

Occupational asthma specific to amoxicillin

A 54-YEAR-OLD, nonatopic woman had worked for 30 years in a pharmaceutical laboratory. Over the last 3 years, she presented nasal obstruction, sneezing, rhinorrhea, pharyngeal itching, dry cough, dyspnea, and wheezing when packing amoxicillin in powder form for 1 day a week, whereas these symptoms improved at home in the evening. The patient reported absence of symptoms at weekends and during holidays. Physical examination at the time of the study was normal. Hematimetry, blood biochemistry, and chest and paranasal sinus

roentgenograms showed no abnormalities. Spirometric parameters were normal. Skin prick tests with a battery of 30 common allergens were negative. Skin tests (prick test and intradermal) were carried out with factory amoxicillin 10 mg/ml, and commercial β -lactams:

amoxicillin 10 mg/ml (Beecham SA, Spain), ampicillin 10 mg/ml (Beecham SA, Spain), benzylpenicillin 50 U/ml (Level SA, Spain), benzylpenicilloyl-polylysine 6×10^{-5} M (Kremers Urban, Wisconsin, USA), and minor determinant mixture 1×10^{-2} M (Allergopharma, Germany). All these tests were negative at 15 min, 8 h, and 24 h. Amoxicillin-specific IgE (CAP System, Pharmacia, Sweden) was 1.38 kU/ml (class 2). Specific IgE antibodies to ampicillin and penicillin V were not detected.

A methacholine inhalation test was done, showing a PC_{20} FEV₁ value of 3.7 mg/ml. Specific bronchial provocation tests with factory amoxicillin and penicillin V (Level SA, Spain) were carried out by the procedure of Pepys & Hutchcroft (1). On the first day, the patient poured lactose from one tray to another for 30 min in a confined space. Pulmonary spirometric recordings were taken under basal conditions and 5, 10, 20, 30, and 60 min after exposure (Fig. 1). Peak flow was subsequently monitored hourly for the rest of the day. On different days, provocation was carried out by the same method for the test materials, added to 250 g of lactose. The starting dose was 10 mg of antibiotic,

A 54-year-old woman presented occupational asthma due to amoxicillin with good tolerance of orally administered and inhaled penicillin.

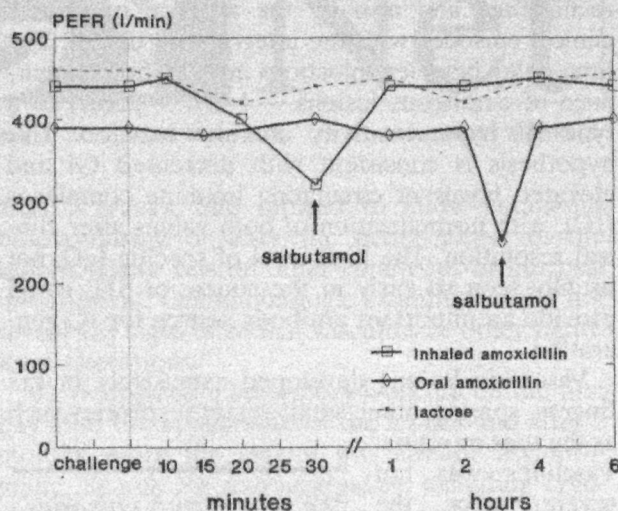


FIGURE 1 PEFR recordings during bronchial provocation and oral challenge with amoxicillin.

increasing 10 times each day. Inhalation challenge with 1 g amoxicillin was followed by a 36% fall in FEV₁ with respect to basal values. The patient failed to show an asthmatic reaction on challenge with 10 g penicillin V.

Oral challenge tests were carried out with commercial amoxicillin (Beecham SA, Spain) and penicillin V (Level SA, Spain). After administration of 250 mg amoxicillin, the patient presented at 210 min a 33% fall in FEV₁ with respect to basal values. The patient tolerated penicillin V at therapeutic doses.

■ In the patient we describe, occupational asthma was confirmed by specific bronchial provocation test with factory amoxicillin powder, which induced an immediate asthmatic reaction. Furthermore, oral challenge with commercial amoxicillin induced a late asthmatic reaction. Although the allergenic activity of contaminants in the factory amoxicillin cannot be excluded, the positive oral challenge with commercial amoxicillin and the detection of specific serum IgE support the role of amoxicillin as the causative agent. We hypothesized that the antigenic determinant responsible for sensitization is probably the aminohydroxyphenyl side-chain of amoxicillin, given that amoxicillin and penicillin share a common nucleus and that our patient became selectively sensitized to amoxicillin, but not to penicillin. To the best of our knowledge, previous reports of occupational asthma due to β -lactams (2, 3) have not demonstrated selective sensitization to amoxicillin with good tolerance of orally administered and inhaled penicillin.

Key words: amoxicillin; asthma; occupational disease.

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Anaphylaxis from linseed

IN the past 6 years, a 40-year-old man with no personal or family history of atopy had had five or six episodes of intestinal/abdominal pain, vomiting, diarrhea, generalized urticaria, acute dyspnea without bronchospasm, hydorrhea, successive sneezing, nasal obstruction, pruritis, and intense general malaise which required emergency treatment. All episodes had occurred within 2-3 min of his consuming multigrain bread. The patient was currently asymptomatic as he had been avoiding contact with or consumption of products that contain linseed.

We prepared an extract of linseed with a concentration of 1:10 w/v, as described previously (1). Skin prick tests (SPT) were performed with the linseed extract and were strongly positive (wheal diameter of 14 mm). Besides a battery of the most common inhalant allergen sources in our area, we performed SPT with cereals, milk, egg, peanuts, and dried fruits (Abelló SA, Madrid, Spain), all of which were negative. SPT with the linseed extract were negative in 10 atopic and 10 nonatopic patients.

Specific IgE for linseed was determined by coating the allergen onto cellulose disks activated by CNBr. A Pharmacia-Trazer kit (Kabi Pharmacia)

A case of IgE-mediated anaphylaxis caused by linseed.

was used during the development process. Specific IgE to linseed was positive with a value of 38 Phadebas RAST units/ml. Specific IgE to linseed was negative in six control subjects.

SDS-PAGE by the technique of Laemmli and SDS-PAGE immunoblotting with the patient's serum were performed, in reducing (the reductor agent breaks the disulfide bonds between polypeptides) and nonreducing conditions, with the linseed extract. Immunoblots of linseed under nonreducing conditions showed intense IgE-binding bands with an apparent high mol. wt. (over 150-175 kDa), as well as discrete bands of about 90-100 kDa. When linseed extract was carried out under reducing conditions, these bands disappeared, and very discrete bands of a broad range (35-100 kDa) were detected. This different reactivity is in agreement with the bands detected in SDS-PAGE (Fig. 1). We also performed immunoblotting with the serum of six patients having total IgE <2 UI/ml that showed no bands.

For ethical reasons, we did not perform the oral provocation with linseed.

Hypersensitivity to linseed was described in 1930 (2, 3). However, cases of linseed hypersensitivity are extremely rare. Currently, due to the use of linseed as a laxative and the rise in consumption of bakery products containing linseed, new cases may appear. Our patient presented with various episodes of anaphylaxis as a result of having consumed linseed. Type I hypersensitivity was strongly suggested by the positivity of SPT and by the demonstration of specific IgE antibodies to linseed in the patient's serum. This case demonstrates the importance of finding the specific cause of an allergic disorder since the patient experienced five or six anaphylactic episodes until linseed was

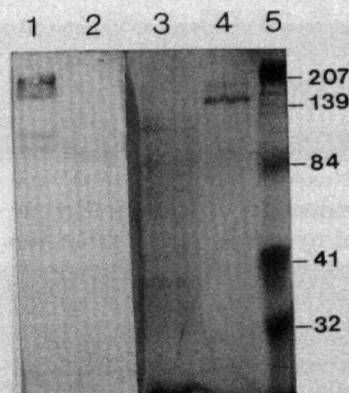


FIGURE 1 Lane 1) Immunoblotting in nonreducing conditions; 2) immunoblotting in reducing conditions; 3) SDS-PAGE in reducing conditions; 4) SDS-PAGE in nonreducing conditions; 5) mol.-wt. standards.

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