Baboon syndrome induced by oral antitussive-decongestant agent in a child

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We present the case of a three-year-old boy who developed a special exanthem after oral intake of an antitussive-decongestant agent. The clinical findings were compatible with baboon syndrome. To our knowledge, this is one of the rare reported cases of baboon syndrome associated with use of an antitussive-decongestant including pseudoephedrine HCl, dextromethorphan HBr and chlorpheniramine maleate.

Key words: baboon syndrome, chlorpheniramine maleate, children, dextromethorphan, pseudoephedrine, systemic contact dermatitis.

Baboon syndrome was first described by Andersen et al. in 1984[1]. It is a systemic allergic contact dermatitis characterized by a widespread exanthema favoring the major flexures, and developing several hours or days after contact with different allergens or drugs[2,3]. Contact dermatitis triggered through systemic exposure to topical allergens, such as mercury, nickel, cobalt, and gold has been well documented previously[2,4,5]. The systemic drugs reported in association with this syndrome during childhood were amoxicillin, penicillin, erythromycin, pseudoephedrine, and risperidone[6-9].

Here, we present a case of baboon syndrome associated with antitussive-decongestant use affecting a three-year-old child.

Case Report

A three-year-old male infant was presented with a pruritic macular erythematous rash of four days duration. This rash started following oral intake of an antitussive-decongestant including pseudoephedrine HCl, dextromethorphan HBr and chlorpheniramine maleate. On physical examination, he had macular erythematous patches affecting the buttocks, anogenital area, inner thighs, pubic-suprapubic regions, neck, and groin (Figs. 1, 2). Oropharynx and tonsils appeared normal and systemic signs were absent. Complete blood count, erythrocyte sedimentation rate, C-reactive protein, and blood biochemistry including liver and renal function tests were normal. Throat culture was negative.

He had no history of topical exposure to this antitussive-decongestant agent, but his medical history was remarkable for having had the same rash develop after oral intake of the same antitussive-decongestant agent four months and nine months previously. He had been diagnosed as scarlet fever and upper respiratory infection and had been given antibiotic, but the rash did not disappear until stopping both the antitussive-decongestant and antibiotic medication. However, because of the mistaken diagnosis, the same agent was again given to the child for the third time and the eruption reappeared in the same anatomic locations 24 hours later. After dermatology consultation, the diagnosis of baboon syndrome was made and the medication was discontinued. The eruption resolved two days after withdrawal of the causative agent and use of oral antihistaminic and topical hydrocortisone.

Discussion

Baboon syndrome is a rare, prognostically benign systemic contact dermatitis with distinct

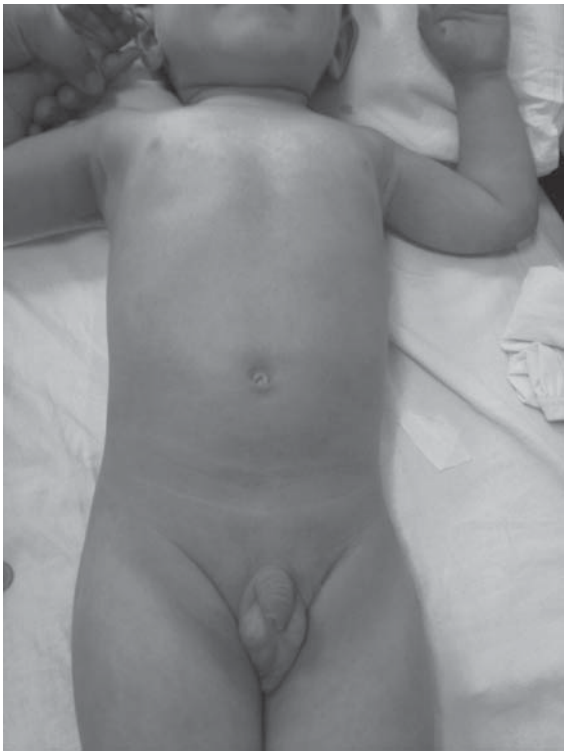

Fig.1. Macular erythematous skin rash affecting inner thighs, groin and pubic-suprapubic regions.

clinical features. It is a special form of systemic contact dermatitis occurring usually within 1 to 8 days after ingestion or systemic absorption of a contact allergen in individuals previously sensitized by topical exposure to the same allergen[7].

It was proposed recently to replace baboon syndrome with the acronym SDRIFE, and in 2004, Arnold et al.[10] proposed the acronym SDRIFE ("symmetrical drug-related intertriginous and flexural exanthema") specifically for cases associated with systemic drugs; it represents a distinct reaction pattern related to systemic drugs. The diagnostic criteria of SDRIFE were: (1) exposure to a systemically administered drug either at first or repeated dose (excluding contact allergens); (2) sharply demarcated erythema of the gluteal/perianal area and/or V-shape erythema of the inguinal and perineal area; (3) involvement of at least one other intertriginous/flexural localization; (4) symmetry of affected areas; and (5) absence of systemic symptoms and signs.

The pathogenesis of systemic contact dermatitis is not very clear. Some studies have concluded that this reaction may be mediated by both a type III and a type IV mechanism[11].

The inhalation of mercury vapor is the most common trigger in patients with baboon syndrome. A large number of drugs such as erythromycin, penicillin, amoxicillin, heparin, immunoglobulins, radiocontrast media, and others have been implicated[3,12].

In conclusion, this syndrome is usually underdiagnosed in children because it is confused with the exanthems of childhood viral and bacterial infections such as scarlet fever and staphylococcal-scalded skin syndrome, and with other distinct drug eruptions such as acute generalized exanthematic pustulosis, toxic epidermal necrolysis and disseminated multilocular fixed drug eruption. In addition, most of the children with drug eruption are not consulted to dermatologists. It should always be taken into account when facing the differential diagnosis of acute exanthem in children, and the clinicians should inquire about the use of drugs in the routine medical history. To our knowledge, this is the first reported case of baboon syndrome or SDRIFE associated with use of an antitussive-decongestant including pseudoephedrine HCl, dextromethorphan HBr, and chlorpheniramine maleate.

Fig. 2. Macular erythematous skin rash affecting the neck.

REFERENCES

1. Andersen KE, Hjorth N, Mene T. The baboon syndrome: systemically-induced allergic contact dermatitis. Contact Dermatitis 1984; 10: 97-100.
2. Audicana M, Bernedo N, Gonzalez I, Munoz D, Fernandez E, Gastaminza G. An unusual case of baboon syndrome due to mercury present in a homeopathic medicine. Contact Dermatitis 2001; 45: 185.
3. Garcia-Menaya JM, Cordobes-Duran C, Bobadilla A, Lamilla A, Moreno I. Baboon syndrome: 2 simultaneous cases in the same family. Contact Dermatitis 2008; 58: 108-109.
4. Dou X, Liu LL, Zhu XJ. Nickel elicited systemic contact dermatitis. Contact Dermatitis 2003; 48: 126-129.
5. Jankowska-Konsur A, Kolodziej T, Szepietowski J, Sikora J, Maj J, Baran E. The baboon syndrome-report of two first cases in Poland. Contact Dermatitis 2005; 52: 289-290.
6. Moreno-Ramirez D, Garcia-Bravo B, Pichardo AR, Rubio FP, Martinez FC. Baboon syndrome in childhood: easy to avoid, easy to diagnose, but the problem continues. Pediatr Dermatol 2004; 21: 250-253.
7. Akay BN, Sanli H. Symmetrical drug-related intertriginous and flexural exanthem due to oral risperidone. Pediatr Dermatol 2009; 26: 214-216.
8. Sanchez-Morillas L, Reano Martos M, Rodriguez Mosquera M, Iglesias Cadarso A, Perez Pimiento A, Dominguez Lazaro AR. Baboon syndrome due to pseudoephedrine. Contact Dermatitis 2003; 48: 234.
9. Sanchez TS, Sanchez-Perez J, Aragues M, Garola-Diaz A. Flare-up reaction of pseudoephedrine baboon syndrome after positive patch test. Contact Dermatitis 2000; 42: 312-313.
10. Arnold AW, Hausermann P, Bach S, Bircher AJ. Recurrent flexural exanthema (SDRIFE or baboon syndrome) after administration of two different iodinated radio contrast media. Dermatology 2007; 214: 89-93.
11. Raison-Peyron N, Guillard O, Khalil Z, Guilhou JJ, Guillot B. Nickel-elicited systemic contact dermatitis from a peripheral intravenous catheter. Contact Dermatitis 2005; 53: 222-225.
12. Dhingra B, Grover C. Baboon syndrome. Indian Pediatr 2007; 44: 937.