Case Report

Occupational rhinitis due to Pepsin

Ll. Marquès,1 S. Lara,1 T. Abós,1 B. Bartolomé2

1 Allergy Unit. Hospital Universitari Arnau de Vilanova. Lleida, Spain
2 R&D Department, Bial-Aristegui, Bilbao. Spain

Summary. A woman presented work-related rhinoconjuntivitis due to inhalation of pepsin used in a slaughterhouse. Prick tests and conjunctival challenge were positive to pepsin. Serum specific IgE to pepsin was 5.58 kU/L and an IgE-binding band of 43 kDa was detected in SDS-PAGE Immunoblotting. Rhinoconjuntivitis improved clearly when the patient was assigned to another place without contact with pepsin. Pepsin has been previously reported to cause occupational allergic asthma on three occasions. As far as we know, this is the first reported case in which an IgE-immunoblot has been performed.

Key words: enzymes, occupational rhinitis, pepsin A

Enzymes are well known sensitizers that often cause occupational respiratory diseases. Pepsin is an enzyme used in different occupational settings. We describe a case of occupational allergic rhinoconjuntivitis due to pepsin in a woman who worked checking meat for trichinae.

A 22 year-old-woman who had been working at a slaughterhouse for 3 years presented work-related rhinoconjuntivitis after 7 months. She did three weekly rotatory tasks at her work: checking meat for trichinae at the laboratory; sanitary control of cattle and sheep, and sanitary control on a pork production line.

She suffered from immediate and delayed rhinoconjuntivitis in the laboratory area. Symptoms were triggered by pepsin powder used to proteolize the samples of meat in order to search for trichinae larvae. A standard method is used to prepare these samples before its examination by the veterinarian: pure pepsin powder is poured in a pan with boiling water and pieces of meat. This is when the patient presented her symptoms.

We prepared a pepsin solution at 10 mg/ml and performed cutaneous and challenge tests on the patient. Serum specific IgE measurement and IgE SDS-PAGE immunoblotting were also carried out.

The prick test was positive to pepsin (wheal 4 mm) (negative in various atopic and non atopic controls). Prick tests to common inhalants were positive for *Dermatophagoides pteronyssinus* and pollens from *Chenopodium album* and *Artemisia vulgaris*. Pepsin conjunctival challenge was positive: at 10 mg/ml she presented conjunctival erythema with epifora and pruritus (negative in three atopic and three non atopic controls). Serum specific IgE was 5.58 kU/L to pepsin (class 3), 1.2 kU/L (class 2) to *Dermatophagoides pteronyssinus*, and 2.8 kU/L (class 2) for cow dander. When the pepsin sample was incubated with patient’s serum in SDS-PAGE Immunoblotting, an IgE-binding band of 43 kDa was detected (Figure 1). Spirometry values were in the normal range (FVC 3.54 L (86%), FEV1 3.24 L (96%), FEV1/FVC 91%). The rhinoconjuntivitis improved clearly when the patient was assigned to another place without contact with pepsin.

The patient’s clinical history suggested occupational
Occupational rhinitis due to Pepsin

rhinitis due to pepsin exposition. Prick test, CAP and immunoblot results showed IgE-mediated reaction to pepsin and the challenge test identified pepsin as the etiologic agent. We consider the sensitization to Dermatophagoides pteronyssinus and cow dander not significant to the rhinoconjunctivitis symptoms observed, as the patient only manifests these symptoms when exposed to pepsin powder, not in any other situation. Moreover, it is quite normal to find simultaneous sensitisations in occupational asthma and rhinitis [1].

Commercial pepsin is a protease usually obtained from pig pepsinogen by post-translational proteolysis and acts as an important digestive enzyme of the gastric juice. There are many allergens with protease activity. In recent years, some authors have suggested the importance of the protease activity of certain allergens (Der p 1, Der p 3, Der p 9, protein extracts from Aspergillus sp., from cockroach, from various species of pollen) to elicit the allergic response. In vitro they demonstrated the proteolytic activity of some allergens on some membrane proteins (CD25, CD23, protease-activated receptor type 2, occludin, claudin) of different types of cells (lung epithelial cells, B cells, dendritic cells) which produce physiological pro-inflammatory or pro-allergic changes in them [2,3]. In vivo, using immunization assays with animal models, they observed a different T cell response depending on the catalytic state of the allergen used to immunize them: when the active form of the enzyme was used, a Th2 response was observed, whereas a Th1 response was detected when the inactive form was used [4].

Pepsin has been previously reported to cause occupational allergic asthma: Cartier et al described a case of occupational asthma in the pharmaceutical industry, with positive cutaneous test at 10 mg/ml [5]; Añibarro et al described a case of asthma in a cheese factory and used pepsin at 1 mg/ml for cutaneous test [6]; in 1997 Drexler et al described a patient with the same job as ours with asthma and used pepsin at 10 mg/ml [7]. As far as we know this is the first reported case in which an IgE-immunoblot has been performed.

Many of the causative agents of occupational asthma are also capable of inducing occupational rhinitis and patients with occupational asthma frequently report symptoms of occupational rhinitis. In a recent study, a higher risk of asthma was found among patients with occupational rhinitis than among those with other occupational diseases [8]. In our patient the eviction of pepsin may possibly prevent the evolution to asthma.

References


Lluís Marquès

Allergy Unit. Hospital Santa María Rovira Roure, 44 25198 Lleida, Spain Tel.: +34 973 72 72 22 Fax: +34 973 72 72 25 E-mail: lluismarques@menta.net