

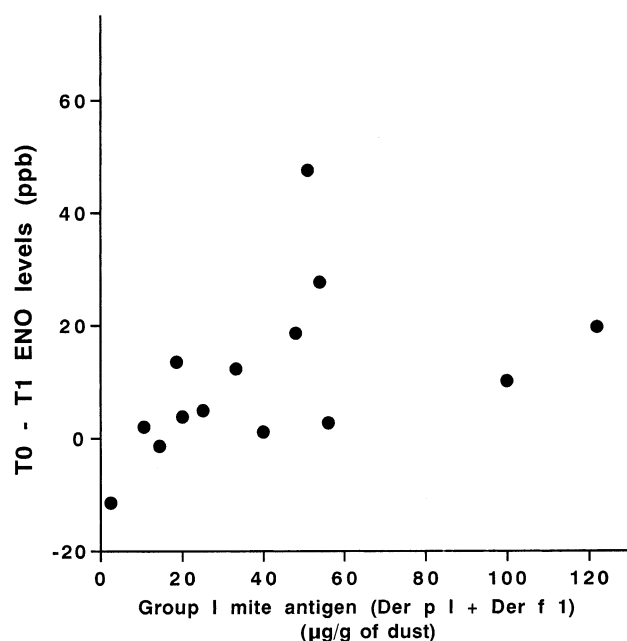


Figure 1. Relationship between difference in ENO level at T0–T1 and HDM group I antigens in study group.

the beginning of the study (T0). The regular treatment with inhaled steroids was gradually withdrawn in all the subjects. None of the subjects had received inhaled steroid treatment for at least 1 month before T1.

The study was approved by the Istituto Pio XII ethics committee, and both children and their parents gave informed consent.

The correlations between the differences of ENO levels at T0 and T1 (T0–T1) and the levels of HDM antigens in the beds of the patients before T0 were tested by Spearman's rank correlation test. The correlation between these values showed a value of $r=0.618$ ($P=0.026$) (Fig. 1).

In this study, we confirm that ENO measurement is a sensitive means to monitor avoidance of antigen exposure in asthmatic children allergic to HDM. The relevance of the effect of the original level of exposure to HDM on ENO reduction after HDM avoidance is even more evident if it considered that this statistically significant level of correlation was achieved in a small group of patients.

The results of this study reinforce the idea that the environmental control measures need to be made as effective as possible to achieve the most complete reduction of HDM in the environment of allergic asthmatic patients, particularly their beds.

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References

1. PIACENTINI GL, BODINI A, COSTELLA S, et al. Allergen avoidance is associated with a fall in exhaled nitric oxide in asthmatic children. *J Allergy Clin Immunol* 1999;**104**:1323–1324.

2. CHAPMAN MD, HAYMANN PW, WILKINS SR, BROWN MB, PLATTS-MILLS TAE. Monoclonal immunoassay for the major dust mite (*Dermatophagoides*) allergens, *Der p 1* and *Der f 1*, and quantitative analysis of the allergen content of mite and house dust extracts. *J Allergy Clin Immunol* 1987;**80**:184–194.
3. KHARITONOV SA, BARNES PJ. Exhaled nitric oxide: a marker of airway inflammation? *Curr Opin Anesthesiol* 1996;**9**:542–548.

Clinical features of cashew allergy

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Key words: allergy; cashew nut; fatal reactions.

● CASHEW has recently become widely available in the form of butter spreads. Cashew causes symptoms in formal food challenges (1) and has caused death (2). A report found 0.08% of British 4-year-olds to be allergic to

cashew (3), and 40% of 142 French peanut-allergic subjects

A potentially life-threatening disorder.

were found to be sensitized to cashew (4). We report here the clinical features of cashew allergy in 29 paediatric and adult subjects whose history of reaction was supported by a positive skin prick test to cashew or a raised serum level of cashew-specific IgE. Two subjects had positive open challenges to cashew. This study was approved by the Southampton Joint Ethics Subcommittee.

The age range of the 29 subjects was 1–30 years (median 7.5 years). Nineteen (65%) of the subjects were female. Twenty-six (89%) subjects were children (under 16 years). The median age of onset was 49 months (range 2 months–27 years), and 96% of subjects had

not knowingly been exposed to cashew before the exposure causing the first reaction. Fourteen subjects (48%) reacted to minimal contact with cashew; that is, smelling, touching, or tasting, but not eating, cashew. No one reported more than four reactions. Twenty-one subjects (72%) had suffered only one reaction to cashew, five (17%) had suffered two reactions, and only three had experienced more than two reactions. Fourteen subjects (48%) reported wheeze after first exposure, and 11 (38%) reported collapse or feeling faint.

This series describes features of nonfatal reactions to cashew. These features include the severity of cashew allergy (at least equal to that of peanut allergy), the importance of which is counterbalanced by the relative rarity of accidental exposure to cashew, compared to peanut (1, 5), and the later age of onset of cashew allergy (5, 6).

The young age of onset of peanut allergy has been attributed to the availability and convenience of peanut butter as a spread. By anecdotal evidence, parents have started to avoid peanut butter, in line with UK government advice, especially if there is a positive family history of allergy. We have seen cashew-allergic families who had been avoiding peanut butter and started using cashew butter instead, unaware of the potential for reactions of at least equivalent severity to peanut-allergic reactions. Cashew butter is now widely available in the UK, and it is reasonable to speculate that a decrease in age of onset of cashew allergy may become evident over time.

Allergists and other physicians must be aware that although cashew avoidance is easier than peanut avoidance, cashew allergy represents a threat to life that is at least as severe as that posed to peanut- or tree-nut-allergic subjects by the respective allergenic foods.

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References

1. BOCK SA, ATKINS FM. The natural history of peanut allergy. *J Allergy Clin Immunol* 1989;**83**:900–904.
2. SAMPSON HA, MENDELSON L, ROSEN JP. Fatal and near-fatal anaphylactic reactions to food in children and adolescents. *N Engl J Med* 1992;**327**:380–384.
3. TARIQ SM, STEVENS M, MATTHEWS S, RIDOUT S, TWISELTON R, HIDE DW. Cohort study of peanut and tree nut sensitisation by age of 4 years. *BMJ* 1996;**313**(7056):514–517.
4. MONERET VD, RANCE F, KANNY G, et al. Food allergy to peanuts in France – evaluation of 142 observations. *Clin Exp Allergy* 1998;**28**:1113–1119.
5. HOURIHANE JO'B, KILBURN SA, DEAN P, WARNER JO. Clinical characteristics of peanut allergy. *Clin Exp Allergy* 1997;**27**:634–639.
6. SICHERER SH, BURKS AW, SAMPSON HA. Clinical features of acute allergic reactions to peanut and tree nuts in children. *Pediatrics* 1998;**102**:e6.

Widespread thromboembolism in allergy

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Key words: allergy; asthma; *Aureobasidium pullulans*; eosinophilia; pulmonary embolism; thromboembolism.

● A 19-year-old man was referred to our department with fever of 38°C, dyspnea, and pleuritic chest pain lasting over 10 days. His past medical history was significant only for mild intermittent asthma, occasionally treated with salbutamol. His physical condition was normal except for tachycardia (120/min) and tachypnea (26/min).

Complete blood count and coagulation assay revealed hemoglobin, 13.8 g/dl; white blood cells, $18.5 \times 10^9/l$ with 40% eosinophils; platelets, $80 \times 10^9/l$; prothrombin time 16 s (control 12 s); activated partial thromboplastin time 32 s (control: 28 s); and D-dimers,

A case of transient hypereosinophilia and embolism.

4000 (normal range: 0–200). Oxygen saturation was 90%. Chest radiography demonstrated diffuse bilateral infiltrates.

A diagnosis of bilateral pulmonary embolism was confirmed by both pulmonary perfusion scintigraphy and angiography. Additional thrombi at the left deep femoral, anterior tibial, and right hepatic veins were detected by ultrasound Doppler. Because of worsening hemoptysis and dyspnea, along with the life-threatening thromboembolic phenomena, a titanium Greenfield filter was implanted in the inferior vena cava to prevent further emboli, followed by IV heparin and glucocorticosteroids. Subsequently, there was gradual fever resolution, respiratory improvement, and normalization of blood tests within the following week. After 2 weeks, the patient was discharged with warfarin and prednisone treatment.

Further examinations revealed no other underlying immunologic or thrombotic diathesis conditions. Blood, urine, and stool cultures were all negative, but sputum obtained from the patient on the day of admission grew black yeast identified as *Aureobasidium pullulans*.

After 1-year follow-up and without further complication, warfarin was discontinued. Prednisone was stopped gradually after 6 weeks, and the eosinophilia did not return.

Allergic reaction to saprophytic fungi may cause three pulmonary manifestations of clinical relevance: hypersensitivity pneumonitis, IgE-mediated asthma, and