CASE REPORT

ATOPY PATCH TEST IN DIAGNOSIS OF FOOD ALLERGY TO EGG IN ADULT PATIENTS SUFFERING FROM ATOPIC ECZEMA. THREE CASE REPORTS

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Introduction

Food allergy and atopic eczema (AE) may occur in the same patient. Besides typical immediate types of allergic reactions (i.e. noneczematous reactions) which are observed in patients suffering from AE, can foods, such as cow's milk and hen's eggs directly provoke flares of AE, particularly in sensitized infants (25).

The role of food allergy remains controversial in adolescents and adult patients suffering from AE, few studies concerning the food allergy in this group of patients are available. The importance of food allergens for eczematous reactions in atopic eczema with a diagnostic algorithm for the elucidation especially of late reactions to foods was given in the position paper of the EAACI and GA2LEN (25). Eczematous reactions to foods can appear isolated or associated with preceding other symptoms.

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), challenge tests (open exposure test – OET, double – blind, placebo – controlled food challenge test – DBPCFC). DBPCFC always remains the golden standard in diagnosis of food allergy (17, 25). The atopy patch test is a skin test that aims to elicit cutaneous cell-mediated immune responses after prolonged (48 h) skin contact with food allergens. According to the ETFAD/EADV eczema task force 2009 position paper on diagnosis and treatment of AE atopy patch tests are performed with self-made food material applied to the back with large test chambers for 48–72 h. Food APT is not standardized for routine use (7). Whereas immediate – type food reactions are associated with SPT positivity; delayed ones are related to positive responses to APTs. However, food challenge is not replaced by patch testing (15).

A general problem is that the APTs with food have mostly been studied in infants and children since food allergy plays a role especially in this age group.

The diagnostic work – up of food allergy in adolescents and adult patients suffering from atopic ecezma is demonstrated in this report. The diagnosis of atopic eczema was confirmed with the Hanifin-Rajka criteria (9) in all these patients at the outpatient department of Dermatology and Venereology, Faculty Hospital and Medical Faculty of Charles University, Hradec Králové, Czech Republic.

The description of the case

Patient No I

The patient, a 19 year old woman, was examined at the Department of Dermatology and Venereology, Faculty Hospital and Medical Faculty of Charles University, Hradec Králové, Czech Republic in September 2005. The moderate form of atopic eczema with frequent exacerbations was the reason for examination.

History

Family history: parents are healthy, no allergy and no signs of atopy in her family.

Personal history: asthma bronchiale from childhood, no other disease.

Allergy: Penicillin – toxoallergic exanthema.

Social: student; contact with animal: cat and dog in the flat.

Medications: salmeteroli xinafoas (Seretide dis aer 50/250) 2x1 breath, salbutamoli sulfas (Ecosal aer) 1x breath, desloratadinum (Aerius tabl) 1x per day.

Allergology: The patient was examined and followed up for moderate perrenial form of asthma bronchiale at out – patient allergological department from early childhood, but no positive results was found in sIgE and SPT for common food and aeroallergens. The patient did not suffer from rhinitis.

Dermatology: The patient suffered from moderate form of atopic eczema from early childhood with exacerbations at predilection localisation with erythema and papules exanthema. The diagnosis of atopic eczema was made with the Hanifin-Rajka criteria in the dermatological out – patient department. The therapy of atopic eczema during last

years consisted of systemic treatment – antihistamines, topical treatment – corticosteroid therapy, topical antibiotic therapy, indiferent therapy with emollients. The worsening of atopic eczema was observed during last one year. Because of frequent exacerbations with impetiginisation the patient was recommended to the detailed examination for diagnostic work – up of food allergy to the Department of Dermatology, Faculty Hospital, Hradec Králové, Czech republic. This examination started in the period with milder symptoms of atopic eczema in November 2005 with the SCORAD of 34 points.

Results of examinations

Personal history: the patient observed pruritus of the skin after seasoned meal.

The examination of total and specific IgE antibodies: The serum level of the specific IgE to the tested foods has been measured with the method of FEIA (Pharmacia CAP system, Uppsala, Sweden). The level of specific IgE higher than 0.35 kU/l was assessed as positive.

Results: total IgE 75 IU/ml, specific IgE was negative to these foods: egg‘s white, yolk, milk, cod, wheat flour, soy, nuts, celery, carp, mustard

Atopy patch tests: Atopy patch tests were performed on non – lesional, non – abraded, untreated skin of the back during a remission.

A technique similar to conventional patch tests was used by performing atopy patch testing – CURATEST (Lohmann Rauscher International GmbH Co. Germany) test with a 12 mm large cup size. After discontinuation of antihistamines and topical steroids for at least 5 days and UV phototherapy for at least 2 months, the atopy patch tests with native foods (egg‘s white, yolk, cow‘s milk, wheat, soy, peanuts) have been applied. Wheat powder and soy powder were used with vehicle – distillated water (1 g of wheat powder or soy powder in 10 ml of distillated water), egg‘s white, yolk, cow‘s milk and peanuts were used in the native form as the fresh foods without any dilution. Single vehicle (distillated water) has been used as a negative control. The occlusion time of atopy patch test was 48 hours, the first results were evaluated 30 min after the removal of the tests and the second results were analysed 72 hours after the application of the tests.

Grading of positive APT reactions was according to the reccomendation of the European task Force on Atopic Dermatitis (EFTAD) Consensus Meetings (6):

?	erythema
+	erythema, infiltration
++	erythema, infiltration, papules (up to 3)
+++	erythema, papules from 4 to many
++++	erythema, many or spreading papules
+++++	erythema, vesicles.

Only reactions from + (erythema, infiltration) onwards were designated positive.

Results: positive results to egg‘s white and yolk in 48 and 72 hours – evaluated as +++, other foods negative.

Skin prick tests: Commercial food extracts Alyostal (Stallergens, France) were used for skin prick tests.

Results: negative reaction to these foods: milk, egg‘s white, yolk, seafish, wheat flour, soy, nuts, celery, carp.

The diagnostic hypoallergenic diet

In the period of four weeks we recommended to the patient the diagnostic hypoallergenic diet. This diet consist from gluten free foods, potatoes, rice, meat – beef, pork, vegetable, and fruits only after thermal modification, but parsley, celery, and seasoning were not allowed. The patient was allowed to drink only drinking water, mineral water, or black tea.

The skin finding improved in the course of such a diet. The patient was without acute skin eczematous lesions, but the postinflammatory red macules in previous numular lesions of atopic eczema lasted. The patient was allowed to treat himself with a low potency topical corticosteroid. No other anti-inflammatory substances, anti-histamines nor UV-therapy were applied.

Consecutively after the elimination diet open exposure tests (OET) was performed with egg. OET took two days with three doses of examined food in the interval of 12 hours also, but one dose was divided into incremental dosages given during 80 minutes. The well – cooked egg was applied at 20 – minute intervals with incremental dosages – 50 mg, 500 mg, 5 g, 50 g, and 100 g. The patient recorded worsening of atopic eczema such as redness and new eczematous papules seven hours after the administration of the first dose of food and this late reaction repeated after the second and third dose, but the papules and redness were more intensive. The open exposure test with the egg was concluded as positive and according to the result the patient was advised to avoid the ingestion of eggs and food products containing eggs. Double – blind, placebo – controlled food challenge test was not performed.

The skin finding is better in this patient for a long time. She has been suffering only from some postinflammatory lesions at the predilection localisation and from dry skin. During the first year of diet the patient was checked every three months, the severity of atopic eczema was evaluated with SCORAD. SCORAD decreased from 34 points to 15 points during the diagnostic hypoallergenic diet. The level of SCORAD 10 points is recorded during the last three years also. During the breaking of the diet regimen the patient observes regularly the worsening of atopic eczema with late eczematous reaction. The patient has been examined at allergologist for asthma bronchiale, but the severity of asthma bronchiale didn’t change after the elimination of an egg. In last one year the patient suffers only from dry skin and she is without any eczematic lesions.

Patient No II

The description of the case

The patient, a 23 year old woman, was examined at the Department of Dermatology and Venereology, Faculty

Hospital and Medical Faculty of Charles University, Hradec Králové, Czech Republic in May 2006. The moderate form of atopic eczema with frequent exacerbations was the reason for examination.

History

Family history: parents are healthy without any allergic or dermatologic diseases, asthma bronchiale in her mother's cousin.

Personal history: healthy, pneumonia in 5 years of age.

Social: teacher.

Medications: hormonal contraception.

Allergology: The patient was examined for the first time at outpatient allergologist department for acute severe worsening of atopic eczema at March 2006. Spirometry examination – the incipient asthma bronchiale was recorded. The therapy with Alvesco 160 mg 1x1 and desloratadinum (Aerius tabl 1x per day) was introduced. The patient has not been suffering from rhinitis. *Skin prick test* was not performed because of severe form of atopic eczema on arms. Total IgE 47 IU/ml, *specific IgE* with negative results for the pollen of grass, pollen of trees, for moulds, Dermatophagoides, fur of cat, dog, rabbit, feathers, poppy sead, spices, egg's white, yolk, milk, seafish, wheat flour, soy, nuts, celery, and carp.

Dermatology: The patient suffered from moderate form of atopic eczema from infancy till 5 years of age. Then she suffered only from mild form till 14 years of age, from this time she has been suffering from moderate form with exacerbations of AE at predilection localisation – face, neck and flexor parts of the body. The therapy of atopic eczema during these last years consisted in systemic treatment – antihistamines, topical treatment – corticosteroid therapy, local antibiotic therapy, indiferent therapy with emollients. Because of frequent exacerbations of atopic eczema in the last 6 months the patient was recommended from allergologist to the Department of Dermatology, Faculty Hospital, Hradec Králové to the detailed examination for severe form of atopic eczema. SCORAD was 51 points at the first examination in May 2006. The patient suffered from erythematous lesions in the face, neck, trunk, in flexor part of arms and legs with lichenification and erosions.

In the systemic therapy the treatment for asthma bronchiale continued without changes and the systemic treatment with antihistaminic medication (desloratadinum, Aerius tabl 1x per day) continued as well.The topical corticosteroid therapy with topical antibiotics and with emolients was used.The skin finding improved during this therapy. Because the patient has recorded the worsening of atopic eczema after ingestion of kiwi and currants, we started the diagnostic work – up of food allergy as the possible cause of the disease. This examination was performed in intervals with milder symptoms of atopic eczema – SCORAD was 33 points. We decided not to repeat the allergological examination from March 2006, but only to complet it with performing of atopy patch testing with food allergens. After discontinuation of antihistamines and topical steroids for at least 5 days the *atopy patch tests* with common food allergens were performed in the same way as described in the patient No I. Results of atopy patch tests: positivity to yolk in 48 and 72 hours as ++, other foods negative.

The same diagnostic hypoallergenic diet was recommended to the patient as well. The skin finding improved in the course of such a diet.

Consecutively after the elimination diet OET was performed with egg in the same way as in previous case. The patient recorded also worsening of atopic eczema such as an erythema, eczematous papules at flexor parts five hours after the administration of the first dose. Pruritus as early reaction appeared after the ingestion of the second and third dose and late reactions appeared after seven hours more intensively then after the administration of the first dose. The open exposure test with the eggs was concluded as positive and according to the result the patient was advised to avoid the ingestion of eggs and food products containing eggs. Double blind placebo controlled food challenge test with egg was not performed. Kiwi and currants were not examined in the diagnostic work-up of food allergy, the patient eliminated them for a long time. The clinical symptoms after ingestion of kiwi and currants were concluded as food hypersensitivity.

The skin finding is better in this patient for a long time. She suffers from some exacerbations of atopic eczema, but the main triggering factor is stress. During the first year of diet the patient was checked every two months, the severity of atopic eczema was evaluated with SCORAD. SCORAD decreased from 51 points to 20 points during the elimination of an egg. The level of SCORAD 20–22 points is recorded every three months during the last three years also. During the breaking of the diet regimen the patient can observe regularly pruritus and the worsening of atopic eczema.

Patient No III

The patient, a 17 years old boy, was examinedat at the Department of Dermatology and Venereology, Faculty Hospital and Medical Faculty of Charles University, Hradec Králové, Czech Republic in Novembre 2008. The severe form of atopic eczema with impetiginisation was the reason for examination.

History

Family history: parents are healthy, no atopic disease at family.

Personal history: healthy.

Social: student.

Medications: desloratadinum (Aerius tabl) 1x per day, salmeteroli xinafoas (Seretide 50/250) 2x breath per day.

Allergology: The patient was followed up for the perrenial asthma bronchiale and for seasonal pollinosis at

outpatient allergologist department from childhood. The allergy to pollen of grass in skin prick test was recorded.

Dermatology: The patient had been suffering from mild form of atopic eczema from two till 15 years of age, it was evaluated as mild form of atopic eczema at out – patient dermatologist department. The worsening of eczema occured from 15 years of age. Because of frequent exacerbations of atopic eczema with impetiginisation, the patient was recommended to be examined at Department of Dermatology and Venereology, Faculty Hospital and Medical Faculty of Charles University, Hradec Králové. Because of severe form of atopic eczema the patient was admitted to hospitalisation in November 2008. The skin of the patient was affected with eczematic lesions, in flexural parts with the sings of impetiginisation. The severity of atopic eczema was evaluated with SCORAD index, SCORAD was 58 points. Internal and neurological examinations were without pathological findings.The hematological and biochemical examinations were all right. The systemic treatment with antihistaminic medication continued. The topical corticosteroid therapy with topical antibiotics and with emolients was used. The skin finding improved during hospitalisation, but some papules and redness in flexural part outlasted.

We started the diagnostic work – up of food allergy as the possible cause of such a torpid course of the disease in last two years. This examination was performed in intervals with milder symptoms of atopic eczema. After discontinuation of antihistamines and topical steroids for at least 5 days the skin prick tests, the atopy patch tests, and the challenge tests were performed.

Results of examinations

Personal history: the patient observed the worsening of atopic eczema after ingestion of citruses and after the sport activity.

The examination of total and specific IgE antibodies:

The serum level of the specific IgE to the tested foods has been measured with the method of FEIA (Pharmacia CAP system, Uppsala, Sweden). The level of specific IgE higher than 0.35 kU/l was assessed as positive.

Results: total IgE 2 574 IU/ml, specific IgE was positive to peanuts 0,79 U/ml, soy 0,74 U/ml, egg‘s yolk 7, 28 U/ml, specific IgE was negative to these foods: egg‘s white, milk, wheat flour.

The specific IgE for some aeroallergens was completed: specific IgE was positive for house dust 8,84 U/ml, Dermathophagoides pteronyssinus 0,73 U/ml, Dermathophagoides farinae 0,59 U/ml, early grass 21, 38 U/ml, late grass 29, 2 U/ml, negative specific IgE was to hay and feathers

Skin prick tests: Commercial food extracts Alyostal (Stallergens, France) were used for skin prick tests.

Results: positive reaction to egg‘s white and yolk negative reaction to to these foods: milk, wheat flour, soy, nuts, celery, carp.

Atopy patch tests: positive results to egg‘s white and yolk in 48 and 72 hours – evaluated as +++, other foods negative.

The same diagnostic hypoallergenic diet was recommended to the patient. The skin finding improved in the course of such a diet.

OET was performed with egg in the same way as in previous cases consecutively after the elimination diet. The patient recorded after ingestion of the first dose of an egg pruritus during 1 hour and new papules and erythema appeared on arms and legs during other 5 hours. Pruritus appeared after the ingestion of the second and third dose and late reactions repeated as well more intensively. The open exposure test with the egg was concluded as positive and according to the result the patient was advised to avoid the ingestion of eggs and food products containing eggs. Double blind placebo controlled food challenge test was not performed.

Because of pollen allergy it was recommended to perform to open exposure test with some common kinds of fruits and vegetables to evaluate the cross- allergy. The patient observed reactions after ingestion of oranges and lemons, but it was concluded as food hypersensitivity.

The patient suffered from allergy to pollen of grass and to house dust and Dermathophagoides pteronyssinus. It was recommended to introduce some anti allergic arrangements at the home setting.

This patient suffers now from mild form of atopic eczema after elimination of an egg. He records less frequent exacerbations of atopic eczema. During the first year of elimination of an egg the patient was checked every two months, the severity of atopic eczema was evaluated with SCORAD. SCORAD decreased from 58 points to 24 points during the elimination of an egg. The level of SCORAD 25–28 points is recorded every three months during the last three years also. During the breaking of the diet regimen the patient describes regularly pruritus and new erythematous and papulous lesions on arms and legs.

Discussion

Food has been discussed as a trigger factor of AE for many years. A problem in the most published clinical evaluations of food allergy in AE is that eczema which usually worsens on the day after the oral food challenge or even later was not scored systematically before and the day after oral food challenges (25). Eczematous reactions to foods can appear isolated or associated with preceding other symptoms. In some previous publications, those reactions were defined as late (i.e. by time) or delayed (i.e. by mechanism) reactions (26). In isolated eczematous reactions, it is often difficult to relate the clinical reaction to a relevant trigger factor that started to cause the reaction many hours before. Therefore, the identification of trigger factors of eczema in AE is usually more difficult than the elucidation of the cause of immediate symptoms in food allergy.

There is no single parameter that can prove the clinical relevance of a sensitizing food in patients with AE. A stepwise procedure addressing individual factors is recommended (25). In patients suffering from AE and in addition from immediate reactions to foods the responsible food can often be identified by taking a careful history, performing skin prick tests and/or determining food-specific IgE in vitro. Since specific IgE, prick tests and the history sometimes do not correlate with clinical observations, food challenges are necessary to show the clinical relevance of the findings. The placebo-controlled, double-blinded oral food challenge is the gold standard in the diagnostic procedure of food-associated reactions (1, 8, 17, 25). The patient's history can be very helpful to identify a relationship between the clinical symptoms and the ingested food. In particular it gives hints to IgE-mediated reactions. Unfortunately, the patient's history is not reliable in isolated eczematous reactions to food and particularly in case of multiple sensitizations to foods. If food allergy is expected tests to prove IgE-mediated sensitization (i.e. skin tests and /or in vitro-investigations – specific IgE in the serum) should be performed. It was suggested previously that decision points in which the predicted probability of a positive challenge outcome exceed, e.g. 95 % may make open exposure test unnecessary in selected children. At present, such a correlation has only been demonstrated in children and only for few foods (3, 5, 20, 21). However, no decision points have been established yet for eczematous reactions to foods. The APT can be considered as an additional diagnostic tool that can be used in specialized institutions. The APT with foods (cow's milk, hen's egg, cereals and peanut) may increase the identification of food allergy in patients with AE in the following cases (23, 24): 1) Suspicion of food allergy without predictive specific IgE levels or positive SPT, 2) Severe and/or persistent AE with unknown trigger factors, 3) Multiple IgE sensitizations without proven clinical relevance in patients with AE. However, APTs are still not generally recommended for routine diagnoses of food-induced eczema. Moreover, more series were studied only in patients of lower age groups (infants and young children with AE). It was concluded from a recent evaluation of a large number of children with AE that the APT does not lead to a significant reduction of the need of open exposure test when food-induced eczema is supposed (15).

Egg allergy is generally considered to have a good prognosis in children, and parents are counseled that their children will outgrow the allergy by the early school-age years. Egg-sensitized children have been shown to have more severe and persistent dermatitis, and the level of sensitization correlated with the severity of disease (11, 12). Delayed exacerbations of atopic dermatitis (>24 h after egg consumption) may occur in children without evidence of sensitization to egg, most likely because of T cell-mediated allergic reactions (4, 13, 16). Recent reports indicate that it is taking longer for children to outgrow their egg allergy, with most developing tolerance in their teenage years rather that in early school – age as previously thought (22). 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 age 6 years, 37 % by age 10 years, and 68 % by age 16 years (22).

Five major allergens have been characterized in hen's egg, which are designated Gal d 1–5 (10). Egg white contains several allergenic proteins, including ovomucoid (Gal d 1; 11 %), ovalbumin or conalbumin (Gal d 2, 55 %), ovotransferrin (Gal d 3, 12 %), lysozyme (Gal d 4, 3 %) and ovomucin (4 %) (23). Ovomucoid, a 28kDa glycoprotein comprising 186 amino acids, has been shown to be the immunodominant protein in egg white. Other egg white proteins, including ovotransferrin and lysozyme, appear to be less important in the pathogenesis of egg allergy. Chicken serum albumin (Gal d 5) is the major allergen in egg yolk and is thought to be involved in the pathogenesis of bird-egg syndrome (19).

The patient suffering from moderate or severe form of atopic eczema does not usually observe the late eczematous reaction on the skin during the common meals. Hen's egg is a versatile ingredient used in the cooking of many cultures, including a wide range of manufactured food products. Elimination and the exposition to egg in suspicion of egg allergy with eczematous reaction seem to be important part in the diagnostic work – up of this food allergy. Although the unequivocal diagnosis of egg allergy requires a double – blind, placebo – controlled food challenge (2), open food challenges, which are less resource – intensive, are generally considered sufficient in clinical practice.

We demonstrate three patients suffering from childhood from atopic eczema. Two of them have the low level of total IgE (75, resp. 47 U/ml) and no positive results were found in SPT or sIgE to common food and aeroallergens. Both patients suffer from asthma bronchiale, one of them from early childhood; asthma bronchiale was diagnosed during the first allergological examination in adulthood in the other patient. Moderate or severe persistent AE with unknown trigger factors were observed in both of them in last year before our examination. The classification in the intrinsic form of atopic eczema is likely difficult; both of them have been suffering from AE from childhood and one of them suffers from asthma bronchiale from early childhood as well. The intrinsic form affecting 20–30 % patients with AE, where non IgE mechanism is involved, is connected with the onset of AE after 20 years of age, with more often occurring positive reaction in atopy patch test and with lower production of cytokines Th2 (14). Extrinsic form involving 70 % to 80 % of patients is associated with

IgE-mediated sensitization to inhallant and food allergens and with increased production of cytokines Th2: Il 4, Il 5, Il 13. This kind of AE is linked with a greater incidence of allergic rhinitis and asthma bronchiale (18). The patient No III could be classed in this form of atopic eczema. The total level of IgE was 2 574 IU/ml. Multiple IgE sensitizations was recorded for aeroallergens in this case and the worsening of atopic eczema was considered as a result of this sensitizations. The examination for food allergy was beneficial in this case – even if the patient did not observe either the early or late reactions after the ingestion of an egg during the common meal, the positive results for egg's yolk was found in sIgE and for egg's white and yolk in SPT and APT. Open exposure test with egg confirmed the food allergy.

Atopy patch test with food allergens was a usefull tool in diagnosing of food allergy to egg in our patients. In all of them the delayed reactions appeared during open exposure test and also the early reactions appeared in the last one. The diagnosis of food allergy was confirmed during the three years period with elimination of an egg and during occasionally exposition at the breaking of diet regimen with the worsening of atopic eczema.

It is a question, if these young patients have been suffering from food allergy to egg from childhood. All these patients have the egg from childhood in their common meal, they did not observe any immediate reactions after the ingestion of an egg, but had been suffering from mild or moderate form of atopic eczema from childhood almost permanently. They did not keep any diet from childhood and they had not any suspicion of egg allergy from previous examinations at outpatient allergologist or dermatologist. But allergy to hen's egg is common in infancy and is closely associated with AE, particularly in infants who develop eczema in the first year of life (11). It is quite rare in adults and even in those cases clinical symptoms began in childhood or early adulthood. There are certain factors affecting the high incidence of food hypersensitivity in childhood such as a lack of immunological oral tolerance, gastric or intestinal enzymes, and immature gastrointestinal mucosa. Immunological barriers together with physiological barriers and undamaged intestinal mucosa are very important in protection from food allergy. On the other hand some studies have shown that physical or psychological stress may alter intestinal microflora, it could be hypothesized that stress might be responsible for disrupting oral tolerance to some antigens (27).

Conclusion

The examination of food allergy in adult patients suffering from atopic eczema may be beneficial. Atopy patch test with food allergens can be useful in the cases of moderate or severe persistent atopic eczema with unknown trigger factors. Egg allergy in adolescents and adults may affect their course of atopic eczema. Elimination of an egg, open exposure test and the long term follow-up of the severity of AE may confirm the role of food allergen in adolescent and adult patients suffering from AE.

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