

Anaphylaxis due to a tick bite

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Key words: anaphylaxis; IgE; tick hypersensitivity; skin tests.

Allergic reactions after tick bites have been previously reported (1). Specific IgE antibodies for tick proteins have been demonstrated in patients for *Ixodes holocyclus* (2), *Ixodes ricinus* (3, 4) and *Ixodes pacificus* (5).

We report a case of anaphylaxis caused by a *Rhipicephalus* sp. bite. A 56-year-old nonatopic man, who works as a goatherd, reported generalized itching and urticaria, upset feeling, hypotension, dizziness and chest tightness after a tick bite. Treatment with adrenaline and parenteral corticosteroids resulted in his recovery. Skin-prick tests were negative for usual aeroallergens and several foods (including *Anisakis simplex*).

Total IgE was 88.2 kU/L (CAP System IgE FEIA). Specific IgE to *Rhipicephalus* sp. whole body extract (WBE) and salivary gland extract (SGE), measured by EAST technique were <0.35 kU/L and 0.72 U/L (Class 2), respectively.

A WBE and SGE of *Rhipicephalus* sp. (R), *Ixodes ricinus* (I) and *Haemaphysalis punctata* (H) were studied by sodium dodecyl sulphate-polyacrylamide gel electrophoresis immunoblotting (Western blotting). The SGE from the three tick species showed protein patterns with bands ranging from 94 to 14 kDa (Fig. 1).

This patient is always exposed to ticks, and he had experienced some tick bites with no or little local reaction before suffering from anaphylaxis. This kind of clinical history seems to be an usual

An occupational anaphylaxis in a goatherd.

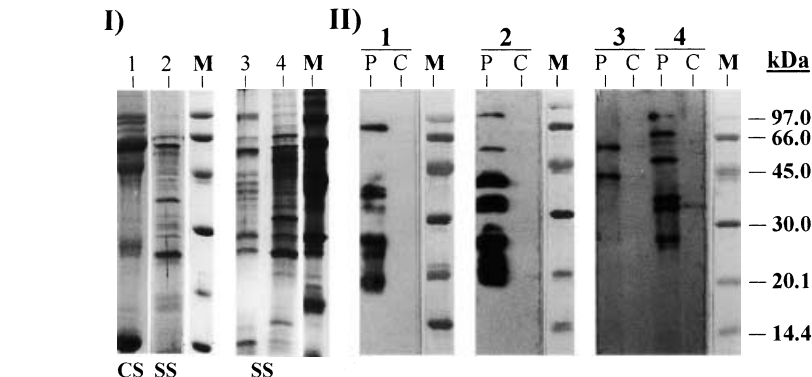


Figure 1. Electrophoretic and Western blotting results of different tick extracts. (I) SDS-PAGE (II) SDS-PAGE immunoblotting incubated with patient's serum. (1) R-WBE; (2) R-SGE (IgE-binding bands of 80, 51, 38, 31.5, 26, 24, 22, 20 and 19 kDa); (3) I-SGE (IgE-binding bands of 60, 44 kDa and faint bands of 34.5 and 28 kDa) (4) H-SGE (IgE-binding bands of 71, 52, 37, 34.5, 27 kDa and faint bands of 99, 63, 58, 45, 40, 31 and 26 kDa). CS, Coomassie Brilliant Blue staining R-250; SS, Silver staining. Lane P, patient's serum; lane C, control serum (pool from nonatopic subject's sera); lane M, molecular mass marker.

pattern in tick systemic reactions (1, 3, 5). The specific IgE value obtained in the patient's serum to tick proteins was higher with R-SGE than with R-WBE and as previously described for ticks (6) and hymenopterans, it is more suitable to use SGE for diagnostic purposes than the WBE.

Comparing the molecular masses of *I. holocyclus* (6), *I. pacificus* (5) and the allergenic proteins obtained in this work with *Rhipicephalus* sp., some common allergenic proteins with molecular masses, 51, 38, 35, 28 kDa, could be pointed out. This coincidence reinforces the previously suggested cross-reactivity among the hard tick allergens (4).

This new case demonstrates that tick anaphylaxis can occur. Tick bites can trigger allergic reactions especially during occupational or recreational exposure. It is noteworthy that anaphylaxis caused by tick bite could be more common than suspected. Although not always possible, the correct establishment of allergy to ticks depends on the identification of the tick and appropriate tick extracts.

I. ricinus is the most common tick in Europe (7), but other ticks can also produce sensitization and allergic symptoms as in the case here.

To our knowledge, this is the first case of anaphylaxis in a human being caused by a *Rhipicephalus* sp. bite, in an occupationally exposed patient. An IgE mechanism has been demonstrated, with immunoblotting detecting the main IgE-binding allergens.

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References

1. BROWN AF, HAMILTON DC. Tick bite anaphylaxis in Australia. *J Accid Emerg Med* 1998;**15**:111–113.

2. SOLLEY GO. Allergy to stinging and biting insects in Queensland. *Med J Aust* 1990;**153**:650–654.
3. BEAUDOUIN E, KANNY G, GUERIN B, GUERIN L, PLENAT F, MONERET-VAUTRIN DA. Unusual manifestations of hypersensitivity after a tick bite: report of two cases. *Ann Allergy Asthma Immunol* 1997;**79**: 43–46.
4. FERNÁNDEZ-SOTO P, DÁVILA I, LAFFOND E, LORENTE F, ENCINAS-GRANDES A, PÉREZ-SÁNCHEZ R. Tick bite-induced anaphylaxis in Spain. *Ann Trop Med & Parasitol* 2001;**95**:97–103.
5. VAN WYE JE, HSU YP, LANE RS, TERR AI, MOSS RB. IgE antibodies in tick bite-induced anaphylaxis. *J Allergy Clin Immunol* 1991;**88**:968–70.
6. GAUCI M, STONE BF, THONG YH. Detection in allergic individuals of IgE specific for the Australian paralysis tick, *Ixodes holocyclus*. *Int Arch Allergy Appl Immunol* 1988;**85**:190–193.
7. FAIN A. Les tiques de Belgique (Acari: Ixodoidea). Document de travail de l'Institut Royal des Sciences Naturelles de Belgique no. 58, p. 34.

A case of coconut allergy

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Key words: children; coconut; *Cocos nucifera* cross-reactivity; food allergy.

The coconut (*Cocos nucifera*) is a monocotyledonous plant of the *Aracaceae* family. Immediate hypersensitivity reactions to coconut fruit are rare (1,2) and have never been reported in children. We report the first case of a child who suffered an Immunoglobulin (Ig) E-mediated reaction to coconut.

A 3-year-old referred boy abdominal pain, vomiting, oral allergy syndrome, and edema of the eyelids immediately after oral contact with a coconut sweet. One year later, after eating a small portion of fresh coconut, he suffered sudden

abdominal pain, vomiting, oral allergy syndrome, and edema of the eyelids. No other food allergies or intolerances were reported. Skin tests to inhalant allergens were positive to house dust mites. Skin prick tests to common foods and tree nuts were negative except for a weak wheal to almond. A prick test with fresh coconut was strongly positive. An open oral challenge test to almonds proved negative. Patch test to coconut was negative. Serum-specific IgE to coconut was positive (RAST class 3). IgE-immunoblotting after SDS-PAGE under nonreducing conditions showed an Ig-E binding protein of about 18 kDa, and two weaker bands of about 25 and 75 kDa (Fig 1).

Allergic reactions to coconut fruit has only occasionally been reported (1, 2). The first report describes immediate hypersensitivity to coconut in two patients aged 50 and 21 years, previously diagnosed of severe sensitivity to nuts (1). In that report, Ig-E binding proteins of 55, 36.5, and 35 kDa were observed on the nonreduced coconut protein immunoblot and these bands were immunologically cross-reactive with seed storage proteins from walnut, almond, and peanut. Furthermore, studies of the major albumin and globulin proteins in coconut endosperm have indicated immunologic cross-reactivity with the soy globulins conglycinin and glycinin (3). Moreover, a coconut 35 kDa protein is known to

share similar physical and biochemical characteristics with other globulins of the legumin group, including soybean glycinin, peas legumin and peanut arachin (4). Thus, these two studies support the cross-reactivity between coconut and legumin-like seed storage proteins in the two coconut allergic adult patients mentioned below. The second report describes a monosensitized adult patient with an IgE-mediated reaction to coconut, which was demonstrated by both *in vivo* and *in vitro* tests, with an IgE immunoblot showing an intense reactivity to a protein of 78 kDa (2).

In conclusion, in the present case, the young age of the child and the lack of other food and pollen allergies suggest coconut hypersensitivity to be a true primary sensitizing agent and not a cross-reactive one. Serum Ig-E binding to an 18-kDa protein would indicate that another allergen, not previously described, could be relevant in patients with single allergy to coconut.

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References

1. TEUBER SS, PETERSON WR. Systemic allergic reaction to coconut (*Cocos nucifera*) in 2 subjects with hypersensitivity to tree nut and demonstration of cross-reactivity to legumin-like seed storage proteins: new coconut and walnut food allergens. *J Allergy Clin Immunol* 1999;**103**: 1180–1185.
2. ROSADO A, FERNÁNDEZ-RIVAS M, GONZÁLEZ-MANCEBO E, LEÓN F, CAMPOS C, TEJEDOR MA. Anaphylaxis to coconut. *Allergy* 2002;**57**:182–183.
3. DE MASON DA, SKHAR CKN. Electrophoretic characterization and immunological localization of coconut (*Cocos nucifera*) endosperm storage proteins. *Bot Gaz* 1990;**151**:302–313.

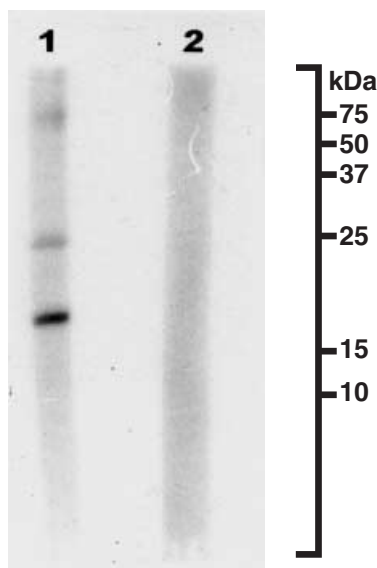


Figure 1. SDS-PAGE immunoblotting. Lane 1: patient serum. Lane 2: negative control.

4. CARR HJ, PLUMB GW, PARKER ML, LAMBERT N. Characterization and crystallization of an 11S seed storage globulin from coconut (*Cocos nucifera*). Food Chem 1990;**38**:11–20.

Side effects to castor oil

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Key words: contact dermatitis; castor oil; drug allergy/reactions.

A 26 year-old woman, suffering from a recurrent vulvovaginitis for 1 year, was treated with a topical pessary of meclocycline sulfosalicylate. After a 7-day treatment the following symptoms appeared: edema

A fixed drug eruption caused by topical medication.

of the labia, widespread irregular abdominal dermatitis in the right iliac fossa (fixed drug eruption), and numerous generalized small wheals. These symptoms were treated with dexchlorpheniramine maleate without results; it was necessary to prescribe prednisone tablets.

After a few days, the majority of the symptoms had disappeared and the fixed abdominal drug eruption had faded.

The patient was not atopic, but in her history she reported, that 1 year earlier she had been treated for acne with minocycline cloridrate and she had been forced to interrupt the treatment because urticaria appeared.

For this reason, and as suggested by the literature (1), we carried out a patch test with meclocycline sulfosalicylate and the other compounds of suppository i.e. D,1-alfa-tocopherol acetate, precipitated silex, sodium metabisulfite, solid semi-synthetic glycerides, but the result was negative.

One week later the patient showed another episode of edema of the pudendal labia provoked by the same lubricating gel used during the same previous symptoms.

We repeated patch tests with the single components of the product: octyl palpi-

tate, cyclomethicone, tocopheryl acetate, hydrogenated castor oil, and hydrossisteic acid.

We removed the patch tests after 4, 12, 24, 48 and 96 h; a positive result to hydrogenated castor oil was observed with erythema, infiltration and papules after 48 h.

Although dry or scaly skin and a burning feeling are usually considered adverse effects of topical treatment with tetracycline, the possibility that the same symptoms may be caused by the use of OTC products should not be ruled out during diagnosis.

There have been several reports of allergy to castor beans (*Ricinus communis*), from which castor oil is derived. Cases of asthma have been found not only in the oil industry (2), but also in exposed seamen and laboratory workers (3). Using Western blotting, Thorpe et al. (4). identified three important allergens from *Ricinus communis*: a 2S storage albumin of 11kDa, a 11S crystalloid protein with five bands at 50 kDa, a protein doublet of 47 and 51 kDa. These allergens have been named Ric c 1, Ric c 2, and allergen 3. Castor oil extractors, fertilizer workers, and farmers may acquire occupational dermatitis from handling plants or beans.

Castor oil may be used in cosmetics, particularly lipsticks, because of its superior moisturising characteristics, and may rarely produce allergic contact dermatitis (5,6). Sporadic case reports of allergy to it have implicated ricinoleic acid as the causative agent (7).

No other reports of pudendal contact dermatitis due to hydrogenated castor oil have been found. Fixed drug eruption is manifested as localized, circumscribed, round or oval plaques that characteristically recur in the same site with each use of the offending drug.

As far as we know, this is also the first report of fixed drug eruption caused by topical medication.

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References

1. KNOWLES SR, SHAPIRO LE, SHEAR NH. Serious adverse reactions induced by minocycline and review of the literature. Arch Dermatol 1996;**132**:934–939.
2. PANZANI R, JOHANSSON SGO. Results of skin test and RAST in allergy to a clinically potent allergen (castor bean). Clin Allergy 1986;**16**:259–266.
3. DAVISON AG, BRITTON MG, FORRESTER JA, DAVIES RJ, HUGHES DTD. Asthma in merchant seamen and laboratory workers caused by allergy to castor beans: analysis of allergens. Clin Allergy 1983;**13**: 553–561.
4. THORPE SC, KEMENY DM, PANZANI RC, MCGURL B, LORD M. Allergy to castor bean II. Identification of the major allergens to castor bean seeds. J Allergy Clin Immunol 1988;**82**:67–72.
5. BRANDE I, BOUJNAH-KHOUDJA A, FOUSSEAU J. Allergy to castor oil. Contact Dermatitis 1983;**9**:121.
6. SAIS I. Contact dermatitis to castor oil in a lipstick. Contact Dermatitis 1983;**9**:75.
7. FISHER AA. Allergic cheilitis due to castor oil in lipsticks. Cutis 1991;**47**:389.

First case of anaphylaxis to iodinated povidone

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Key words: anaphylaxis; basophil activation test; CAST; povidone; skin test.

A 32-year-old man underwent surgery in February 2002.

Induction of the anesthesia, took place at 8:10 hours with diprivan, sufenta and tracrium.

An infusion of cefazoline, pro-paracetamol and ketoprofene was started at 8:30 hours. At 9:30 hours, some minutes

Anaphylaxis to povidone could explain allergy to betadine®.

after the surgical wound was treated with a small quantity of betadine® the patient presented with grade 4 anaphylactic shock with acute respiratory distress syndrome requiring intensive resuscitation for 24 h. Sampling performed immediately after the incident showed the tryptase level to be 39 µg/l and serum histamine level 22.4 nmol/l.

Personal history included a previous surgery for peripheral neurolysis in 2000, during which acute urticaria of the left arm spreading to the neck and face was observed 20 min after swabbing with betadine®.

Skin tests carried out 9 months after the anaphylactic shock on skin reactive to codeine (4 mm) were negative for all curarizing agents. Prick test to betadine® was positive: 5 mm, intradermal test (IDT) was positive at 10^{-4} (10 µg/ml); 7.5 mm (injection papula 3 mm), prick test to povidone; 3 mm, IDT to povidone was positive at 10^{-4} ; 7 mm (injection papula: 2.5 mm).

Total IgE value was 5000 kIU/l. The results of the leukocyte histamine release test could not be interpreted because cellular histamine was lower than normal (280 nmol instead of 600). Also, the iodinated povidone interfered with the histamine assay. The basophil activation test (BAT) was positive to betadine® (14% CD3+, CD63+ and to povidone: 18% for a negative control at 0.5% and a positive anti-IgE control at 23%). The leukotriene release test was positive to betadine® and to povidone: 180 and 140 pg/ml (normal limit ≤100 pg/ml). Both tests were negative to atracurium.

The radioimmunoassay (RIA) to quaternary ammonium ions was positive at 4.8% but the inhibition test with atracurium was negative. RIA to penthotal was positive. Serum tryptase values were normal, thus eliminating the acceptable clinical explanation that the severity of the shock may be related to latent mastocytosis.

Anaphylaxis induced by iodinated povidone is anaphylaxis to povidone, the carrier molecule for iodine atoms. Anaphylaxis is documented by positive skin tests, BAT and cysteinyl-leukotriene release test (CAST), (1, 2). Positive RIA to quaternary ammonium ions is of no

value: there is no RIA inhibition with atracurium and the skin tests to curares are negative. Only 89% specificity has been documented (3–5). Similarly, RIA to penthotal may produce false positive results (6).

Povidone was previously used via the IV route as a plasma replacement or as a solubilizing agent for injectables. Doxycycline IV that contained povidone was withdrawn from the Pharmacopoeia because of anaphylactic reactions. Iodinated povidone is a skin antiseptic but the case reported here shows the risk of intra-vascular passage if a little quantity is introduced in a surgical wound. When the patient was questioned later, it was found that he had had a less serious reaction earlier. This is the second case of allergy reported to povidone (7).

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References

1. SAINTE-LAUDY J. Application of flow cytometry to the analysis of activation of human basophils. Immunologic validation of the method. *All Immunol (Paris)* 1998;**30**:41–43.
2. LEBEL B, MESSAAD D, KVEDARIENE V, RONGIER M, BOUSQUET J, DEMOLY P. Cysteinyl-leukotriene release test (CAST) in the diagnosis of immediate drug reactions. *Allergy* 2001;**56**:688–692.
3. GUÉANT JL, MATA E, MONIN B, MONERET-VAUTRIN DA, KAMEL L, NICOLAS JP, LAXENAIRE MC. Evaluation of a new reactive solid phase for radioimmunoassay of serum specific IgE against muscle relaxant drugs. *Allergy* 1991;**46**:452–458.
4. GUILLOUX L, RICHARD-BLUM S, VILLE G, MOTIN J. Histamine release assay and radioimmunoassay for the detection of IgE antibodies against neuromuscular blocking drugs. *Ann Fr Anesth Reanim* 1993;**12**:182–186.
5. MONERET-VAUTRIN DA, RENAUDIN JM, KANNY G, WIDMER S, HANESSE B, DEZ-FOULIAN B. Prospective study of the latent sensitization to muscle relaxants. In: OEHLING AK, HUERTA LOPEZ JG, editors. *Progress in allergy and clinical immunology Proceedings of the XVth ICACI*, 19–24 Oct, 1997, Seattle: Hogrefe & Huber 1997:361–367.
6. HARLE DG, BALDO BA, FISHER MM. The molecular basis of IgE antibody binding to thiopentone. Binding of IgE from thiopentone-allergic and non-allergic subjects. *Mol Immunol* 1990;**27**:853–858.
7. MONERET-VAUTRIN DA, MATA E, GERARD H, TRECHOT M. Probable allergy to povidone, responsible for a reaction to iodinated contrast medium: a case of asthma after hysterosalpingography. *All Immunol (Paris)* 1989;**21**:196,198–199.

Allergy to local anesthetics of the amide group with tolerance to procaine

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Key words: local anesthetics; drug allergy; ropivacaine; procaine; cross-reactivity.

Local anesthetics (LA) are widely used drugs in dentistry and ambulatory surgery. Adverse reactions to LA are frequently reported but the incidence of hypersensitivity reactions, like IgE-mediated reactions are rare, being probably <1% (1, 2). LA are grouped, depending on their chemical structure, into two categories: benzoate esters and amides; the latter group is usually well tolerated. In some exceptional reports of allergic reactions caused by LA of the amide group, mainly to lidocaine, other amide drugs, such as mepivacaine and ropivacaine, were usually well tolerated (3).

We present a 46-year-old woman, referred to us for evaluation of tolerance to LA before the performance of deep skin biopsies and dental procedures; in

Allergy to amide group local anesthetics including ropivacaine.

brief, she had anaphylactic reactions after the administration of an unknown local anesthetic agent 20 years ago and after lidocaine administration 12 years ago. Five years ago the patient also reported a large local urticaria after topical use of lidocaine (EMLA® anesthetic disc, AstraZeneca, USA). After obtaining the patient's consent, the diagnostic approach was carried out. Skin tests were performed with commercial LA drugs. Positive skin-prick test results with full-strength drug were obtained for lidocaine (AstraZeneca) with a wheal diameter of 5×4 mm, bupivacaine (AstraZeneca) with 6×4 mm and mepivacaine (Laboratórios Inibsa, Portugal) with 4×3 mm. Skin-prick test for ropivacaine (AstraZeneca) was negative, but the intradermal (ID) test with 1 : 100 dilution was positive (12×10 mm). Then, we performed skin tests (prick and ID testing) with procaine (Labesfal, Portugal), the LA belonging to the ester group, which were negative. On the next day, the patient was admitted to an intensive care unit for challenge procedures. The subcutaneous challenge with 1 ml of procaine (2%) was negative, (three biopsies – forearm and thigh), with no adverse reactions. In order to ensure that the positive skin test results were relevant, we carried out placebo controlled (saline), single-blind subcutaneous challenge test with 0.1 ml of ropivacaine (0.2%, preservative-free solution), and it was strongly positive (severe systemic reaction with laryngospasm, treated with parenteric epinephrine), confirming the diagnosis of allergy to LA of the amide group. The immediate hypersensitivity reaction was proved in this case and, as far as we know, this is the first report of severe hypersensitivity to ropivacaine, a drug unique among LA because it is prepared as an isomer (the S-enantiomer of the propyl homologue of bupivacaine) rather than a racemic mixture. The therapeutic alternative was procaine, the ester anesthetic, not routinely used nowadays. In conclusion, although ropivacaine is a first-choice drug for the majority of LA allergic patients, cross-reactivity must be thought within the amide group, in patients with positive skin tests (4); in these cases, allergy evaluation must be accomplished in a reference centre in order to identify safe alternatives (3).

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References

- GALL H, KAUFMANN R, KALVERAM CM. Adverse reactions to local anesthetics: analysis of 197 cases. *J Allergy Clin Immunol* 1996;**97**:933–937.
- FISHER MM, BOWEY CJ. Alleged allergy to local anaesthetics. *Anaesth Intensive Care* 1997;**25**:611–614.
- SOTO-AGUILAR MC, DESHAZO RD, DAWSON ES. Approach to the patient with suspected local anesthetic sensitivity. *Immunology and Allergy Clinics of North America* 1998;**18**:851–865.
- CUESTA-HERRANZ J, DE LAS HERAS M, FERNANDEZ M, LLUCH M, FIGUEROA E, UMPIERREZ A, LAHOZ C. Allergic reaction caused by local anesthetic agents belonging to the amide group. *J Allergy Clin Immunol* 1997;**99**:427–428.

Comparison of skin prick test and atopy patch test with dust mite extracts in patients with respiratory symptoms or atopic eczema dermatitis syndrome

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Key words: atopy patch test; mite allergy; skin prick test.

The atopy patch test (APT) was introduced to assess sensitization to inhalant allergens in patients with atopic eczema dermatitis syndrome (AEDS), but its diagnostic role in subjects with respiratory allergy has been only scantily investigated. We

Skin prick and atopy patch test in mite allergy.

sought to evaluate the response to mite extracts using APT and to skin prick tests (SPT) in subjects with persistent respiratory symptoms, AEDS, and with both the diseases.

Eighty-nine patients were included in the study, 75 (84.3%) children and 14 (15.7%) adults, 54 (60.7%) males and 35 (39.3%) females. They were divided in to three groups of 47, 15, and 27 subjects, with only respiratory symptoms, only AEDS, or both, respectively. They underwent usual SPTs with mite extracts and APTs by mite extract in Finn chambers, which were removed after 48 h, with readings after 20 min and 24 h.

Of the 89 patients, 24 showed a positive SPT and 69 a positive APT; in 17 both SPT and APT were positive, while 13 were negative to both the tests. The APT was more frequently positive than SPT not only in the two groups with AEDS – 32 of 42 (86.5%) vs eight of 42 (21.6%) – but also in the group with only respiratory symptoms – 37 of 47 (78.7%) vs 16 of 47 (34%).

These results confirm the high value of APT in patients with mite-induced AEDS and suggest that its routine use might improve the diagnosis of respiratory allergy to house dust mites also.

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Contact dermatitis due to a 'de Quervain' splint

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Key words: allergic contact dermatitis; 'de Quervain' splint; orthopaedic splint; polyethylene.

Several components of splints, such as rubber or neoprene, have been implicated

in contact dermatitis related to orthopaedic conditions (1, 2).

A 'de Quervain' splint is designed

to immobilize the wrist and avoid ulnar movement; this kind of splint maintains the thumb in its functional position.

A rare case of allergy contact dermatitis from a 'de Quervain' splint made of polyethylene is described.

A polyethylene 'de Quervain' splint was fixed to the left wrist a 35-year-old white woman because of a minor traumatism. Two days later she experienced itching and erythematous-squamous, oedematous plaques appeared in the skin region that had been in contact with the splint. The patient had been suffering from pellitory pollinosis since the age of 21, but otherwise her previous history was nil.

Patch testing with the European standard series, as well as splinter-related series, including rubber, plastics, glues, methacrylate and textile resins gave negative results. Skin prick test to natural rubber latex extract was also negative.

Patch test with a sample of the patient's splint gave a positive result (+ + +) at 48 and 72 h after. So allergic contact dermatitis due to this splint was diagnosed. With removal of the splint and topical betamethasone valerate, all symptoms disappeared.

Orthopaedic conditions require prolonged use of splints. Several components of splints, such as rubber or neoprene, have been implicated in contact dermatitis so far (1, 2). We report a case of allergic contact dermatitis provoked by a 'de Quervain' or half-length radial splint made of Plastazote® (Zotefoams, Croydon, UK).

Plastazote foam is a closed cell cross-linked polyethylene foam available in sheet form. Polyethylene is a condensation of polymers of ethylene. This product is malleable and flexible, but at the same time it is resistant to chemical substances, heat and biodegradation (3).

Polyethylene is a polymer too large to be responsible for contact sensitization, but other products are not included in the Plastazote® composition, and patch test with a sample of the splint gave a positive result. Nevertheless, polyethylene is easily oxidized, and antioxidants are used dur-

Skin problems related to polyethylene likely to increase.

ing manufacturing process to avoid such oxidation (4). Recently, a contact urticaria caused by polyethylene gloves has been described. In this case, the authors assume that some of the additives used as antioxidants caused urticaria (3). But, in the present case, patch test with plastic additives, including antioxidants, were negative, and although this splint is widely used, previous allergy contact dermatitis has not been reported.

Skin problems related to polyethylene are very likely to be more prevalent in the future, because polyethylene products are being increasingly used.

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References

1. VILLAREAL BALZA DE VALLEJO O. Contact dermatitis from diphenylthiourea in a knee brace. *Contact Dermatitis* 1997;**36**:166–167.
2. THOMSON KF, WILKINSON SM, CHALMERS RJ, BECK MH. Allergic contact dermatitis from a neoprene elbow splint. *Contact Dermatitis* 1998;**38**:179.
3. SUGIURA K, SUGIURA M, SHIRAKI R, HAYAKAWA R, SHAMOTO M, SASAKI K, ITOH A. Contact urticaria due to polyethylene gloves. *Contact Dermatitis* 2002;**46**:262–266.
4. Occupational hazards of polyethylene and polypropylene processing. *Prog Clin Biol Res* 1984;**141**:313–317.

Aerobiologic diagnosis of brassicaceae-induced asthma

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Key words: aerobiological sampling; asthma; brassicaceae; rapeseed.

In respiratory allergy, clinical history, skin prick tests, radioallergosorbent test

(RAST) assay and specific provocation tests can determine the causal agent in the majority of cases (1). In a

minority of cases the routine tests are unable to identify the causal allergen and, therefore, it is not possible to operate an effective allergen avoidance or to prescribe a specific immunotherapy when it is indicated. In those cases, a detailed environmental study can be helpful and provide suggestions for the diagnosis. We describe herein one case of unexplained asthma, diagnosed by means of the aerobiologic sampling of the patient's environment.

A 46-year-old woman from Bari (southern Italy) suffered from seasonal asthma for 6 years, the symptoms being present only during April and May, predominantly when she stayed at home. When she moved to Liguria (northern Italy) during the relevant months, the symptoms invariably disappeared. She had been treated in the past only with inhaled albuterol. Skin prick tests showed only a borderline positivity for parietaria, and this was confirmed by a class 1 Phadiatop RAST. Thus, parietaria could not be responsible for asthma, as this plant is ubiquitous in northern and southern Mediterranean regions of Italy and its pollen season is almost perennial.

An aerobiologic study of the environment was then performed with a portable sampler (Partrap FA52; Coppa, Biella, Italy). The sampler is battery-powered and provides a suction flow of about 5 l/min (2) and the samples are collected in a disposable chamber where they can be easily studied. The sampler was put on a window of the house for three consecutive days, when the patient was symptomatic. The samples were stained with acid fuchsin and analyzed by optical microscopy. The count showed 33 pollen grains of Brassicaceae and 18 of parietaria for cube meter of air. After an accurate inspection a culture of oilseed rape was discovered a couple of kilometers from the patient's house. These cultures had been established in the region about 7 years before. Although commercial Brassicaceae extracts for

In situ aerobiologic sampling detects allergen where routine tests fail.

diagnosis are not available, rapeseed was identified as the culprit allergen. It was suggested that the patient move away, if possible, during the pollen period and take a standard pre- and co-seasonal therapy with long-acting bronchodilators and inhaled corticosteroids.

Brassica napus (rapeseed, oilseed rape), belonging to Brassicaceae, is cultivated in many central and northern European regions for the production of rapeseed oil, whereas it is not typical of the Mediterranean area. Brassica is not considered a relevant allergen, but some reports point to its possible etiologic role in respiratory allergy (3). In our case, rapeseed was identified as the culprit allergen, and it was possible to establish, by clinical history, that symptoms appeared after about 1 year of seasonal exposure.

To make a precise etiologic diagnosis of respiratory allergy may be difficult in some cases, when clinical history, skin test and specific immunoglobulin E assay provide no conclusive or discordant results. In these cases, the presence of multiple sensitizations usually makes the diagnostic process more complex and leaves behind uncommon allergenic species. In such cases, an aerobiological sampling, performed directly in the environment where the patient lives, allows detection and quantification of the allergenic burden. In particular, sampling a restricted area can evidence those 'proximity (neighbourhood) allergens' which remain confined near their source (4). In the above-mentioned case, only the use of a portable sampler was able to identify the responsible allergen. Therefore, we suggest that in difficult cases, local aerobiologic sampling by portable devices should be part of the diagnostic process.

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References

1. BOUSQUET J, VAN CAUWENBERGE P. Allergic rhinitis and its impact on asthma. *J Allergy Clin Immunol* 2001;**108** (5 Suppl.):S208–S216.
2. PORTNOY J, LANDUYT J, PACHECO F, FLAPAN S, SIMON S, BARNES C. Comparison of the Burkard and Allergenco MK-3 volumetric collectors. *Ann Allergy Asthma Immunol* 2000;**84**:19–24.
3. D'AMATO G, SPIEKMA FT, LICCARDI G, JAGER S, RUSSO M, KONTOU-FILI K, NIKKELS H, WUTHRICH B, BONINI S. Pollen-related allergy in Europe *Allergy* 1998;**53**:567–578.
4. HEMMER W, LOCKE M, WANTKE F, JAGER S, GOTZ M, JARISCH R. Oilseed rape pollen is a potential relevant allergen. *Clin Exp Allergy* 1997;**27**:156–161.

Identification of common polymorphisms in the mitochondrial genome

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Key words: bronchial asthma; mitochondria; oxidative stress; polymorphisms.

The mitochondrial genome is inherited solely maternally and varies highly between different ethnic populations because of its rapid evolutionary change (1). Many mitochondrial polymorphisms have already been identified and some have been shown to be functional and in association with different diseases (2). Many of these 'mitochondrial' diseases have an inflammatory component in common. Mitochondria are the main source of reactive oxygen species (ROS) of the cell. An abnormal function of the mitochondria because of polymorphisms can cause an excess of ROS which in turn leads to inflammation, recruitment of immune effector cells by chemotactic factors and to an increased production of pro-inflammatory

cytokines like eicosanoids (3). These features are also the main characteristics of bronchial asthma. In fact, the important role of ROS in airway inflammation has already been discussed several years ago (4).

The genetic basis of bronchial asthma is highly complex, and many different chromosomal regions and candidate genes have been described to be in linkage or association with asthma (5). Until now, no study has investigated the potential involvement of the mitochondrial genome in the genetics of bronchial asthma. However this would be interesting for at least two reasons: first, it could be one explanation of the predominantly maternal transmission of asthma. Secondly, because of the involvement of mitochondria in the detoxification of pollutants, polymorphisms within mtDNA could be responsible for an enhanced reaction of asthmatic patients to environmental stimuli. Thus mitochondrial polymorphisms could even act as predictors of the individual response to pollutants.

We are interested to know whether common mitochondrial polymorphisms are associated with the development of bronchial asthma in our population. Thus the aim of this study was to identify polymorphisms within the mtDNA in the south-western German population.

By direct genomic sequencing of the whole mitochondrial genome in three DNA pools and a single DNA we were able to identify 29 common mitochondrial polymorphisms, two of those for the first time.

The search for polymorphisms within mtDNA is complicated by the fact that during the course of evolution many mitochondrial sequences were incorporated in the nuclear genome where they persist as pseudogenes. Therefore an amplification of mtDNA by polymerase chain reaction (PCR) might result in a co-amplification of these nuclear pseudogenes and thereby false 'mitochondrial' polymorphisms are identified. In fact, some 'pathogenic' mitochondrial polymorphisms which had been described a few years ago have already been shown to be due to such co-amplification. To avoid such co-amplification of nuclear DNA during PCR we used primer pairs which

Possible role of mitochondria in asthma genetics.

have previously been shown to solely amplify mtDNA.

The important role of mitochondria in the pathogenesis of inflammation and in the development of bronchial asthma has been described above. However, the possible impact of mitochondrial polymorphisms on the pathogenesis of asthma has been neglected so far. This study identified common polymorphisms within the mitochondrial genome in a population of the south-western part of Germany. These polymorphisms are now available for association studies with bronchial asthma, using the same population and primer sets.

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References

1. ANDERSON S, BANKIER AT, BARRELL BG, DE BRUIJN MH, COULSOU AR, DROUIN J et al. Sequence and organization of the human mitochondrial genome. *Nature* 1981;**290**:457–465.
2. DiMAURO S, SCHON EA. Mitochondrial DNA mutations in human disease. *Am J Med Genet* 2001;**106**:18–26.
3. MARTINEZ J, MORENO JJ. Influence of superoxide radical and hydrogen peroxide on arachidonic acid mobilization. *Arch Biochem Biophys* 1996;**336**:191–198.
4. BARNES PJ. Reactive oxygen species and airway inflammation. *Free Rad Biol Med* 1990;**9**:235–243.
5. HEINZMANN A, DEICHMANN KA. Genes for atopy and asthma. *Curr Opin Allergy Clin Immunol* 2001;**1**:387–392.