

Practical Clinical Solutions

Peanut Allergy: Case of an 11-Year-Old Boy with a Selective Diet

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HISTORY AND REVIEW OF PEANUT ALLERGY

Current statistics estimate the prevalence of food allergy in the United States to be approximately 6% in children and 3% to 4% in adults (1). Although eight food proteins are considered responsible for 90% of all allergic reactions (cow's milk, soybean, egg, wheat, peanut, tree nut, fish, shellfish [2]), allergy to peanut requires special consideration for several reasons (3). The age of onset of peanut allergy is young (median age of onset is approximately 24 months), and reactions are immediate and often severe. When combined with tree nut allergy, peanut allergy is responsible for the 80% of the 100 to 200 food-induced anaphylactic fatalities every year in the United States (4,5). Sicherer and colleagues recently reported that the prevalence of peanut allergy in the United States has more than tripled between 1997 (0.4%) and 2008 (1.4% of the US population, or more than 3 million Americans) (6). Peanut allergy is potentially life-long, although there is evidence that approximately 20% of individuals with peanut allergy will become clinically desensitized (7). It must be noted, however, that up to 8% of those who have established tolerance may become re-sensitized after a negative prick skin test (PST) and negative food challenge, especially without regular exposure to peanuts (8). Of additional concern are quality-of-life issues and behavioral and emotional repercussions that can result from having a potentially life-threatening condition (9,10).

PSYCHOSOCIAL IMPACT OF PEANUT ALLERGY

It has been reported that children with peanut allergy have a poorer health-related quality-of-life compared with children with diabetes, likely due to anxiety (11). Other data confirm that food allergy has a psychosocial impact on both the affected child and the family. Marklund and colleagues found that parents of these children experience more distress and worry and report more interruptions in usual family activities than a sample US

population (9,11). Children with food allergies can experience fear and anxiety about the potential of having an adverse reaction. Although food-induced gastrointestinal symptoms reportedly cause the most worry in families, food-induced breathing difficulties may actually improve health-related quality of life in two aspects: well-being and family cohesion. Sicherer and colleagues showed that family cohesion was stronger in families of children with peanut allergy than in a population sample (9). A lower health-related quality-of-life seems to be a result of the fear or anticipation of having a food-induced reaction and the efforts used to avoid a reaction, rather than from the actual clinical reaction to the allergen intake (11).

The emotional/behavioral influence of having a peanut allergy may also affect nutritional status over time if fear of having a reaction impacts willingness to eat. Fear of an adverse event may lead to more anxiety about eating and more restriction as a result (12). Avery and colleagues have concluded that this may result from individuals with peanut allergy and their families living with the ever-present potential for a reaction and the need for vigilance in every aspect of life involving food (13). It must be added, however, that the more concrete information the family has about preventing exposure and early treatment of accidental exposure, the more reassured they feel (14). It is worth noting that in one study children felt safer when carrying epinephrine (12). An appropriate level of anxiety may contribute to the desire to acquire information and maintain vigilance, and typically subsides over time (15). However, a sustained intense level of anxiety is considered maladaptive. Either experiencing or witnessing a near-fatal anaphylactic reaction can result in post-traumatic stress disorder, especially in patients or family members with asthma (16,17). There have been isolated case reports of individuals with food allergy whose food restrictions were so extreme as to meet the psychiatric criteria for an eating disorder (18). This possibility needs to be considered when extreme self-restriction is identified and results in unwarranted weight loss in an individual with food allergy.

PATHOPHYSIOLOGY OF PEANUT ALLERGY

There are different mechanisms by which food-induced clinical reactions can take place: IgE-mediated reactions that occur immediately or within 2 hours of allergen ingestion; non-IgE (cell)-mediated reactions that occur >2 hours after ingestion; or mixed IgE- and non-IgE-mediated (19). The mechanism of a peanut-induced reaction is IgE-mediated, occurring rapidly, within minutes of ingestion and in some cases requiring only trace amounts of peanut protein (substantially less than the amount of protein found in one peanut) (20). In these reactions,

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peanut-specific IgE antibodies bind to high-affinity receptors on mast cells and basophils in the gastrointestinal tract, skin, and respiratory tract mucosa (21). When peanut allergens cross the mucosal barrier, cell-bound IgE and peanut allergens crosslink, which causes degranulation of preformed allergic mediators and subsequent cell activation at the mucosal site, resulting in clinical symptoms. These cells may then produce a variety of cytokines and chemokines, which recruit other inflammatory cells and contribute to the IgE-mediated late-phase allergic response (reactions that can occur 1 to 8 hours after the initial symptoms have resolved) (20,22). Clinical manifestation may involve cutaneous, cardiovascular, gastrointestinal, genitourinary, and/or respiratory systems. Progressive upper or lower respiratory symptoms, hypotension, and arrhythmias typically develop in fatal and near-fatal cases of anaphylaxis (22). Factors that seem to contribute to a fatal outcome include a concomitant diagnosis of asthma, a delay in the administration of epinephrine, previous severe allergic reactions to peanut, and not recognizing the presence of peanut in the meal (23).

DIAGNOSIS AND TREATMENT OF PEANUT ALLERGY

Along with obtaining a thorough history, serum-specific IgE and PST are recognized as the most reliable methods for diagnosing peanut allergy. The results, however, predict only the likelihood of experiencing a food-specific reaction and cannot predict the type or severity of a reaction (19,24). Serum IgE testing (eg, ImmunoCAP, Quest Diagnostics, Madison, NJ) carries a 95% predictive value for a peanut-specific reaction with an IgE level of 15 kUA/L or more (2,25). A negative PST has predictive value of 95%, but a positive PST carries only a 60% predictive value in patients who already have a convincing history of peanut allergy (2). Roberts and Lack have demonstrated that a positive skin-prick test with a wheal diameter of 8 mm or more had a predictive value of 95% for a positive challenge (2,25). Still considered the “gold standard” for diagnosing food allergy is the double-blind placebo-controlled food challenge. However, for an individual who has had a reaction based on known ingestion of peanut, an oral challenge is not indicated. Acute reactions can be confirmed by history and ImmunoCAP and/or PST (3).

To date, the only treatment for peanut allergy is strict avoidance of peanut protein. Due to the potential severity of reactions and the often difficult task of avoiding hidden sources of peanuts, precautions must be taken to improve the outcome in the case of accidental exposure. These precautions include blunting the severity of the reaction with availability and early onset use of epinephrine (26). For individuals with peanut allergy and concomitant asthma, well-controlled asthma may play a role in preventing fatality from peanut ingestion (26).

Current studies are investigating ways to desensitize individuals with peanut allergy to at least minimize a reaction from an accidental exposure or even allow them to eventually ingest peanuts freely without clinical symptoms (3). Therapies under investigation include oral peanut desensitization and immunotherapy, monoclonal anti-IgE, and use of Chinese herbs, probiotics, and heat-killed *listeria monocytogenes* (3). These therapies are still under

investigation and are not yet being used in clinical practice, but are showing promising results (3).

NUTRITIONAL IMPLICATIONS OF PEANUT ALLERGY

Peanuts are botanically classified as legumes rather than nuts. The legume family also includes soybeans, peas, lima beans, green beans, other beans, chickpeas, and lentils (3). Peanuts are a nutritionally dense food. They are a good source of fat, protein (but limited in the essential amino acid methionine), chromium, magnesium, manganese, niacin, and vitamin E. They also contain some biotin, copper, folacin, phosphorus, potassium, and vitamin B-6. In spite of their nutritional contribution to the diet, these nutrients can be obtained by eating other foods, primarily other legumes and whole grains (27).

CASE REPORT

BR is an 11-year 8-month-old male who was referred to a registered dietitian (RD) for outpatient nutrition counseling in June 2009 because of his extremely limited dietary intake, decreasing velocity of weight gain, and allergy to peanut.

Family History

BR lives with his mother, father, and 7-year-old and 20-year-old brothers. BR's family history was significant for his 20-year-old brother's recent diagnosis of eosinophilic esophagitis. He had a lifelong history of being a cautious eater and his diagnosis was prompted by a choking episode. It is not known if his eosinophilic esophagitis is food-induced. BR's younger brother has a history of congenital heart disease and transient vomiting episodes, especially related to ingestion of milk products. His tolerance to low-lactose milk may be indicative of lactose intolerance rather than a true hypersensitivity to cow's milk.

Medical History

BR denied having any of the symptoms experienced by his brothers except for his extreme cautiousness and self-restriction related to eating. His mother reported that he has previously expressed a fear of unknowingly ingesting peanuts and having a serious reaction. BR was exposed to peanut butter when he was an infant. His mother reported that she had attempted to feed him a peanut butter sandwich but when she grazed his mouth with the sandwich, his lips started to swell and he progressed to having a reaction. It is not known what medical intervention he received for this reaction. BR's peanut allergy was subsequently confirmed with PST. He has had only one accidental exposure to peanut since his diagnosis. When he was 7 years old he ate a chocolate bar that did not list peanut on the label. His reaction at that time involved shallow breathing and itching. Medical intervention for that reaction is also not known. His mother reported that he seemed to suffer trauma from having that reaction and this may have been the beginning of his period of extreme self-restriction of food. BR has not had a reaction since this episode and has never been re-evaluated to determine his current level of sensitization.

Table 1. Serial anthropometric data for a patient (from 8 to 12 years old) with a peanut allergy who was evaluated for an increasingly self-selective diet

Age	Weight (kg)	Weight-for-age percentile	Height (cm)	Height-for-age percentile	IBW ^a (kg)	% IBW	BMI ^b	BMI-for-age percentile
8y ^c	28.0	70th	130.0	64th	26.75	105	16.6	67th
11y ^c	32.0	26th	144.0	53rd	36.25	88	15.4	16th
11y8m ^d	34.9	28th	146.8	49th	39.0	90	16.2	24th
12y ^c	36.3	28th	150.0	55th	41.0	88	16.2	20th

^aIBW=ideal body weight.
^bBMI=body mass index; calculated as kg/m².
^cMeasurements obtained from primary care physician's office at 8-y, 11-y, and 12-y well-child check-ups.
^dMeasurements obtained at outpatient nutrition consultation June 2009.

During the previous school year BR expressed dissatisfaction with his body weight. He was concerned that he was gaining weight too fast because he did poorly on a fitness test in physical education at school. At the time of the visit with the RD, he was pleased with his body weight because he believed he was in better physical condition than he was the previous year. His mother described BR as self-sufficient and a perfectionist who does well in school but struggles with anxiety. BR had a history of constipation severe enough to require medical treatment in the past. There were no currently reported stooling complaints. His medications included Claritin (Schering-Plough Healthcare Products, Kenilworth, NJ) and Epipen (Dey Pharma, Napa, CA) as needed. BR had no sleep problems and no history of snoring, but was occasionally grinding his teeth while sleeping.

BR's mother reported that both parents prepared and ate adequate amounts of fruits and vegetables on a daily basis, but all three of their sons refused to eat them. They sat down as a family to eat once per day, but the sons did not eat what the parents prepared. BR typically prepared his own meals, which consisted mostly of convenience foods that could be heated, such as pre-prepared pasta sauce. There was a history of stressful mealtimes when BR's mother was previously trying to increase his intake and improve the nutritional quality of his diet. His mother was no longer trying to force him to eat, so the stress level at meals had resolved. But his mother was still concerned about the nutritional quality of his diet. She was convinced that BR would rather not eat than eat something he did not want to eat. Recently, he had become more self-restrictive and even started to refuse foods that he previously liked, such as pizza. His diet did not vary much from day to day. His mother described BR's activity level as high on weekends and moderate during the week. He watched 1 hour of television per day on weekdays and 2 to 3 hours per day on weekend days.

24-Hour Dietary Recall

Breakfast: 1 cup MultiGrain Cheerios (General Mills, Minneapolis, MN), 8 oz low-fat Lactaid milk (McNeil Nutritionals, Ft Washington, PA).
Lunch: 1 cup macaroni and cheese, 1 can (8 oz) Ensure (Abbott Laboratories, Abbott Park, IL), 1 bagel.

Dinner: 1 cup pasta with ½ cup Prego marinara sauce (Campbell Soup Company, Camden, NJ), 8 oz low-fat chocolate milk.

After-dinner snack: 1 cup Quaker Oats Squares (Quaker Oats Company, Chicago, IL), 8 oz low-fat Lactaid milk.

Nutrition Assessment

Although BR's body mass index percentile and percentage ideal body weight were within normal limits, a review of his growth records revealed that his weight gain and linear growth velocity had decreased considerably between ages 8 and 11 years. Specific anthropometric measurements from these visits are presented in Table 1 (one measurement taken after the visit is included). This growth pattern is indicative of probable inadequate energy intake over time and/or excessive energy expenditure. It was not clear whether this growth deceleration coincided with BR's accidental exposure to peanuts approximately 5 years ago. By the time he was seen by the RD at age of 11 years 8 months, his rate of weight gain was improving, but his previous linear growth velocity had not resumed.

Nutrition Intake and Intervention

After the evaluation by the RD, it was determined that BR's extremely limited diet was likely psychosocial in origin. It was most likely rooted in underlying baseline anxiety that was exacerbated by his fear of having an allergic reaction. Distorted body perception or risk for an eating disorder was ruled out by the RD based on his self-reported satisfaction with his current body shape, weight, and height. Because of the significant gastrointestinal symptoms experienced by his siblings, the possibility of a physiologic cause was not ruled out. The RD referred the family to a child psychologist and, given his family history, the primary physician was asked to consider a referral to a gastroenterologist to rule out eosinophilic esophagitis or another gastrointestinal disorder.

The nutrition intervention included providing nutritional guidelines to enhance both his energy intake and the nutritional quality of his diet and a clinical analysis of his 24-hour dietary recall (computerized nutrient analysis took place after the visit). At the time of the consultation, BR's diet was deficient in fruit, vegetables, and

Table 2. Nutrient analysis of a 24-hour dietary recall and estimated macro- and micronutrient needs, Dietary Reference Intakes (DRIs), Adequate Intakes (AIs), and Recommended Dietary Allowances (RDAs) for an 11-year-old boy with allergy to peanut and a self-selective diet

Macro/micronutrient	24-Hour intake	Estimated needs/DRIs	% Estimated needs/DRIs	Mean daily intake for boys 12-19 y ^a
Calories ^{bc}	1,636 kcal (46.9/kg)	2,277 kcal (65.2/kg) ^{bc}	72%	
Protein ^{bd}	65.75 g (1.99/kg)	33.2 g	199%	
Dietary fiber ^b	11 g	31 g†	36%	14.9 g
Vitamin A ^e	550 µg	600 µg	92%	680 µg
Thiamin (vitamin B-1) ^f	3.07 mg	0.9 mg	341%	1.88 mg
Riboflavin (vitamin B-2) ^f	4.07 mg	0.9 mg	452%	2.58 mg
Niacin (vitamin B-3) ^f	34.63 mg	12 mg	289%	28.9 mg
Vitamin B-6 ^f	3.25 mg	1.0 mg	325%	2.29 µg
Vitamin B-12 ^f	9.84 µg	1.8 µg	547%	6.68 µg
Biotin ^f	90.4 µg	20 µg†	452%	NA
Vitamin C ^g	69.1 mg	45 mg	154%	86.6 mg
Vitamin D ^h	475 IU/11.9 µg	600 IU/15 µg	79%	236 IU/5.9 µg
Vitamin E ^g	23.1 mg	11 mg	210%	7.7 mg
Folate ^f	765 µg	300 µg	255%	198 µg
Vitamin K ^e	21.6 µg	60 µg†	36%	76.5 µg
Pantothenic acid ^f	14.4 mg	4 mg†	359%	NA
Calcium ^h	2,034 mg	1,300 mg	157%	1,173 mg
Chromium ^e	30 µg	25 µg†	120%	NA
Copper ^e	0.91 mg	700 µg	130%	1.3 mg
Iodine ^e	221.5 µg	120 µg	185%	NA
Iron ^e	28.3 mg	8 mg	354%	16.6 mg
Magnesium ⁱ	301 mg	240 mg	125%	282 mg
Manganese ^e	2.73 mg	1.9 mg†	144%	NA
Molybdenum ^e	52.9 µg	34 µg	156%	NA
Phosphorus ⁱ	1,674.2 mg	1,250 mg	134%	1516 mg
Selenium ^g	63.8 µg	40 µg	159%	125.2 µg
Zinc ^e	22 mg	8 mg	275%	13.2 mg

^aUS Department of Agriculture Research (29).

^bInstitute of Medicine (31). RDAs are shown in bold type. AIs in ordinary type with a dagger (†).

^cEstimated Energy Requirements (EER)=88.5–[61.9×age (y)]+physical activity coefficient×[26.7 ×wt (kg)+903×ht (m)]+25.

^dEstimated protein requirements: ages 4 to 13 years (both sexes)=0.95 g/kg/day.

^eInstitute of Medicine (32).

^fInstitute of Medicine (33).

^gInstitute of Medicine (34).

^hInstitute of Medicine. DRIs/RDAs for vitamin D and calcium newly established, 2010 (28).

ⁱInstitute of Medicine (35).

whole grains, with presumed inadequacies in fiber and associated micronutrients.

The nutrient analysis (ESHA Research Food Processor Nutrition and Fitness Software version 8.8, 2006, Salem, OR) revealed that his 24-hour intake met the minimum Dietary Reference Intakes for all analyzed micronutrients except vitamin K, vitamin D, and dietary fiber (Table 2). It was presumed that his intake of Ensure contributed to meeting these minimum micronutrient requirements in spite of his apparently nutritionally deficient diet. He met and even exceeded the newly reviewed and established Dietary Reference Intake (DRI)/Recommended Dietary Allowance (RDA) for calcium, but his dietary vitamin D intake was less than the newly established DRI/RDA (28). His ultraviolet sun exposure, and therefore its contribution to meeting his vitamin D requirements, was not known. It is likely that his low intakes of vitamin K and fiber were related to his lack of fruit and vegetable intake. Compared with National Health and Nutrition

Examination Survey data collected on intakes of 12- to 19-year-old boys in 2007-2008, BR's fiber and vitamin K intakes were lower, but his vitamin D and calcium intakes were higher (Table 2) (29). BR's mother was provided with behavioral and environmental guidelines for mealtime structure and stress reduction along with an eating schedule to maximize his appetite. The discussion included suggestions for encouraging BR to become more involved in family meal preparation rather than preparing foods only for himself. The goal was to increase his exposure to and interest in new foods and to increase his confidence in knowing that what he is eating is safe.

Follow-Up

An initial consultation was arranged with a child psychologist 2 months after the outpatient nutrition consultation (August 2009). The family attended the intake session and the psychologist determined that BR suffered from

an anxiety disorder that was having an impact on his eating patterns. Relaxation exercises were provided along with other techniques for managing stress and anxiety. There has been no subsequent follow-up with the psychologist. The parents discussed with their primary care physician the need for an evaluation by a gastrointestinal specialist, but it was determined that no diagnostic tests were needed at that time. At BR's 12-year well-child visit in October 2009, his rate of weight gain was appropriate and his linear growth velocity had increased (Table 1).

The most recent report from the mother was September 2010, approximately 15 months after the first visit. She reported that the variety in BR's diet has greatly expanded. He is now eating fruits and vegetables daily and the quantity of food he is eating has substantially increased. In particular, he is eating more meat. He no longer expresses a fear of accidentally ingesting peanut, but occasionally asks his mother whether something he is about to eat contains peanut. Although he is still anxious about other aspects of his life, his anxiety related to eating seems to have subsided. BR's mother was informed that research suggests that approximately 20% of people with peanut allergy outgrow their clinical symptoms. She was encouraged to consider meeting with an allergist in the future to re-measure BR's peanut sensitivity.

DISCUSSION

It is important for nutrition practitioners to understand the psychosocial impact of living with a food allergy, especially if it is potentially life-threatening. It is not enough to learn only about the technical and clinical aspects of treating a patient with a food allergy. It is important to understand that the knowledge and support offered to a family may have a profound effect on their ability to cope with the consequences of the diagnosis. The more concrete knowledge that can be provided (about reading labels, safely eating at restaurants, risks of cross-contamination, potential sources of hidden allergens, support groups, and online resources), the more families are armed for survival. A thorough diet history and nutrition assessment are important for identifying any growth disturbances or unusual patterns of self-restriction resulting from anxiety or deliberate withholding of food with the intention of losing weight (or gaining control over an uncontrollable situation). If these issues are identified, especially if accompanied by an expression of a distorted body perception, a referral to a psychologist is warranted.

Using a 24-hour diet recall to analyze nutritional intake can be beneficial as it is inexpensive, quick, and accurate because it requires only short-term memory (30). The limitations of its use include the possibility that the subject withholds or alters information about intake, and a record of a single day's intake can be a poor descriptor of an individual's usual intake (30). In the case of BR, a 24-hour diet recall was considered a useful tool in determining usual intake based on his mother's report that his diet was so limited that his food intake did not vary much from day to day. The cause of his limited diet was not clear. It may have coincided with an accidental exposure to peanut, which may have exacerbated or precipitated an underlying anxiety disorder. He expressed

past dissatisfaction with his body, but by the time of the consultation this seemed to have resolved. There was no indication that his restricted diet was an attempt to lose weight. His mother was supportive and sought the follow-up care that was suggested. Based on his most recent anthropometric measurements and his mother's report of his intake, BR's feeding problems have substantially improved or resolved. This case is an example of how beneficial it is to learn to identify patients who are suffering from fear or anxiety to individualize the intervention to provide the help and support that will best inform and comfort patients and their families.

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