

significant reaction to intradermal hydrocortisone sodium succinate, methylprednisolone sodium succinate, and prednisolone sodium succinate, whose structure is similar. Therefore, we considered that steroid sodium succinate induced adverse reactions, because steroid sodium succinate has induced adverse reactions more often than did steroid sodium phosphate (3). However, hydrocortisone sodium phosphate was also positive in the skin prick test. On the other hand, the test results for betamethasone disodium phosphate and dexamethasone sodium phosphate were negative. Therefore, no consistent stoichiometric arrangement was associated with predictable reactivity. Skin test results in this case could not rule out allergy to sodium succinate. No published cases of steroid allergy have shown allergy to succinate (5); hence, the possibility of succinate allergy may have been very low in this case.

Three years previously, the patient received hydrocortisone sodium succinate d.i.v., which was effective for his dyspnea. He had never received both methylprednisolone sodium succinate and hydrocortisone sodium phosphate d.i.v. before this episode. Unexpectedly, his adverse reactions of steroid occurred. In this case, skin tests were very useful in predicting the safe administration of alternative steroid agents for future use. At

present, betamethasone disodium phosphate d.i.v. is effective for his asthma attack. However, it remains unknown whether betamethasone disodium phosphate d.i.v. may induce any adverse reaction in future.

In conclusion, we present a patient who had allergy to both steroid sodium succinate and sodium phosphate and had never been administered these steroids except for hydrocortisone sodium succinate. It is unknown whether cross-reactivity between these steroids exists and how these steroids sensitized him. Physicians should be aware of the possibility of an adverse reaction to steroid even though its previous use has been effective.

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Progressive eosinophilia and elevated IgE in enterobiasis

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Key words: enterobiasis; *Enterobius vermicularis*; eosinophilia; IgE; pinworm.

● PARASITOSIS due to *Enterobius vermicularis* (pinworm) is very common especially among children. This small white nematode inhabits the cecum and adjacent gut. The gravid females migrate at night through the large intestine up to the anal sphincter and the perineal region for oviposition. It is very rare to find their eggs in feces, and the most reliable method of diagnosis is by examination of an adhesive cellophane tape pressed against the perianal

Table 1. Results of intradermal, prick, and patch tests

Tested material	Concentration (mg/ml)	Intradermal test	Prick test	Patch test
Betamethasone disodium phosphate (Rinderon from Shionogi)	4	–	nt	nt
Prednisolone sodium succinate (Predonine from Shionogi)	4	2+	nt	nt
Methylprednisolone sodium succinate (Solu-Medrol from Pharmacia & Upjohn)	62.5	3+	nt	nt
Hydrocortisone sodium succinate (Solu-Cortef from Pharmacia & Upjohn)	50	3+	nt	nt
Hydrocortisone sodium phosphate (pure dissolved in water)	5	nt	+	–
Hydrocortisone sodium succinate (Saxizon from Nikken Chemical)	5	nt	+	–
Dexamethasone sodium phosphate (Orgadrone from Organon-Sankyo)	0.38	nt	–	–
Solvent of Solu-Cortef (paraben)		nt	+	+
Solvent of Sol-Medrol		–	nt	nt
Saline		–	–	–

nt: not tested.

region early in the morning. Pinworm infection is very contagious, and its mode of transmission is fecal-oral. Autoinfection is frequent. The clinical presentations are related largely to perianal and perineal pruritis, but a large proportion of pinworm infections are essentially asymptomatic. The infection may cause right iliac cavity discomfort, diarrhea, and, rarely, appendicitis. The migration from the anus to the vagina may cause vaginitis, leukorrhea, and even urinary infection. Blood eosinophilia is rarely detected or is medium, and the IgE level is normal.

A 19-year-old woman was referred to us for blood eosinophilia. She had been diagnosed with synovial chondromatosis and hepatitis in childhood. She

had no toxic exposure and was not receiving medical treatment. Eosinophilia had been detected 2

months before, and this was confirmed by a second determination. In clinical anamnesis, she had a 1.5-year history of perennial rhinitis, nonproductive cough, and exercise-related dyspnea. There was no geographic, indoor-outdoor, nor seasonal influence in symptomatology. The patient reported absence of anal pruritus, and digestive and cutaneous symptoms. She had two pets (a cat and a dog) and tolerated antibiotics, foods, and aspirin. She denied foreign travel and watercress ingestion.

Physical examination was normal. Rhinoscopy showed no polyps. Hematologic determinations 2 months before showed 1200 eosinophils (18%). Another test in the last month showed eosinophilia of 1600 (21%).

We performed the following examinations and tests when we first saw her.

- Hematimetry with differential count showed 1900 eosinophils (32%). Erythrocyte sedimentation rate and urinalysis were normal.

Asymptomatic enterobiasis caused eosinophilia and increased IgE in a 19-year-old woman.

- Serum biochemistry: cholesterol, 237 mg/dl; other parameters, normal.
- Thyroid hormones, complement levels, autoantibodies, and vitamin B₁₂: normal; hydatidosis, syphilis, and hepatitis B and C serologies: negative.
- Serum IgA and IgG: normal; IgM: 25.6 mg/ml; IgE: 4240 kU/l (IRMA, Immunotest, Italy).
- Specific IgE (CAP System, Pharmacia, Sweden): *Ascaris*, 14.40 kU/l; *Anisakis simplex*, 4.2 kU/l; *Aspergillus fumigatus*, <0.35 kU/l; *Equinococcus*, <0.35 kU/l.
- Skin prick tests with house-dust mites, molds (*Aspergillus* included), pollens, danders (cat and dog), and *Anisakis* were negative.
- Chest and paranasal sinus radiographs showed no abnormalities.
- Spirometry was normal, and the bronchodilation test negative.
- Mantoux test, sputum acid fast smears, and Löwenstein-Jensen cultures were negative. Sputum cytology revealed absence of eosinophils.
- Stool examination for parasites (three determinations) consistently showed massive presence of *E. vermicularis*. The adhesive cellophane tape test was also positive for this nematode.

Treatment with mebendazol (Lomper®) was initiated by the patient and her family. A test for parasites in stools and the cellophane tape test performed later were negative. At this time, the total of blood eosinophils was 600 (9.2%). After 6 months, the intestinal parasite stool tests remained negative, total serum IgE was 1825 kU/l (decrease 57%), and eosinophils totaled 700 (8.3%). Two months later, this value fell to 590 (8.7%) eosinophils (decrease 69%).

The relationship among blood eosinophilia, serum level of IgE, and some parasite infections by helminths is well known. In uncomplicated enterobiasis, the pinworm usually remains in the intestinal lumen; therefore, *E. vermicularis* has very rarely caused these hematologic effects.

Recently, there have been several case reports of eosinophilic gastroenteritis with

concomitant blood eosinophilia, eosinophilic salpingitis, epididymitis, eosinophil cystitis, perianal granuloma, appendicitis, and even ileum perforation due to pinworm infection. In our patient, neither digestive nor genitourinary symptoms were present.

Moreover, our patient was atypical since abundant eggs of *E. vermicularis* in feces are very rare. This suggests that the massive infection, although asymptomatic in our patient, may have induced eosinophilia and high serum IgE. This is supported by the rapid fall, after treatment of the enterobiasis, of the total eosinophil count and IgE level, which was maintained 8 months later. Extensive examination of our patient showed no additional causes of eosinophilia.

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Erroneous diagnosis of idiopathic anaphylaxis

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Key words: connubial; dipyrone; idiopathic anaphylaxis; nontherapeutic exposure.

● ANAPHYLAXIS is a potentially fatal immediate-type reaction due to the release of bioactive mediators from mast cells and basophils. It can be caused by acute hypersensitivity or by non-IgE-mediated mast-cell mediator liberation. Both mechanisms produce the same clinical picture, which includes anxiety, urticaria, angioedema, respiratory distress, and cardiovascular collapse.

The diagnosis of idiopathic anaphylaxis (IA) is an exclusion diagnosis that is made when all known causes of anaphylaxis have been excluded. In some cases, a "hidden" allergen may be responsible and an intense effort may be required to identify it.

This report describes the assessment of one case of anaphylaxis in which a diagnosis of IA was made erroneously.

A 30-year-old woman had two episodes in the last year of intense pruritus of the palms and soles followed by generalized itching with urticaria, dysphagia, cough, and wheezing.

The episodes occurred in the absence of any identifiable inciting antigen, exercise,

or other stimulus known to cause anaphylaxis. They occurred hours after the last food ingestion, a fact which suggests food was not the source of an allergen. The patient did not take any prescription before or during the events. In both episodes, there had appeared urticaria that does not occur with hereditary angioedema or carcinoid syndrome (1). She did not have urticaria pigmentosa, a disorder which indicates systemic mastocytosis, and she never showed the typical finding of pheochromocytoma: pallor, hypertension, and tachycardia.

The patient had a history of rhinitis and conjunctivitis caused by sensitization to *Dermatophagoides pteronyssinus*. Skin tests to foods, including spices, and other aeroallergens were negative. Total serum IgE values were at 107 IU/ml (normal values in adults are less than 100 IU/ml). The patient had normal routine laboratory test results such as serum chemistries, liver enzymes, thyroid function tests, stool assays for ova and parasites, and complement level. The diagnosis of IA was made at that point of the study by exclusion.

Four months later, she took one Optalidon® dose (propyphenazone plus dihydroergotamine plus caffeine) for headache. A few minutes after taking it, she suddenly exhibited generalized erythema with urticaria, facial angioedema with loss of voice (she was not able to ask for help), dysphagia, and circulatory collapse with hypotension. She had tolerated Aspirin® (AAS) and Termalgyn® (paracetamol) in this period and after. The cutaneous test with dipyrone was positive (10×15 mm), in the intradermal test at dilution 1/100. The results of prick by prick tests with Optalidon (propyphenazone plus dihydroergotamine plus caffeine) and Cafergot® (ergotamine plus caffeine) were negative. The oral challenges with Aspirin and Cafergot were negative.

The only possible association that could explain the appearance of the symptoms in the

two first episodes was that the patient had developed symptoms within a few minutes after being kissed by her husband. She reported that her husband frequently took Optalidon for headache. He remembered having taken Optalidon at that time.

Antibiotics (penicillin) present in foods (2) and the environment (3, 4), or excreted by animals or humans (5) can be a source of challenge to individuals who are sensitized. There are cases of sensitized individuals who are vulnerable to nontherapeutic exposure. Cases of postcoital urticaria, lip angioedema, rash, and systemic pruritus have been reported, (5, 6). We cannot explain the mechanism of these reactions and their relationship to medicament excretion. We presume that active metabolites of propyphenazone are excreted by saliva, as occurs with dipyrone (7), or by breathing, and this must be the challenge mechanism responsible for "false idiopathic anaphylaxis" in our case.

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