Duck Egg Allergy in an Adult Patient Without Allergy to Chicken Egg

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Key words: Uncommon food allergy. Duck egg. Ovalbumin.

Egg is a basic ingredient in our diet because it provides essential nutrients of high biological value that are easily assimilated by the human body. Duck egg allergy is an uncommon food allergy that has been commonly associated with allergies to other types of egg, usually chicken [1]. We present a case of food allergy after ingestion of duck egg in an adult patient with no previous allergy to chicken egg. The patient was a 25-year-old woman with a medical history of urticaria to dog dander who experienced abdominal pain, diarrhea, and loss of consciousness within 60 minutes after ingestion of a fried duck egg. She tolerated hen egg with no problems. No symptoms of rhinitis or asthma were reported.

Skin prick tests were performed with commercially available extracts of common inhalant allergens (house dust mites, molds, animal dander, and pollens) and hen egg proteins (egg white, egg yolk, ovalbumin, and ovomucoid) (Leti). Prick-prick testing was performed with fresh homemade uncooked white and yolk from duck egg (Anas domesticus) according to the Dreborg and Foucard method [2].

Specific and total IgE were determined using ImmunoCAP (ThermoFisher Scientific) to hen egg proteins (yolk, white, ovalbumin, and ovomucoid) according to the manufacturer’s instructions, with negative results (ImmunoCAP <0.35 kU/L). Fresh homemade extracts of white and yolk of duck egg were prepared in phosphate buffer at 10% (wt/vol) and kept for 90 minutes at 4°C with magnetic shaking. They were then centrifuged, the resultant supernatant was filtered through a 0.2-µm membrane, and glycerin was added up to 50% before use. Commercial extracts of ALK from hen’s egg were also used. The extracts and the molecular weight markers were analyzed using glycine SDS-PAGE (acrylamide concentration, 16%) under nonreducing conditions. The extract proteins separated by SDS-PAGE were transferred onto nitrocellulose membranes as described by Towbin et al [3]. Immunoblotting of IgE-binding protein was achieved by enhanced chemiluminescence according to the manufacturer’s instructions (ECL-Amersham Bioscience). As negative controls, the blots were also incubated with dilution buffer.

Skin prick tests were positive for pollens of grass, olive, Plantago, cypress, and dog dander and negative to the remaining inhalant extracts and hen’s egg. Prick-prick testing was only positive to duck egg white, with a wheal measuring 22 mm in diameter. The Figure shows the result of IgE Immunoblotting for the different egg extracts. Duck egg allergy is an uncommon allergy. Allergy to egg is associated with different species, although 2 cases of specific allergy to duck egg in patients with no hen egg allergy have been reported. In both, a lysozyme and ovalbumin were identified as the responsible allergens [4,5]. In the case presented in this study, the protein detected by immunoblotting has a molecular weight that suggests that ovalbumin could be the allergen responsible for the allergic reaction. The patient’s IgE recognized this protein in duck egg but not in hen egg. These findings seem to indicate that the patient’s IgE can recognize specific epitopes of duck egg ovalbumin.

Funding
The authors declare that no funding was received for the present study.

Conflicts of Interest
The authors declare that they have no conflicts of interest.

References
Autoimmune Diseases and Asthma

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Cellular immune mechanisms comprise 2 types of response, namely the T1 response and the T2 response [1]. Asthma is a heterogeneous disease characterized by chronic inflammation of the airways. Various cells and cytokines, mainly those of the T2 profile [2], intervene in the pathogenesis of asthma, whereas the T1/T17 profile predominates in autoimmune diseases. The immunologic paradigm TI/T2 predicts a negative association between autoimmune (T1) and allergic diseases (T2) [1]. However, some authors propose autoimmunity as the key pathological mechanism of so-called intrinsic asthma, while others consider it an additional phenomenon to allergy in the development of asthma. Co-occurrence of allergy and autoimmunity in the same patient and the presence of autoantibodies in both entities support the hypothesis of autoimmunity in asthma [3-5]. Other factors that corroborate this hypothesis are the role that T-cell dysregulation and mast cells have in both diseases [2-5].

With the aim of analyzing the inflammatory profile of patients with asthma and autoimmune diseases, we describe a series of consecutive asthmatic patients (diagnosed according to GINA criteria [2] at least 1 year before inclusion in the study) with a known concomitant autoimmune disease attended during the year 2016 in a certified multidisciplinary severe asthma unit.

After signing the informed consent document, patients were included in the study and phenotyped as T2-high or T2-low according to the following criteria proposed by Woodruff et al [6] and Kraft [7]: the T2-high phenotype was defined as total IgE >100 IU/mL and peripheral blood eosinophilia >140/mm³; the phenotype was considered T2-low if only 1 or neither of these 2 criteria was met. Pulmonary function parameters and other inflammatory biomarkers (fractional exhaled nitric oxide [FeNO] and serum periostin) were registered. Asthma control was assessed based on the presence of at least 1 exacerbation during the previous year and on the results of the Asthma Control Test (ACT).

References:

**Manuscript received August 21, 2018; accepted for publication January 4, 2019.**

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