

Anaphylaxis in a child playing in tall grass

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Key words: anaphylaxis; dyspnea; nasal allergy; orchard grass; urticaria.

An 8-year-old male presenting with stridor and urticaria was admitted to our emergency room. One hour prior to admission, he had been playing with friends in an area of overgrowth next to a river. Fifteen minutes after entering the area, he suddenly felt something stuck in his throat and an itchy urticaria papulosa appeared on his body.

On examination, urticaria papulosa was observed over the patient's entire body. He also complained of a slight dyspnea with stridor. He could not inhale through his nose because it was obstructed by nasal edema, and an examination with a laryngeal fibroscope revealed slight vocal cord and tracheal edema. The patient's oxygen saturation was within the normal range, and a lateral tracheal X-ray film showed mild edema on the anterior surface of his trachea, similar to the findings for croup. An auscultation study did not reveal any abnormal breath sounds other than stridor during both inhalation and exhalation. A chest X-ray film of the patient's lungs was normal. Both the patient and his mother had a history of atopic dermatitis and nasal allergy. We suspected an allergic reaction causing urticaria and laryngeal edema. The patient was treated with an inhalation of epinephrine, but his stridor was unaffected. The

Anaphylaxis after exercise in grass in children.

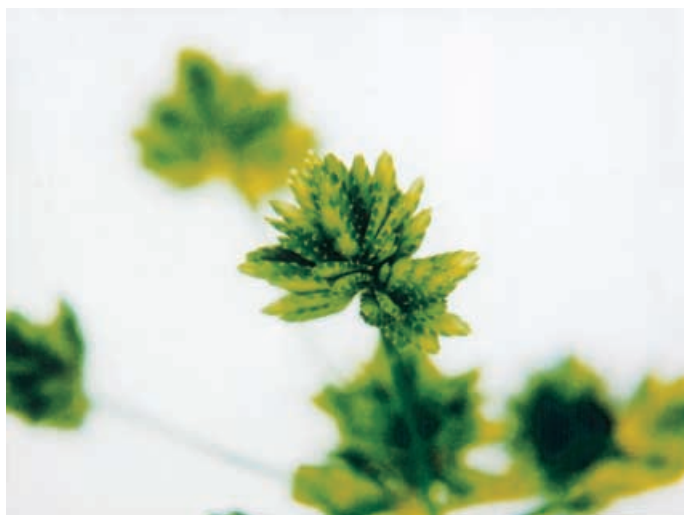


Figure 1. Orchard grass.

patient was then intravenously treated with 150 mg of hydrocortisone. Six hours after the injection, the edema and the urticaria had completely disappeared and his hoarseness had resolved.

The patient's father was given eye protection and a respiratory mask and asked to provide a sample of the plants growing in the area where the boy had been playing. The plant sample was subsequently identified as orchard grass (Fig. 1). A serum allergic reaction test (UniCAP® specific-IgE, Pharmacia Diagnostics AB, Uppsala, Sweden) produced a strongly positive reaction (67.5 UA/ml, normal range <0.34 UA/ml) to orchard grass. The serum IgE concentration and eosinophil count were also elevated at the time of the patient's admission. Several other inhalants and food allergens (multiple antigen simultaneous test) tested negative, with the exception of Japanese cedar pollen.

The patient had a history of nasal allergy (Japanese cedar pollen; peak season, February through April). The present attack occurred in mid-June,

during the peak season of orchard grass pollen. The patient had played in the same area during the previous 2 years. According to his mother, the patient had taken anti-allergic medicine for Japanese cedar pollen from the beginning of February through to the end of April for the last 2 years. According to his father, only orchard grass was present in the area where the boy had been playing. Similar critical allergic reactions caused by plants have been reported (1, 2). Anaphylaxis caused by the direct exposure of abraded skin to grass was reported in a patient with grass pollen allergy and a previous history of contact urticaria (3). Also, a case of anaphylaxis causing respiratory arrest after running in a wheat field (4) and a case of anaphylaxis while on an alpine slide (5) have been reported. In the present case, we could not find any injuries or bites from insects on his skin. We did not ask the patient to undergo an orchard grass pollen inhalation test because of the risk involved.

This is the first report of a critical allergic reaction induced by the inhala-

tion or direct contact with the pollen of orchard grass.

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Oral wheat flour anaphylaxis related to wheat α -amylase inhibitor subunits CM3 and CM16

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Key words: α -amylase inhibitor; anaphylaxis; food allergy; IgE-immunoblotting; wheat allergens.

Wheat α -amylase inhibitor subunits related to food allergy.

Several 12–16 kDa salt-soluble proteins from wheat flour, members of the α -amylase/trypsin inhibitor family, have been implicated as major allergens associated with baker's

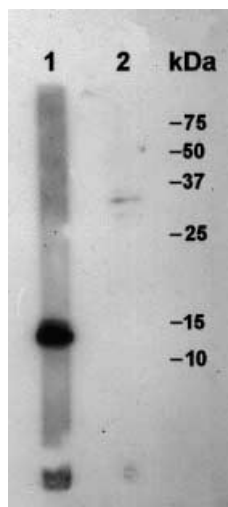


Figure 1. IgE-immunoblotting of saline wheat flour extract. (1) patient's serum. (2) negative control. The molecular weight (kDa) of pre-stained markers run in parallel are indicated.

asthma (1) and more rarely with food allergy (2). A child who had anaphylaxis after ingestion of a five-cereal baby food when he was 9 months old has been reported here. The involvement of the wheat α -amylase inhibitor subunits, CM2, CM3 and CM16, was studied.

The 9-month-old patient tolerated milk and nongluten cereals (rice and corn).

Three days after introducing gluten cereals in his diet (five-cereal baby food), the patient suffered an anaphylactic reaction after eating some spoonfuls of the baby food.

Skin prick test (SPT) with flours (ALK-Abelló, S.A. Madrid, Spain) were positive for wheat (10 × 5 mm) and negative for rye, barley, corn and rice. SPT with a standard battery of aeroallergens were all negative. Specific IgE (Pharmacia CAP System, Uppsala, Sweden) was 17.9 kU/l for wheat flour, 0.71 kU/l for barley and 1.88 kU/l for rye. Prick by prick carried out directly with the five-cereal baby food was positive (7 × 5 mm). Provocation test was clearly positive – 40 min after eating four spoonfuls of baby food, the patient presented trunk urticaria, eyelid angioedema, cough, wheezing and vomiting, which was resolved with dexchlorfeniramine injection.

Figure 1 shows the result of the IgE-immunoblotting carried out with wheat

flour and the patient's serum. A broad band with a molecular weight close to 15 kDa is mainly detected in the wheat flour extract. This band(s) is probably a member of the wheat α -amylase inhibitor family. To further investigate the nature of the allergens involved, the 12–16 kDa wheat tetrameric inhibitor subunits CM2, CM3 and CM16, which are allergens involved in baker's asthma (1), were purified and used in skin prick test. A positive reaction was obtained with CM3 (10 × 5 mm) and CM16 (7 × 4 mm) and a negative reaction with CM2.

These results suggest that CM3 and CM16 may be the sensitizing allergens in patients experiencing hypersensitivity reactions after the ingestion of wheat flour. A similar role for CM3 has been proposed in patients with atopic dermatitis related to wheat (3).

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Occupational asthma and allergic rhinitis due to xerographic toner

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Key words: methacrylates; occupational asthma; sick building syndrome; xerographic toner.

Acrylates are described as occupational asthmogens (1, 2), but to our knowledge, there are no published data on xerographic toner-induced allergic asthma.

Black xerographic toner basically consists of carbon black in binder resins, such as polystyrene-*n*-butyl methacrylate, polystyrene-*n*-butyl acrylate, etc. While taking photocopies, ozone, magnetizable metallic oxides, and components of thermally activated toner are released.

We present the case of a 44-year-old nonsmoking woman, who had been working for 8 years as a secretary in a primary school. For the past 2 years, she had been suffered from rhinorrhea, dyspnea and cough attacks, occurring 15–20 min after beginning of taking black and white photocopies, using toner containing polystyrene-*n*-butyl methacrylate. The patient reported only work-related symptoms.

Clinical examinations, as well as routine laboratory parameters, remained normal. The total IgE (CAP System, Pharmacia, Uppsala, Sweden) level was low (18.04 ku/l). Skin prick tests to common allergens (Allergopharma, Hamburg, Germany) were negative. The histamine provocation test showed bronchial hyperreactivity ($PC_{20} = 2.16$ mg/ml).

The patient was subjected to subsequent single-blind bronchial challenge tests with: thermally activated (80°C) methyl methacrylate, thermally activated (80°C) polystyrene, placebo (potato flour), and test at work-place-like conditions (taking photocopies for 30 min) monitored by spirometry and peak

A case of occupational asthma and rhinitis caused by xerographic toner, confirmed by specific bronchial provocation.

expiratory flow rate measurements. At the 18th minute of taking photocopies clinical symptoms of dyspnea, and a 21% fall in forced expiratory volume (FEV_1), followed by 24% fall at 1 h, and 19% decrease at 4 h were observed. The provocation with methyl methacrylate induced a 30% fall in FEV_1 at 1 h, with 24% decrease 4 h later. Tests with polystyrene and placebo did not cause any clinical or spirometric changes. Nasal lavage fluid was obtained 10 min before, and 30 min, 2, 4, and 24 h after the challenge. Only the work-place-like test and challenge test with methyl methacrylate, induced cellular and biochemical changes in nasal lavage. The increase in eosinophil proportion, up to 6% (work-place like) and 8% (methyl methacrylate) (Fig. 1), and permeability index (from 6.5% to 16.1% and from 9.1% to 19.7%, respectively) at 24 h were observed.

The reason for recognition of methyl methacrylate-induced asthma and rhinitis in this patient was the clinical response during specific challenge tests accompanied by changes in nasal lavage fluid. Asthma caused by xerographic toner is characteristic of the 'office microenvironment' and may be treated as one of those diseases related to 'sick building syndrome'.

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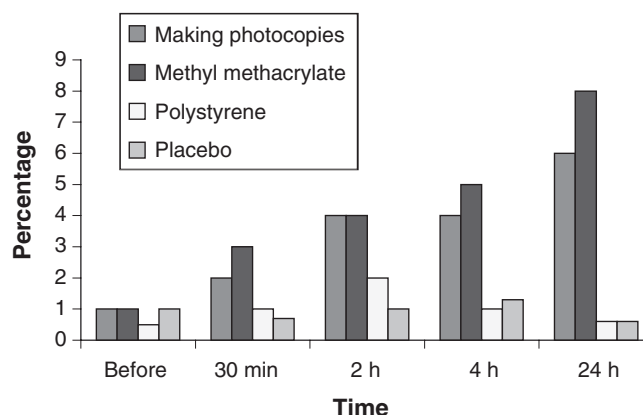


Figure 1. The proportion of eosinophils in nasal lavage fluid after challenge tests.

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Occupational contact allergy to ceftiofur

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Key words: ceftiofur; cephalosporins; contact allergy; contact dermatitis; cross reactivity; occupational.

Ceftiofur sodium (Excenel®, Pharmacia & Upjohn, Kalamazoo, MI) is a third-generation cephalosporin meant for exclusive veterinary use. This is mainly used for treating respiratory infections in pigs and cows

A case of extensive dermatitis for occupational contact allergy to ceftiofur (without cross-reactivity to other cephalosporins) is reported.

Table 1. Summary of skin tests results

Allergens	C/p	PhT	PT	ID
Ceftiofur	5%	+	+	+
PPL		nd	–	–
MDM		nd	–	–
Penicillin G	5%	–	–	–
Ampicillin	25%	–	–	–
Amoxycillin	20%	–	–	–
Cephalexin	20%	–	nd	nd
Cefixime	20%	–	nd	nd
Cephadroxile	20%	–	nd	nd
Cefamandole	20%	–	–	–
Cefuroxime	25%	–	–	–
Cephalotin	20%	–	–	–
Ceftriaxone	20%	–	–	–
Cephotaxime	25%	–	–	–
Ceftazidime	20%	–	–	–

C/p, concentration in petrolatum; PhT, patch test; PT, prick test; ID, intradermo-reaction; PPL, penicilloyl polylysine; MDM, minor determinant mixture, Allergopen®.

* Only delayed reaction; nd, not done.

All antibiotics have been tested in concentrations of 1 and 2 mg/ml except for penicillin, ranging from 0.1 to 10 000 IU/ml.

and for prevention of colibacillosis in chicks.

A 46-year-old woman reported a 6-month history of severe hand eczema and extensive itchy dermatitis with erythema, maculopapular eruption and plaques, spreading to exposed forearm skin, face, eyelids and neck.

Her personal history was negative for atopy and related diseases. The skin patch tests with Italian standard series and prick tests with a panel of common inhalant and food allergens were negative.

The patient worked as a chick vaccinator and the dermatitis seemed work-related. She usually handled vaccines for the prophylaxis of the infectious bursal disease and fowl pox. Ceftiofur was added to the vaccine diluent to prevent infections in vaccinated chicks.

The patch tests were performed with diluent (as it is) and ceftiofur (5% in petrolatum). The former was negative, while ceftiofur produced a strong cutaneous reaction. Ceftiofur patch tests in 10 healthy controls yielded negative responses.

The patient did not report any adverse reactions to penicillin and cephalosporin. The skin tests – performed by patch, prick and intradermal methods with penicilloyl polylysine (PPL), minor determinant mixture (MDM), penicillin G, ampicillin,

amoxycillin and a panel of cephalosporins ranging from first to third generation, so as to cover the main chemical subgroups – were all negative. Only the intradermal test with ceftiofur showed a positive delayed reaction (Table 1).

Ceftiofur was first introduced in the USA in 1987 and is being widely used in many countries. Only three cases of occupational contact allergy have been reported so far, all of them in Spain (1, 2). In Italy, ceftiofur was introduced in 1995, and this is the first case reported.

Allergic contact dermatitis to cephalosporins is unusual and mainly occurs in professional exposed workers. Only a few cases have been reported so far, and cross-reactivity is normally found (3, 4). Rare cases of simultaneous allergy to cephalosporin and penicillins have been explained by sensitization to the β -lactam ring. Cross-reactivity between different cephalosporins usually depends on sensitization to amino-thiazolic or tetrazolic groups (4), but sensitization to an individual allergenic group (side chains) can also occur.

Ceftiofur has an exclusive radical, the furanyl-carbonyl-thio-methyl group, not present in other cephalosporins. Sensitization to this radical could explain the lack of cross-reactivity found in the

present case as in the cases previously reported (1–2).

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Nickel sulphate, food additives and seronegative arthritis: is there any relationship?

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Key words: food additive; seronegative arthritis; double-blind placebo-controlled challenge test; nickel sulphate.

Seronegative spondilo-arthropathies are characterized by particular clinical features such as sacroileitis, spondylitis and asymmetric peripheral arthritis mainly involving the lower limbs

Nickel sulphates are triggers or aggravating factors for seronegative arthritis.

and negative rheumatoid factor. In most cases, they are associated with the HLA class I antigen B 27 (1). We have recently reported that in few selected cases of seronegative arthritis, articular symptoms may be triggered by food antigens (2). Therefore, as a complement to the above study (2), we discuss here the association between food additives, other substances like nickel sulphate and seronegative spondilo-arthropathies. In this study, we have enrolled 88 subjects, 54 females and 18 males, with ages ranging from 12 to 60 years, affected by seronegative arthritis and with suspected food additive intolerance on the basis of an improvement of symptoms after a food additives-free diet.

Fifty-two out of 88 patients presented a concomitant recurrent urticaria. After an additive-and nickel-free diet for 4 weeks, the patients were made to perform double blind, placebo-controlled challenges (DBPC) with food additives and nickel sulphate. The following substances were tested: tartrazine (E102), erythrosine (E127), sodium benzoate (E211), p-hydroxybenzoate (E218), sodium metabisulfite (E223), monosodium glutamate (E621) and nickel sulphate. The placebo and the substances could not be distinguished. Only one DBPC was performed per day. Each substance under investigation was challenged with at least 1-week interval. Only the appearance of arthritis (arthralgia and swelling of joints) and the possible appearance of urticaria (itch and hive) were considered as a positive response. The DBPC challenge test resulted positive in 13 patients, four males and nine females; the median number of joints involved was three (range 1–10). Ten patients (66.7%) presented a concomitant urticaria. With regard to the relapse of arthritis, seven patients were positive to only one substance and six patients to two substances. More precisely, six patients were positive to nickel and one patient to erythrosine. With reference to the patients with positive DBPC to two substances three of six were positive to nickel and to monosodium glutamate, two of six were positive to nickel and to tartrazine, and one out of six was positive to monosodium glutamate and to sodium metabisulfite. No patient presented arthralgia or joint swelling after the placebo challenge.

All patients with DBPC positive to nickel sulphate presented a positive patch test.

Seronegative arthritis constitutes a broad spectrum of diseases of unknown origin. Food antigens have been suggested to be involved in few selected cases of seronegative arthritis (2). Data from our study suggested that nickel sulphate, an ubiquitous metal, can induce an exacerbation of arthralgias in patients with previous sensitization to nickel itself. Contact with nickel may occur not only through the skin but also through gastrointestinal mucosa, by the ingestion of certain food items (3). The oral DBPC and the results of the free diet, suggested that in some patients the nickel and food additives, introduced with the daily diet, may be the cause of the perpetuation of the articular symptoms. We think that nickel sulphates and food additives are triggers or aggravating factors, instead of simply being etiologic factors of such symptoms.

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Serial blood and urine levels of EDN and ECP in eosinophilic colitis

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Key words: EDN; ECP; IgE; eosinophilic colitis.

Eosinophil-derived neurotoxin (EDN), also called eosinophil protein X (EPX), and eosinophil cationic protein (ECP) have been widely

accepted as markers of eosinophilic inflammation including inflammatory bowel diseases.

Higher levels of fecal and serum ECP and EPX were found in eosinophilic gastroenteritis than in other inflammatory bowel diseases (1), such as ulcerative colitis and Crohn's disease. However, no previous study has investigated subsequent levels of blood and urine EDN and ECP in eosinophilic colitis in parallel with the clinical course. This is the first report of eosinophilic colitis, in which blood and urine levels of EDN and ECP were measured serially.

A 28-year-old man suffering from consistent diarrhea visited a hospital in February 2002. He was introduced to our clinic because of peripheral blood eosinophilia. No remarkable finding was observed on physical examination, and stool culture was negative for pathological bacteria and no parasite infections were noted too. Diagnosis of eosinophilic colitis was made with histological findings from biopsy of colon, which showed numerous eosinophil infiltrations in the interstitium and edematous change of the tissue. Conservative medication was given to him, and his clinical symptoms improved.

Blood and urine EDN and ECP was measured by the method which we have developed recently (2) and the Pharmacia ECP-radioimmunoassay (RIA) kit (Pharmacia, Uppsala, Sweden), respectively. Urinary EDN concentration was expressed as microgram per millimol of creatinine (Cr) corrected for urine volume.

Blood and urine EDN is high in a symptomatic condition of eosinophilic colitis.

The laboratory data on 18 March 2002 showed the peripheral eosinophil count to be 7590/ μ l and the total serum IgE level to be 590 IU/ml. The levels of serum and urine EDN were 265 ng/ml and 790.2 μ g/mmolCr, respectively, and the serum ECP level was 3.3 ng/ml. On 26 March, the laboratory data showed the peripheral eosinophil count to be 2800/ μ l. The levels of serum and urine EDN were 260 ng/ml and 309.4 μ g/mmolCr, respectively, and the serum ECP level was 2.75 ng/ml. After improvement of his clinical symptoms, the laboratory data on 8 April showed the peripheral eosinophil count to be 400/ μ l and total serum IgE level to be 350 IU/ml. The levels of serum and urine EDN were down to 48 ng/ml and 98.9 μ g/mmolCr, respectively, and serum ECP level was also down to 1.1 ng/ml. Urine ECP was not detectable throughout the study. ECP is a very sticky and extremely charged protein, making the urine ECP undetectable.

Although Bischoff et al. (1) reported that the levels of serum and fecal ECP and EPX in eosinophilic gastroenteritis

were higher than in other inflammatory bowel diseases, no study has investigated the eosinophil-derived proteins serially in eosinophilic colitis. Furthermore, as far as we know, no previous study has been carried out which showed urine levels of eosinophil-derived proteins in eosinophilic colitis.

In this report, we measured the levels of EDN and ECP in blood and urine serially and the levels of serum EDN, ECP and urine EDN, but not urine ECP were higher in his symptomatic condition than in his remission, suggesting that measurements of blood and urinary EDN concentrations may be a candidate for a useful and easily accessible marker for eosinophilic inflammation in eosinophilic colitis.

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