

allergens. The presentation was eczema/dermatitis (92.3%). The symptoms were referred with an evolution time of less than or equal to one year (82.9%).

The head was most commonly affected (79.4%) (the eyelids in 32.5%). The implicated cosmetics were facial creams (32.4%), haircare products – especially dyes, (26.4%), make-up products (23.5%), perfumes and, to a much lesser extent, sunscreen (2.1%). The causal allergenic groups were fragrances (44.1%), preservatives (32.3%), and para-phenylenediamine (23%). Fragrances were the allergen which most frequently justified the ACED to cosmetics in men (85.6%) and the most common sensitizers were oak moss absolute (14.7%), balsam of Peru, jasmine and isoeugenol (11.7%). The main preservatives causing allergy hypersensitivity in this group were formaldehyde (16.5%), Euxyl K 400 (14.6%), parabens (12.1%) and Kathon CG (11.75%).

In the last two decades there has been a significant increase in hypersensitivity reactions to cosmetic products, which is explained by the greater number of users and the increasing number of products offered (2,3).

Current reviews demonstrate that the most frequent allergenic groups are fragrances, preservatives, and para-phenylenediamine, showing effects in similar proportions to those found in our study (4,5).

Preservatives constitute the second most frequent allergenic group, and formaldehyde continues to be the most frequent allergen (6). A decrease can be observed in ACED caused by parabens, possibly due to replacement by preservatives with a more complex molecule, which in the not too distant future will also play a role as a cause of allergy. Although it has only recently been incorporated, Euxyl K 4000 is becoming more important within this group due to its extensive use (7).

Para-phenylenediamine is involved in the majority of hypersensitivity reactions due to contact hair dyes. It can present cross-reactions with other chemically related hair dyes such as *p*-toluendiamine, *p*-aminophenyldiamine, and even Henna (8).

It is recommended that similar studies be conducted periodically in order to observe the diverse variations in allergens responsible for hypersensitivity to cosmetic products.

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T cell mediated allergy to abciximab

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Key words: abciximab; epicutaneous test; immunohistology; T cell mediated eruption.

• A NONATOPIC woman, 54 years old, was referred for unstable angina. Coronary angiography revealed critical stenosis for which stenting was indicated, and which was performed under i.v. heparin and abciximab, infused for 12 h.

Nine days later the patient underwent a generalized exanthema with a cervicofacial swelling that

persisted for several days. A fall in platelet count to $64 \times 10^9/l$ was observed, in

parallel with a rise of eosinophilia from $123/mm^3$ to $1000/mm^3$. The test for heparin antibodies was negative. The anomalies subsided during the following days and the patient was discharged. Since that time two coronary angiographies have been carried out without any incident.

Skin tests were carried out nine months and 11 months later. Prick tests, intradermal tests (IDT) and epicutaneous tests were performed and read at 20 min, 24 h, 48 h, 72 h for iodinated contrast media, heparins, danaparoid and low molecular weight (LMW) heparins, beta-blockers, perindopril and Reopro[®], tested at 0.2 and 2 mg/ml.

At 48 h, only the IDT to Reopro[®] were positive to both concentrations. The histology showed a dense perivascular inflammatory infiltrate, mainly composed of lymphocytes. The whole population was CD45Ro⁺. Seventy percent of the cells were CD45Ra⁺, 60% were CD8⁺, 40% were CD4⁺. Numerous cells in the epidermis were CD1a⁺ with some identical cells sheathing the vessels. There were no deposits of immunoglobulins or of complement.

Abciximab is a platelet glycoprotein IIb/IIIa inhibitor. It is the fragment antigen binding portion of a chimeric human–murine monoclonal antibody. The binding to platelets is irreversible, lasting

A case with skin eruption and eosinophilia.

for the lifetime of the platelet, accounting for an extended biological half-life of more than 10 days (1). The immunogenicity of this presentation of abciximab may be postulated. The immunohistological study is compatible with a T cell mediated allergy. Seventy percent of cells are activated T cells, with a predominance of CD8⁺ cells. These features have been observed in drug skin reactions (2,3).

Thrombocytopenia is an adverse effect of abciximab, with an incidence of 4.2% to 5.2%, starting in the first 24 h (4,5). It occurred eight days later, unrelated to heparin: the search for heparin antibodies was negative, and besides this Reopro[®] inhibits heparin-induced aggregation (6,7). The eruption and the transient eosinophilia may favour an immunoallergic mechanism of this thrombopenia.

Sparse publications draw attention to an allergic risk of abciximab, despite the fact that anti-abciximab antibodies have been observed in 5.8% of cases receiving it (8). This is the first documented case of T cell mediated allergy to abciximab (9).

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Pneumothorax following insect sting anaphylaxis

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Key words: anaphylaxis; *Hymenoptera*; insect; pneumothorax; respiratory distress.

● REACTIONS TO *Hymenoptera* stings are usually local pain and dermal manifestations, but may develop into venom-induced hypersensitivity and anaphylaxis (1,2). The skin may manifest erythema, pruritus, urticaria, or angioedema over a large area, whereas the cardiovascular and respiratory systems may manifest potentially lethal consequences, such as laryngeal edema, bronchospasm (i.e. severe asthma), and cardiovascular collapse (i.e. anaphylactic shock) (1,2).

We present a 25-year-old nonsmoking man, without a history of pneumothorax, *Hymenoptera* sting or venom allergy. While hiking in the mountains in Cyprus

during his vacation, he experienced abrupt pain on the left upper back. Within 10 min local angioedema and edema became visible, and he shortly lost consciousness. Subsequently, signs of generalised erythema and urticaria, dyspnoea, swollen tongue, and a sense of fullness in the throat became apparent. He was immediately transferred to the local hospital where an embedded *Hymenoptera* stinger was removed from the left upper back. Forty-five minutes after the initial symptoms had appeared, the persisting dyspnoea increased acutely, with ipsilateral chest pain. A radiograph of the chest revealed a tension pneumothorax that was subsequently punctured.

On the 10th hospital day the patient was transferred to our hospital. Physical examination showed a minimal pectus excavatum, but no signs of Marfan or Ehlers–Danlos syndromes (i.e. dislocation of the lens, joint hypermobility, arachnodactyly, or dolichostenomelia). The patient was tall and thin (height 1.995 m, weight 73 kg; body mass index 18.3 kg/m²), with two brothers taller than 1.9 m. Laboratory tests indicated some muscle injury (elevated creatinine kinase and aspartate aminotransferase, but normal creatinine kinase MB isoenzyme and other liver enzymes), which normalised over the subsequent days. Increased eosinophils were found in all white cell counts. Levels of total IgE and wasp-, bee-, bumblebee-, hornet-, and phospholipase-specific IgE titres were normal. Subsequent radiographs of the chest showed a gradual reduction of the residual space. The chest tube was removed on the 17th hospital day and he was discharged with instructions to take prophylactic measures, to carry an epinephrine pen as emergency medication, and to wear an alert device identifying his risk for repeat anaphylaxis.

Primary, spontaneous pneumothorax has an incidence of 7–18 cases per 100 000 of the population per year among men, and 1–6 cases among women, with a recurrence rate of approximately 30% (3). In this patient, a ruptured subpleural bleb or bulla may have acted as a one-way valve, causing tension pneumothorax, which is a complication of one to two percent of cases of idiopathic spontaneous pneumothorax.

What could explain the sequence of events: a *Hymenoptera* sting resulting in an acute anaphylaxis that evolved into a tension pneumothorax? The venom from a

A rare hazardous sequel to a Hymenoptera sting.