Published in final edited form as:

J Allergy Clin Immunol Pract. 2015 ; 3(4): 612–614. doi:10.1016/j.jaip.2015.03.003.

Management of Acquired Peanut Allergy Following Solid-Organ Transplant

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Keywords

solid-organ transplant; peanut allergy; anaphylaxis; Ara h 1; Ara h 2

TO THE EDITOR:

Passive transfer of peanut-specific IgE following 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 management of acquired peanut allergy following transplantation. Testing for specific IgE and skin prick reactivity to peanut are discussed with focus on mitigating risk for food-anaphylaxis in the post-transplant period.

A 66 year-old woman with history of hepatocellular carcinoma and steatohepatitis, with no history of atopy or food allergy, received a deceased donor liver transplant from a 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 post-operative day five 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, lightheadedness and collapsed. The patient's condition stabilized following administration of epinephrine and antihistamines by EMS. Serum tryptase, obtained 16 hours after the acute event, was normal at 4.6ng/mL. She was discharged with an epinephrine pen, anaphylaxis action plan and recommendations for strict peanut and tree nut avoidance.

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

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Passively acquired peanut allergy was suspected based on the donor history. To test this, we measured allergen-specific IgE in stored serum specimens available from the liver donor and recipient prior to 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 to serum from the liver donor (ImmunoCAP; Phadia, Uppsala, Sweden). Similar to the donor profile, the recipient IgE antibody profile showed elevated specific serum IgE to peanut, Ara h 1, -2 and -3 at the time of anaphylaxis which had not been present prior to transplant (Table 1). IgE and percutaneous skin prick testing (SPT) to peanut and tree nuts were followed over the course of 6 months. At 2 months post-transplantation, serum IgE to peanut, 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, and walnut to reduce the patient's risk for another allergic reaction in an unsupervised setting. After passing challenges, tree nuts were re-introduced into the patient's diet with continued peanut avoidance. At 6 months post-transplant, SPT to peanut extract was non-reactive and the patient underwent open peanut challenge without adverse reaction. The patient currently maintains peanut 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 post-transplant must be distinguished from *de novo* food allergies reported in 6-17% 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 (2,3,4). IgE profiling in donor and recipient serum combined with skin prick testing has also helped to safely guide the re-introduction of dietary peanut for organ recipients presenting with peanut anaphylaxis after transplant (2, 3, 4).

Comparing serial component IgE titers from our patient to cases from the transplant literature shows that among affected recipients from different solid organs, titers of IgE ab to peanut may be low compared to the organ donor and decline at variable rates over the first year post-transplant (Figure 1). IgE to Ara h 2 of 1.43 kU_A/L, compared to 133 kU_A/L in the organ donor, was associated with near life-threatening anaphylaxis in our patient. Though the average half-life of serum IgE ab 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 to peanut components up to 10 months following 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 (2,3,4), recurrent adverse clinical reactivity with peanut ingestion was prevented by patient education and delaying peanut re-entry into the transplant recipient's diet until both serum IgE and skin prick reactivity to peanut were negative. While 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 if the serum sensitization profile in the organ recipient reflects organ donor sensitization. However, serum IgE testing without follow-up skin prick testing before dietary peanut re-introduction 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, in order to identify recipients at risk for anaphylaxis from passive IgE transfer. Education of identified “at risk” organ recipients regarding food avoidance, the signs and symptoms of anaphylaxis, and how to treat an allergic reaction with epinephrine auto-injectors 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 post-transplant period.

Acknowledgments

Funding Sources: This work was supported by the following grants from NIH/NIAID: R01 AI 020565-29 (TAE Platts-Mills); and by an AAAAI/Food Allergy Initiative Howard Gittis Memorial Fellowship Award (J. Wisniewski).

Abbreviations

Ab	antibody
SPT	skin prick testing

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Figure 1.
Titers of IgE to peanut, Ara h 1 and Ara h 2 in solid organ donors and recipients following transplantation.

Table 1**IgE and Skin Prick Test Results**

	Donor	Liver Transplant Recipient			
Clinical Event	Pre-transplant	Pre-transplant	Hospitalized Anaphylaxis	Allergy clinic Follow-up	Allergy clinic Follow-up
Time from Transplant	0	0	2 weeks	2 months	6 months
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	
Hazel Nut	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	
Skin Prick Testing (wheal/flare mm)					
Peanut				10mm/8mm	0/0
Almond				4mm/6mm	0/0
Brazil nut				0/0	0/0
Cashew nut				5mm/8mm	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				4mm/6mm	0/0
Walnut				0/0	0/0
Histamine control				7mm/10mm	5mm/6mm
Negative control				0/0	0/0