

Report

Egg allergy in patients over 14 years old suffering from atopic eczema

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Abstract

Objective The aim of this study was to evaluate the occurrence of egg allergy in patients over 14 years old suffering from atopic eczema and especially to evaluate if egg allergy can deteriorate the course of atopic eczema in this group of patients.

Materials and methods Altogether 179 patients suffering from atopic eczema were included in the study: 51 men and 128 women, with an average age of 26.2 years (SD 9.5 years), with median SCORAD 31.6 (SD 13.3) points. A complete allergological and dermatological examination was performed on all patients, including diagnostic work-up of food allergy to egg [skin prick tests, atopy patch tests (APTs), measurement of specific IgE level to egg yolks or whites]. Open exposure test (OET) with egg was performed in patients with positive results in some of these diagnostic methods. Food allergy to egg was determined according to positive results in the OET or according to sufficient anamnestic data about the severe allergic reaction after the ingestion of an egg.

Results An allergy to egg was confirmed in 11 patients out of 179 (6%). Of these patients, only six (3.3%) had a clear improvement in the SCORAD after the elimination of egg. Other triggering factors may cause exacerbation of the atopic eczema in the patients enrolled in the study. Twenty-eight percent of patients were only sensitized to egg without clinical symptoms. APTs were a useful tool in the diagnosis of food allergy to egg in patients without IgE reactivity.

Conclusion Egg allergy may play an important role in the worsening of atopic eczema acting as a triggering–exacerbating factor in a minority of patients. The diagnostic work-up may comprise the challenge tests to confirm the food allergy to egg.

Introduction

Egg allergy is one of the most common food allergies in infants and young children, affecting 1–2% of young children.¹ In a recent meta-analysis,² the prevalence of egg allergy was estimated to be between 0.5 and 2.5%. Egg sensitization is closely associated with atopic dermatitis, particularly in infants who develop eczema in the first year of life.^{3–5} Egg-sensitized children have been shown to have more severe and persistent dermatitis, and the level of sensitization correlated with the severity of disease.^{3,5} Delayed exacerbations of atopic dermatitis (>24 hours after egg consumption) may occur in children without evidence of sensitization to egg,^{6,7} most likely because of T-cell mediated allergic reactions.⁸ The risk of sensitization to aeroallergens⁹ and asthma¹⁰ is also increased in egg-allergic children. In an international multicenter study of children

with atopic dermatitis, a close association between early-onset, moderate-to-severe eczema and egg sensitization was found.³ A small number of children with an egg allergy presented with gastrointestinal symptoms, including allergic proctocolitis or eosinophilic esophagitis.^{11,12} Egg can cause severe allergic reactions in sensitized children, and egg avoidance can cause significant dietary limitations.¹³ Egg allergy is generally considered to have a good prognosis, and parents are typically counseled that their children will outgrow the allergy by early school-age years; however, recent reports indicate that it is taking longer for children to outgrow their egg allergy, with most developing tolerance in their teenage years rather than in early school-age as previously thought.^{14,15} Diagnosis of food allergy is based on personal history, measurement of specific IgE (serum-specific IgE level – sIgE, skin prick tests – SPT), atopy patch tests (APTs), and exposure tests (open

exposure test – OET). Double-blind, placebo-controlled food challenge (DBPCFC) remains the gold standard in diagnosis of food allergy.¹⁶ Although the unequivocal diagnosis of egg allergy requires a DBPCFC,¹⁷ open food challenges, which are less resource-intensive, are generally considered sufficient in clinical practice.

The aim of this study is assessment of the egg allergy in patients with atopic eczema over 14 years old and especially the evaluation of whether food allergy to egg can deteriorate the course of atopic eczema. Our other aim is the long-term follow-up of patients with confirmed food allergy to egg with assessment of the severity of atopic eczema for 12 months after the elimination of the food allergen.

Materials and methods

Patients over 14 years old with atopic eczema examined at the Department of Dermatology at the Faculty Hospital in Hradec Králové (Czech Republic) from January 2005 to June 2009 were included in the study.

The diagnostic work-up of food allergy to egg (SPT, APT, sIgE) in intervals with milder symptoms of atopic eczema (evaluated with SCORAD) was performed.

Complete dermatological and allergological examination was performed in all included patients, with the evaluation of the occurrence of asthma bronchiale at the allergological outpatient department.

Scoring of atopic eczema

The diagnosis of atopic eczema was made using the Hanifin-Rajka criteria¹⁸ at the outpatient department of Dermatology and Venereology, Faculty Hospital and Medical Faculty of Charles University, Hradec Králové, Czech Republic.

The severity of eczema was scored in agreement with the SCORAD score: as a mild form – 20 points; moderate – over 20–50 points; severe – over 50 points. The severity of atopic eczema was evaluated by this system at the beginning of the study and then at the end of the specific hypoallergenic diet before the OET and during the OET until 48 hours after the challenge. The severity of atopic eczema was evaluated in patients with a confirmed food allergy three, six, nine, and 12 months after elimination of egg from the diet. In patients with negative reactions in the OET, egg was introduced in a common meal, and the severity of atopic eczema was also evaluated over a period of three months in one year.

Personal history

The detailed personal history of possible food allergy was taken in all included patients.

The patients answered if they suffered from immediate or late food adverse reactions affecting the skin, gastrointestinal tract, or respiratory tract not only after the ingestion of egg but also after ingestion of other foods.

After the discontinuation of antihistamines and topical steroids for at least five days and systemic steroid therapy and phototherapy for at least two months, SPT, APT, and challenge tests were performed. The serum level of the specific IgE to yolk and white has been examined. The patient was allowed to treat himself with a low-potency topical corticosteroid. No other anti-inflammatory substances or UV-therapy were applied.

SPT

Commercial food extracts Alyostal (Antony, Stallergens, France) with yolk and white were used for SPTs.

SPTs were placed on the volar side of the forearm according to the extent of atopic eczema. SPTs were carried out by a standardized method using lancets with a 1-mm tip. The results were read after 15 minutes, and were assessed by comparison with the wheal induced by histamine (10 mg/ml) and negative control. A wheal with a diameter >3 mm in comparison with a negative control was scored as positive.

Specific IgE

The serum level of the specific IgE to yolk and white has been measured using the CAP method (system FEIA; Pharmacia Diagnostics, Uppsala, Sweden). A level of specific IgE higher than 0.35 U/ml was assessed as positive.

APTs

APTs were performed on non-lesional, non-abraded, untreated skin of the back during a remission.

A technique similar to conventional patch tests has been used for performing ATPs – CURATEST F strip (Lohmann & Rauscher International GmbH, KG D-56579, Rengsdorf, Germany) with 12-mm cup size. APTs were performed with native egg whites and yolks. The occlusion time of APTs was 48 hours, the first results were evaluated 30 minutes after removal of the tests, and the second results were analyzed 72 hours after application of the tests. Grading of positive APT reactions was similar to the criteria used in conventional contact allergy patch testing, with the modifications of the European task Force on Atopic Dermatitis (EFTAD) Consensus Meetings.¹⁹ Only reactions from erythema, infiltration onwards were designated positive.

Recommendation of performing the OET with egg after the diagnostic hypoallergenic diet

The OET with egg after the diagnostic hypoallergenic diet was recommended to be performed in patients with suspicion of egg allergy according to the positive results in sIgE, SPT, and APT to egg whites or yolks.

The diagnostic hypoallergenic diet

The diagnostic hypoallergenic diet was introduced following the patient's informed consent. We recommended the following foods to the patient for a period of four weeks: gluten-free

foods; potatoes; rice; meat – beef, pork; vegetables; and fruits only after thermal modification; but parsley, celery, and seasoning were not allowed. The patients were allowed to drink only drinking water, mineral water, or black tea.

Patients recorded symptoms of atopic eczema (the extent of involved skin, itching, sleep disorder) and other potential health problems in the special forms.

OETs

Consecutive OETs were performed after the elimination diet. This test was performed in intervals without symptoms or during a consistent period with regard to atopic eczema. When it was impossible to avoid steroids, the patient was allowed to treat himself with a low-potency topical corticosteroid. No other anti-inflammatory substances, antihistamines or UV therapy were applied.

OETs took two days with three doses of examined food in the interval of 12 hours, but one dose was divided into incremental dosages given over 80 minutes. The well-cooked egg was applied at 20-minutes intervals with incremental dosages – 50 mg, 500 mg, 5 g, 50 g, and 100 g. On the first day, the patient consumed a dose of egg at 08:00 hours, the reaction was observed, and in the case of no response, a second dose of food was consumed at 18:00 hours. If no response resulted, the last dose challenge was administered the next day at 08:00 hours. The patient also recorded the reaction during the 48 hours after the last dose. The skin was scored by the SCORAD system before the elimination diet, during the elimination diet, before OET, and then 24 and 48 hours after OET.

The food challenge results were scored as positive if one or more of the following objective and subjective clinical reactions were noted: urticaria, angioedema, vomiting, wheezing, abdominal pain, diarrhea, oral allergic syndrome, pruritus, erythema, rashes, or worsening of atopic eczema (evaluated with SCORAD). Early reactions were defined as clinical

symptoms within two hours after ingestion of the dose in OET and late symptoms if occurring after more than two hours.

In the case where the physician or the patient recorded worsening of atopic eczema or some other reactions during the test with egg, the patient continued in the elimination of egg, and the severity of atopic eczema was evaluated every three months for one year.

If the test with egg was negative, the patient introduced egg in the diet regimen. The severity of atopic eczema was evaluated during the average daily intake of food over a period of three months for one year.

Determination of allergy to egg

1 Patients with a positive result in the OET with egg and with a positive result in at least one of the diagnostic methods (sIgE, APT, SPT) were considered to have an egg allergy.

2 Patients with an early reaction after the ingestion of an egg in their history from their childhood and with a positive result in at least one of the diagnostic methods (sIgE, APT, SPT). The OET was not performed in these patients because they could suffer from severe allergic reaction.

Results

Patients

Altogether 179 persons suffering from atopic eczema were included in the study: 51 men and 128 women entered the study with an average age of 26.2 years (SD 9.5 years); min. 14 years, max. 63 years; with the median SCORAD 31.6 points, SD 13.3 (max. 79.5 points; min. 12.5 points) at the beginning of the study.

Clinical outcomes of challenges

All 179 patients were subjected to SPTs, APTs, sIgE, to egg whites and yolks. A positive result in at least one of

Table 1 Patients divided according to the results of OET, and according to the results of APT, SPT and sIgE

Result of OET	Number of patients	Number of positive SPT	Number of positive sIgE	Number of positive APT
OET positive	7	Yolk 1 White 2	Yolk 5 White 2	Yolk 3 White 1
OET not performed because of early allergic reaction	4	Yolk 4 White 4	Yolk 3 White 4	Yolk 0 White 0
OET negative (but elimination of egg)	4	Yolk 0 White 2	Yolk 1 White 3	Yolk 0 White 0
OET negative	46	Yolk 8 White 11	Yolk 25 White 9	Yolk 1 White 3
Whole	61	Yolk 13 White 19	Yolk 34 White 18	Yolk 4 White 4

Table 2 Patients with egg allergy (11 out of 179) according to the positive results in OET and according to history with early allergic reaction

Patient	History – egg allergy	SPT	sIgE yolk IU/ml	White IU/ml	APT yolk/white	AB	OET early reaction	OET late reaction	SCORAD I	SCORAD II
1	–	–	8.10	–	–	–	Pruritus, rashes, erythema	Exacerbation of eczema	20...15	9...12...9
2	–	–	2.15	0.83	–	–	–	Exacerbation of eczema	48...40	32...30...31
3	OAS	Yolk white	–	2.37	–	+	n.d.	n.d.	47...39	35...37...32
4	–	White	8.03	4.09	–	+	Pruritus, diarrhea, erythema	Exacerbation of eczema	54...34	18...20...15
5	Breathless	Yolk white	0.6	0.89	–	–	n.d.	n.d.	23...15	23...25...20
6	–	–	–	–	++/–	+	–	Exacerbation of eczema	20...12	9...11...10
7	OAS	White yolk	0.59	0.74	–	–	n.d.	n.d.	33...32	31...30...32
8	OAS	White yolk	1.05	1.3	–	–	n.d.	n.d.	44...30	30...35...37
9	–	White yolk	7.38	–	++/–	+	Pruritus, rashes	Exacerbation of eczema	40...15	17...20...19
10	–	–	0.77	1.10	–	+	Pruritus, OAS	–	55...40	43...45...48
11	–	–	–	–	++/+	+	–	Exacerbation of eczema	33...20	15...10...11

SCORAD I – before diagnostic hypoallergenic diet and at the end of this diet before OET with egg.

SCORAD II – after elimination of egg and egg-containing products for 3, 6, 9 months in patients with positive results in OET or in patients with positive history, who eliminated egg for a long time because of early allergic reaction.

n.d. – not done because of severe allergic reaction danger.

APT – evaluation of the results according to EFTAD: – negative results; ? erythema; + erythema, infiltration; ++ erythema, infiltration, papules (up to 3) The reactions evaluated only as + and ++ were recorded in our study, no stronger reactions were recorded. OAS – oral allergy syndrome AB – asthma bronchiale.

these methods was recorded in 61 patients; altogether 92 reactions. The OET with egg was recommended to be performed in patients with positive results. The OET with egg was performed in 57 patients; in four patients it was not performed because of an early allergic reaction after ingestion of an egg in their history. Negative results in APT, sIgE, and SPT to egg whites and yolks were recorded in 118 patients; the OET with egg was not performed in these patients.

A positive result in OET with egg was recorded in seven patients (4%); OET with egg was negative in 50 patients (28%).

Table 1 shows positive results of examinations to egg whites and yolks in SPT, sIgE, and APT with comparison to the confirmed food allergy in the OET.

Sixty-one patients with suspicion of egg allergy according to the results of APT, SPT, and sIgE are divided according to the results in OET and are shown in Table 1.

1 Positive results in OET – seven patients (4%).

2 OET not performed because of early allergic reaction – four patients (2%).

3 Negative results in OET in patients who eliminated egg – four patients (2%).

4 Negative results in OET, but positive results in sIgE, SPT or APT – 46 patients (26%).

Table 2 shows 11 patients with an egg allergy (seven patients with positive results in OET and four patients

with early reaction in their history after ingestion of an egg). The results of the examination in sIgE, APT, and SPT to egg whites and yolks, the reaction in OET and the occurrence of asthma bronchiale are mentioned here. SCORAD I is recorded before the elimination diet and at the end of this diet before the OET. SCORAD II is recorded after elimination of egg every three months for 1–3 years in these patients with confirmed food allergy to egg.

Early skin reaction as rashes, erythema, and pruritus was recorded in patients no. 1, 4, and 9, one hour after the ingestion of the first dose. These patients continued with exposition, and the same early reaction appeared after the ingestion of the second and third dose. Oral allergic syndrome and pruritus of the skin was recorded in patient no. 10 during the first dose in OET. Late reaction as new eczematous papules with exacerbation of atopic eczema was seen in patients no. 1, 4, and 9, for 7–10 hours after ingestion of each dose. A positive result in sIgE was recorded in all these patients, in one of them there was also a positive result in APT (no. 9). A late reaction on the skin (pruritus, eczematous papules with exacerbation of atopic eczema for 6–10 hours after ingestion of the first dose in OET) was only seen in three other patients (no. 2, 6, and 11). The same reaction, but more intensive, was recorded in all of them after ingestion of the second and third dose in OET for 6–10 hours. A positive result in APT was recorded in two of these patients without positive results in sIgE or

SPT; in one patient a positive result in sIgE to egg whites and yolks was recorded.

OET was not performed in four patients (no. 3, 5, 7, and 8) because of anaphylactic reaction danger. All these patients have rigorously eliminated egg from meals for a long time; three of them described oral allergic syndrome, and one patient suffers from breathlessness after ingestion of an egg. Positive results to egg whites and yolks in sIgE and SPT were recorded in all patients; APTs were negative in these patients.

The severity of atopic eczema in patients with positive results in OET to egg was evaluated after elimination of egg for 3 months for 1–3 years; the skin improved in six patients (3.3%). These patients also suffered from exacerbations of atopic eczema, but this was a consequence of other triggering factors, as they described (stress, inhalant allergens, pollen-associated foods). These patients eliminated egg from their meals, and during a mild breaking of the diet regimen they observed, after the ingestion of egg or egg-containing products, reactions on the skin, such as pruritus, erythema and new eczematous lesions.

Elimination of egg did not influence the severity of atopic eczema in patient no. 10 with early reaction in OET, and also did not influence the severity of atopic eczema in another four patients (no. 3, 5, 7 and 8) with a history of egg allergy.

The severity of atopic eczema and the occurrence of other allergic reactions were evaluated in patients with negative results in OET, but with positive results in sIgE, SPT or APT to egg whites and yolks after introducing egg in a common meal. No reactions were observed in these patients after ingestion of egg and egg-containing products.

Statistics

The probability that patients with a suspicion of food allergy according to the results of the examinations will in reality have clinical symptoms is $11/61 = 18\%$.

The sensitivity of examinations in sIgE, SPT and APT in patients with early reactions after ingestion of an egg in their history and in patients with a positive result in OET was calculated. The high sensitivity (100%) for egg whites and yolks in sIgE and SPT was recorded for early reactions. On the contrary, in the cases of late reactions with worsening of atopic eczema was sensitivity of SPT to egg yolks (4%) and whites (28%); sIgE to yolk (71%) and white (28%); and APT to yolk (42%) and white (14%).

Discussion

Atopic eczema is a chronic intermittent inflammatory skin disease that may in some cases be associated with food

allergies. Other numerous trigger factors for atopic eczema have been identified, including inhalable respiratory allergens, irritative substances and infectious microorganisms, such as *Staphylococcus aureus*. Psychogenic and climatic factors may also cause exacerbation of atopic eczema.

The epidermal barrier plays a crucial role in protecting the body against infection and other exogenous insults through both minimizing transepidermal water loss and conferring immunological protection.²⁰ Filaggrin gene defects substantially increase the risk of atopic eczema. The increased skin permeability may increase the risk of sensitization to food and other allergens, this pointing to the possible role of cutaneous allergen avoidance in early life to prevent the onset of atopic eczema and food allergy.²⁰

Food allergy appears to be an important factor in exacerbation of atopic eczema in only a subset of children (and a much smaller subset of adults) where non-IgE mediated mechanisms are sometimes implicated in atopic eczema flare-ups associated with ingestion of the foods in question, and that food allergy appears to have little or no role in children with non-atopic eczema.^{21–23} According to a questionnaire survey of schoolchildren's perceptions in dealing with factors influencing atopic eczema,²⁴ an extremely small proportion (8%) of atopic eczema respondents believed certain foods or drinks have an effect on their eczema. A survey of adults in High Wycombe showed that 20% perceived adverse reactions to specific foods, but only 1% had confirmed food allergies.²⁵ According to Sicherer, both atopic eczema and food allergy are, in the majority of individuals, transitory conditions that improve with increasing age.²⁶

In patients with suspected food allergy, that is thought to be aggravating eczema, it is therefore important that any food avoidance measures suggested are based on a detailed history and supportive evidence from objective allergy tests. The key here is the role of SPT and measure of specific IgE. Patch testing may have a role in those in whom delayed cell-mediated hypersensitivity reactions are thought to be implicated.²⁷ In cases of diagnostic doubt, an oral food challenge may be performed. In this provocation test, early clinical responses are those reactions observed within 2 hours after the last administered dose; late-phase reactions are those occurring between 2 and 48 hours after the last administered dose. Food challenges can cause three different patterns of clinical reactions to foods in patients with atopic eczema:²³ (1) Non-eczematous reactions: immediate-type reactions. Clinical symptoms include cutaneous symptoms, such as pruritus, urticaria, and rashes, and/or non-cutaneous gastrointestinal or respiratory symptoms or even anaphylaxis. (2) Isolated eczematous reactions: late reactions, e.g. flare-up after hours to days. (3) A combination of non-eczematous

and eczematous reactions. In some previous publications, the eczematous reactions were defined as late (i.e. by time) or delayed (i.e. by mechanism) reactions.²⁸

In order to distinguish eczematous responses clearly from early cutaneous reactions, the suspected food should be given over a period of two days. This provocation scheme was recommended for the detection of "true" eczematous late-phase reactions by the German Society of Allergology and Clinical Immunology.²⁹ The skin must be scored by an established eczema score (SCORAD) before OET and at least after 24 hours. It is not sufficient to score eczema just before and immediately after OET, as eczematous reactions will be missed in many cases. A difference of at least 10 SCORAD points is usually considered a positive reaction. OET should always be performed by persons with experience in OET, and emergency equipment must be available at all times.

Egg allergy is one of the most common food allergies in infants and young children and is closely associated with atopic eczema. According to the results of the Savage study,¹⁵ egg allergy is generally considered to have a good prognosis, and parents are typically counseled that their children will outgrow the allergy by early school-age years. Results from this analysis of more than 850 patients with IgE-mediated egg allergy support the idea that the majority of children with egg allergy will develop tolerance over time. However, the data suggest that this does not happen as early as previously thought. Furthermore, they have identified an egg IgE level ≥ 50 kU/l as a marker of persistent egg allergy. They found that 4% developed tolerance by age 4 years, 12% by 6 years, 37% by 10 years, and 68% by 16 years.

Five major allergens have been characterized in hen's eggs, which are designated Gal d 1–5.¹ Ovomucoid-Gal d 1, a 28-kDa glycoprotein comprising 186 amino acids, has been shown to be the immunodominant protein in egg white. Chicken serum albumin (livetin, Gal d 5) is the major allergen in egg yolk and is thought to be involved in the pathogenesis of bird-egg syndrome.^{30–32}

Food allergy to egg was confirmed in our study in 11 patients out of 179 (6%). Of these patients, only six (3.3%) had a clear improvement of the SCORAD after elimination of egg (patients 1, 2, 4, 6, 9, 11), a very low number. Our results have shown that the majority of patients with positive results in SPT and sIgE and a negative result in OET (28% in our study) are only sensitized to egg whites or yolks, without clinical symptoms.

For a comparison of our results, there are no studies dealing with the same question in adolescents and adults, patients with atopic eczema. In children, some authors correlated allergen-specific serum IgE levels with challenge outcomes.^{33,34} Benhamou³⁵ found that egg-specific IgE levels increased significantly in concordance with the

severity of allergic reactions. Decision points for specific IgE levels were not established for isolated eczematous reactions yet. In our study, a level of specific IgE from 0.6 to 2.37 IU/ml was recorded in four patients with early reaction in their history. On the contrary, a level of specific IgE from 7.38 to 8.01 IU/ml was recorded in patients without anamnestic suspicion of allergy to egg but with a positive result in OET. In patients with negative OET results to egg, we recorded serum specific IgE positivity to yolk in 26 patients (14%) and to white in 12 patients (6%). These patients introduced egg in a common meal without the occurrence of early or late allergic reactions. The average level of specific IgE in these patients was: to egg yolk – 3.3 IU/ml (SD 2.4); to egg white – 1.06 IU/ml (SD 0.2).

SPTs in patients with a negative result in OET with egg were positive in eight (4%) patients to yolk and in 13 (7%) to white. These patients also introduced egg in a common meal without the occurrence of early or late allergic reaction. All these reactions in SPT were recorded as mild. In children, Knight *et al.*³⁶ retrospectively analyzed the diagnostic utility of SPT in 74 children and concluded that an egg white SPT wheal diameter of 3 mm corresponded to a 50% probability of tolerating egg.

The evaluation of a possible food allergy in the patient's history is an important step in the diagnostic work-up of food allergy. Especially important is the statement about early reaction after the ingestion of some food in the patient's history. But the late eczematous reaction on the skin in patients with a persistent moderate or severe form of atopic eczema can be observed with difficulty. The worsening of atopic eczema that occurs several hours after ingestion of food allergen and other factors can contribute to the deterioration of atopic eczema as well at the same time. Patch testing may have a role in those in whom delayed cell-mediated hypersensitivity reactions are thought to be involved. In our study, the positive reactions in APT to yolk were the only positive reactions in two young women suffering from a moderate form of atopic eczema. The OET confirmed food allergy to egg in both of them. These patients eliminated egg from meals and observed an improvement of atopic eczema. They suffer from exacerbations of atopic eczema, but the main triggering factor is stress. The APTs were a valuable tool in the diagnosis of food allergy to egg in these two patients without positive sIgE and SPT. The positive result in APT together with positive results in SPT and sIgE were recorded in another patient with a confirmed egg allergy in OET. The APT is a skin test that aims to elicit cutaneous cell-mediated immune responses after prolonged (48 hours) skin contact with food allergens.³⁷ Clinical applications of the APT in patients with immediate egg allergy are limited. However, APT may

have a role in defining non-IgE-mediated cutaneous manifestations to egg in SPT-negative patients with eczema flares after ingestion of egg-containing foods or breast milk.¹⁶ APT may also be helpful in delineating underlying food allergies in patients with eosinophilic esophagitis.³⁸

The interesting case in our study was a 58-year-old woman (no. 4) with late onset of atopic eczema and with a confirmed food allergy to egg in OET. She had been suffering from allergic asthma bronchiale from early childhood and from acute allergic rhinitis and shortness of breath in contact with birds, such as a canary or parrot, and animals, such as a horse, a cat or a dog. This case was concluded according to all results of examinations as a bird-egg syndrome. This syndrome is related to the cross-reactive IgE antibody able to bind the yolk livetin and bird feather allergens.^{30–32} The initial symptoms are usually asthma with or without rhinoconjunctivitis due to contact with birds. Individuals first become sensitized to bird proteins (feathers, excrements, serum, meat) and subsequently develop an egg allergy.

Although the unequivocal diagnosis of egg allergy requires a DBPCFC,¹⁷ open food challenges, which are less resource-intensive, are generally considered sufficient in clinical practice. The European Academy of Allergology and Clinical Immunology proposed a starting dose of 1 mg of egg protein, followed by doubling the dose every 15–30 minutes.³⁹ Some centers recommend against the use of uncooked egg because of the risk of contamination with enteric pathogens.⁴⁰

Conclusion

Egg allergy may play an important role in the worsening of atopic eczema (acting as a triggering-exacerbating factor) only in a minority of adolescents and adult patients.

Moreover, other triggering factors may cause exacerbation of the atopic eczema in the patients enrolled in the study. The majority of patients with positive sIgE and SPT are only sensitized to egg without clinical reactions. ATPs were a useful tool in the diagnosis of food allergy to egg in patients without IgE reactivity. The diagnostic work-up may comprise the challenge tests to confirm the food allergy to egg in patients without severe allergic reaction in their history.

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