

unmeasurable) and unconsciousness within a few minutes after administration of the drug. There were no signs of respiratory failure at that moment. After another few minutes, asystole developed. The patient was successfully resuscitated, and hemodynamics were monitored in the intensive care unit for 1 day. After 1 day, he recovered without further complications. There were no signs of either acute myocardial infarction or pulmonary embolism. No tryptase level was measured.

Several weeks after discharge, cutaneous tests were performed. A prick test with omeprazole (4 mg/ml) resulted in a wheal and erythema at the test site within 10 minutes, comparable with the results of a histamine prick test. Omeprazole prick tests in control subjects did not cause a reaction.

DISCUSSION

Because the patient had urticaria, angioedema, and hypotension, an anaphylactic reaction seems to be the probable cause of the described symptoms. According to the temporal relationship, the positive reaction to rechallenge and the positive cutaneous test results, anaphylaxis was probably due to use of omeprazole in this patient. To our knowledge, this adverse effect of omeprazole has never been reported in the literature.

Anaphylaxis is an acute reaction, which occurs within 1 hour after exposure to a substance and which can involve the cardiovascular system (e.g., hypotension), the respiratory system (e.g., bronchospasm), the skin (e.g., urticaria), and the gastrointestinal system (e.g., diarrhea). In The Netherlands, drug-induced anaphylaxis is probably most frequently caused by penicillins, analgesics, and nonsteroidal antiinflammatory drugs.⁵

Because of the rapid occurrence of anaphylaxis after administration of omeprazole and the occurrence of urticaria, we believe that this case represents a type I or IgE-mediated allergic reaction. Omeprazole is almost completely metabolized after absorption and rapidly eliminated in the urine. However, because the reaction occurred immediately after intravenous administration of omeprazole and because the results of prick tests were positive, the allergic reaction was probably to omeprazole itself and not to a metabolite.

Although omeprazole-induced anaphylaxis is probably rare, physicians should be aware of this possibility, especially in a patient with urticaria who is treated with omeprazole. Early recognition of an anaphylactic reaction is important, because a serious reaction, or even death, can occur within minutes to hours after the first symptoms appear.

REFERENCES

1. Clissold SP, Campoli-Richards DM. Omeprazole: a preliminary review of its pharmacokinetic, and therapeutic potential in peptic ulcer disease and Zollinger-Ellison syndrome. *Drugs* 1986;32:15-47.
2. Sölvell L. Clinical safety of omeprazole. *Can J Gastroenterol* 1989;3(suppl A):91A-7A.
3. Meding B. Contact allergy to omeprazole. *Contact Dermatitis* 1986;15:36.
4. Haeney MR. Angio-oedema and urticaria associated with omeprazole. *BMJ* 1992;305:870.
5. Van der Klauw MM, Stricker BHC, Herings RMC, Cost WS, Valkenburg HA, Wilson JHP. A population based case-cohort study of drug-induced anaphylaxis. *Br J Clin Pharmacol* 1993;35:400-8.

Allergy to supplemental lactase enzyme

Karen E. Binkley, MD, FRCPC *Toronto, Ontario, Canada*

Lactose intolerance results from deficient production of lactase enzyme, so ingested lactose is available for fermentation by gut bacteria, which

results in cramping and diarrhea. This affects approximately 18% to 20% of the general population. Supplemental lactase enzyme obtained from fungal sources is commercially available for treatment. Milk pretreated with lactase enzyme (Lactaid and Lacteeze brand milk) is also available. This is the first case report of a patient who had allergic symptoms after the ingestion of supplemental lactase enzyme.

From the University of Toronto, Toronto, Ontario, Canada.
Reprint requests: Karen E. Binkley, MD, FRCPC, 30 St. Clair Ave. West, Suite 201, Toronto, Ontario, M4V 3A1, Canada.
J Allergy Clin Immunol 1996;97:1414-6.
Copyright © 1996 by Mosby-Year Book, Inc.
0091-6749/96 \$5.00 + 0 1/54/70626

CASE REPORT

The patient, a 35-year-old man, experienced his first reaction in the summer of 1994. He had minor gastrointestinal upset and took two Lactaid pills. Within half an hour, he experienced burning and swelling of his throat with difficulty swallowing, but there were no other symptoms. He went to the emergency department, and his throat appeared to be swollen. Oral corticosteroids were prescribed, and his condition improved. His second reaction occurred in the spring of 1995 when he took two Lactaid pills for dyspepsia and almost immediately experienced a burning sensation in his mouth and throat and difficulty swallowing but no other symptoms. The patient had not previously taken Lactaid tablets. Nonetheless, he had consumed Lacteeze brand milk while visiting his in-laws. No other cause for these reactions could be identified. He had seasonal rhinoconjunctivitis during the fall but no other atopic symptoms.

Allergy testing was performed (prick method) with extracts (Bencard Laboratories, Mississauga, Ontario, Canada) with positive and negative controls. The patient showed a trace reaction to *Aspergillus* species (a mixture of *A. terreus*, *A. niger*, *A. anstelodami*) but no reaction to other common inhalant allergens.

The patient had a 5⁺ reaction to a slurry prepared from the crushed Lactaid tablets (approximately 10% wt/vol). A control subject showed no reaction.

The manufacturer of Lactaid tablets confirmed that the tablets contained an enzyme derived from *Aspergillus oryzae*. Two enzyme preparations had been used, which they supplied. The patient had a 5⁺ reaction to 10% wt/vol slurries of each.

One nonatopic control subject, two atopic subjects not allergic to mold, and three of four subjects allergic to *Aspergillus* species showed no reaction to either enzyme preparation. One of the four subjects allergic to *Aspergillus* species had a history of allergic reactions of unidentified cause. This subject had intermittent exposure to lactase-treated milk and showed equivocal reactions to both preparations of enzyme.

Lacteeze milk contains lactase from *Saccharomyces fragilis*, whereas Lactaid milk contains enzyme from *Kluyveromyces lactis*, a yeast. The index patient was skin tested with samples of each type of milk and showed a 1⁺ reaction to Lacteeze but no reaction to the Lactaid milk. One control subject showed no reaction to either product.

The patient has been advised to avoid lactase products and has had no further reactions. Because there is the potential for inadvertent consumption of milk or foods containing the enzyme, the patient has been instructed in the use of injectable epinephrine.

DISCUSSION

This patient likely experienced IgE-mediated reactions to the ingestion of *A. oryzae*-derived lactase. Sensitization may have occurred either through exposure to the cross-reacting *S. fragilis*-

derived lactase enzyme in the milk he consumed while visiting his in-laws or as a result of his inhalant *Aspergillus* allergy, analogous to the "oral allergy syndrome."

Although the patient's reaction was limited to the oropharynx, the potential for anaphylaxis can readily be appreciated. The thermal stability of these enzymes is unknown; hence, the patient was advised to avoid all cooked foods and frozen goods that might contain the lactase-supplemented milk.

The patient had seasonal rhinoconjunctivitis, which peaked in the fall, and showed a trace reaction to a standard *Aspergillus* mixture. Cross-reactivity between *Aspergillus* species and between *Aspergillus* species and other fungi has been demonstrated for some but not all epitopes.¹⁻⁶ This patient showed reactivity to lactase from *Aspergillus* species and *S. fragilis*. Identified antigenic determinants from *Aspergillus* species include α -amylase⁷ and lipase.⁸ Thus it is not clear whether individuals with a history of inhalant *Aspergillus* allergy are at increased risk of allergy to ingested lactase derived from *Aspergillus* species or other fungi.

This is the first description of allergic reaction after oral ingestion of *Aspergillus*-derived lactase although α -amylase has been suspected of causing symptoms of asthma after ingestion.⁹ Given the allergenicity of *Aspergillus* species and the wide availability of lactase enzyme, it is possible that other allergic reactions have previously been unrecognized. Thus specific enquiry as to the consumption of these products should be made when investigating the cause of allergic reactions. Patients sensitized to lactase should be made aware of the full range of products they should avoid.

I thank Dr. Jim Swan and McNeil Consumer Products Company of Guelph, Ontario, Canada, for their enthusiastic assistance and supply of enzyme preparations.

REFERENCES

1. Kappe R, Schulze-Berge A. New cause for false-positive results with the Pastorex *Aspergillus* antigen latex agglutination test. J Microbiol 1993;31:2489-90.
2. Kumar A, Kurup VP. Murine monoclonal antibodies to glycoprotein antigens of *Aspergillus fumigatus* show cross-reactivity with other fungi. Allergy Proc 1993;14:189-93.
3. Lundgren B, Kovacs JA, Nelson NN, Stock F, Martinez A, Gill VJ. *Pneumocystis carinii* and specific fungi have a common epitope identified by a monoclonal antibody. J Clin Microbiol 1992;30:391-5.
4. Shen HD, Choo KB, Chen JH, Lin WL, Chang ZN, Han SH. Characterization of a monoclonal antibody (P40) against the 68kD major allergen of *Penicillium notatum*. Clin Exp Allergy 1992;22:485-90.

5. Sakamoto T, Ito K, Yamada M, Torii S, Matsuda Y. Allergenic relationships between *Aspergillus restrictus* and *Aspergillus fumigatus* determined by radio allergosorbent test inhibition [in Japanese]. *Arerugi-Jpn J Allergol* 1991;40:1320-6. English abstract.
6. Puente P, Ovejero MC, Fernandez N, Leal F. Analysis of *Aspergillus nidulans* conidial antigens and their presence in other *Aspergillus* species. *Infect Immun* 1991;59:4478-85.
7. Baur X, Chen Z, Sander I. Isolation and denomination of an important allergen in baking additives: α -amylase from *Aspergillus oryzae* (Asp o II). *Clin Exp Allergy* 1994;24:465-70.
8. Hilton J, Dearman RJ, Basketter DA, Kimber I. Serological responses induced in mice by immunogenic proteins and by protein respiratory allergens. *Toxicol Lett* 1994;73:43-53.
9. Kanny G, Moneret-Vautrin D. α -Amylase contained in bread can induce food allergy. *J Allergy Clin Immunol* 1995;95:132-3.

Occupational asthma caused by sarsaparilla root dust

Olivier Vandenplas, MD,^a Suzanne Depelchin, MSc,^b
Gaelle Toussaint, MSc,^b Jean-Pierre Delwiche, MSc,^a
Raymond Vande Weyer, MD,^c and Jean-Marie Saint-Remy, MD, PhD^b
Yvoir and Brussels, Belgium

Occupational asthma has been documented in workers exposed to a wide variety of plant-derived materials including grains, beans, seeds, leaves, roots, exudates (latex and gums), and wood dusts.¹ An IgE-mediated mechanism has been demonstrated in most cases, with the exception of wood dusts.¹

We describe a case of occupational asthma resulting from exposure to sarsaparilla (*Smilax*; botanical family, Liliaceae) in an herbal tea worker.

CASE REPORT

A 36-year-old man first experienced rhinitis and asthma 6 months after starting employment as a foreman in a small factory where various herbal teas were manufactured. Plants were manually unloaded from bags and fed into rollers where they were ground into 0.8 to 1.5 mm pieces. The components from a variety of

Abbreviation used

SPT: Skin prick test

plants were manually blended to obtain the desired medicinal properties and were then packed into small paper bags.

The symptoms occurred at work and improved within about 30 minutes, when the subject moved away from the production area. The subject noticed persistence of nasal obstruction and wheezing in the evening, but he never experienced nocturnal asthma attacks. Only on occasion did he use an inhaled bronchodilator at work, and he had no symptoms on weekends or holidays. He noticed that his symptoms were specifically related to the production of a depurative herbal tea, and he believed that the reddish dust from sarsaparilla roots was the causal agent. Twenty-one months after the onset of symptoms, he was removed from work by his attending physician and became free of symptoms except for wheezing on exercise. Six months later, he was asked to manage the transfer of the herbal tea production facility to another factory. When re-exposed to plant dust, he noticed recurrence of rhinitis and asthma symptoms. Seven months later, he lost his job because the production of herbal teas was discontinued.

The subject was investigated in our center 15 months after his last workplace exposure. He reported no personal or familial history of atopic disease. He

From ^athe Department of Chest Medicine, University Hospital of Mont-Godinne, Yvoir; ^bExperimental Medicine Unit/Allergy and Clinical Immunology Unit, Catholic University of Louvain, Brussels; and ^cFonds des Maladies Professionnelles, Brussels.

Reprint requests: Olivier Vandenplas, MD, Department of Chest Medicine, University Hospital of Mont-Godinne, 5530-Yvoir, Belgium.

J Allergy Clin Immunol 1996;97:1416-8.

Copyright © 1996 by Mosby-Year Book, Inc.

0091-6749/96 \$5.00 + 0 1/54/70872