

Fatalities due to anaphylactic reactions to foods

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Fatal anaphylactic reactions to foods are continuing to occur, and better characterization might lead to better prevention. The objective of this report is to document the ongoing deaths and characterize these fatalities. We analyzed 32 fatal cases reported to a national registry, which was established by the American Academy of Allergy, Asthma, and Immunology, with the assistance of the Food Allergy and Anaphylaxis Network, and for which adequate data could be collected. Data were collected from multiple sources including a structured questionnaire, which was used to determine the cause of death and associated factors. The 32 individuals could be divided into 2 groups. Group 1 had sufficient data to identify peanut as the responsible food in 14 (67%) and tree nuts in 7 (33%) of cases. In group 2 subjects, 6 (55%) of the fatalities were probably due to peanut, 3 (27%) to tree nuts, and the other 2 cases were probably due to milk and fish (1 [9%] each). The sexes were equally affected; most victims were adolescents or young adults, and all but 1 subject were known to have food allergy before the fatal event. In those subjects for whom data were available, all but 1 was known to have asthma, and most of these individuals did not have epinephrine available at the time of their fatal reaction. Fatalities due to ingestion of allergenic foods in susceptible individuals remain a major health problem. In this series, peanuts and tree nuts accounted for more than 90% of the fatalities. Improved education of the profession, allergic individuals, and the public will be necessary to stop these tragedies. (*J Allergy Clin Immunol* 2001;107:191-3.)

Key words: Food allergy, anaphylaxis, food induced fatality, peanut allergy, tree nut allergy

In 1992 Sampson et al¹ reported on 13 subjects who either died of or nearly died of anaphylactic reactions to foods. A reporting registry, through the American Academy of Allergy, Asthma, and Immunology with the assistance of the Food Allergy and Anaphylaxis Network, was established to determine the occurrence of further fatalities from anaphylactic reactions to foods. The objective of this article is to report the deaths that continue to be recorded by the registry.

METHODS

Between 1994 and 1999, 32 fatalities due to food-induced anaphylaxis were identified through several sources. This was not a formal epidemiologic study; rather, cases were reported to the registry (publicized in *The JACI*) by physicians, the media, and the Food Allergy and Anaphylaxis Network (a national nonprofit educational organization). One of the authors (A. M.-F.) then contacted the family of the deceased individual whenever possible (20 families). A structured questionnaire was used to interview family members about the details of the fatality and prior history of allergic reactions to food.

The structured questionnaire was used to obtain the following information if available: demographic details, sequential events of the food ingestion and subsequent symptom progression, treatment at the time symptoms started (specifically epinephrine), transport to a hospital, emergency medical response treatment, information about the food ingested, asthma and allergy history (especially previous reactions to the food), current medication, associated factors (exercise), and prior education about food allergy severity. From the information gathered, we were able to divide the 32 individuals into 2 groups.

Subjects in group 1 had enough information to answer the following questions: (1) Was death from this food a certainty, and was the food responsible known with certainty? (2) Did the subject have asthma? (3) Was epinephrine given in a timely fashion? (4) Where did the reaction occur? (5) Had the individual reacted to the same food previously? For subjects in group 2, we could determine which food was responsible; however, we could not always be certain about the presence of asthma, use of epinephrine, and history of a reaction to the same food.

RESULTS

Tables I and II present the details of the 32 fatalities. The age range was 2 to 33 years. However, only 3 subjects were younger than 10 years. Sixteen of the individuals (50%) were female. Peanut accounted for 20 (63%) of the fatalities, with nuts accounting for 10 (31%). The 2 deaths from other foods (milk and fish) occurred in 2 of the younger children. At the time of the fatal reaction and before it was eaten, no individual was aware that the food about to be ingested contained the food that had previously provoked an allergic reaction. One young man, with peanut allergy, knowingly ate pistachio nuts because he did not think they would cause a reaction. One fatality occurred in a 2-year-old child not known to have any food allergy, asthma, or other atopic condition.

Group 1

In group 1, 14 (67%) of 21 subjects died of anaphylactic reactions to peanut, and the other 7 (33%) subjects died of reactions to tree nuts. All but 1 of the subjects had active asthma at the time of death. Only 2 subjects did not have a history of symptomatic reaction to the food that caused

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0091-6749/2001 \$35.00 + 0 1/87/112031

doi:10.1067/mai.2001.112031

TABLE I. Group 1

Patient No.	Age (y)	Sex	Food	Asthma	Prior history	Food	Location	Epinephrine
1	2	M	Brazil nut	No	No	Mixed nuts	Home	No
2	15	F	Peanut	Yes	Yes	Cake	Friend's home	No
3	19	M	Pecan	Yes	Yes	Dip	Country club	Unknown
4	14	F	Peanut	Yes	Yes	Egg roll	Restaurant	No
5	18	M	Peanut	Yes	Yes	Candy bar	School	No
6	13	F	Walnut	Yes	Yes	Candy	School	Late
7	29	M	Peanut	Yes	Yes	Mexican food	Restaurant	Late
8	16	M	Peanut	Yes	Yes	Candy	Home	Late
9	17	F	Peanut	Yes	Yes	Snack mix	Video store	No
10	12	F	Peanut	Yes	Yes	Cookie	Home	No
11	20	M	Walnut	Yes	Yes	Veggie burger	Friend's home	Late
12	14	F	Peanut	Yes	Yes	Peanut sauce	Home	No
13	14	F	Peanut	Yes	Yes	Peanut butter contamination	Camping trip	No
14	18	F	Nut	Yes	Yes	Dessert	University cafeteria	Late
15	19	M	Peanut	Yes	Yes	Cookie	College dorm	No
16	21	F	Peanut	Yes	Yes	Cake	Banquet	Late
17	20	M	Peanut	Yes	Yes	Chinese food	College dorm	Late
18	28	F	Brazil nut	Yes	Yes	Ice cream	Restaurant	Yes
19	18	M	Pistachio	Yes	No*	Nuts	College camp	Yes
20	33	F	Peanut	Yes	Yes	Peanut sauce	Restaurant	Late
21	20	F	Peanut	Yes	Yes	Peanut	Camp	No

*History of reaction to peanut but not to pistachio.

TABLE II. Group 2

Patient No.	Age (y)	Sex	Food	Asthma	Prior history	Meal	Location	Epinephrine
22	6	M	Fish	Yes	Yes	Lunch	School	Unknown
23	3	F	Milk	Unknown	Yes	Milk	Day care	Unknown
24	18	F	Pecan	Unknown	Yes	Dinner	College	Late
25	13	F	Walnut	Unknown	Unknown	Cookie	Dance class	Unknown
26	20	F	Peanut	Unknown	Yes	Cookie	College apartment	Yes
27	28	M	Nut	Unknown	Yes	Nuts in bowl	Hotel bar	Unknown
28	27	M	Peanut	Unknown	Yes	Peanut sauce	Friend's home	No
29	26	M	Peanut	Unknown	Yes	Unknown	Restaurant	Yes
30	19	M	Peanut	Yes	Yes	Egg roll	College	No
31	32	M	Peanut	Yes	Yes	Chinese food	Home	Unknown
32	17	M	Peanut?	Yes	Unknown	Burrito*	Restaurant	Late

*Burrito never proven to contain peanut.

their deaths. Subject 3 had never been exposed to nuts, was not known to have any allergic disease, and did not have asthma. Subject 19 was known to be allergic to peanut but thought that pistachio nuts would not provoke a reaction.

In this group, 2 subjects (10%) received intramuscular epinephrine within minutes of the onset of their symptoms; however, they died of fatal anaphylaxis. In all of the other subjects, epinephrine was either not given or was not administered until a significant time interval had elapsed since the onset of their symptoms.

One subject deserves special mention because he was so exceptional. Subject 1 was a 2-year-old child with no previous history of asthma or atopic disease. He had never had any type of food reaction. He was eating Brazil nuts with his father. He put one in his mouth, immediately he spit it out, and complained that his tongue hurt. He abruptly became flushed, developed hives, and vomited. Then his face became puffy, and he started having difficulty breathing. The reaction

rapidly progressed to the point at which it shut off his airway, and he could not be resuscitated. It is certain that he was not choking, and in retrospect, he had obvious features of an allergic reaction that were unfamiliar to the family.

Group 2

For the 11 subjects in group 2, the information available was less complete. In 5 cases peanut was the most likely culprit. Tree nuts were probably the cause of death in 3, and fish and milk were almost certainly the causes in 2 other subjects. The last subject was known to be allergic to peanut, but the contents of the burrito he ate were never determined. The status of asthma in each individual in this group is also less clear; the information was not obtainable. Two subjects in this group did receive intramuscular epinephrine promptly, but it was not life saving.

The locations where the fatal reactions occurred were documented in all cases. In 5 the reaction occurred at home,

in 4 it occurred at school or day care, in 18 it occurred at a restaurant or at the home of a friend, and in 5 it occurred at other miscellaneous places. Numerous types of food and meals contained the food that caused the fatality (Tables I and II). In all cases for which there are adequate data, the individuals were immediately aware that they had ingested food containing a substance to which they were allergic.

In 2 subjects the initial symptoms seemed to subside, and normal activities were resumed. Then within 30 minutes in subject 9 and in more than an hour in subject 14, the symptoms returned and were rapidly fatal.

DISCUSSION

The tragic demise of these individuals provides us with a number of lessons. First, anaphylactic reactions to foods affect both sexes equally. Second, the large majority of the victims were adolescents or young adults. (The group reported by Sampson et al¹ was slightly younger.) Third, all but 1 of these individuals were known to have food allergy with a prior history of some type of reaction to the food that caused the fatal reaction (except the subject with a history of peanut allergy who died after eating pistachio nuts). Fourth, in 24 (96%) of 25 subjects for whom we have data, all but 1 was known to have asthma. Fifth, we know that very few (3/32 [10%]) of these individuals had epinephrine available for use at the time of their reaction. (The fourth individual who received epinephrine promptly was given the medication from the camp emergency box.) Sixth, the 4 subjects who appeared to have received injected epinephrine in a timely fashion did not survive. (There are too few data to draw any important conclusion from this information.) Seventh, peanuts or tree nuts caused most (94%) of the fatalities.

The cases reported here strongly implicate the association of asthma with the fatal reactions. Several individuals were known to be on "as needed" bronchodilators, whereas others were reportedly taking asthma medication regularly. It is important to determine whether the food-allergic reaction triggers asthma symptoms as well as laryngospasm and to determine whether this makes treating the episode more difficult. Data from Sampson et al¹ strongly suggest that if the bronchospasm is exacerbated during the allergic reaction, reversing the episode will be more difficult.

Peanut and tree nuts are responsible for the vast majority of fatalities due to food-induced anaphylaxis. Therefore we must improve the education of subjects with a history of allergic reactions to these foods and the education of manufacturers producing products containing peanut and tree nuts. We must provide better instructions on how to avoid these foods (eg, reviewing unexpected sources and possible cross-contamination [cookies, candies]). Further, we must improve the response time of all allergic individuals to the accidental ingestion of allergens by teaching them to recognize early symptoms, providing them with self-injectable epinephrine, and providing them with an "action" plan including transport to the nearest emergency department.

A number of recent reports of food-induced anaphylaxis and food-related fatalities provide insight into the magnitude of this problem.²⁻¹³ From 1983 to 1987, investigators²⁻⁴ found an anaphylaxis occurrence rate of 30 per 100,000 person-years in a population-based study of Olmstead County, Minn. In their emergency department, allergic reactions to food were the leading identifiable cause of anaphylactic reactions. Extrapolating from these data would predict that there are about 29,000 anaphylactic episodes due to food allergy in the United States each year, resulting in about 150 deaths. A Danish study reported a fatality rate of 5%.⁷

It is our opinion that only more stringent food-labeling requirements and improved education can bring an end to these tragic fatalities. Ultimately this can be accomplished only by ongoing attention to the subject of food-induced anaphylaxis by the medical profession, especially at the primary care level, and through the education of the public by professional and lay organizations with expertise in this field. First, patients must be given extensive instructions on how to avoid foods to which they are allergic. Second, patients at risk for food-induced anaphylaxis (ie, those with previous reactions involving the airway or those with asthma and food allergies) must be educated to recognize the early signs of anaphylaxis, given and trained in the use of self-injectable epinephrine, and provided with an emergency plan in case of accidental ingestion. In addition to education of the allergic individual, it is imperative that we improve the knowledge of families, friends, caregivers, schools, restaurants, and the general public.

REFERENCES

1. Sampson HA, Mendelson L, Rosen JP. Fatal and near-fatal anaphylactic reaction to food in children and adolescents. *N Engl J Med* 1992;327:380-4.
2. Yocum MW, Butterfield JH, Klein JS, Volcheck GW, Schroeder DR, Silverstein MD. Epidemiology of anaphylaxis in Olmstead County: a population-based study. *J Allergy Clin Immunol* 1999;104:452-6.
3. Yocum MW, Khan DA. Assessment of patients who have experienced anaphylaxis: a 3-year survey. *Mayo Clin Proc* 1994;69:16-23.
4. Melton LJ. History of the Rochester Epidemiology Project. *Mayo Clin Proc* 1996;71:266-74.
5. Sicherer SH, Muñoz-Furlong A, Burks AW, Sampson HA. Prevalence of peanut and tree nut allergy in the US determined by a random digit dial telephone survey. *J Allergy Clin Immunol* 1999;103:559-62.
6. Golbert TM, Patterson R, Pruzansky JJ. Systemic allergic reactions to ingested antigens. *J Allergy* 1969;44:96-107.
7. Sorensen HT, Nielsen B, Nielsen JO. Anaphylactic shock occurring outside hospitals. *Allergy* 1989;44:288-90.
8. Kemp SF, Lockey RF, Wolf BL, Lieberman P. Anaphylaxis: a review of 266 cases. *Arch Intern Med* 1995;155:1749-54.
9. Pumphrey RSH, Stanworth SJ. The clinical spectrum of anaphylaxis in north-west England. *Clin Exp Allergy* 1996;26:1364-70.
10. Stewart AG, Ewan P. The incidence, aetiology and management of anaphylaxis presenting to an accident and emergency department. *Q J Med* 1996;89:859-64.
11. Bock SA. The incidence of severe adverse reactions to food in Colorado. *J Allergy Clin Immunol* 1992;90:683-5.
12. Novembre E, Cianferoni A, Bernardini R, Mugnaini L, Caffarelli C, Cavagni G, et al. Anaphylaxis in children: clinical and allergologic features. *Pediatrics* 1998;101:e81-8.
13. Yunginger JW, Sweeney KG, Sturmer WQ, Giannandrea LA, Teigland JD, Bray M, et al. Fatal food-induced anaphylaxis. *JAMA* 1988;260:1450-2.