suggest different cut off values with levels as low as 1.2 kU_A/l.[7]

IgE levels for egg white and yolk of our patient were low, while the wheals formed with the prick test and prick by prick test were significantly larger. We note that in our patient, SPT and serum Ig E measurement suggested opposite provocation test results.

Egg white is the major source of allergens in egg. Ovomucoid (Gal d 1), ovalbumin (Gal d 2), ovotransferrin/ conalbumin (Gal d 3) and lysozyme (Gal d 4) have been identified as the major allergens. Ovomucoid has been identified to be the most important egg protein clinically, presumably because of its ability to maintain allergenicity despite extensive heating. Lemon-Mule et al. suggest that extensive heating (e.g baked) diminishes the allergenicity of egg white protein and that subjects with egg allergy differ in their ability to mount IgE antibody responses against heated (denatured) egg white proteins[8] It has been hypothesised that the food body, consisting of fats, carbohydrates, and other proteins, may affect allergenic potential of allergens. For example, the fats may protect proteins during the digestion process, or they may influence the activation of immune cells, which may result in enhanced allergenicity of a protein.

Those studies could explain why our patient could not tolerate regular heated scrambled egg but she can eat egg products with a different food matrix and extensive heating, such as cookies. The ingestion of extensive heated egg product in our patient could contribute to the outgrowning of allergy to regular heated egg.

It is considered that the natural evolution of food allergy has good tolerance prognosis but the estimated exact probability of tolerance varies greatly between studies. Savage found that only 12% of children with egg allergy have tolerance at six years age[9] while another study indicates that 66% of children fail to develop tolerance until five years of age.[10] Our patient who ate egg presented with anaphylaxis and required emergency care at the age of six. We perform the oral provocation test and confirmed the tolerance of the patient at the age of eight, contrary to the predictive results of prick test.

We seek to show an unusual and severe case of egg allergy and that despite the great efforts being made to predict tolerance through Ig E levels or skin prick as to whether a patient can eat eggs or not, it is still under discussion when we can be safe. The oral challenge and medical history remain the gold standard tools for diagnosis and prognosis of patients with egg allergy.

References

1. Rona RJ, Keil T, Summers C, Gislason D, Zuidmeer L, Sodergren E, et al. The prevalence of food allergy: a meta-analysis. J Allergy Clin Immunol. 2007;120:638—46.
2. Palmer DJ, Gold MS, Makrides M. Effect of cooked and raw egg consumption on ovalbumin content of human milk: a randomized, double-blind, cross-over trial. Clin Exp Allergy. 2005;35:173—8.
3. Vance GH, Grimshaw KE, Briggs R, Lewis SA, Mullee MA, Thornton CA, et al. Serum ovalbumin-specific immunoglobulin G responses during pregnancy reflect maternal intake of dietary egg and relate to the development of allergy in early infancy. Clin Exp Allergy. 2004;34:1855—61.
4. Sporik R, Hill DJ, Hosking CS. Specificity of allergen skin testing in predicting positive open food challenges to milk, egg and peanut in children. Clin Exp Allergy. 2000;30:1540—6.
5. Sampson HA. Utility of food-specific IgE concentrations in predicting symptomatic food allergy. J Allergy Clin Immunol. 2001;107:891—6.
6. Ando H, Movérare R, Kondo Y, Tsuge I, Tanaka A, Borres MP, et al. Utility of ovomucoid-specific IgE concentrations in predicting symptomatic egg allergy. J Allergy Clin Immunol. 2008;122:583—8.
7. Crespo JF, Pascual C, Ferrer A, Burks AW, Diaz Pena JM, Martin Esteban M. Egg white-specific IgE level as a tolerance marker in the follow up of egg allergy. Allergy Proc. 1994;15:73—6.
8. Lemon-Mulé H, Sampson HA, SichererSH, Shreffler WG, Noone S, Nowak-Wegrzyn A. Immunologic changes in children with egg allergy ingesting extensively heated egg. J Allergy Clin Immunol. 2008;122, 977—83e1.
9. Savage JH, Matsui EC, Skripak JM, Wood RA. The natural history of egg allergy. J Allergy Clin Immunol. 2007;120:1413—7.
10. Boyano-Martínez T, García-Ara C, Díaz-Pena JM, Martín-Esteban M. Prediction of tolerance on the basis of quantification of egg white-specific IgE antibodies in children with egg allergy. J Allergy Clin Immunol. 2002;110:304—9.

J. Sanchez*, R. Ramírez, C. Chinchilla , R. Cardona

Unit of Alergology, University of Antioquia, Medellín Colombia

* Corresponding author.
E-mail address: jotamsc@yahoo.com (J. Sanchez).

doi:10.1016/j.aller.2010.05.005

Phytodermatitis caused by *Agave americana*

To the Editor:

Agave americana (Figure 1) is a popular ornamental plant. Irritant contact dermatitis induced by *Agave americana* has rarely been reported.

We present a case of allergic contact dermatitis associated with the use of this plant.

A 58 year-old woman developed an acute dermatitis on her face and neck after application of *Agave americana*. Due to confusion she had used this plant as a treatment for rheumatism instead of *Aloe vera*. A few minutes after using this plant she developed an intense pruritus on the contact zone. This area was intensive washed with water and soap. Twelve hours later she developed erythema, pruritus, oedema and suppurative lesions on her face and neck. The symptoms disappeared after treatment with systemic antihistamines and corticosteroids some weeks later.

Figure 1 Agave americana.

Patch testing was carried out according to the International Contact Dermatitis Research Group recommendations with a European Standard Battery and a European Plants Battery.

An open test (sap was applied on the wrist) with *Agave americana*, and then an open test with the plant sap in dilution (2 g was dissolved in 2 ml of saline solution, and 1/10 and 1/100 of this dilution) was carried out in the patient and in five controls.

The patch tests were positive at 48 and 96 hours to nickel sulphate and negative to any other allergens tested. The open test on the patient was intensive positive. A few minutes after the first contact on the wrist, she developed pruritus, erythema, papules and oedema. Treatment with occlusion topical corticosteroids was recommended. The open test on controls was mild positive; immediate pruritus with some late papules without pruritus were presented in two of them. The open test with the plant sap in dilution was negative in the patient and in the controls to all concentrations.

Agave americana belongs to the *Agaveceae* family[1] and is widely used in ornamental gardens in the Mediterranean area. The species of *Agave americana* have irritant properties due to the presence of calcium oxalate crystals (raphides),[2] volatile oil, saponins, agave gum and other components in its sap.[3] Calcium oxalate crystals seem to be the irritant main component.[4] In some case systemic symptoms have been described.[5]

Due to the mild irritant reaction observed in the controls, compared with the severe reaction in the patient we diagnosed this case as allergic contact dermatitis. Probably the previous contact causing sensitivity could have been caused when the patient pruned the plant.

Because of the onset of symptoms, which appeared in the morning and the patient had used the plant at night, photodermatitis was ruled out. A nickel sulphate contact allergy without present relevance was diagnosed.

Our patient had an important local reaction with open test. Due to the potential of plants to cause severe dermatitis, physicians should be careful with the use of plants for the diagnosis of phytodermatitis. To the best of our knowledge we recommend that in case of unknown products an open test must be the first procedure, and when this open test is negative, a patch test will be performed.

References

1. Segura JJ, Calzado Flores C, Del Valle Cantu J. Agavaceae, section IV, chapter 16. In: Dermatologic Botany, eds Avalos J, Maibach HI, crc Press, Boca Raton, 1999: 253—260.
2. Salinas NL, Ogura T, Soffuchi L. Irritant contact dermatitis caused by needle-like calcium oxalate crystals, raphides, in agave tequilana among workers in tequila distilleries and agave plantations. Contact Dermatitis. 2001;44:94—6.
3. High WA. Agave contact dermatitis. Am J Contact Dermat. 2003;14:213—4.
4. Brazelli V, Romano E, Balduzzi A, Borroni G. Acute irritant contact dermatitis from *Agave americana*. Contact Dermatitis. 1995;33:60—1.
5. Brenner S, LandauM, Goldberg I. Contact dermatitis with systemic symptoms from Agave Americana. Dermatology. 1998;196:408—11.

T. Linares[a,*], A. Fernández[b], E. Escudero[a], T. Soto[a]

[a] *Sección de Alergologia, Hospital Provincial de Pontevedra*
[b] *Servicio Aparato Digestivo, Hospital POVISA. Vigo, Spain*

* Corresponding author at: Sección de Alergología, Hospital Provincial de Pontevedra, C/ Doctor Loureiro Crespo 2, 36001 Pontevedra. Spain.
E-mail address: tania.linares.mata@sergas.es (T. Linares).

doi:10.1016/j.aller.2010.05.003