibited IgE binding to HSA alone.

Earlier, Pryse-Phillips et al. (3) detected specific IgE antibodies to methyl prednisolone in a patient who developed anaphylaxis during Solu-Medrol infusion. It may seem contradictory that although patient 2 tolerated 5–30 mg of prednisolone *per os*, she had a clearly positive intradermal test reaction to the same drug. However, this is in keeping with earlier findings

demonstrating that a patient with contact allergy to a corticosteroid may tolerate moderate or even high oral or parenteral doses of the same drug (4). The anti-inflammatory and anti-allergic properties of corticosteroids may mask their allergic effect. Patient 2 was also positive to the hydrocortisone/HSA conjugate on immunospot. These results indicate that a true, IgE-mediated allergy mechanism may underlie adverse reactions to corticosteroids.

*North Karelia Central Hospital
Tikkamaentie 16
FIN-80210 Joensuu
Finland
Fax: +358-13-1713206

Accepted for publication 9 January 2001
Allergy 2001; 56:352–353
Copyright © Munksgaard 2001
ISSN 0105-4538

References

1. KAMM GL, HAGMEYER KO. Allergic-type reactions to corticosteroids. *Ann Pharmacother* 1999;**33**:451–460.
2. MAKINEN-KILJUNEN S. Banana allergy in patients with immediate-type hypersensitivity to natural rubber latex: characterization of cross-reacting antibodies and allergens. *J Allergy Clin Immunol* 1994;**93**:990–996.
3. PRYSE-PHILLIPS WEM, CHANDRA RK, ROSE B. Anaphylactoid reaction to methylprednisolone pulsed therapy for multiple sclerosis. *Neurology* 1984;**34**:1119–1121.
4. RASANEN L, TUOMI M-L, YLITALO L. Reactivity of tixocortol pivalate-positive patients in intradermal and oral provocation tests. *Br J Dermatol* 1996;**135**:931–934.

Honeymoon rhinitis

J. Monteseirin*, M. J. Camacho, I. Bonilla, C. Sánchez-Hernández, M. Hernández, J. Conde

Key words: allergy; honeymoon; mite; pollen; postcoital; rhinitis; rhinoconjunctivitis; seminal plasma; sexual.

● **ATTACKS** of asthma and rhinitis can be provoked by a variety of stimuli, including infections, allergens, exercise, and emotional stress. Sexual intercourse, too, has rarely been reported to trigger attacks of asthma and

rhinitis (1, 2). Although studies have documented the

Postcoital exacerbation of allergic rhinoconjunctivitis.

impact of chronic obstructive pulmonary diseases and cystic fibrosis on sexual activity, little attention has been paid to the effect of asthma and rhinitis on adult sexual life. We present here several patients with postcoital exacerbation of rhinitis (honeymoon rhinitis) (2).

Twenty-three allergic subjects (nine female and 14 male; mean age, 39.47 years; range 24–51 years) had a history of symptoms of rhinoconjunctivitis. Ten had seasonal rhinoconjunctivitis (March to May) with positive skin prick test (Bial-Arístegui, Bilbao, Spain) and specific IgE (HYTEC 288, Hycor Biomedical, Inc., Irvine, CA, USA) to grass pollen (*Phleum pratense*, *Lolium perenne*, *Poa pratensis*, and *Dactylis glomerata*) and *Olea europaea* pollen. Thirteen had perennial rhinoconjunctivitis with positive skin prick test (Bial-Arístegui) and specific IgE (HYTEC 288) to *Dermatophagoides pteronyssinus* and *D. farinae*. None had a history of asthma, were smokers, had sinusitis or other cause of rhinitis, or had previously received immunotherapy. Since sexual activity involves exercise equivalent to climbing two flights of stairs (3), nasal