

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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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

allergic bronchopulmonary fungal disease. Asthmatic patients are susceptible to allergic fungal reactions, and eosinophilia is one of the characteristic features of this syndrome. Although *Aspergillus* species are the main causative organisms, it is now recognized that many other fungi can be associated with this syndrome (1).

A. pullulans is a yeast-like fungus found in leaves, soil, and water. Although widely prevalent in the world, the level of its spores in air is low. It is a rare human pathogen, and it has been found to be the responsible allergen in few outbreaks of allergic pulmonary reactions, mostly due to contamination of heating, cooling, and ventilation units by this fungus (2).

Thromboembolism is a well-known manifestation of the idiopathic hypereosinophilic syndrome (IHS). Cardiac involvement in IHS is the main cause of death in this syndrome, and mural thrombosis is characteristic of IHS. Other thromboembolic events have been described in this syndrome, affecting the brain, lungs, retina, and intra-abdominal veins (such as the hepatic vein) (3).

Thromboembolic events were also found in transient, unsustained conditions of eosinophilia such as allergic reactions to food, drugs, or aeroallergens (4). The association between eosinophilia and thrombophilia was first suggested by Fauci et al. in 1982 (3). The proposed pathogenesis was the direct cytotoxic effect of the eosinophils on the endothelium or endocardium. More recent evidence suggests that eosinophil-derived mediators can modulate the coagulation cascade to promote thrombosis (5). It was found that eosinophil-specific granule proteins, particularly major basic protein (MBP), inhibit the anticoagulant effect of thrombomodulin (6, 7).

In summary, the patient presented here had severe eosinophilia most probably caused by an allergic reaction to *A.*

pullulans. This severe reaction, accompanied by marked eosinophilia, caused a thrombotic diathesis and widespread thromboembolism. This case illustrates the association of allergic conditions in general and eosinophilia in particular with thromboembolism. Hence one should bear in mind that even moderate eosinophilia may lead to thrombophilia resulting in thromboembolic manifestations.

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CD124 on monocytes in grass-pollen allergy

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Key words: allergy; IL-4; IL-12; monocytes.

● ALLERGIC individuals display a still unrecognized failure of IFN- γ production and/or excessive production of IL-4 that may affect the Th1-Th2 balance (1). Decreased production of IFN- γ by allergic PBMC could depend on defective production of other regulatory cytokines, such as monocyte (M ϕ)-derived

IL-4 receptor overexpression could explain IFN- γ decrease.

IL-12, a strong stimulator of IFN- γ production. Indeed, there have been reports that the M ϕ of allergic patients produced a smaller amount of IL-12 (2). Recently, IL-4 has been found to suppress IL-12 production (3). Therefore, this study aimed to compare the M ϕ response to IL-4 of allergic and healthy individuals *in vitro*.

The study involved monoallergic grass-pollen-sensitive patients ($n=16$) with positive skin tests, total IgE of >400 IU/ml, and moderate seasonal allergic symptoms. The patients were examined in a grass-