

Asthma induced by latex from 'Christmas flower' (*Euphorbia pulcherrima*)

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Key words: asthma; Christmas flower; cross-reactivity; *Euphorbia pulcherrima*; IgE-mediated; poinsettia; rhinitis; rubber latex.

Christmas flower or poinsettia [*Euphorbia pulcherrima* (Ep)] is widely used as an ornamental plant during Christmas season in many countries. It belongs to the family Euphorbiaceae that includes other allergenic plants such as *Hevea brasiliensis* (Hb) or rubber tree. There are few reports of contact dermatitis induced by this plant (1, 2). We studied a 6-year-old male who presented with rhinitis and asthma upon exposure to several poinsettia plants for two consecutive Christmas seasons. He clearly improved when he was out of his apartment. At the time of the first study he did not suffer from any other respiratory symptoms. Three years after the first Christmas-related episode, he developed mild rhinoconjunctivitis during spring time. He was able to eat banana, kiwifruit and chestnut without any ill effects. He had never eaten avocado.

Red and green leaves and latex from Ep, and natural rubber latex were extracted in phosphate-buffered saline (PBS), dialysed in a 3.5-kDa cut-off dialysis membrane and freeze-dried, that was later reconstituted at a concentration of 10 to 1 mg/ml (25 mg of protein per 100 mg of raw material). Healthy, atopic and rubber latex (*H. brasiliensis*)

allergic patients were studied as controls.

The patient had positive skin-prick test (SPT) to grass pollen and to Ep red leaves (5 mm) and latex extracts (7 mm), whereas it was negative to green leaf extract and other common aeroallergens in our area. Natural rubber latex extract from Hb elicited a 2-mm wheal. Prick-prick tests with kiwifruit, banana, avocado and chestnut were negative. In five healthy control patients, SPT with Ep extracts were negative, but were positive in five of eight patients diagnosed with rhinitis and asthma induced by latex from Hb.

Baseline PC₂₀ methacholine was 14.3 mg/ml. Pre-challenge induced sputum samples showed no eosinophils. Both performed as previously described (3). After parent's consent, bronchial challenge by tidal volume method with Ep extract at a concentration of 0.31 mg/ml elicited an isolated immediate asthmatic response, with a 20% fall in forced expiratory volume (FEV)₁.

Twenty-four hours after the specific bronchial challenge, PC₂₀ methacholine decreased to 2.5 mg/ml and 5% of eosinophils was seen in induced sputum sample. Rubbing and latex glove use test were negative.

In one patient diagnosed with rhinitis and asthma induced by Hb latex, who also had a positive SPT to Ep, the bronchial challenge test with Ep extract was negative.

Total IgE was 703 kU/l. Specific IgE (Pharmacia CAP System, Uppsala, Sweden) was positive to Ep latex (6.65 kU/l), Hb latex (8.24 kU/l), banana (6.28 kU/l) and chestnut (9.3 kU/l). ImmunoCAP inhibition experiments demonstrated a high degree of allergenic cross-reactivity between latex extracts of Hb and Ep.

IgE immunoblots performed under reducing conditions revealed major IgE-binding bands in the Ep latex extract (Fig. 1). The most intense reaction was observed at 36, 25 and 19 kDa bands. Patient's serum also revealed several IgE-binding bands in the Hb extract (Fig. 1).

Case reports of IgE-mediated rhinitis and asthma induced by latex from ornamental plants, and dust samples from the floor, such as *Ficus benjamina*, have been previously described (4). To the best of our knowledge, this is the first case report of IgE-mediated rhinitis and asthma induced by latex of Christmas flower or poinsettia. Although extensive *in vitro* cross-reactivity with natural rubber latex and related fruits was demonstrated, the patient had no symptoms with these products.

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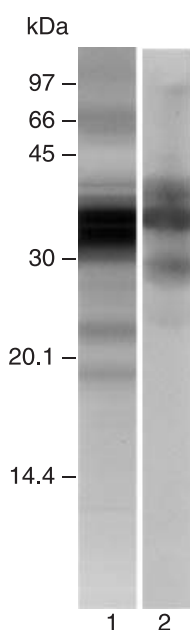


Figure 1. IgE immunoblot with patient's serum. Lane 1: latex of *Euphorbia pulcherrima*; lane 2: rubber latex of *Hevea brasiliensis*.

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References

1. MASSMANIAN A. Contact dermatitis due to *Euphorbia pulcherrima* Wild, simulating a phototoxic reaction. *Contact Dermatitis* 1998;**38**:113–114.
2. PAULSEN E, SKOV PS, ANDERSEN KE. Immediate skin and mucosal symptoms from pot plants and vegetables in gardeners and greenhouse workers. *Contact Dermatitis* 1998;**39**:166–170.
3. QUIRCE S, FERNÁNDEZ-NIETO M, BARTOLOMÉ B, BOMBÍN C, CUEVAS M, SASTRE J. Glucoamylase: another fungal enzyme associated with baker's asthma. *Ann Allergy Asthma Immunol* 2002;**89**:197–202.
4. BIRCHER AJ, LANGAUER S, LEVY F, WAHL R. The allergen of *Ficus benjamina* in house dust. *Clin Exp Allergy* 1995;**25**:228–233.

Hypersensitivity pneumonitis caused by triglycidyl isocyanurate

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Key words: epoxy compound; hypersensitivity pneumonitis; triglycidyl isocyanurate.

Polyester powder paints are extensively used in metal painting. Exposure to these powder paints may give rise to hypersensitivity pneumonitis (1) and late asthmatic reactions accompanied by fever and leucocytosis (2), which are thought to be caused by acid anhydrides (1, 2). Triglycidyl isocyanurate (TGIC), a synthetic epoxy compound that is often used as a curing agent in these paints, has not been previously reported as a causative agent of hypersensitivity pneumonitis.

We describe a 22-year-old man, a smoker of 1 pack-year, who developed recurrent episodes of fever, cough, dyspnoea, shivers and nocturnal sweating 4 months after he started to work in a powder-coating factory. In his workplace, metallic boards were treated with

an electrostatic powder paint containing 2.5–10% TGIC. This paint was sprayed onto the metallic boards through injectors in an open tunnel and they were then heated. He was first admitted in the emergency room after a typical workshift of 8 h. Physical examination revealed bilateral crackles, fever (39°C), and hypoxemia. Bilateral reticulonodular shadowing was seen on chest radiography. Diffuse micronodular infiltrates were observed in the high-resolution computed tomography. Lymphocytosis (88%) and an increased number of CD8+ cells (93%) were observed in the bronchoalveolar lavage fluid with an inverse CD4+/CD8+ ratio (0.05). A transbronchial lung biopsy sample revealed a diffuse interstitial infiltrate comprised of T lymphocytes, with a striking predominance of CD8+ lymphocytes. Results of serologic tests to common respiratory microorganisms were negative. Precipitating antibodies to a panel of moulds and thermophilic actinomycetes were not detected in the patient's serum.

Only supportive treatment was given and within 24 h the fever waned and the patient improved in a few days. Spirometry revealed a forced vital capacity (FVC) of 80% predicted, forced expiratory volume (FEV)₁ of 83% predicted and FEV₁/FVC 88%. Lung volumes determined by plethysmography disclosed a total lung capacity of 81% predicted and residual volume of 92% predicted. Diffusion capacity of the lung for carbon monoxide was reduced to 68% predicted.

After 1 month of being away from work, the patient was symptom-free, the diffusing capacity was normal and a methacholine inhalation test revealed no airway hyper-responsiveness. Specific inhalation challenges with toluene and hexamethylene diisocyanates and with phthalic anhydride yielded negative results. Specific IgG antibodies to TGIC were determined by enzyme-linked immunosorbent assay (ELISA). TGIC was conjugated with human serum albumin (HSA) as previously described (3). A positive IgG-ELISA result of 0.70 optical densities (OD) was obtained against TGIC–HSA conjugate with patient's serum, whereas serum from an unexposed control subject had a value of 0.20 OD.

A workplace 'realistic' challenge was carried out by asking the patient to perform his usual duties over a standard workshift of 8 h. By the end of the workshift, the patient experienced an acute episode of hypersensitivity pneumonitis.

The results of the investigations carried out in this worker point out that he had developed hypersensitivity pneumonitis caused by sensitization to TGIC contained in polyester powder paint. Allergic contact dermatitis (4) and occupational asthma caused by TGIC have been previously described (4, 5).

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References

1. PIIRILÄ P, KESKINEN H, ANTILA S, HYVÖNEN M, PFÄFFLI P, TUOMI T et al. Allergic alveolitis following exposure to epoxy polyester powder paint containing low amounts (<1%) of acid anhydrides. *Eur Respir J* 1997;**10**:948–951.
2. CARTIER A, VANDENPLAS O, GRAMMER LC, SHAUGHNESSY MA, MALO JL. Respiratory and systemic reaction following exposure to electrostatic polyester paint. *Eur Respir J* 1994;**7**:608–611.
3. HOWE W, VENABLES KM, TOPPING MD, DALLY MB, HAWKINS R, LAW JS et al. Tetrachlorophthalic anhydride asthma: evidence for specific IgE antibody. *J Allergy Clin Immunol* 1983;**71**:5–11.
4. MEULEMAN L, GOOSSENS A, LINDERS C, ROCHETTE F, NEMERY B. Sensitization to triglycidylisocyanurate (TGIC) with cutaneous and respiratory manifestations. *Allergy* 1999;**54**:752–756.
5. PIIRILÄ P, ESTLANDER T, KESKINEN H, JOLANKI R, LAAKKONEN A, PFÄFFLI P et al. Occupational asthma caused by triglycidyl isocyanurate. *Clin Exp Allergy* 1997;**27**:510–514.