



HAL
open science

Occupational asthma to dried tobacco leaves: a very delayed diagnosis

E. Penven, Mathias Poussel, Isabelle Thaon, Christophe Paris

► **To cite this version:**

E. Penven, Mathias Poussel, Isabelle Thaon, Christophe Paris. Occupational asthma to dried tobacco leaves: a very delayed diagnosis. *Journal of Investigational Allergology and Clinical Immunology*, 2015, 25 (2), pp.144-145. hal-01713335

HAL Id: hal-01713335

<https://hal.univ-lorraine.fr/hal-01713335v1>

Submitted on 20 Feb 2018

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

2. Sánchez-Madrid F, Morago G, Corbi AL, Carreira J. Monoclonal antibodies to three distinct epitopes on human IgE: their use for determination of allergen-specific IgE. *J Immunol Methods*. 1984;73:367-78.
3. Duffort OA, Polo F, Lombardero M, Díaz-Perales A, Sánchez-Monge R, García-Casado G, Salcedo G, Barber D. Immunoassay to quantify the major peach allergen Pru p 3 in foodstuffs. Differential allergen release and stability under physiological conditions. *J Agric Food Chem*. 2002;50:7738-41.
4. Kitano A, Miyazaki T, Yoshioka K, Kurono T, Kurono S, Matsumoto T. Facial rash and palmoplantar pruritus in an infant after first contact with kaki. *J Investig Allergol Clin Immunol*. 2009;19:237-8.
5. Anliker MD, Reindl J, Vieths S, Wüthrich B. Allergy caused by ingestion of persimmon (*Diospyros kaki*): detection of specific IgE and cross-reactivity to profilin and carbohydrate determinants. *J Allergy Clin Immunol*. 2001;107:718-23.
6. Martínez JC, Armentia A, Bartolomé B, Callejo A, Fuentes MJ, Fernández A. Anaphylaxis after ingestion of sharon fruit. *Allergol Immunopathol*. 2001;29:69-71.
7. Prandini M, Marchesi S. Anaphylaxis to persimmon. *Allergy*. 1999;54:897.
8. Bolhaar ST, van Ree R, Ma Y, Bruijnzeel-Koomen CA, Vieths S, Hoffmann-Sommergruber K, Knulst AC, Zuidmeer L. Severe allergy to sharon fruit caused by birch pollen. *Int Arch Allergy Immunol*. 2005;136:45-52.
9. Salcedo G, Sanchez-Monge R, Diaz-Perales A, Garcia-Casado G, Barber D. Plant non-specific lipid transfer proteins as food and pollen allergens. *Clin Exp Allergy*. 2004;34:1336-41

■ *Manuscript received October 11, 2013; accepted for publication, May 28, 2014.*

Beatriz Rodríguez Jiménez
 Unidad de Alergología
 Hospital Universitario de Getafe
 Carretera de Toledo Km 12,500
 28905 (Getafe) Madrid, Spain
 E-mail: brodriguezjimenez@hotmail.com

Occupational Asthma to Dried Tobacco Leaves: A Very Delayed Diagnosis

Penven E^{1,2}, Poussel M^{3,4}, Thaon I^{1,2}, Paris C^{1,2}

¹*Occupational Diseases Department, Bâtiment Philippe Canton, CHU Nancy, Vandoeuvre-lès-Nancy, France*

²*EA-7298 INGRES, Université de Lorraine, Vandoeuvre-lès-Nancy, France*

³*Department of Pulmonary Function Testing and Exercise Test, CHU Nancy, Vandoeuvre-lès-Nancy, France*

⁴*EA 3450 DevAH - Development, Adaptation and Disadvantage, Cardiorespiratory regulations and motor control, Université de Lorraine, Vandoeuvre-lès-Nancy, France*

Key words: Occupational asthma. Tobacco. Specific inhalation challenge.

Palabras clave: Asma ocupacional. Tabaco. Prueba específica de provocación por inhalación.

A 59-year-old woman who had never smoked and had no known history of atopy was seen in our department in 2011 for a possible diagnosis of occupational asthma. The patient had not been employed since 2000 and had worked as a production agent at a cigarette manufacturing facility between 1986 and 2000. Her work primarily consisted of manually filling small bags with dried, milled tobacco leaves. She described the factory as very dusty, particularly during the early years, but no atmospheric measurements were available. The patient reported the appearance of rhinitis, cough, dyspnea, and wheezing, closely related to work periods, some months after starting to work at the factory. An initial check-up in 1991 led to a diagnosis of asthma, but a skin prick test (SPT) to tobacco leaves yielded a wheal of just 2 mm in diameter and was considered doubtful. The patient continued to work until 2000 without any change in her exposure to tobacco leaves; she described progressive worsening of her asthma, despite short-acting β_2 -agonist treatment. The patient stopped working at the factory in 2000 and was no longer exposed to respiratory allergens or irritants. Her respiratory symptoms decreased, but did not disappear completely. In 2008, she experienced worsening of dyspnea and received inhaled corticosteroid therapy, with only slight improvement due to poor treatment adherence. In 2011, the patient was referred to our department for a possible diagnosis of occupational asthma. Clinically, she had bronchial hyperresponsiveness, but no wheezing under treatment. SPTs to airborne allergens and tobacco leaves (after humidification) were negative. The blood count was normal and total IgE was 1451 IU/mL. Specific IgE levels were 0.21 kU_A/L for tobacco leaves and 0.12 kU_A/L for eggplant. The results were negative for latex, tomato, and potato allergens. The baseline functional respiratory test demonstrated a slight reversible obstructive syndrome (forced expiratory volume in the first second [FEV₁], 2.11 L; 91% of predicted; FEV₁/forced vital capacity [FVC], 70%; forced expiratory flow at 50% relative to FVC, 1.97 L/s; 54% of predicted). A methacholine challenge was

positive with a 36% decrease in FEV₁ for a cumulative dose of 160 µg of 1% methacholine (approximately 0.5 mg/mL). An inhalation control test to lactose powder (stepwise handling of lactose powder for 1, 2, 3, 4 and 5 minutes) was strongly negative. In the specific inhalation challenge (SIC) to tobacco, the patient was asked to pour 2 cups of 20 g of tobacco leaf powder according to the same schedule as that used for lactose powder. A strong immediate positive reaction appeared after 10 minutes of cumulative exposure, with a 42% decrease in FEV₁ relative to baseline. No delayed symptoms were observed before discharge from our department 8 hours after the SIC, but the general practitioner reported some wheezing the following morning. To rule out nonspecific irritant reactions, we performed SICs with tobacco leaves in the same conditions in 2 volunteers, using a positive methacholine test as a control. These 2 SICs were strongly negative. A possible diagnosis of occupational asthma to tobacco leaves was established.

Occupational asthma to tobacco dust was first described by Gleich et al [1] in 1980. Since then, many authors have reported cases of occupational asthma as well as alterations in respiratory function in cigarette facilities. In 1988, for instance, Lander et al [2] reported a significant change in daytime peak flow expiratory in tobacco workers compared with controls. More recently, Mustajbegovic et al [3], following the systematic examination of 121 tobacco workers, reported 6 cases of occupational asthma to tobacco dust, interestingly all in women (total women, 97). To date no tobacco allergens have been identified. Although contamination of tobacco by fungi was initially hypothesized, more recent findings suggest that a profilin-like protein, or a villin-like protein [4] belonging to the cytoskeletal of plants, may be involved, as there have been several (but inconsistent) reports of cross-reactivity between several allergens from the Solanaceae family [5-6] as well as latex [7] in individuals with tobacco leaf asthma. Although our case is consistent with previously reported cases, the diagnosis of occupational asthma is questionable considering that the source of occupational exposure was eliminated a long time ago. The absence of atopy or previous asthma, intense occupational exposure to tobacco leaves for more than 10 years, and the patient's clinical history all support this potential diagnosis but may not be sufficient. The strongest diagnostic evidence is the positive SIC to tobacco leaf powder. However, this may also correspond to a simple immediate reaction to a nonspecific irritant. The absence of a reaction to the control test to lactose powder using the same procedure, the severity of the specific response (fall of 42% in FEV₁ relative to baseline), and the existence of a slight delayed reaction at 24 hours all support a diagnosis of occupational asthma rather than a simple immediate irritant reaction to tobacco leaf dust. Nevertheless, specific IgE to tobacco leaves was low, but this might be explained by the long period without exposure. We therefore believe that a diagnosis of occupational asthma to tobacco leaves is the most plausible diagnosis. Consequently, even though end of exposure is often proposed as an explanation for a negative SIC, our observation suggests that positive reactions may still occur many years later. Clinicians should also be aware that functional respiratory reactions could still be severe in such cases.

Funding

The authors declare that no funding was received for the present study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

1. Gleich GJ, Welsh PW, Yunginger JW, Hyatt RE, Catlett JB. Allergy to tobacco: an occupational hazard. *N Engl J Med* 1980;302:617-9.
2. Lander F, Gravesen S. Respiratory disorders among tobacco workers. *Br J Ind Med* 1988;45:500-2.
3. Mustajbegovic J, Zuskin E, Schachter EN, Kern J, Luburic-Milas M, Pucarin J. Respiratory findings in tobacco workers. *Chest* 2003;123:1740-8.
4. Mittermann I, Voronin V, Heberle-Bors E, Valenta R. Identification of a villin-related tobacco protein as a novel cross-reactive plant allergen. *FEBS Lett* 2005;579:3807-13.
5. Armentia A, Bartolomé B, Puyo M, Paredes C, Calderón S, Asensio T, del Villar V. Tobacco as an allergen in bronchial disease. *Ann Allergy Asthma Immunol* 2007;98:329-36.
6. Ortega N, Quiralte J, Blanco C, Castillo R, Alvarez MJ, Carrillo T. Tobacco allergy: demonstration of cross-reactivity with other members of Solanaceae family and mugwort pollen. *Ann Allergy Asthma Immunol* 1999;82:194-7.
7. Armentia A, Dueñas-Laita A, Bartolomé B, Martín-Gil FJ, San Miguel A, Castrodeza JJ. Clinical significance of cross-reactivity between tobacco and latex. *Allergol Immunopathol (Madr)* 2010;38:187-96.

■ Manuscript received February 14, 2014; accepted for publication, June 2, 2014.

Christophe Paris

Centre de consultations de pathologies professionnelles
 CHU Nancy
 Bâtiment Philippe Canton
 Rue du Morvan
 Vandoeuvre-lès-Nancy, F-54511, France
 E-mail: christophe.paris@inserm.fr