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Post liver’s transplant acquired food allergy

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Transplant-acquired allergy (TAA) was firstly described as transplant-acquired food allergy (TAFA) after bone marrow transplantations and mostly observed in a transient form. The development of allergy to a specific food in the recipient of blood products from a donor allergic to the culprit food is easily explained by the passive transfer of food allergen–specific IgE. Identifying the mechanisms responsible for the development of food allergy in liver transplant recipients is more complex, as there are several different clinical scenarios related to the immunological function of the liver.

One of the assumptions of the TAA, is linked to the use of immunosuppressive drugs used in prevention of graft rejection. An increased prevalence of food allergy noted specifically in children receiving tacrolimus immunosuppression supports the hypothesis that selective suppression of Th1 lymphocytes by the interleukine (IL)-2 inhibitor immunosuppressants promotes Th2 lymphocytes and an allergic immune response (Ozdemir 2013).

We describe a case of a 68 years old woman that received liver transplantation from a donor with food allergy who died for anaphylactic shock. In view of the clinical features of the donors and of the reports found in the literature (Wisniewski 2012), the patient was in therapy post transplantation with tacrolimus, mycophenolate methyl and oral prednisone.

Specific IgE to were sought by ImmunoCAP. The patient scored positive for casein, beta lactoglobulin and negative result of skin test to foods (milk, nut, egg, LTP ecc, ecc.). A Basophil activation test (BAT) (CD63) was carried out one month after the liver transplantation and scored positive for casein alpha-lactoglobulin, beta lactoglobulin and egg yolk. While it was negative for other food allergens. If CD203c was used as a marker in BAT, all tested allergens scored positive.

In view of the recent case (Vagefi 2009), of a patient who received a transplant from a donor aged 15 with allergy to peanuts, kiwi, fish and wheat, which had died of anaphylaxis who developed anaphylactic shock after eating peanuts one week after the transplantation.

Milk- and egg-free diet was started and continued for one month. Then oral provocation tests with both cow’s milk and hen’s egg (were carried out). Cow’s milk challenges were performed using pasteurized milk (Calvani M. 2012). First, a drop of milk was put on the tongue, then increasing doses (0.1, 0.3, 1, 3, 10, 30, 100 ml) were given at 20-min intervals or until a reaction was noted. OFC with raw egg boiled was performed by administering an emulsion of one both raw yolk and white mixed with a tolerated juice, starting with a drop and roughly doubling doses until the whole egg was given. Oral provocation test was negative both for egg both for milk.

The patient continued eating nuts without symptoms. 4 months after the reintroduction of milk and egg the basophil activation test became negative; also the measurement of specific IgE to food allergens was negative and also the skin prick test still remained negative.

Showing that most patients developed food allergy by one yr post transplant, the latency between time of transplant and diagnosis of FA varied from 1.2 months to 17.6 yr in patients, suggesting that different
mechanisms of sensitization may be involved in different individuals. Donor allergic status is already an established risk factor for de novo allergy among solid organ transplant recipients. Passive transfer of allergy from IgE in blood of an allergic donor to naive recipient has been demonstrated but is only a plausible explanation for those recipients who developed symptoms within two months following transplantation and does not account for eosinophilic disease. Various mechanisms of sensitization transfer have been suggested. One possibility is passive transfer of donor IgE, although Legendre pointed out that the half-life of circulating IgE is only a few days. However, reactions have been reported soon after transplantation and might be explained by retention of specific IgE in donor liver sinusoids, leading to recipient mast-cell sensitization. Long-term binding of donor-derived specific IgE to recipient mast cell and basophil high-affinity receptors increases its half-life and could thus lead to mediator release with clinical repercussions during a period of several months after transplantation. In our patient, circulating IgE specific to casein and cow milk protein was probably detectable as early as 24 h after transplantation and was when tested 3 and 6 months after the transplantation. This case demonstrates that the transmission of food allergies in liver transplantation should be considered especially in the case of death of the donor shock anaphylaxis.