



Acquired Donor Peanut Allergy From Lung Transplantation Resulting in Respiratory Failure: A Case Report

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ABSTRACT

This case report describes a patient who acquired a donor peanut allergy after lung transplantation. A 53-year-old woman with alpha-1 antitrypsin deficiency underwent left-sided lung transplant from a donor with a history of anaphylaxis to peanut. Two weeks after the transplant, the patient developed acute respiratory failure immediately after consuming a peanut butter and jelly sandwich. The donor's serum confirmed high titers of peanut-specific immunoglobulin E (IgE). The recipient patient had never had allergies to peanuts or other nuts before her transplant. After the transplant, she had negative serology but positive skin testing to peanuts. This case illustrates the importance of considering donor food allergies when caring for solid organ transplant recipients.

CASE REPORT

A 68-YEAR-OLD woman with alpha-1 antitrypsin deficiency and resulting emphysema underwent a unilateral left-sided orthotopic lung transplant in late 2017. On postoperative day 14, the day before her planned discharge, she developed acute hypoxic respiratory failure. The patient's postoperative course was initially unremarkable. She was induced with basiliximab and methylprednisolone, with no primary graft dysfunction. She was extubated on postoperative day 1 and put on room air for 8 days before her acute respiratory failure. The donor was a 22-year-old man with anoxia from a polydrug overdose. The donor lung grew *Klebsiella pneumoniae*, *Aspergillus*, and *Candida albicans*, for which the patient was on appropriate antimicrobials for 2 weeks. Her immunosuppression at the time was tacrolimus, prednisone 12.5 mg daily, and mycophenolate mofetil.

The patient developed acute onset chest tightness immediately followed by hypoxic respiratory failure with oxygen saturation (SpO₂) of 61% on room air. On physical exam, the patient was in visible respiratory distress, with decreased breath sounds bilaterally. No rashes, facial neck swelling, or gastrointestinal signs or symptoms were appreciated. She was immediately placed on a nonrebreather mask, with SpO₂ improving to 85%-90%; she was then placed on bilevel positive airway pressure ventilation machine, with a fractional inspiration of oxygen of 100%, with SpO₂ improving to 90%-94%. A portable chest radiograph had no acute pneumothorax, pleural effusion, or new lung infiltrates. Because of her continued respiratory distress on the bilevel positive airway pressure ventilation machine, the patient was intubated. Laryngeal swelling and foreign bodies were not seen during direct laryngoscopy. Computed tomography scan (with contrast) of the chest

revealed a small left upper subsegmental pulmonary embolism but no bronchial anastomotic stricture or new lung infiltrates. Ultrasound of the lower and upper extremity were negative for deep vein thrombosis. Bronchoscopy found minimal thin secretions, no foreign bodies or mucus plugs, and normal left anastomosis site without strictures, dehiscence, or black eschar. Her transthoracic echocardiogram showed normal cardiac function, without right or left ventricular dysfunction, and improving pulmonary artery pressures from before the transplant of 40 mmHg to 30 mmHg. She was successfully extubated about 18 hours after her respiratory failure event. After extubation, the patient revealed that she was completely asymptomatic leading up to her respiratory failure. Immediately before the event, she had eaten a peanut butter and jelly sandwich and subsequently started feeling chest tightness.

The transplant agency confirmed that the donor had known anaphylaxis to peanut. The donor's serum was subsequently positive for peanut IgE (Table 1). The IgE concentrations for the donor's peanut IgE and Ara H2 were highly elevated and moderately elevated for Ara H8. The recipient patient's nut IgE panel was negative for all nuts including peanut. Tryptase level 2 days after respiratory failure was normal at 3.2 ug/L. At discharge, the patient was advised to avoid peanuts and tree nut products, and she was prescribed an epinephrine autoinjector. Upon follow-up at the Allergy and Immunology Clinic, the patient reported no allergic

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Table 1. Lung Donor's Serum Immunoglobulin E Concentration to Peanut and Peanut Components

Whole Peanut IgE 9.74 kU/L	Reference Interval: ≤ 0.34
Peanut component Ara H1 IgE 0.13 kU/L	Reference interval: ≤ 0.09
Peanut component Ara H2 IgE 6.97 kU/L	Reference interval: ≤ 0.09
Peanut component Ara H3 IgE < 0.10 kU/L	Reference interval: ≤ 0.09
Peanut component Ara H9 IgE 0.33 kU/L	Reference interval: ≤ 0.09
Peanut component Ara H8 IgE 2.79 kU/L	Reference interval: ≤ 0.09

Ara H1, H2, H3, H8, and H9 indicate the seed storage proteins from *Arachis hypogaea* (peanuts) with roles in peanut allergy.

Abbreviation: IgE, immunoglobulin E.

reactions while continuing to avoid peanuts and tree nuts. The patient's nut serology done approximately 3 months from the first serology was negative again for all nuts and peanut (including component testing). Skin prick testing, however, was positive to peanut, almond, cashew, coconuts, and hazelnut.

DISCUSSION

Our case illustrates a lung transplant wherein the donor's allergic reaction to peanut was transferred to the recipient patient. The acquisition of food allergies has been established in the literature in the cases of liver, kidney, lung, bone-marrow, heart, and kidney transplants but most reported cases have been liver transplants in pediatric patients. The food allergies reported have included but are not limited to nuts, milk, egg, wheat, and fish that develop over days to months posttransplant [1]. There is an increased frequency of food allergies in children receiving tacrolimus after liver transplants from 6% to 20% [2]. Our patient also had been on tacrolimus. There have been 3 case reports with acquired peanut allergies with anaphylaxis after a lung transplant. One reported in 2005 described a woman who had 4 episodes of anaphylaxis in the first 4 weeks postoperatively, with negative recipient peanut-specific IgE but positive skin prick testing that declined over a year [3]. Another case in 2009 described a donor who had passed away from anaphylaxis to peanuts, and the recipient had wheezing and dyspnea while eating peanut-containing products starting 4 days after transplant and subsequently experienced anaphylaxis 7 months after the transplant. This recipient had positive skin prick tests and IgE after the transplant [4]. A case in 2009 also had anaphylaxis 3 months

after lung transplant in a patient with negative peanut-specific IgE serologies before transplant. After the transplant, the patient's peanut specific IgE turned positive, and the patient also had positive peanut skin-prick tests. In our case, although the recipient IgE has been negative during both evaluations, this may be a false negative in the setting of chronic steroids, which can suppress in vitro but not in skin prick testing. Our patient's skin testing, which is more sensitive compared to in vitro testing, has been repeatedly positive for peanuts [5]. Given that systemic corticosteroids are essential in transplant management, sole reliance on in vitro testing would not be recommended. There are unfortunately no valid biomarkers to predict kinetics of transferred sensitization, but available case reports document instances of transient sensitization. Our approach will be to monitor peanut sensitization over the year after transplant to determine whether oral graded challenges to peanut can be safely performed.

CONCLUSIONS

Lung transplants can transfer donor peanut allergies to their recipients, but donor transferred allergies may be inherently underreported owing to lack of awareness of this phenomenon. Avoiding donor food allergies after transplant would be prudent to minimize the risk of anaphylaxis until the recipient patient is evaluated for the allergy in a controlled setting with serology, skin testing, and oral challenges.

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