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Occupational asthma due to allergy to spinach powder in a pasta factory

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Key words: food allergy; food industry; inhalation fever; occupational asthma; spinach; vegetable.

Occupational asthma caused by exposure to animal proteins is well documented. Allergic risk, with vegetable proteins inhalation is actually scarce. We report a case of asthma induced by spinach dust.

A 30-year-old man, with no atopy, presented with chronic rhinitis and an asthma which had occurred over the last few months. He had worked for 9 years as a technician in a pasta factory and was exposed to the inhalation of dust and various food proteins. The patient's symptoms were exacerbated when spinach pasta was being prepared. Rhinitis and dyspnea occurred in the work place with delayed symptoms occurring at home later.

The clinical examination performed 2 days after occupational exposition revealed no abnormality, and the chest CT scan too revealed nothing. The respiratory function test showed distal obstructive ventilatory disorder and a reduced diffusing capacity to carbon monoxide transfer (COT) coefficient at 79%.

Pricktest (PT) and intradermal tests to molds were negative both immediately and on delayed reading. The PT to native foods showed multiple sensitizations to various ingredients of the pasta, like native spinach powder: 6 mm, with a positive codeine control at 2.5 mm. Spinach-specific IgE (Cap-RAST Pharmacia, Upssala, Sweden) were at a level of 15.4 kIU/l.

Ten minutes after handling the dried spinach powder, there was an early onset of dyspnea with sibilant and a 25% decrease in forced expiratory volume per second (FEV₁). Eight hours later, there was a delayed onset of recurrent dyspnea and another decrease in FEV₁ accompanied by fever and arthralgia.

Increase of blood eosinophilia, eosinophil cationic protein (ECP), circulating immune complexes (CIC), tumor necrosis factor (TNF)- α and interleukin (IL)6 were noted at 24 h.

Analysis of bronchoalveolar lavage (BAL) fluid carried out 72 h after the challenge demonstrated no hypercellularity: macrophages 80%, lymphocytes

10%, polymorphonuclear neutrophils 0% and eosinophils 10%. The microbiological analysis revealed a few colonies of *Candida albicans*, *Aspergillus fumigatus* and *Penicillium* sp.

Spinach powder culture revealed contamination by *A. niger* and *Penicillium* sp.

The lymphocyte activation test (LAT) with flow cytometry analysis of S-phase percentage at 72, 96 and 120 h, showed a significant activation of lymphocytes after incubation with spinach at 96 h (1).

Thus Occupational asthma was established.

Spinacia oleracea belongs to the Chenopodiaceae family which includes other comestible plants like quinoa. Food allergy to spinach is extremely rare (2), because of the rapid denaturing during digestion of the principal allergens (3). Cases of cross-allergy (spinach-latex or -mold) have been reported (4, 5).

In this case, the spinach used as a natural green coloring in the pasta was responsible for occupational asthma. The early symptoms, the PT results, positive IgE values to spinach, increase in serum ECP and the BAL fluid eosinophilia indicate an IgE-dependent asthma with delayed phase. However, the febrile arthralgia, the late onset of dyspnea, the slight decrease of diffusing capacity of lungs for CO and the results of the LAT with spinach powder tend to suggest an associated immunologic mechanism like in hypersensitive pneumonitis (CIC formation, and T lymphocytes activated by antigens that were either fungi or vegetable proteins).

The increase in serum IL6 and TNF α , after the challenge as well as the contamination of the spinach powder by *A. niger* and *Penicillium*, and the delayed onset dyspnea with the accompanying fever and arthralgia suggest an organic dust toxic syndrome (ODTS) that could be related to mycotoxins.

This case report documents the particular risk of respiratory sensitization to food proteins which are usually non-allergenic by ingestion. The complexity of the mechanism is well documented, perhaps due to molds, which may contribute to the development of a hypersensitive pneumonitis but also, via their mycotoxins, to the development of an ODTS.

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Severe venous thromboembolic disease in Churg–Strauss syndrome

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Key words: Churg–Strauss syndrome; deep vein thrombosis; eosinophilia; pulmonary embolism; vasculitis.

Churg–Strauss syndrome (CSS) is a systemic vasculitis, characterized by severe asthma, blood and tissue eosinophilia and diffuse organ involvement (1). Venous thromboembolic disease (VTED) has exceptionally been reported in CSS.

We report three cases of severe VTED in a cohort of 10 CSS patients.

A 58-year-old man had a diagnosis of CSS based on the association of steroid-dependent asthma, blood eosinophilia (21% of WBC, $3.3 \times 10^9/l$), transient pulmonary infiltrates, sinusitis, profound asthenia, and weight loss. No tissue biopsy was performed. Antineutrophil cytoplasmic antibodies (ANCA) were negative. Cyclophosphamide pulses and increased steroid therapy improved markedly his condition. One month later, the patient presented right calf pain and an episode of syncope. Ultrasonography revealed right femoro-popliteal venous thrombosis and spiral computed tomography (CT) of the chest demonstrated bilateral proximal pulmonary embolism (Fig. 1). There was no blood hypereosinophilia at that time. No coagulation abnormalities were found.

A 37-year-old woman was admitted in intensive care unit for dyspnea, fever, and congestive heart failure confirmed by echocardiography and right-heart catheterization. She had a 1-year history of difficult-to-control asthma and sinusitis with recent worsening of her respiratory symptoms with profound asthenia.

Three patient boxes of severe venous thrombo-embolic diseases are described in a cohort of ten patients with Churg–Strauss syndrome.

Blood counts showed 19% of eosinophils ($1.94 \times 10^9/l$). The ANCA were negative. A clinical diagnosis of CSS was established, but no tissue biopsy was performed because of the patient's critical condition. Spiral CT of the chest showed a right posterobasal pulmonary embolism confirmed by pulmonary angiography. Doppler ultrasonography revealed bilateral deep vein thrombosis.

A 72-year-old man with a history of steroid-dependent asthma, chronic sinusitis, marked blood eosinophilia (28% of WBC, $3.2 \times 10^9/l$), pericardial effusion, abdominal pain, arthralgia and asthenia had a high clinical suspicion of CSS. No tissue biopsy was performed. The ANCA were negative. During clinical follow up, he experienced an episode of idiopathic lower limb thrombosis (left common femoral vein). Eosinophil blood count was normal at that time. No coagulation abnormalities were found.

Coronary involvement leading to myocardial infarcts and heart failure is a known complication of CSS (2). A report of coronary artery stenosis repermeabilization after corticosteroid therapy in CSS highlights that vasculitis presumably plays a significant role in these patients (2). VTED is believed to be a rare occurrence in the course of CSS. There are only a few cases in the literature, including a report of deep vein thrombosis and pulmonary infarction in a 13-year-old boy, and a case of thrombosis of the left subclavian vein (3). In



Figure 1. Spiral computed tomography of the chest showing proximal pulmonary embolism in a patient with Churg–Strauss syndrome.