infant. Severe anaphylactic reaction is a medical emergency requiring immediate recognition and treatment, particularly in young infants. We present the case of a near fatal non IgE-mediated anaphylactic reaction due to cefotaxime in a 4-month-old infant. This case shows that it is very important to control the rate of administration of cephalosporins in very young infants.

References

1. Bock SA, Munoz-Furlong A, Sampson HA. Fatalities due to anaphylactic reactions to foods. J Allergy Clin Immunol. 2001;107:191–3.
2. Sampson HA, Munoz-Furlong A, Campbell ML, Adkinson Jr NF, Bock SA, Branum A, et al. Second symposium on the definition and Management of anaphylaxis: summary report – Second National Institute of Allergy and infectious Disease/Food Allergy and Anaphylaxis Network symposium. J Allergy Clin Immunol. 2006;117:391–7.
3. Moneret-Vautrin DA, Morisset M, Flabbee J, Beaudouin E, Kanny G. Epidemiology of life-threatening and lethal anaphylaxis: a review. Allergy. 2005;60:443–51.
4. Carder KR. Hypersensitivity reactions in neonates and infants. Dermatol Ther. 2005;18:160–75.
5. Baumgartner-Bonnevay C, Choquet-Kastylevsky G, Putet G, Bleyzac N, Vial T, Descotes J. Anaphylactic shock associated with ceftriaxone therapy in a newborn. Arch Pediatr. 2002;9:1050–2.
6. Bradley JS, Wassel RT, Lee L, Nambiar S. Intravenous Ceftriaxone and Calcium in the Neonate: Assessing the Risk for Cardiopulmonary Adverse Events. Pediatrics. 2009;123:609–13.
7. Soyer OU, Ozen C, Tiras U, Dallar Y. Anaphylaxis in a neonate caused by ceftazidime. Allergy. 2010;65:1486–7.

Á. Moreno-Ancillo[a,*], A.C. Gil-Adrados[b]

[a] *Servicio de Alergia. Hospital Nuestra Señora del Prado. Talavera de la Reina, Toledo, Spain*
[b] *Centro de Salud La Solana, Talavera de la Reina, Toledo, Spain*

* Corresponding author.
E-mail addresses: a.morenoancillo@gmail.com (Á. Moreno-Ancillo), alanaro@telefonica.net (A.C. Gil-Adrados).

doi:10.1016/j.aller.2011.02.011

Allergic contact dermatitis to cocamidopropyl betaine in Colombia

To the Editor,

Shampoos, soaps and intimate hygiene products have been considered infrequent causes of allergic contact dermatitis (ACD) because they are preparations eliminated with water and their permanence on the skin is very brief. Allergens usually contained have a low sensitising capacity due to their low concentration and brief contact. An exception to this rule is cocamidopropyl betaine (CAPB), a non-ionic tensoactive agent that has been a relatively frequent cause of ACD to shampoos and other products that are eliminated with water in Europe and the US in the last 20 years.[1] Currently, the advantages of synthetic detergent based products have gradually resulted in their greater popularity over common soaps. Recent studies in the US, Australia and Israel, suggest that CAPB allergy persists as a clinical problem, and that such compounds should be included among extracts used in standardised cutaneous patch tests.[2] Detergents in general contain tensoactive agents which are believed to decrease water's superficial tension. On the other hand, surfactants are classified by their ionic properties in water as anionic, cationic, non-ionic or amphoteric.[3] Amphoteric surfactants, of which betaine is the classical example, contain elements with both positive and negative charges within a same molecular structure, producing less irritant effects than those anionic tensoactive agents. CAPB is the main non-ionic tensoactive agent that contains ammonia and was originally introduced in personal hygiene products by Johnson & Johnson® in 1967 with the ''no more tears'' characteristic, mainly in children's shampoo ingredients. CABP is composed of a combination of fatty acids obtained from coconut oil with 3-dimethylamine propylamine (DMAPA). The initial substance obtained is cocamidopropyl dimethylamine, which is an amidoamine derivative (AA). The AA is then processed with sodium monochloroacetate, obtaining the final product: CABP (Fig. 1). The purpose of CABP addition to personal hygiene products is as a foam booster, thickener and softener.[3] Since the beginning of the 1980s, a series of reports have appeared, indicating the CABP may act as a contact allergen. The sensitisation prevalence to this substance is currently unknown in our country, however, a high frequency of sensitisation is known to present in hair dressers and those people who use shampoos, liquid soaps, hair dyes, contact lens solutions, shower gels and skin cleansers, given the presence of this component in these products.[2,4] It must be highlighted that various studies have demonstrated that the true sensitising agents could be intermediate products in the synthesis of CABP such as DMAPA and AA, more than CAPB itself. During many years this issue has been highly controversial and numerous North American studies[5–7] have demonstrated that AA was the cause of DCA while numerous European studies[8,9] show that DMAPA

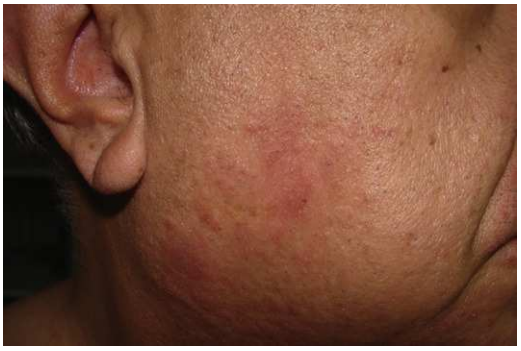

Figure 1 Erythematous scaly plaques in cheek.

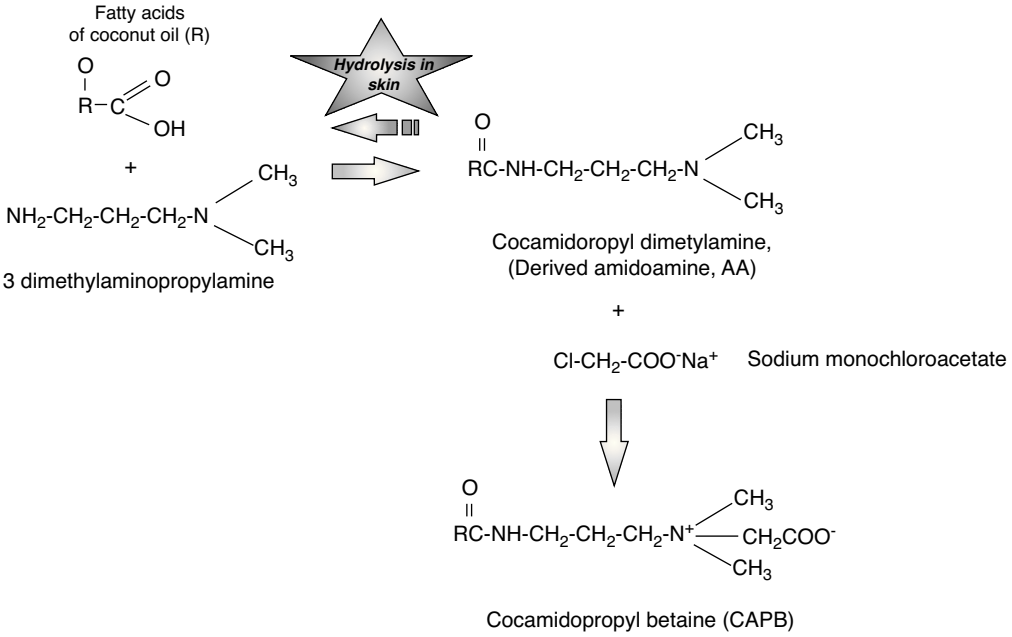

Figure 2 Synthesis of cocamidopropyl betaine (CAPB).

is the true sensitising substance. In our milieu, in which we probably have an under-registration of sensitisation to this substance, the purpose of the present study is to describe the role of CABP as a cause of ACD.

A 61-year-old patient presented with a six month history of pruriginous lesions on the cheeks and chin (Fig. 2), without occupational related risk factors and no history of atopia. Topical steroids had been used with clinical response, but presented with frequent relapses consisting of desquamative erythematous lesions at the sites previously described. An epicutaneous test (patch test) was applied on the upper back with the American standard series (Trolab® Patch Test Allergens) and improved quality chamber (Finn Chambers® for Patch Testing), showing a ++ positive reaction the D1 to CABP, balsam of Peru, balsam of Tolu and mixed fragrances, which persisted until D2 reading. The interview following the tests allowed for identification of a daily use of shaving shampoo containing CABP. Avoidance of this product during the skin care regimen resulted in resolution of the skin lesions and associated symptoms. The patient was finally diagnosed with an ACD to CABP present in the commercial CABP (shampoo).

To date, this is the first Colombian case report to describe ACD produced by sensitisation to CABP in a patient who routinely used liquid soap during shaving. Sensitisation to CABP clinically presents as a recurrent chronic dermatitis that involves the head (scalp, face and eyelids) and neck. International case reports exist which describe occupational ACD in hair dressers and health care personnel who present with forearm and hand involvement[10]. However, more diffuse presentations may present when liquid soaps, shampoos and shower gels are used, as in our case. With respect to CABP sensitisation, various studies have used allergenic extracts in the intermediate product of CABP synthesis patch test in patients allergic to this substance. Of these, DMAPA has been concluded to possibly have a significant role in CABP allergy.[8] In our country, these intermediate substances are not available, which limits the determination of these elements (DMAPA or AA) as causative agents in the clinical manifestations in our case. Given the current conditions of contact dermatitis knowledge in our environment, as is the absence of prevalence studies, this study provides a better understanding of this type of pathologies and specifically with respect to CABP.

Finally, in light of international literature, even though it is important to perform this type of studies, which aid the scientific population in the understanding of allergic cutaneous pathology, we must stress the need for more research studies that provide information regarding the frequency and prevalence of contact allergens in our population with the purpose of intervening with public health measures that impact not only in the health-disease process of our patients, but also in the health-related quality of life.

References

1. Hervella M, Yanguas JI, Iglesias ME, Larrea M, Ros C, Gallego M. Contact allergy to 3-dimethylaminopropylamine and cocamidopropyl betaine. Actas Dermosifiliograficas. 2006;97(April):189–95.
2. Li LF. A study of the sensitization rate to cocamidopropyl betaine in patients patch tested in a University Hospital of Beijing. Contact Dermatitis. 2008;58(January):24–7.
3. Jacob SE, Amini S. Cocamidopropyl betaine. Dermatitis. 2008;19(May–June):157–60.
4. Armstrong DK, Smith HR, Ross JS, White IR. Sensitization to cocamidopropylbetaine: an 8-year review. Contact Dermatitis. 1999;40(June):335–6.
5. Fowler JF, Fowler LM, Hunter JE. Allergy to cocamidopropyl betaine may be due to amidoamine: a patch test and product use test study. Contact Dermatitis. 1997;37(December):276–81.
6. Fowler Jr JF, Zug KM, Taylor JS, Storrs FJ, Sherertz EA, Sasseville DA, et al. Allergy to cocamidopropyl betaine and

amidoamine in North America. Dermatitis. 2004;15(March): 5–6.
7. Brey NL, Fowler Jr JF. Relevance of positive patch-test reactions to cocamidopropyl betaine and amidoamine. Dermatitis. 2004;15(March):7–9.
8. Foti C, Bonamonte D, Mascolo G, Corcelli A, Lobasso S, Rigano L, et al. The role of 3-dimethylaminopropylamine and amidoamine in contact allergy to cocamidopropylbetaine. Contact Dermatitis. 2003;48(April):194–8.
9. Angelini G, Rigano L, Foti C, Rossi P, Vena GA. Pure cocamidopropylbetaine is not the allergen in patients with positive reactions to commercial cocamidopropylbetaine. Contact Dermatitis. 1996;35(October):252–3.
10. Fowler Jr JF. Cocamidopropyl betaine: the significance of positive patch test results in twelve patients. Cutis; Cutaneous Medicine for the Practitioner. 1993;52(November):281–4.

J.J. Yepes-Nuñez[a,*,1], F.E. Gómez Rendón[b], R. Nuñez-Rinta[c]

[a] *Clinical Allergology Service, Academic Group of Allergology and Clinical Experiment (GRACE), University of Antioquia, Medellín, Colombia*
[b] *Dermatology Institute, Medellín, Colombia*
[c] *Faculty of Medicine, Universidad Pontificia Bolivariana, Medellín, Colombia*

* Corresponding author.
E-mail address: juanjoseyepesnunez@une.net.co (J.J. Yepes-Nuñez).
[1] Master of Clinical Science with emphasis in Clinical Epidemiology. Third-year resident of Clinical Allergology. Member of Academic Group Allergology and Clinical Epidemiology (GRACAE), Group of Clinical Allergology and Experimental (GACE), and Academic Clinical Epidemiology Group (GRAEPIC).

doi:10.1016/j.aller.2011.02.006

New pets, new allergies

To the Editor,

During recent years some exotic animals have been introduced as laboratory animals[1] or pets in domestic environments, increasing the risk of exposure to many unknown potential allergens which could cause respiratory allergy symptoms in the owners.[2]

In the case of hamster, there are various species with the same generic name but belonging to different rodent genus coming from different regions of the world without evidence of a clear cross reactivity among their allergens.[2–4]

Now in Spain it is possible to find different types of hamsters as pets, the most common is the golden or Syrian hamster *(Mesocricetus auratus)*, there are dwarfs hamsters: Chinese hamster *(Cricetus griseus)*, Siberian or Russian hamster *(Phodopus sungourus)*, Roborowski *(Phodopus roborowskii)*, and apparently the cross reactivity found among their epithelium allergens is very low.

We present three cases with different sensitisations:

First case: A 41-year-old woman with well-controlled pollinic asthma who began to suffer from daily asthmatic episodes and bad response to treatment with inhaled corticosteroids and B2, after buying a Russian hamster *(Phodopus sungorus)* (RH) for her child.

Skin prick test (SPT) with extract from RH epithelium was positive (8x7 mm), however it was negative against Syrian hamster (SH) epithelium. Histamine control: 4 x 5 mm. Serum specific IgE level was very high against RH epithelium 90.1kU/L and urine 86.3 kU/L, and very low against SH allergenic sources (epithelium: 0.7 kU/L; urine: 0.5 kU/L). SDS-PAGE-Immunoblotting showed an intense IgE binding band of ca. 21 kDa in RH epithelium extract and a high IgE binding area of ca. 18 - 21 kDa in RH urine extract. Some other high molecular mass IgE binding bands were observed in both extracts. No bands were revealed in extract from SH epithelium and very faint ones in SH urine (Fig. 1)

Second case: An 18−year-old woman who suffered from asthma with sensitisation to grass pollen, and horse and cat epithelium, she started with perennial asthma after buying a RH as a pet. Skin prick test with extract from RH epithelium gave a positive result (5x5 mm), with negative against SH epithelium. Histamine control: 4 x 5 mm.

Serum specific IgE level was positive against RH epithelium: 1.8 kU/L and urine: 1.7 kU/L, and very low against SH allergenic sources (epithelium: 1.2 kU/L, urine: 0.5 kU/L)

SDS-PAGE-Immunoblotting showed IgE binding band of ca. 21 kDa in RH epithelium extract, and 17.5 - 16 kDa in RH urine extract. (Fig. 1)

Third case: A 40-year-old woman with asthma with sensitisation to grass and olive pollen who developed perennial asthma when introducing a new pet (RH) to home. Skin prick test with RH epithelium was positive (6 x 5 mm) and negative for SH epithelium. Histamine control (4 x 4 mm)

SDS-PAGE-Immunoblotting showed IgE binding band of ca. 21 kDa in RH epithelium extract, and 18-21 kDa in RH urine extract. (Fig. 1)

Patients' symptoms improved after the hamsters were removed from their house and now they are well controlled using treatment only for spring symptoms, all of them improved the spirometric values (FEV1 and the FEV1%FVC), and the asthma was controlled only with the animal removal.

In the last years two main allergens have been described in rat *(Rattus norvegicus)*: Rat n 1A (20-21 kDa) and Rat n 1B (16-17 kDa), as well as in mouse *(Mus musculus)* Mus m 1 (19 kDa), Mus m 2 (16 kDa), all of them are lipocalins.

There are reports on allergy to Syrian Hamster *(Mesocricetus auratus)* in patients who work in laboratories with animals, and in pet owners. All these publications described an allergen between 15 to 21 kDa, a range of size similar to that of the lipocalins, however the identity of these hamster allergens has not been assessed.

Torres JA et al. described the presence of several Russian hamster *(Phodopus sungoris)* allergens with molecular mass between 18 − 23 kDa in various allergenic sources from RH (epithelium, faeces and urine).

There is a case report of anaphylaxis after hamster bites (Lim et al. and Nitsuma et al.)[5,6] where a specific IgE-binding component of 21 kD was detected in the hamster saliva.