

Nonpigmenting fixed exanthema induced by pseudoephedrine

PSEUDOEPHEDRINE is a sympathomimetic drug widely found in remedies for the common cold due to its nasal decongestant action. In this report, we describe a female patient who presented nonpigmenting fixed exanthema after administration of pseudoephedrine.

■ The patient was a 19-year-old woman with allergic rhinoconjunctivitis due to cat dander. One year before coming to our clinic, she had taken Narine (loratadine 5 mg plus pseudoephedrine 60 mg) for acute rhinoconjunctivitis. Several hours after the first dose, she presented erythematous plaques with burning on the palms and soles. The drug was discontinued and the reaction resolved 8 days later with scaling of the involved sites, but without residual pigmentation. Ten months later, she had an upper airway infection, for which her physician prescribed Inistón (pseudoephedrine 60 mg plus triprolidine 2.5 mg); 8 h after she took the first dose, an identical sequence of events occurred at exactly the same sites as her previous attacks.

Skin prick tests with common aeroallergens yielded negative results except for cat dander. Patch tests were performed with Narine, Inistón, epinephrine, phenylephrine, and pseudoephedrine (1% and 10% pet.). All of them elicited negative reaction at 48 and 96 h except for phenylephrine, which elicited slight erythema at 96 h.

The patient was then challenged orally with Inistón; no immediate response was observed. At 8 h, the patient complained of burning in the palms and soles followed by the appearance of tender red plaques with erythema and edema in the palms and soles. The erythema spontaneously remitted within 2 days, without residual pigmentation, but this was followed by scaling of the involved sites.

Controlled challenge is the only certain method to diagnose nonpigmenting fixed exanthema to pseudoephedrine.

The patient was then challenged orally with 60 mg of pseudoephedrine. Within 12 h, the same erythematous eruption appeared at exactly the same sites as in her three former attacks. A 3-day tapered course of prednisone (40 mg/day) led to the resolution of the lesions within this time. Subsequent separate challenges with the other component of Inistón (triprolidine), loratadine, epinephrine, and phenylephrine showed negative results.

■ In our patient, specific symmetric areas of the skin were sensitized to pseudoephedrine, which proved to be the etiologic agent in the challenge test. The clinical characteristics of the reaction (it recurs in the same symmetric areas of the skin without postinflammatory hyperpigmentation at the fixed sites) are diagnostic data of nonpigmenting fixed exanthema.

Nonpigmenting fixed drug eruption is a distinctive, clinically recognizable entity, as proposed by Abramowitz & Noun (1). Characteristically, the lesions are large, symmetric, well-circumscribed, tender, erythematous plaques that suddenly appear and reappear in exactly the same sites. They fade without pigmentation or any other trace. The epidermis is not disturbed; thus, no pigmentation occurs. The dermis is the primary site of the drug sensitivity response in this dermatitis (2-3).

Sympathomimetic drugs must be rare causes of drug eruptions in general, considering their frequent use in over-the-counter medications for the common cold. To our knowledge, there are few published cases of adverse reactions to this drug (2-11). However, it would appear that fixed drug eruptions, particularly the nonpigmenting variety (2-5), may be the most common cutaneous reaction to pseudoephedrine hydrochloride.

Patch tests are not a useful method for the diagnosis of this reaction. Only by using oral challenge can the true prevalence of the nonpigmenting form be revealed.

Finally, in this patient, we could not demonstrate any cross-sensitivity to the other sympathomimetic drugs tested (epinephrine and phenylephrine), as in the other cases previously investigated (2, 4, 9, 11). Only cross-sensitization between pseudoephedrine and ephedrine has been previously reported (4, 10, 11).

Key words: fixed eruption; pseudoephedrine; sympathomimetic drugs.

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Oral allergy syndrome to bird meat associated with egg intolerance

THE occurrence of allergic reactions related to the ingestion or inhalation of bird products has been previously described. Egg intolerance, mainly due to egg-yolk livetins, in association with respiratory sensitization to bird-serum antigens, has been designated the "bird-egg syndrome" (1-5). Here we report a child with oral allergy syndrome due to sensitization to bird meat, in association with egg intolerance.

■ An 8-year-old boy with family history of atopy and personal history of fish allergy was referred to our service for allergologic investigation. He experienced itching of his mouth and throat immediately after the ingestion of bird meat (chicken, quail, and

turkey). Furthermore, the ingestion of egg immediately resulted in nausea and vomiting. He said that he had never experienced respiratory symptoms, and birds were not present in his usual environment.

Physical examination in our patient was unremarkable. The blood count and chemistry were in the normal range. Skin prick-by-prick tests performed with egg yolk, egg white, chicken meat, quail meat, and turkey meat were negative except for quail meat. Skin prick tests (SPT) with extracts of feather mix, quail meat, turkey meat, chicken meat, and partridge meat (ALK-Abelló, Madrid, Spain) were all positive. No oral challenge test was attempted.

The total IgE serum level was above 300 IU/ml. Specific IgE serum levels (Pharmacia Phadebas RAST, Uppsala, Sweden) were determined by using BrCN-activated disks and then sensitized, as previously described (6), to different extracts (quail meat, turkey meat, partridge meat, chicken meat, chicken feathers, egg white, and egg yolk). In our case, the results appeared to be negative for chicken feathers and egg yolk, and positive for quail meat (1.5 PRU/ml), turkey meat (1.5 PRU/ml), partridge meat (1.8 PRU/ml), chicken meat (1.9 PRU/ml), and egg white (2.6 PRU/ml).

■ The bird-egg syndrome is mainly described in adults (1, 3, 4), but also in children (2). This syndrome is elicited when exposure to a bird causes respiratory symptoms and egg intolerance appears secondarily. It has been suggested that egg intolerance in adults is mainly due to sensitization to egg-yolk livetins and can be provoked by inhalation of pet bird dander. Thus, it must be distinguished from the common egg-white allergy of atopic children

(1). Añibarro et al. (2) described a case of bird-egg syndrome in a child in whom egg allergy differed from the typical forms of egg allergy in children (late onset, greater prevalence of digestive and respiratory

than cutaneous symptoms, and greater sensitization to egg yolk than to egg white). It is assumed that α -livetins (chicken serum albumin) leads to cross-sensitization and consequently to the bird-egg syndrome (3, 4), and that albumin involved in the bird-egg syndrome also induces sensitization to bird meat (1, 2, 5).

In spite of the heterogeneity of SPT and serum specific IgE levels, our results suggest an IgE-mediated mechanism for oral allergy syndrome elicited by bird meat; in the same way, allergy to

A child's allergic reaction to poultry meat may have been caused by IgE antibodies to egg white.

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