

Management of acquired peanut allergy following solid-organ transplant

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Clinical Implications

- Recipients of solid-organ transplants from donors with high peanut IgE antibodies are at risk for anaphylaxis. Effective strategies to mitigate the risk for adverse food reactions should include delaying dietary peanut after transplant, education, and prospective screening with IgE and skin prick test.

TO THE EDITOR:

Passive transfer of peanut-specific IgE after solid-organ transplant is associated with risk for life-threatening anaphylaxis. Prospective identification of organ recipients at risk for passively transferred allergy is not standard of care. We present a case and review the literature on the management of acquired peanut allergy after transplantation. Testing for specific IgE and skin prick test (SPT) reactivity to peanut are discussed with a focus on mitigating the risk for food anaphylaxis in the posttransplant period.

A 66-year-old woman with a history of hepatocellular carcinoma and steatohepatitis, with no history of atopy or food allergy, received a donor liver transplant from a deceased 14-year-old boy. The donor's history was significant for asthma, allergic rhinitis, and nut allergy. The transplant surgery was uncomplicated, and the liver recipient was discharged on postoperative day 5 on mycophenolate, prednisone, and tacrolimus. She was given instructions to maintain adequate nutrition and resume her regular diet without restriction.

Two weeks after surgery, the recipient ingested peanut butter and developed immediate oral and facial swelling, generalized pruritus, and lightheadedness and collapsed. The patient's condition stabilized after the administration of epinephrine and antihistamines by Emergency Medical Services. Serum tryptase level, obtained 16 hours after the acute event, was normal at 4.6 ng/mL. She was discharged with an epinephrine pen, anaphylaxis action plan, and recommendations for strict peanut and tree nut avoidance.

Passively acquired peanut allergy was suspected on the basis of donor history. To test this, we measured allergen-specific IgE in stored serum specimens available from the liver donor and recipient before transplantation and followed these tests prospectively in our patient. We compared the IgE antibody profiles for peanut and peanut components (Ara h 1, -2, -3, -8, and -9) from our patient with serum from the liver donor (ImmunoCAP;

Phadia, Uppsala, Sweden). Similar to the donor profile, the recipient IgE antibody profile showed elevated specific serum IgE levels to peanut and Ara h 1, -2, and -3 at the time of anaphylaxis that had not been present before transplant (Table 1). Results of the specific IgE tests and percutaneous SPT to peanut and tree nuts were followed over the course of 6 months. At 2 months posttransplantation, serum IgE level to peanut and Ara h 1, -2, and -3 returned to negative (<0.1 kU_A/L); however, SPT to peanut and tree nuts to which the donor was also sensitized revealed reactivity (Table 1). We recommended graded food challenges to almond, cashew nut, and walnut to reduce the patient's risk for another allergic reaction in an unsupervised setting. After passing challenges, tree nuts were reintroduced into the patient's diet with continued peanut avoidance. At 6 months posttransplant, SPT to peanut extract was nonreactive and the patient underwent open peanut challenge without adverse reaction. The patient currently maintains peanuts and tree nuts in her diet. This time course supports passive transfer of IgE from donor to recipient at the time of transplantation.

Food anaphylaxis from passive IgE in the first year posttransplant must be distinguished from *de novo* food allergies reported in 6% to 17% of certain solid-organ transplant populations.¹ Examination of affected patients' peanut IgE component profile relative to donor serum can help to support the diagnosis of a transient food allergy.²⁻⁴ IgE profiling in donor and recipient serum combined with SPT has also helped to safely guide the reintroduction of dietary peanut for organ recipients presenting with peanut anaphylaxis after transplant.²⁻⁴

Comparison of serial component IgE titers from our patient with cases from the transplant literature shows that among affected recipients from different solid organs, titers of IgE antibody to peanut may be low compared with the organ donor and decline at variable rates over the first year posttransplant (Figure 1). IgE level of 1.43 kU_A/L to Ara h 2, compared with 133 kU_A/L in the organ donor, was associated with near life-threatening anaphylaxis in our patient. Although the average half-life of serum IgE antibody is 3 days, the kinetics of passive IgE catabolism in recipient serum are influenced by the titer of IgE in donor serum, recipient IgE, as well as IgE bound to mast cells and basophils.^{5,6} There are undetermined variables that affect IgE catabolism after transplant; previous reports have detected elevated IgE levels to peanut components up to 10 months after some organ transplants, suggesting that solid organs serve as a reservoir of IgE entering circulation over time.² Given the unknown kinetics of free peanut IgE transfer from different solid organs, affected organ recipients should be followed closely with the recommendation of peanut avoidance until testing is complete.

In current practice, prospective screening for acquired peanut allergy based on donor history is rare,⁴ leading to periodic case reports of anaphylaxis.^{2,3,7} In this case, and 3 others,²⁻⁴ recurrent adverse clinical reactivity with peanut ingestion was prevented by patient education and delaying peanut reentry into the transplant recipient's diet until both serum IgE and SPT reactivity to peanut were negative. Although testing for serum IgE to nut components provides clinical utility to guide food challenges in certain patient situations,^{8,9} we propose that its main value in acquired peanut allergy is to evaluate whether the serum

TABLE I. IgE and SPT results

Results	Donor		Liver transplant recipient		
	Pretransplant	Pretransplant	Hospitalized anaphylaxis (2 wk posttransplant)	Allergy clinic follow-up (2 mo posttransplant)	Allergy clinic follow-up (6 mo posttransplant)
Total IgE (IU/mL)		648	146	68.1	
Peanut IgE (kU _A /L)	387	0.16	4.39	<0.1	
Peanut components (kU _A /L)					
rAra h1	102	<0.1	1.2	<0.1	
rAra h2	133	<0.1	1.43	<0.1	
rAra h3	30.2	<0.1	0.39	<0.1	
rAra h8	2.09	0.36	<0.1	<0.1	
rAra h9	<0.1	<0.1	<0.1	<0.1	
Tree nut IgE (kU _A /L)					
Almond	4.99	<0.1	<0.1	<0.1	
Brazil nut	2.35	<0.1	<0.1	<0.1	
Cashew nut	4.27	<0.1	<0.1	<0.1	
Hazelnut	4.79	<0.1	<0.1	<0.1	
Pecan nut	0.45	<0.1	<0.1	<0.1	
Pistachio	5.30	<0.1	<0.1	<0.1	
Sesame seed	3.01	<0.1	<0.1	<0.1	
Black walnut			<0.1	<0.1	
Walnut	0.25	<0.1	<0.1	<0.1	
SPT (wheal/flare) (mm)					
Peanut				10/8	0/0
Almond				4/6	0/0
Brazil nut				0/0	0/0
Cashew nut				5/8	0/0
Hazelnut				0/0	0/0
Pecan nut				0/0	0/0
Pistachio				0/0	0/0
Sesame seed				0/0	0/0
Black walnut				4/6	0/0
Walnut				0/0	0/0
Histamine control				7/10	5/6
Negative control				0/0	0/0

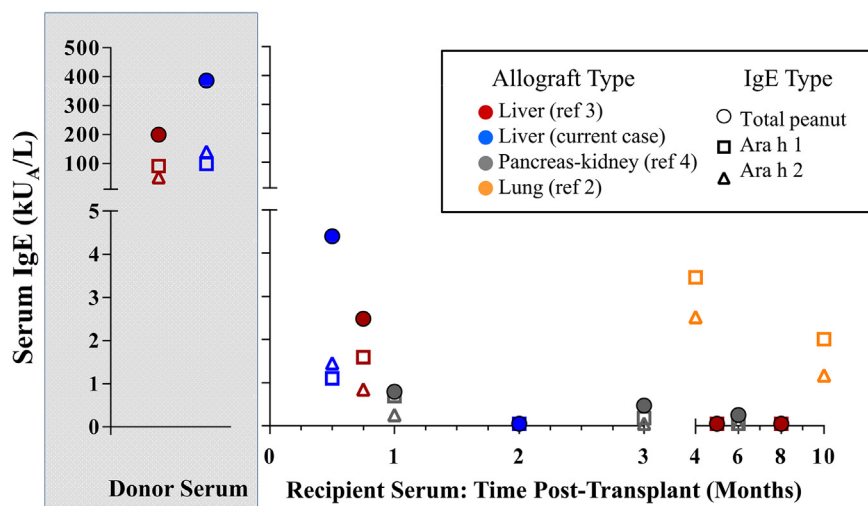


FIGURE 1. Titers of IgE to peanut, Ara h 1, and Ara h 2 in solid-organ donors and recipients after transplantation.

sensitization profile in the organ recipient reflects organ donor sensitization. However, serum IgE testing without follow-up SPT before dietary peanut reintroduction may place transplant patients at risk for anaphylaxis.

This case, taken together with a larger body of recent reports, emphasizes the need for standardized assessment of solid-organ recipients, beginning with donor allergy history, to identify recipients at risk for anaphylaxis from passive IgE transfer. Education of identified “at-risk” organ recipients regarding food avoidance, signs and symptoms of anaphylaxis, and how to treat an allergic reaction with epinephrine autoinjectors could prevent potentially fatal food-related anaphylaxis. This case highlights the phenomenon of passive acquisition of IgE from solid-organ transplant and the need to perform serial allergy assessments when introducing food allergens during the posttransplant period.

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