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CASE REPORT

Peanut allergy as a trigger for the deterioration of atopic dermatitis and precursor of staphylococcal and herpetic associated infections – case report

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Abstract

Atopic dermatitis (AD) is a multifactorial and chronic disease, with genetic, environmental, immunological and nutritional origins. AD may be aggravated by allergies associated with infections. This study aims to describe a paediatric case of AD in which the peanut allergy was the triggering factor to aggravate the disease, and was also the concomitant precursor of staphylococcal (methicillin-sensitive *Staphylococcus aureus*, carrier of the Panton-Valentine leukocidine (PVL) genes) and herpetic (Herpes Simplex – HSV) infections. The clinical management approach and nursing strategies promoted a favourable evolution during the hospitalization period, besides the family approach, which was essential to control any flare-up of the disease. Adherence to a recommended diet and the use of strategies to prevent any recurrent infections were important to ensure the patient's quality of life.

Key words

atopic dermatitis, allergy, infection

INTRODUCTION

Atopic Dermatitis (AD) is a multi-factorial chronic disease involving genetic, immunologic and environmental factors that culminate in a dysfunctional skin barrier and a deregulated immune system [1]. The occurrence of AD in paediatric patients is 20–30% in the USA and 10–15% in Brazil [2, 3]. Diagnosis is made clinically; skin biopsy or other tests may be performed only to rule out other skin conditions [4]. The Scoring Atopic Dermatitis (SCORAD) and serum lactate dehydrogenase (LDH) levels [5] are parameters used to assess the level of AD. Some factors may be involved in its aggravation, such as food allergies [6] and colonization and/or infection by microorganisms [7]. There are some treatment options for patients with severe or refractory AD. These include phototherapy and systemic drugs, e.g. cyclosporine and azathioprine. Mycophenolate mofetil and gamma-interferon may be considered as alternative drugs [8]. This study describes a case in which peanut allergy was a triggering factor that aggravated AD, and was the forerunner of concomitant staphylococcal and herpetic infections in a paediatric patient.

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CASE REPORT

Early in 2011, a female patient aged 3 years and 7 months was brought for clinical assessment and monitoring at the Dermatology and Pediatric Allergy Ambulatory of a Pediatric Hospital in Rio de Janeiro, Brazil, and was diagnosed with moderate atopic dermatitis (SCORAD 47.2). Abnormal laboratory results were lactate dehydrogenase (LDH) 530 U/L; cholesterol 845 mg/dl, triglycerides 85, ESR 4 mm, IgE 1000 kU/L. Methylprednisolone 0.9 mg/kg/day, hydroxyzine 0.7 mg /day and applications of mineral oil on the skin were prescribed. She has class 4 food sensitization/allergy to beta-lactalbumin and egg, and class 3 to wheat. An exclusion diet was prescribed. After 4 months, it became clear that the family had difficulty in adhering to the diet, and despite several attempts

to discontinue corticosteroid therapy, the clinical status of the patient deteriorated. Cyclosporine (3 mg/kg) was introduced in order to reduce methylprednisolone progressively, and on 25 November 2011 the patient presented a SCORAD of 19.9 and the oral corticosteroids were discontinued.

However, on 16 March 2012, the patient showed a deterioration in her clinical condition and was admitted to the hospital with irritability, afebrile with erythroderma, *excoriation*, and oozing on the face, trunk and limbs, but without any infection or secondary manifestation of systemic involvement (SCORAD 78.9). The child's caretaker attributed the worsening due to the ingestion of peanuts. Abnormal findings were leukocytes 18,800 µL; eosinophils 7.144 mm3; platelets 606.000 /uL; reticulocytes 1.8. and LDH 2,786 U/L. Peanut-specific IgE level was 3.14 kU/L. Based on laboratory results, the cyclosporine treatment was stopped and methylprednisolone (2 mg/kg) and antihistamine were started and used throughout the hospitalization period. Abdominal ultrasonography, echocardiography and radiography of the skull and long bones showed no changes. After skin biopsy, histopathological findings suggested chronic spongiotic dermatitis with signs of abrasions. A haematological evaluation described macrocytosis, microcytosis, hypochromia and the presence of numerous neutrophils plurisegmented with left desviation compatible with chronic inflammation. During this stage, the patient developed a secondary infection on the skin due to methicillin-susceptible *Staphylococcus aureus* (MSSA). She was treated with sulfamethoxazole/trimethoprim which produced a clinical improvement within 10 days. All the other possible causes of the increase of LDH had been ruled out and the patient was discharged after 24 days in hospital.

Four days after being discharged, the patient returned to the hospital with a worsening of the skin lesions, intense itching, multiple oozing and rounded excoriations, with vesicles and pustules, located on the face and upper and lower limbs (SCORAD 74.3), suggesting a clinically-associated bacterial and herpetic infections (Fig. 1). The patient was once again hospitalized. Abnormal laboratory results were leukocytes 18,200 µL (segmented 39% and lymphocytes 50%), platelets 680,000 /µl; Cr 0.40 mg/dl and LDH 1,130 U/L. Serology for Herpes Simplex was positive (IgM) and the culture of the swab of the cutaneous lesion showed MSSA positive for Panton-Valentine leukocidin (PVL) genes. Erythromycin (50 mg/kg/day for 7 days) and acyclovir (200 mg/5X/day for 5 days) were then administered orally. Cyclosporine was discontinued but methylprednisolone 1.6 mg/kg/day was maintained. The patient improved with a remission of the cutaneous lesions, a decrease of the pruritus and a regression of the erythroderma (SCORAD 36). She was discharged with a gradual reduction of methylprednisolone and cyclosporine (3 mgkg/dia) on 24May 2012 (Fig. 2). Currently, the patient is under monthly out-patient monitoring without presenting any significant clinical change.

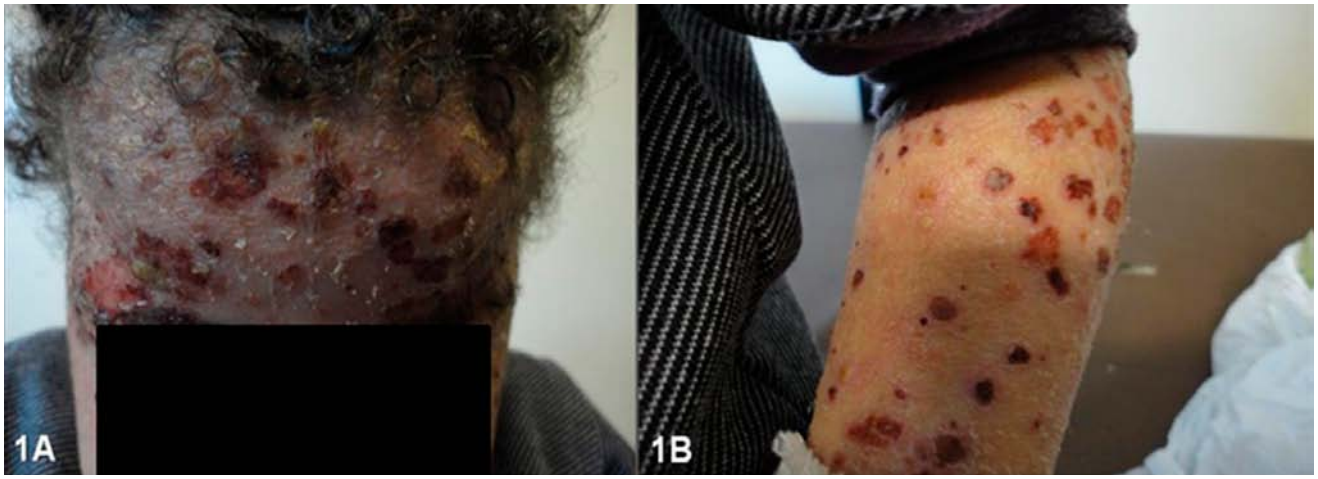

Figure 1. Picture 1A: flaking, ex-ulceration areas and hematic crusts; Picture 1B: Hematic crusted lesions and melicerica scabs, some with an erythematous halo, located on the right upper limb

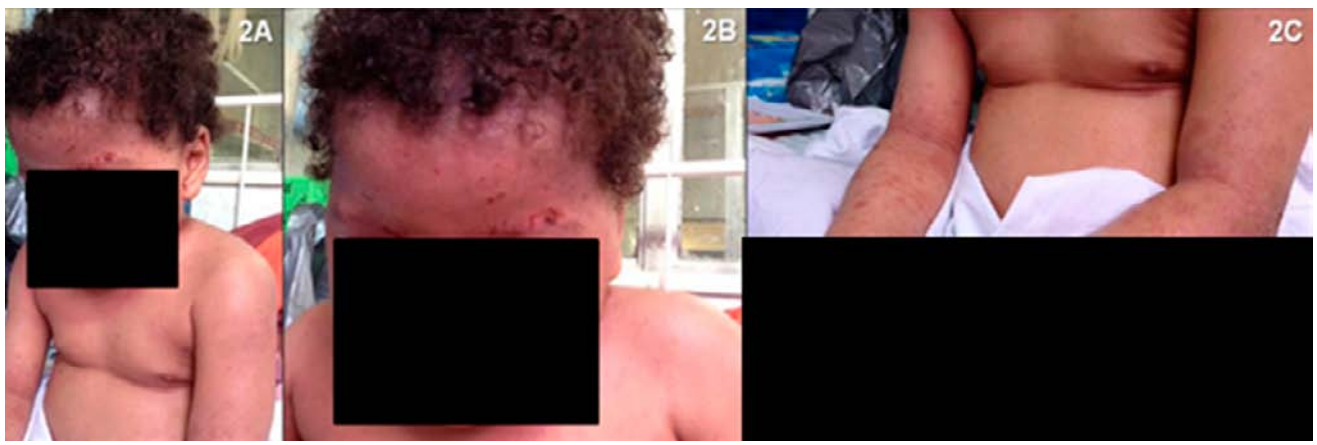

Figure 2. Picture 2A: Lesions with hypochromic center and hypochromic halo, residuals on the face and upper limbs; Picture 2B: Rounded hypochromic lesions on the forehead; Picture 2C: Patient with hypochromic center and hypochromic halo lesions

The patient described in the presented case report is one of a group of patients who belong to a cohort study with clinical monitoring for a year. Her guardian parent consented to the use of the images published in this study.

DISCUSSION

Faced with the multifactorial aspects of AD, clinical management of these patients becomes a challenge due to possible complications. This includes the resurgence of the disease, requiring hospitalization and rigorous management. The patient in this study was treated as an out-patient for about 14 months, with no history of previous hospitalization. However, in the last 4 months, her clinical status was very unsettled and investigation of the nutritional aspect confirmed the non-adherence to diet and peanut consumption. These data are in line with those of other authors who observed that patients with AD may experience a deterioration of their clinical status when such foods are consumed [6]. Moreover, the results of the investigation of peanut-specific IgE in the patient showed the presence of high levels of this immunoglobulin. SCORAD values for children with AD and food allergy have been shown to be significantly higher in comparison to those without food allergy to the allergen [9].

Another important factor identified in this patient was an increase of LDH. With recrudescence of AD (SCORAD 74.3) in which there were concomitant bacterial and viral infections, LDH was 1.130 U/L, i.e. about 20 times the initial level. However, to-date, no biomarker has shown reliable sensitivity or specificity for AD for disease monitoring [10]. Morishima et al. [5] correlated AD severity with LDH levels, eosinophil counts and total IgE, and found that LDH levels increased with the worsening of a patient's clinical condition, and decreased with its improvement, suggesting that this is a factor with poor prognostic value.

The patient in this study also developed clinical instability and deterioration of the SCORAD, with associated bacterial infection and herpetic infection. Beck et al. [11] demonstrated that a history of infection with *S. aureus* was a predisposing factor for future cases of eczema herpeticum in patients with AD. Moreover, an association of bacteria/viruses has already been suggested in skin diseases, including AD [12, 13]. Furthermore, Bin et al. [13] showed that α-toxin of *S. aureus* is capable of increasing the viral load of HSV in keratinocytes. The fact that the patient was colonized by a PVL^+ isolate may have contributed to the worsening of the patient's clinical condition. The expression of PVL leads to the disruption of cells and formation of necrotic spots, causing an efflux of immune cells to sites on the skin.

This case report sought to demonstrate that food allergy should be considered relevant in patients with severe AD. Here, peanut consumption aggravated the clinical condition, forming excoriations in response to itching, which secondarily evolved into bacterial infection. This, as the patient was colonized by MSSA PVL^+, culminated into an associated herpes infection, which suggests that herpes viruses may contribute to the pathogenesis of AD lesions.

Concerning the limitations of the presented study, it is worth highlighting that the authors did not collect clinical samples from the skin lesions during the exacerbation period. Also, they did not collect clinical samples from anterior nares of contacts, because they could be colonized by the same clone of the patient. This leaves open the possibility of re-colonization after the use of antibiotics by the patient.

The patient under discussion underwent appropriate clinical management, use of antibiotics, antiviral drugs and care strategies for patients with AD. This approach succeeded in improving the clinical condition of the patient who is currently under outpatient follow-up.

CONCLUSION

The clinical management approach and nursing strategies promoted a favorable evolution during the hospitalization period, next to the family approach, which was essential for the control of any flare-up of the disease. Adherence to a recommended diet and the use of strategies to prevent recurrent infections were important to ensure the patient's quality of life. This report sought to demonstrate that food allergy should be considered relevant in terms of the clinical condition, especially in patients with severe AD. This case report reveals the probability of the occurrence of superimposed *Staphylococcus* and herpes infections during the crisis (exacerbations) of atopic dermatitis.

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REFERENCES

1. Kabashima K. New concept of the pathogenesis of atopic dermatitis: interplay among the barrier, allergy, and pruritus as a trinity. J Dermatol Sci. 2013; 70(1): 3–11.
2. Eichenfield LF, Ellis CN, Mancini AJ, Paller AS, Simpson EL. Atopic dermatitis: epidemiology and pathogenesis update. Semin Cutan Med Surg. 2012; 31(3 Suppl): S3–5.
3. Castro AP. Calcineurin inhibitors in the treatment of allergic dermatitis. J Pediatr. 2006; 82(5 Suppl): S166–172.
4. Eichenfield LF, Hanifin JM, Luger TA, Stevens SR, Pride HB. Consensus conference on pediatric atopic dermatitis. J Am Acad Dermatol. 2003; 49(6): 1088–1095.
5. Morishima Y, Kawashima H, Takekuma K, Hoshika A. Changes in serum lactate dehydrogenase activity in children with atopic dermatitis. Pediatr Int. 2010; 52(2): 171–174.
6. Wisniewski JA, Agrawal R, Minnicozzi S, Xin W, Patrie J, Heymann PW, et al. Sensitization to food and inhalant allergens in relation to age and wheeze among children with atopic dermatitis. Clin Exp Allergy. 2013; 43(10): 1160–1170.
7. Lübbe J. Secondary infections in patients with atopic dermatitis. Am J Clin Dermatol. 2003; 4(9): 641–654.
8. Sidbury R, Davis DM, Cohen DE, Cordoro KM, Berger TG, Bergman JN, et al. Guidelines of care for the management of atopic dermatitis: section 3. Management and treatment with phototherapy and systemic agents. J Am Acad Dermatol. 2014; 71(2): 327–349.
9. Rosinska-Wieckowicz A, Czarnecka-Operacz M. Disease extent and severity in patients with atopic dermatitis and food allergy. Post Dermatol Alergol. 2011; 5: 382–388.
10. Eichenfield LF, Tom WL, Chamlin SL, Feldman SR, Hanifin JM, Simpson EL, et al. Guidelines of care for the management of atopic dermatitis: section 1. Diagnosis and assessment of atopic dermatitis. J Am Acad Dermatol. 2014; 70(2): 338–351.
11. Beck LA, Boguniewicz M, Hata T, Schneider LC, Hanifin J, Gallo R, et al. Phenotype of atopic dermatitis subjects with a history of eczema herpeticum. J Allergy Clin Immunol. 2009; 124(2): 260–269.
12. Bakaletz LO. Immunopathogenesis of polymicrobial otitis media. J Leukoc Biol. 2010; 87(2): 213–222.
13. Bin L, Kim BE, Brauweiler A, Goleva E, Streib J, Ji Y, et al. *Staphylococcus aureus* α-toxin modulates skin host response to viral infection. J Allergy Clin Immunol. 2012; 130(3): 683–691.