5-19-2011

Anaphylaxis from Passive Transfer of Peanut Allergen in a Blood Product

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TO THE EDITOR: Anaphylactic reactions to blood transfusions are rare and their causes often remain elusive.[1] The inducement of clinically relevant allergic reactions by means of the passive transfer of IgE in blood products has been well documented.[2] In an editorial comment written in 2003, Erick speculated on the possibility that allergic transfusion reactions could be induced by the passive transfer of food allergens.[3] We present such a case.

A 6-year-old boy with acute lymphoblastic leukemia had an anaphylactic reaction while receiving a leukoreduced pooled buffy-coat product with ABO-identical platelets. During transfusion, rash, angioedema, hypotension, and difficult breathing occurred. The patient recovered within 30 minutes after resuscitation with adrenaline. His serum level of mast-cell tryptase, measured directly after the reaction, was 24 μg per liter (normal level, <5), which confirmed the clinical pic-

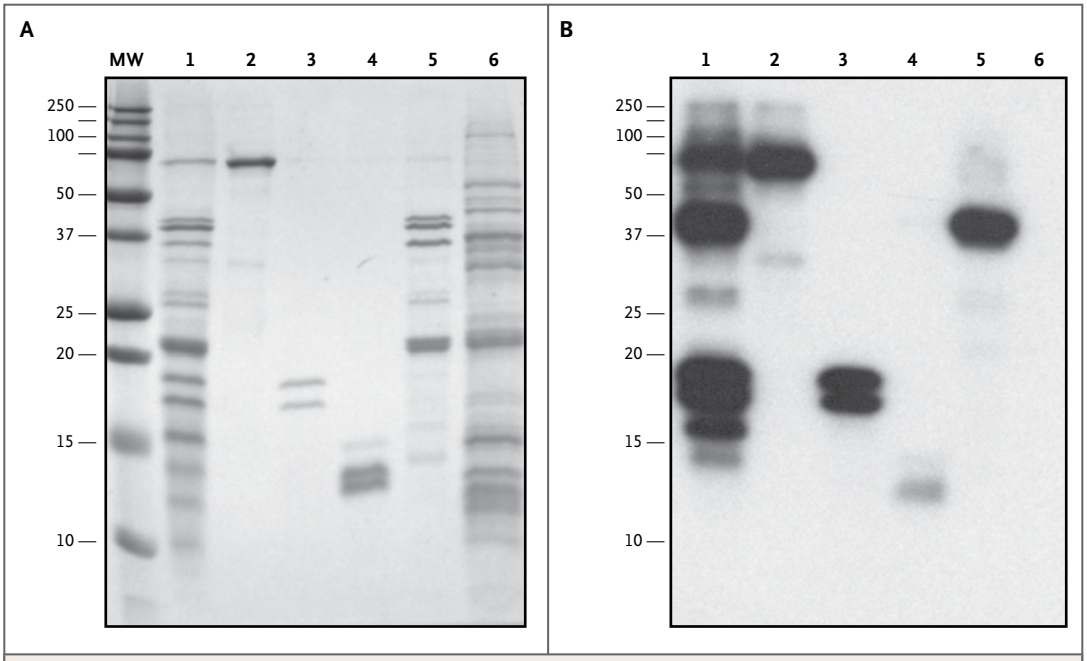

Figure 1. IgE Reactivity of a 6-Year-Old Boy to Peanut Allergens.

Panel A shows the results of sodium dodecyl sulfate–polyacrylamide-gel electrophoresis and Panel B IgE immunoblotting with use of the patient's serum. In both panels, peanut protein (5 μg) appears in lane 1; the peanut allergens Ara h1 (0.5 μg) and Ara h2 (0.5 μg) in lanes 2 and 3, respectively; digestion-resistant peptide from Ara h2 (1 μg) in lane 4; peanut allergen Ara h3 (0.5 μg) in lane 5; and a buckwheat protein–negative control (5 μg) in lane 6. The standard marker for molecular weight (MW) is shown for comparison.

ture of a type I allergic reaction. No conventional mechanism could explain this transfusion reaction. Detailed laboratory analyses ruled out the possibilities of deficiencies in IgA, C4, or haptoglobin, allergies to drugs or latex, the presence of HLA antibodies, and transfusion-related acute lung injury.

The patient's mother stated that her son had had a similar reaction after eating peanuts at the age of 1 year. Since that time, peanuts had been excluded from his diet. Three of the five blood donors, contacted shortly after the transfusion reaction, recalled eating several handfuls of peanuts the evening before donation. The major peanut allergen, Ara h2, is extremely resistant to digestion[4]; therefore, we investigated whether the peanut allergens ingested by the donors could have been administered in the transfusion. In contrast with the intact peanut protein, the digestion-resistant peptide from Ara h2 (DRP–Ara h2) can be detected in serum for up to 24 hours after ingestion,[5] and it is sufficiently large (with a mass of 10 kD) to bind IgE and to elicit allergic reactions. We corroborated the recipient's peanut allergy with an ImmunoCAP assay (Phadia) that revealed a serum level of peanut-specific IgE of 72.5 kU per liter (normal level, <0.35). We also determined that IgE antibodies reactive to both Ara h2 and DRP–Ara h2 were present (Fig. 1).

These data are consistent with the hypothesis that the consumption of peanuts by the donors before blood donation provided the trigger for this patient's transfusion reaction. It is possible that allergens transferred in blood products to other patients have led to reactions that have gone unexplained and unreported. This case highlights the need to consider donor-ingested allergens as a source of reactions in sensitized recipients.

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Disclosure forms provided by the authors are available with the full text of this letter at NEJM.org.

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