Anaphylaxis Due to *Pachycondyla goeldii* Ant: A Case Report

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Palabras clave: Alergia a himenópteros. Anafilaxia. Alergia a hormiga.

Most cases of anaphylaxis caused by ants are attributed to ants from the *Solenopsis* genus [1] but there are many ant species of different genera and their distribution around the world varies. In Brazil, where there are over 2000 catalogued species of ants, all the cases of ant-induced anaphylaxis reported to date have been attributed to *Solenopsis* species [2,3]. We describe the first case of anaphylaxis caused by *Pachycondyla goeldii*, an ant from the *Ponerinae* subfamily.

A 52-year-old man developed anaphylaxis after being stung on the hand by an unidentified insect while removing grass from his garden. He felt a tingling sensation and developed generalized urticaria and shortness of breath. He received treatment for anaphylactic shock at the emergency department and recovered within a few hours.

Five days later, the patient had recovered completely. At the site of the sting, there was a dark spot surrounded by a red ring. He was receiving treatment for diabetes and hypertension (glimepiride, metformin, and enalapril [5 mg/day]) and he stated that he had not taken any other drugs or eaten any unusual foods. The patient reported that he had been stung twice (40 and 15 days previously) by a black insect like a wasp on his shoulder, which had become swollen and painful. No *Solenopsis* ants or wasps nests were found during a visit to the patient's house but black ants measuring 1 cm in length (and popularly known as *gambá*) were found in the garden.

The ants were identified as *P goeldii* by the Center for the Study of Social Insects (CEIS) at São Paulo State University. The venom was obtained by removing the venom reservoirs with microscissors and squeezing the content into twice-distilled water. The crude venom solution was then lyophilized and maintained at –20ºC until needed.

The investigation of specific immunoglobulin E (IgE) in the patient's serum (ImmunoCAP, Phadia Uppsala, Sweden) yielded positive results for *Solenopsis invicta* (class 3) and negative results for *Vespula* species, *Polistes* species and *Apis mellifera*. The total IgE was 49 IU/mL.

A positive intradermal skin test was observed with *P goeldii* venom at a concentration of 0.1 µg/mL and with *S invicta* extract (IPI-ASAC Laboratory São Paulo, Brazil) at a concentration of 10 µg/mL. The tests performed in 2 nonatopic healthy controls were negative up to 10 µg/mL for both venoms.

The venom of *P goeldii* was subjected to two-dimensional sodium dodecyl sulfate polyacrylamide gel electrophoresis, with the detection of approximately 45 proteins, with a molecular weight of 10 to 66 kDa and an isoelectric point of between 4 and 10 (Figure, A). Immunoassays with the patient's serum revealed 4 IgE-reactive spots of between 30 and 45 kDa (Figure, B).

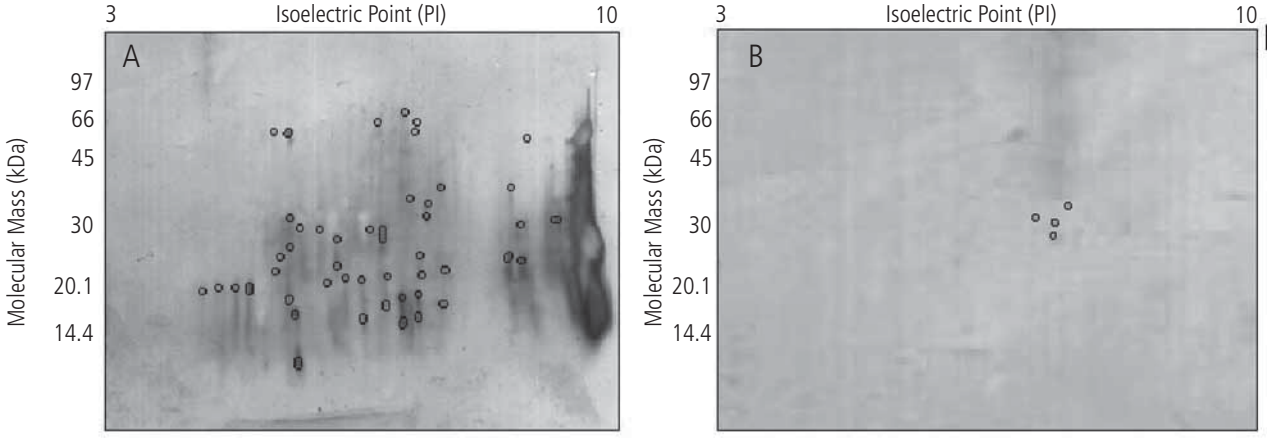

Figure. A, Profile of *Pachycondila goeldii* venom after two-dimensional sodium dodecyl sulfate polyacrylamide gel electrophoresis using a strip of 13 cm; isolectric point, 3 to 10. B, Membrane showing immunoglobulin E- reactive *Pachycondila goeldii* venom proteins with a molecular weight of 30 and 45 KDa with the patient's serum.

We were thus able to demonstrate that the patient had specific IgE to *P goeldii* venom and conclude that the anaphylaxis had been caused by the sting of this ant. The 2 previous stinging incidents also probably involved *P goeldii* because these ants have wings and fly during their reproductive period.

Another interesting observation in this case was the specific-IgE detected to *S invicta* both in the ImmunoCAP and intradermal skin test. We do not know whether this was due to a cross reaction or dual sensitization.

Pachycondyla species are predatory ants belonging to the *Ponerinae* subfamily. They are found throughout tropical and subtropical regions, feed on larvae and insects, have black bodies about 1 cm in length, and are found walking alone in gardens and forests.

Allergy to *Pachycondyla* species has been well described in Korea (*Pachycondyla chinensis*) and the United Arab Emirates (*Pachycondyla sennaarensis*) [4,5]. In Korea, one study demonstrated that 23% of the population was sensitized to *P chinensis* and that 1% had anaphylactic reactions [4] while another showed that *P chinensis* venom was cross-reactive with bee venom but not with *S invicta* venom [6].

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A Hidden Cause of Perioperative Anaphylaxis

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Key words: Povidone-iodine allergy. Perioperative anaphylaxis. Drug allergy. General anesthesia. Anaphylaxis.

Palabras clave: Reacción a povidona iodada. Anafilaxia perioperatoria. Reacción a medicamentos. Anestesia general. Anafilaxis.

The reported risk of perioperative anaphylaxis in patients requiring general anesthesia ranges from 1 in 3500 to 1 in 13 000 in French series [1,2] and from 1 in 10 000 to 1 in 20 000, with a mortality rate of between 3% and 6%, in an Australian study [3]. The most common drugs or substances involved are neuromuscular relaxant drugs, latex, and antibiotics. Nevertheless, it is often difficult to determine the cause of anaphylaxis in such patients because they receive multiple medications within a short time, there are other exposures (eg, latex), and allergy testing has limitations. Identification of the culprit agent can thus be a challenge to the allergist, and close cooperation between anesthetists and allergists is essential to establish the cause of the reaction and prevent future anaphylactic reactions.

We report the case of a 42-year-old nonatopic man who developed anaphylaxis during removal of metalwork in his ankle. The patient was referred to our department by the anesthetist. An hour and a half after induction of anesthesia, he had developed generalized urticaria, tongue swelling, upper airway breathing difficulty, and a drop in systolic blood pressure from 124 to 94. The symptoms improved immediately after administration of intravenous hydrocortisone and chlorpheniramine. The patient had tolerated general anesthesia in 2004, although the medications he received are not known.

The information contained in the anesthesia chart provided by the anesthetist is shown in the Table.

In the allergy assessment carried out 3 months after the reaction, the patient reported having tolerated codeine, paracetamol, and dextropropoxyphene since the reaction. Following the European Network Drug Allergy guidelines, we performed intradermal testing for midazolam (1/1000, 1/100, 1/10 dilution), propofol (1/1000, 1/100, 1/10 dilution), morphine (1/10 000, 1/1000 dilution), and fentanyl (1/1000, 1/100, 1/10) [4]. The results were negative for propofol and fentanyl but equivocal for midazolam and morphine. Additional subcutaneous incremental tests with midazolam up to 1 mg and morphine up to 5 mg were both well tolerated. An oral challenge test with ketorolac up to 30 mg was also well tolerated. Skin prick tests with latex and neat iodine were also negative.

In view of the negative results, we contacted the anesthetist, who confirmed that the patient had received povidone-iodine (Videne) rather than iodine. The solution had been swabbed

Table. Drug Administration and Onset of Reaction Times

9.30 AM	Induction of anesthesia (midazolam, fentanyl, propofol), iodine skin preparation
9.45 AM	Ketorolac
9.50 AM	Paracetamol and morphine
10.00 AM	Ketorolac and morphine
10.45 AM	Crepe bandage applied to ankle
11.00 AM	Anaphylaxis

over the surgical wound at the beginning of surgery (9.30 AM) and at the end of surgery (10.45 AM), 15 minutes before the onset of the generalized reaction. We performed a skin prick test with neat povidone-iodine and obtained a positive result (wheal 22 mm, flare 10 mm). Identical tests performed on a control group of 10 adults (5 atopic and 5 nonatopic) were all negative. We therefore suspected that the patient had reacted to the povidone-iodine applied at the end of the surgery and believe that a small quantity was probably introduced into the intravascular passage through the surgical wound. The patient was diagnosed with perioperative anaphylaxis secondary to povidone-iodine. We recommended that the patient should avoid contact with povidone-iodine antiseptics, particularly in the case of incisions or mucosal exposure. As an alternative, we recommended antiseptic agents such as chlorhexidine. Because there is no cross-reactivity with iodine, we reassured the patient that he could tolerate antithyroid drugs, contrast media, and seafood [5].

In summary, we present an exceptional case of anaphylaxis secondary to povidone-iodine applied to a surgical wound. Even though povidone-iodine is widely used as an antiseptic in surgery, type I immunoglobulin E-mediated reactions are extremely unusual. We found only 5 such reports in the literature, 3 of which were related to the application of povidone-iodine in the mucosa [6-10]. Allergic contact dermatitis is more common with povidone-iodine. This case report confirms the importance of checking anesthesia charts and considering povidone-iodine and other antiseptics when investigating possible causes of perioperative anaphylaxis. We recommend including the study of antiseptics in the routine diagnostic approach to perioperative reactions.

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2-Phenoxyethanol-Induced Contact Urticaria and Anaphylaxis

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Key words: Anaphylaxis. Contact. Cosmetics. Phenoxyethanol. Urticaria.

Palabras clave: Anafilaxia. Contacto. Cosméticos. Fenoxietanol. Urticaria.

A 42-year-old woman presented with a 6-month history of flares of hives lasting 30 minutes. The hives, which were self-resolving, developed on skin to which any of a large number of products had been applied. These products included moisturizers (Natural Honey Body Milk Primavera Verano and Rosaliac Hydratant Perfecteur), sun care products (After sun Isdin Pediatrics, Vichy Capital Soleil Après-soleil, Delial Ambre Solaire Niños 50+), skin cleansing solutions (Shiseido pureness Eau Dèmaquillante), and other products such as shampoos

and toothpaste. Three months before referral, on applying a moisturizer (Natural Honey Body Milk Primavera Verano) to her arms and legs, the patient immediately developed hives in the contact zone, followed by rhinorrhea, dyspnea, and dizziness without loss of consciousness. Spontaneous recovery occurred within approximately half an hour. She also reported perennial and intermittent nasal symptoms following direct exposure to house dust only.

A skin prick test battery performed with airborne allergens (*Dermatophagoides pteronyssinus, Lepidoglyphus destructor, Tyrophagus putrescentiae, Lolium perenne, Parietaria judaica, Plantago lanceolata, profilin, Alternaria alternata, Cladosporium herbarum, cat epithelium, dog epithelium*, and latex) showed positivity to *D pteronyssinus* only, leading to a diagnosis of intermittent allergic rhinitis.

Interestingly, immediately after using the blue ink pen (the brand name was not recorded) to mark the skin test, the patient developed hives in the area of these marks. We rubbed the skin on the patient´s back to test for dermographism but the result was negative.

We conducted an open test using the moisturizer in question and the patient developed an immediate urticarial reaction confined to the application area.

On studying the labels of all the cosmetics the patient had used (those that had caused reactions and those that had not), we suspected that 2-phenoxyethanol (CAS 122-99-6) might have been responsible for the reactions.

To test this theory, we conducted an open test on the volar aspect of the forearm with Euxyl-K 400 (80% methyldibromo glutaronitrile, 20% 2-phenoxyethanol) 0.1% in petrolatum—which showed minimal positivity—and with 2-phenoxyethanol 1% in petrolatum—which showed clear, immediate positivity followed by spontaneous resolution in about 30 minutes. The low positivity observed with Euxyl-K 400 may be related to the fact that 2-phenoxyethanol is present at a very low concentration (0.02%) in this compound.

As 2-phenoxyethanol is an ingredient in many vaccines, a prick test was performed with Infanrix-Hib (2-phenoxyethanol 5mg/mL) in our patient (positive) (Figure) and in 5 controls (negative).

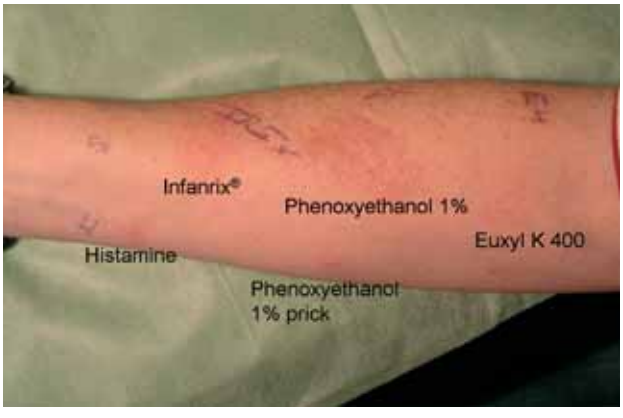

Figure. Open test with Euxyl K-400, prick and open test with 2-phenoxyethanol and prick test with Infanrix.

The patient was diagnosed with 2-phenoxyethanol-induced contact urticaria and anaphylaxis. Phenoxyethanol, a colorless oily liquid, is a glycol ether that is commonly used in cosmetics for its antibacterial and antifungal properties. It is increasingly being used in vaccines as a substitute for thiomersal [1] and is also a component of pen inks and more rarely ear drops [2,3]. Moreover, 2-phenoxyethanol is one of the ingredients of Euxyl-K 400, which is widely reported to cause contact dermatitis, although most of the cases have been attributed to methyldibromo glutaronitrile [4]

The nature of the reaction, the cutaneous test results, and the fact that the patient was atopic (*D pteronyssinus* sensitization) all suggest that the reaction could be IgE-mediated, although we did not investigate this possibility.

There are 3 reports of 2-phenoxyethanol-induced contact urticaria in the literature [5,6,7] The presence of specific IgE was investigated in one of them (negative), but the authors did not explain the method employed [7].

This new case of 2-phenoxyethanol-induced contact urticaria is particularly interesting in that it actually led to an anaphylactic reaction. 2-Phenoxyethanol should be considered in cases of contact urticaria with cosmetics and pen ink. We advised our patient to carefully read the labels of all cosmetics before using them and suggested that she perform an open test on a small area of skin in case of doubt. We also advised her to avoid vaccines containing 2-phenoxyethanol since it is unclear how she would tolerate these.

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Garlic-Induced Severe Anaphylaxis in a Nonatopic Patient

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Key words: Garlic. Allergy. Anaphylaxis. Food allergy. *Allium sativum*.

Palabras clave: Ajo. Alergia. Anafilaxia. Alergia alimentaria. *Allium sativum*

Garlic is a well-known cause of occupational allergy [1], allergic contact dermatitis [2,3], rhinitis, and asthma [4]. However, it rarely causes immunoglobulin (Ig) E–mediated reactions such as anaphylaxis [5] and acute urticaria [6].

A 52-year-old man visited our department for evaluation in May 2009, reporting 2 anaphylactic episodes that had occurred in the past 10 months, both in association with the ingestion of garlic.

The first episode had occurred 8 months before the visit. Approximately 15 minutes after eating mackerel with string beans and a yogurt, cucumber, and garlic sauce, he developed erythematous, pruritic palms and angioedema of the upper lip, followed by dizziness and finally, loss of consciousness. He was transferred immediately to the emergency department and hospitalized for 24 hours with total remission of symptoms.

The second episode occurred 40 days prior to the visit while the patient was eating cod with a garlic-based sauce. The symptoms were erythema and pruritus of the palms and angioedema of the upper lip about 30 minutes after ingestion. He was transferred to the emergency department, where he received intravenous dimethindene and corticosteroids, with remission of symptoms after 1 hour.

The patient stated that he had never experienced any reactions on eating garlic in the past and had subsequently tolerated onion, leek, and various types of fish. He had no history of atopy and the only medication he had received was colchicine to treat elevated uric acid and on occasions nonsteroidal antiinflammatory drugs, without adverse reactions.

We tested the patient's serum using the CAP-FEIA method (Phadia Laboratories, Uppsala, Sweden) for specific IgE to the most common airborne allergens (all of which were negative) and to specific food, namely fish (-), salmon (–), cabbage (–), walnut (–), cucumber (–), onion (0.75 kU/L, class 2) and garlic (0.81 kU/L, class 2). Total serum IgE was 56.0 kU/L and serum tryptase levels were normal.

We carried out skin prick tests (SPTs) with commercial extracts of the most common airborne allergens (all negative) and food allergens such as garlic (+), cucumber (–), cod (–), sardine (–), tuna (–), white fish (–), blue fish (–), shrimp (–), and crustacean (–).

We conducted prick-to-prick tests with garlic, onion, and leek in our patient (Table) and in 3 atopic and 4 nonatopic controls (to exclude the possibility of an irritant reaction). For ethical reasons, we did not conduct oral provocation tests with garlic.

Table. Skin Prick Test and Prick-to-Prick Test With Garlic and Other Members of the Liliaceae Family

	SPT Wheal Diameter, mm	Prick-to-Prick Wheal Diameter, mm
Commercial garlic extract	7×8	ND
Commercial onion extract	Negative[a]	ND
Garlic clove	4×6	4×4
Young garlic	4×5	3×3
Garlic clove germ	9×9	6×6
Cooked young garlic	3×3	Negative[a]
Cooked garlic clove germ	3×4	ND
Onion bulb	ND	Negative[a]
Young onion	ND	Negative[a]
Cooked young onion	ND	Negative[a]
Leek	ND	3×3
Cooked leek	ND	Negative[a]

Abbreviation: ND, not done.
[a]Wheal diameter, <3 mm

The SPTs and prick-to-prick tests with extracts and fresh foods, respectively, proved to be the most reliable methods for diagnosing garlic allergy and locating the culprit allergen (garlic germ induced the greatest wheal reaction).

The CAP specific IgE test for onion and garlic yielded the same low positive results (class 2) but only the ingestion of garlic had caused symptoms in our patient. This finding calls into question the specificity of the CAP test for onion and/or garlic. The result could possibly be explained by the presence of garlic-specific IgG antibodies occupying the same epitopes against which garlic-specific IgE is directed (as was demonstrated in a case of garlic-induced urticaria, where immunoblotting tests became intensely positive after IgG was removed from the patient's serum sample [6]).

In the single published case of garlic-induced anaphylaxis, the patient had a strong history of atopy and episodes of food-dependent, exercise-induced anaphylaxis [5]. She was allergic to young garlic and to various seeds and nuts. The causative allergen was found to be heat-sensitive. Our patient, who had a near-fatal anaphylactic reaction to garlic, was monosensitized and had no history of atopy or occupational exposure to garlic.

Although garlic-induced anaphylaxis is extremely rare, our case shows that it can occur in middle-aged, nonatopic patients without occupational exposure. The culprit allergen seems to be largely heat-labile and mostly located in the germ of the garlic clove. Cross-reactivity between garlic and other members of the Liliaceae family (onion and leek) was not observed in our patient.

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Duration of Breastfeeding and the Risk of Childhood Asthma in Children Living in Urban Areas

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Key words: Asthma. Breastfeeding. Child.

Palabras clave: Asma. Lactancia materna. Niño.

Asthma is one of the most common chronic diseases during childhood, and the role of breastfeeding in its development remains controversial [1-4]. Our objective was to quantify the association between the duration of breastfeeding and the occurrence of asthma in infancy.

We invited 5736 children randomly selected from elementary schools in the city of Porto (Portugal) and their parents to participate in the study: 3327 parents signed and returned the consent form. Anthropometric measurements were taken and parents answered a questionnaire on

Table. Prevalence of Physician-Diagnosed Asthma in Children by Categories of Possible Confounders.

	Girls			Boys		
	N	n	%	N	n	%
Total	978	72	7.4	968	108	11.2
Age, y						
5	15	2	13.3	16	4	25.0
6	212	12	5.7	240	20	8.3
7	265	20	7.5	246	29	11.8
8	210	17	8.1	209	29	13.9
9	236	16	6.8	214	23	10.7
10	40	5	12.5	43	3	7.0
	P for trend=.498			*P* for trend=.942		
Body mass index						
Not overweight	579	37	6.4	566	62	11.0
Overweight	263	19	7.2	243	26	10.7
Obese	106	13	12.3	135	16	11.9
	P for trend=.058			*P* for trend=.833		
Atopic mother						
Yes	246	32	13.0	245	38	15.5
No	702	36	5.1	683	61	8.9
	P<.001			*P*=.004		
Atopic father						
Yes	169	18	10.7	162	28	17.3
No	723	43	5.9	710	68	9.6
	P=.029			*P*=.005		
Mother's age at birth						
1st tertile (10-27 y)	365	27	7.4	332	42	12.7
2nd tertile (27-32 y)	344	23	6.7	324	31	9.6
3rd tertile (32-52y)	262	21	8.0	305	33	10.8
	P for trend=.813			*P* for trend=.446		
Smoking inside the house						
Yes	295	18	6.1	259	30	11.6
No	677	54	8.0	708	78	11.0
	P=.305			*P*=.805		
Breastfeeding						
Never	54	6	11.1	61	8	13.1
< 1 mo	152	11	7.2	125	19	15.2
1 to 3 mo	267	22	8.2	227	29	12.8
3 to 6 mo	210	13	6.2	234	25	10.7
6 to 12 mo	187	13	7.0	206	17	8.3
> 12 mo	95	6	6.3	104	8	7.8
	P for trend=.329			*P* for trend=.022		

sociodemographic characteristics, asthma, and breastfeeding. Overall, 2462 questionnaires were returned and 486 children were excluded, leaving a final sample of 1976 children aged 5 to 10 years. Asthma was defined as self-reported and physician-diagnosed. Unconditional logistic regression models adjusting for confounders were applied to estimate the association between asthma and breastfeeding.

Asthma was more prevalent in boys than in girls (11.2% vs 7.4%, *P*=.004) and in children who had an atopic mother (13.0% vs 5.1%, *P*<.001 for girls; 15.5% vs 8.9%, *P*=.004 for boys) or father (10.7% vs 5.9%, *P*=.029 for girls; 17.3% vs 9.6%, *P*=.005 for boys) (Table). In boys, increased duration of breastfeeding was associated with a lower probability of a medical diagnosis of asthma (*P*=.022) (Table). Breastfeeding for more than 6 months was inversely related to the presence of asthma in boys (odds ratio, 0.554; 95% confidence interval, 0.323-0.949). The results observed for other cutoffs (3 and 12 months) were similar.

Breastfeeding is important for the development of the immune system in infancy and, therefore, may influence the incidence and severity of asthma [2]. Until some time ago, the role of breastfeeding in preventing asthma and allergy was unquestionable. However, in 2000, data from a cohort in Tucson (Arizona, USA) showed that breastfeeding was generally protective against atopy, although breastfeeding mothers with high immunoglobulin E levels had children with higher rates of asthma and allergy [4]. The results from a New Zealand cohort study showed that children who were breastfed for at least 4 weeks were more likely to have asthma as young adults [3]. The methodology of both studies was criticized by Peat et al [5]. The duration of follow-up and age of onset of asthma are the main reasons why in some studies breastfeeding protects against atopy and asthma, whereas in others the risk increases. If breastfeeding delays the onset of asthma, current prevalence of asthma would be lower in breastfed babies than in nonbreastfed babies, although it would be similar later in life [6]. In the present study, occurrence of asthma at the age of 5 to 10 years was an outcome measure. Other reasons for the heterogeneity of these results are as follows: duration of breastfeeding in the study population, which varies considerably; immunological complexity of breast milk, which contains allergens that may be sensitizing in some cases and protective in others; exposure to diverse environmental factors; use of different cutoffs for established breastfeeding; and different confounding factors. The reasons for the gender-specific differences we found could be numerous, because asthma is usually a multifactorial disease in which genetic, environmental, pathophysiologic, and immunologic aspects play a part.

We concluded that prolonged breastfeeding (≥6 months) could reduce the risk of asthma in boys who live in the city.

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Selective Immunoglobulin M Deficiency in a Patient With Refractory Giardiasis

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Key words: *Giardia lamblia*. Giardiasis. Refractory. Selective IgM deficiency.

Palabras clave: *Giardia lamblia*. Giardiasis. Refractoria. Deficiencia selectiva de IgM.

Giardia lamblia is one of the most common gastrointestinal parasites in the world. Failure of treatment in patients with giardiasis is infrequent and usually occurs in immunosuppressed patients such as those with HIV infection and common variable immunodeficiency [1]. Giardiasis has only been found in 1 case of selective immunoglobulin (Ig) M deficiency [2]. The present report describes a patient with selective IgM deficiency who first presented with refractory giardiasis.

In 2007, a 16-year-old Thai boy presented with a 2-year history of chronic diarrhea and weight loss. His medical and family histories were unremarkable, as was a physical examination. Stool examination revealed persistent

G lamblia cysts and trophozoites despite multiple courses of metronidazole and imidazole. Esophagogastroduodenoscopy revealed diffuse multiple small white nodules and ulcers in the duodenum. Histopathology testing revealed acute duodenitis with *G lamblia* trophozoites.

Routine laboratory tests were unremarkable. HIV-1 antibody testing was negative. Serum Ig levels were within the normal range except for IgM, which was constantly below the normal range. A low isohemagglutinin titer was also observed. Flow cytometry for lymphocyte subsets and the lymphocyte proliferation test with phytohemagglutinin were normal. The hepatitis B antibody titer was initially below the protective level, although the patient was able to make an antibody response after vaccination. The results of the immunological workup are summarized in the Table.

Based on the above findings, primary selective IgM deficiency with refractory giardiasis was diagnosed. Combination treatment with metronidazole and albendazole was administered, as quinacrine is not available in Thailand. The regimen failed to eradicate *G lamblia*, but reduced the frequency of diarrhea and enabled the patient to gain weight. The patient remained healthy despite having mild intermittent diarrhea and persistent low serum IgM levels 1 year after the initial diagnosis.

Selective IgM deficiency is a rare disorder characterized by isolated low levels of serum IgM with no other identifiable immunodeficiency [3]. Serum IgM levels are usually less than 20 mg/dL or 2 SD or 10% below the age-adjusted mean [3-5]. To date, there have been only 200-300 reports of selective IgM deficiency worldwide [3].

Selective IgM deficiency is classified as primary and secondary, the latter being more common. Diagnosis of primary selective IgM deficiency requires the exclusion of factors such as autoimmune disease, malignancy, hematologic disorders, gastrointestinal disease, and immunosuppressive treatment [4-6].

The clinical significance of selective IgM deficiency is uncertain, as the pathogenesis and clinical manifestations of this rare condition are heterogeneous [6]. Furthermore, genetic abnormalities have not yet been identified [7,8]. The condition may be complicated by bacterial, viral, fungal, and parasitic infections. The most common are recurrent respiratory tract infections such as otitis media, chronic rhinosinusitis, bronchitis, and pneumonia [3-5]. However, skin infection, diarrhea, meningitis, septicemia, and death have also been reported [5-7].

In patients with selective IgA deficiency, mucosal IgM may be the primary mucosal antibody, although its importance in healthy individuals remains unknown [5]. It may play an important part in mucosal immunity to giardiasis. To the best of the author's knowledge, this is the first report of refractory giardiasis with selective IgM deficiency.

The pathogenesis of selective IgM deficiency has yet to be firmly established. The likely mechanisms are heterogeneous

Table. Results of the Immunological Workup

Date	Sep 2007	Nov 2007	Nov 2008	Reference Range
Immunoglobulin level				
IgM	<21.4	<17.4	<18.6	40-230 mg/dL
IgA	185	153	136	70-400 mg/dL
IgE		6		<100 IU/mL
IgG	1060	995	910	700-1600 mgdL
IgG1		633		370-1280 mgdL
IgG2		207		106-610 mg/dL
IgG3		51.6		18-163 mg/dL
IgG4		8.4		4-230 mg/dL
Lymphocyte subsets				
CD3, %		1989 (67%)		960-2430/μL
CD4, %		950 (32%)		470-1404/μL
CD8, %		890 (30%)		360-1250/μL
CD19, %		20		7.7-25.4
CD56, %		17		3.9-38.5
Antibody tests				
Anti-HBs		Negative	189	>10 mIU/mL
Isohemagglutinin titer		Anti A (1:4)		
Antinuclear antibody		<40		<40
Antithyroglobulin		0.58		<4.11 IU/mL
Antithyroid peroxidase		0		<5.61 IU/mL

Abbreviation: Ig, immunoglobulin.

lymphocyte abnormalities, including functional defects of B lymphocytes in terminal differentiation and T lymphocyte dysfunction (decreased helper T cell activity or excessive regulatory T-cell function) [3-8].

Management of patients with selective IgM deficiency includes antibiotic prophylaxis and immunoglobulin replacement therapy in cases of recurrent infections or specific antibody deficiency. Ig levels should be recorded periodically to monitor disease progression [3].

In conclusion, selective IgM deficiency should be considered a possible cause of refractory giardiasis.

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Immunoglobulin E–Mediated Anaphylaxis to Rabeprazole

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Key words: Anaphylaxis. Rabeprazole. Proton pump inhibitors. Cross-reactivity. Drug allergy.

Palabras clave: Anafilaxia. Rabeprazol. Inhibidor de la bomba de protones. Reactividad cruzada. Alergia medicamentosa.

Proton pump inhibitors (PPIs) are used to treat peptic ulcers. They are generally well tolerated, and the few available published case reports describe anaphylaxis to omeprazole [1], lansoprazole, and pantoprazole, but not to rabeprazole [2,3].

A 66-year-old woman consulted for a generalized allergic reaction approximately 30 minutes after taking her medication (nebivolol, levothyroxine) and rabeprazole (only on the day the reaction occurred). Her symptoms were generalized itching, angioedema of the upper lip, and flushing on the upper trunk. She was immediately given a levocetirizine tablet and intramuscular methylprednisolone was administered. Nevertheless, she lost consciousness and experienced urinary incontinence.

She was admitted to hospital and received dimethindene, hydrocortisone, ranitidine, and normal saline. Two hours later, all her symptoms had resolved.

She continued to take levothyroxine and nebivolol, but not rabeprazole or any other PPI. She had previously taken rabeprazole for short periods due to chronic peptic ulcer.

Nine months previously, she had experienced an episode of generalized itching, vomiting, and fainting shortly after taking her daily medication and a rabeprazole tablet. The symptoms resolved within 2 hours with no treatment. She had no history of atopy. Total serum immunoglobulin (Ig) E was 43 IU/mL and serum tryptase was 3.2 μg/L.

The result of a skin prick test (SPT) with common aeroallergens (Stallergènes, Antony, France) and food allergen extracts was negative, and the patient had no history of respiratory or food allergy. SPT with omeprazole, pantoprazole, lansoprazole, and esomeprazole tablets diluted in 1 mL normal saline were negative while only SPT with rabeprazole was positive (Table).

All the above tests were negative in 5 healthy controls. Histamine (10 mg/mL) and normal saline were used as positive and negative controls.

Intradermal testing with 10-fold dilutions of intravenous omeprazole, pantoprazole, and esomeprazole was negative. Intradermal testing was not performed with rabeprazole or lansoprazole, because the intravenous solution is not commercially available in Greece. Histamine at 0.1 mg/mL and normal saline administered intradermally were used as positive and negative controls.

Graded challenge to omeprazole (placebo and a dose of

Table. Results of Skin Prick Testing and Intradermal Testing With Proton Pump Inhibitors

	SPT Wheel/Flare Diameter, mm	ID 1/1000	ID 1/100	ID 1/10
Omeprazole (20-mg cap)/1 mL NS 0.9% (SPT) Omeprazole IV 4 mg/mL (ID)	Neg Neg	NP Neg	NP Neg	NP Neg
Pantoprazole (40 mg tab)/1 mL NS 0.9% (SPT) Pantoprazole IV 4 mg/mL (ID)	Neg Neg	NP Neg	NP Neg	NP Neg
Esomeprazole (40-mg tab)/1 mL NS 0.9% (SPT) Esomeprazole IV 2 mg/mL (ID)	Neg Neg	NP Neg	NP Neg	NP Neg
Lansoprazole (30-mg tab)/1 mL NS 0.9% (SPT)	Neg	NP	NP	NP
Rabeprazole (20-mg tab)/1 mL NS 0.9% (SPT)	12 × 5/16 × 11	NP	NP	NP
Histamine	12 × 7/27 × 15	18 × 11/26 × 41		
NS 0.9%	Neg	Neg		

Abbreviations: cap, capsule; ID, intradermal; NEG, negative; NP, not performed; NS, normal saline; SPT, skin prick test; tab, tablet.

2, 6, and 20 mg every hour) and lansoprazole (placebo and a dose of 3, 9, and 30 mg every hour) were well tolerated with no reaction. Esomeprazole was not tested. Informed consent was obtained.

Our patient experienced 2 IgE-mediated anaphylactic reactions to rabeprazole, as confirmed by her history and positive SPT results. Other causes of anaphylaxis, such as food, other drugs, and insect stings, were ruled out.

A review of the literature reveals this to be the first reported case of anaphylaxis induced by rabeprazole. There are only 2 published studies on anaphylaxis to lansoprazole and cross-reactivity to rabeprazole. One reported positive intradermal test results with rabeprazole and the other a negative SPT result but a positive challenge with rabeprazole [2,3].

SPT and intradermal testing are accurate and simple methods to identify the cause of an allergic reaction [1,4]. We conclude that the allergen must have been the drug itself (rabeprazole) and not a metabolite of this drug, as confirmed by positive skin test results [4]. It was noteworthy that our patient had negative skin test results (SPT and intradermal tests) to all the other PPIs and that a graded challenge to 2 of them was well tolerated.

We observed no cross-reactivity with other members of this group of drugs, and these results are consistent with those of our previous report, in which sensitization to omeprazole did not extend to all the members of the PPI group [4]. However, some authors have reported extended cross-reactivity [5,6].

Graded challenge to PPIs for which the skin test result is negative seems to be a reliable and safe approach when choosing an alternative drug [6]. Cross-reactivity to the whole group of PPIs should not be assumed without first performing skin tests.

Anaphylaxis to PPIs, especially rabeprazole, is rare. However, health care professionals should be aware of this possibility when attempting to establish a diagnosis.

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Exposure to Risky Concentrations of *Dermatophagoides* Allergens in a High-Altitude Population (Quito, 2800 m Above Sea Level in the Andean Mountains)

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Key words: Quito-Ecuador. Andes Mountains. High-altitude populations. Dermatophagoides group 1 allergens. Rhinitis and asthma.

Palabras clave: Anafilaxia. Rabeprazol. Inhibidor de la bomba de protones. Reactividad cruzada. Alergia medicamentosa.

In our study on domestic mite fauna in high-altitude cities (2500-2800 m above sea level) in Ecuador, we identified 23 mite species and showed that all mattresses studied contained detectable levels of Der p 1, Der f 1 or both [1]. In another study, we demonstrated that up to 79% of asthmatics in Quito were sensitized to *Dermatophagoides pteronyssinus* and *Dermatophagoides farinae* [2]. Quito (the capital of Ecuador, annual mean relative humidity of 75%, and average temperature of 15ºC) is located in the Andes Mountains, 2800 m above sea level. As part of an investigation into risk factors for asthma, we performed an exploratory pilot study to determine what percentage of a group of 65 randomly selected individuals were exposed to risky quantities of *Dermatophagoides* allergens during a 1-year period. To obtain natural levels of allergens, we selected nonallergic households in order to rule out mattresses undergoing dust mite eradication. Participants were aged 1 to 82 years (mean, 34.8 y); 26 were males and 39 were females. Samples were collected from 5 different homes each month (5 samples per month) beginning in January and ending in December 2005. In February, we collected 10 samples from 10 houses. A trained technician collected the samples from the entire surface of each mattress during a 2-minute period using a 1.4-kW portable vacuum cleaner (Trio 2, Electrolux, Brazil) and a Mitest Collector (Indoor Biotechnologies, Charlottesville, Virginia, USA). All samples were frozen immediately and for at least 48 hours to prevent mite proliferation. Der p 1 and Der f 1 levels were quantified using an enzyme-linked immunosorbent assay (Indoor Biotechnologies). Information on relative humidity was obtained from http://espanol.wunderground.com/history/airport. Statistical analyses were performed using the StatView program, version 4.53 (Abacus Concept Inc. Berkeley, California, USA).

Dermatophagoides allergen levels (geometric mean) and relative humidity (percentage) are shown in the Figure. Samples collected in houses with obvious signs of humidity (Der f 1) from mattresses with >10 years of use (Der p 1), and from owners aged <20 years (Der p 1) contained larger quantities (P<.05) of mite allergen. Der p 1 allergen levels >2 µg/g were detected in 26 (40%) samples. Five (7.6%) of the 65 samples had Der p 1 levels >10 µg/g. Levels of Der f 1 allergen >2 µg/g were present in 17 (26.1%) samples. Six (9.2%) of the 65 samples had Der f 1 levels >10 µg/g.

The 2 allergens only appeared together in 8 (12.3%) samples, and in both cases levels were >2 µg/g. One of the samples (1.5%) had levels >10 µg/g. Therefore, 35 (53.8%) of the individuals studied were exposed to >2 µg/g of *Dermatophagoides* group 1 allergen (Der p 1 and/or Der f 1) and 10 (15.3%) to >10 µg/g of *Dermatophagoides* group 1 allergen. While we are aware that a sample of 65 individuals is not statistically representative of the entire population of Quito, the results of this pilot study suggest that a large proportion of people living in Quito could be exposed to quantities of dust mite allergens capable of inducing sensitization and respiratory symptoms [3]. Susceptible patients should take control measures (eg, cleaning, humidity control, mite-proof covers),

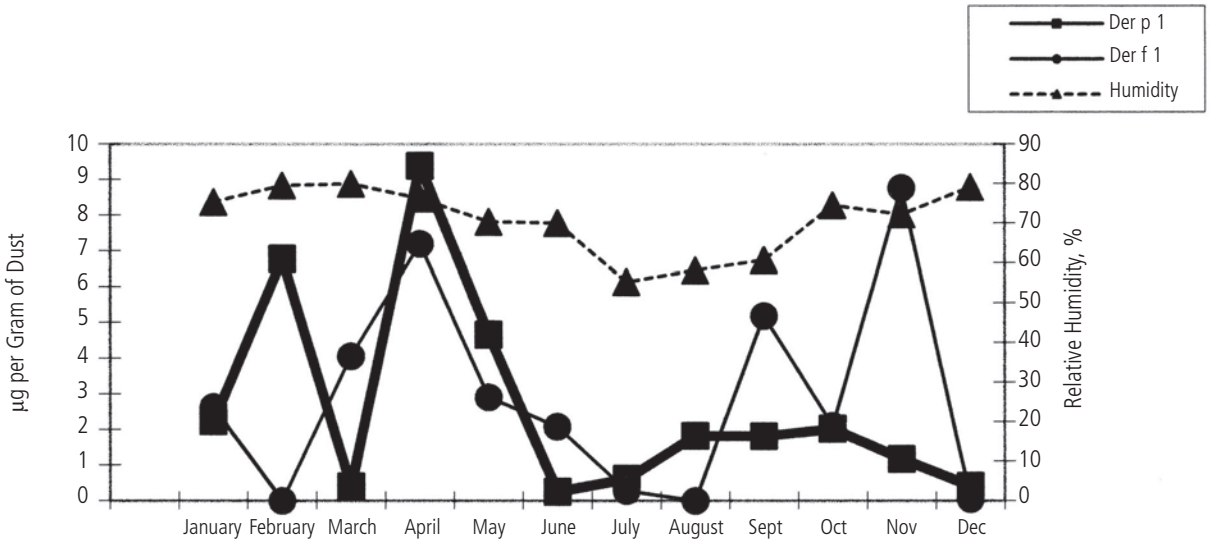

Figure. Levels of humidity (%) and levels of mite allergen (geometric mean) obtained from 65 samples of mattress dust collected over 12 consecutive months. Differences between the monthly levels of allergen or between the variations of humidity and the levels of allergen were not significant.

especially in the case of people with allergic airway diseases or nonallergic children with an atopic background. Unlike high-altitude cities in the northern hemisphere [4-8], the temperature and humidity in Quito may contribute to the presence of dust mites and their allergens throughout the year.

Data have not been presented in abstract or poster form at conferences.

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ERRATUM

Leukotriene B4 and 8-Isoprostane in Exhaled Breath Condensate of Children With Episodic and Persistent Asthma

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- In the list of authors, JB Ramírez should have read J Belda Ramírez.
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