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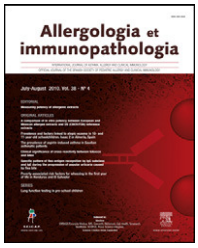

RESEARCH LETTERS

Cefotaxim induced a near fatal anaphylactic shock in an infant

To the Editor,

Severe anaphylactic reactions are potentially life-threatening.[1,2] In the literature, foods, venom and drugs are the most commonly reported exogenous causative agents.[3] Symptoms vary widely and can involve multiple organ systems, with skin, gastrointestinal, respiratory and cardiovascular symptoms.[2] In the paediatric population, allergic disorders have reached epidemic proportions, and anaphylaxis is an increasingly common event. However, drug induced life-threatening anaphylactic shock is still very rare in infants under six months of age.[4–7]

We report a 4-month-old girl who had a severe anaphylactic reaction within one minute after an intravenous administration of the third dose of cefotaxime (200 mg dissolved in 10 ml of specific solvent) given for a bronchial infection. Drug was administered by intravenous ''push'' over two to four minutes, rather than by a more prolonged intravenous infusion of 30 minutes. At the same time, she had just finished the intake of 180 ml of cow's milk. It was her second admission at the hospital. She had tolerated intravenous cefotaxime two months before, in her first admission. Symptoms included facial flush with swelling of the lips, urticarial rash on her trunk which progressed to generalised urticaria, intense dyspnoea, shortness of breath, wheezing and cyanosis with severe hypotension and collapse. After adequate treatment with intense anti-shock therapy, which was high flow oxygen, intravenous crystalloid fluid 20 mL/kg, intravenous hydrocortisone 25 mg, and three doses of intramuscular adrenaline, these symptoms were considerably reduced within one hour; and completely resolved after six hours. The patient had no other medical history.

Skin prick tests were performed with whole cow's milk extract (5 mg/ml), with isolated cow's milk proteins: α -lactalbumin (5 mg/mL), β -lactoglobulin (5 mg/mL), and casein (10 mg/mL); and with cefotaxime (2 mg/ml after dilution in 9%ClNa). Histamine dihydrochloride (10 mg/ml) was used as a positive control, and glycerosaline was used as a negative control. Reactions were read at 15 minutes. A net wheal diameter 3 mm larger than that produced by the negative control was considered positive. All skin prick tests were negative.

Assays for serum specific IgE to milk, α-lactalbumin, β-lactoglobulin, casein, penicilloyl G, penicilloyl V, ampicilloyl, amoxicilloyl, and latex were performed according to the manufacturer's instructions with UniCAP™ (CAP-FEIA; Pharmacia Diagnostics, Uppsala, Sweden). All these tests were negative. Total serum IgE was 10 IU/ml. The levels of serum tryptase, C3 and C4 were also assessed two months after the reaction and they were normal.

Open controlled challenge test with cow's milk was carried out with a formula of cow's milk adapted to the age of the patient. She tolerated the cow's milk without any problem. Her parents rejected the carrying out of any other diagnostic evaluation, including intradermal testing or challenge test with drugs. Latex environment was well tolerated.

There is a lack of information on the prevalence and characteristics of anaphylaxis in young infants. Food is the most common eliciting factor of anaphylactic reactions and furthermore a rising prevalence of food hypersensitivity has been reported during the last decades.[1,2] The negative results of the diagnostics tests and the low levels of total IgE supported our decision on performing a challenge test with milk. The tolerance of cow's milk and the chronology of the reaction suggested the implication of the cephalosporin.

Although a previous administration of cephalosporin existed, the severity of the reaction, the low levels of total IgE and, maybe, the inadequate velocity of the drug administration in the little body of our patient suggest an unspecific mechanism. It is probably a non IgE-mediated anaphylactic reaction. Several cases of life-threatening reaction due to cephalosporin have been reported.[4–7] Some of the reactions have been related to rapid administration of intravenous ceftriaxone coincidentally with a calcium solution.[5,6] In another case caused by cefazidime[7] an IgE-mediated mechanism was suggested, but it could not be demonstrated. We think that most of these reactions should be at least in part associated with histamine release from basophils and mast cells due to a direct membrane effect related to the osmolarity of the drug solution[5,6] with a possible activation of the complement system. Contributing factors for the infant in our report may include the use of a high dosage and intravenous ''push'' administration, and administration of the total daily dosage as a single infusion.

In our case, an adequate, quickly and intense treatment with several doses of adrenaline avoided the death of the

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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.

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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

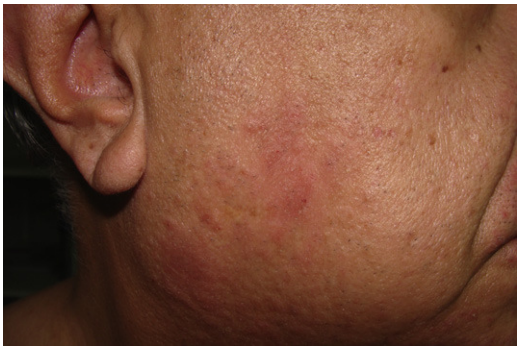

Figure 1 Erythematous scaly plaques in cheek.