

Key words: challenge tests; meloxicam; NSAID; intolerance.

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Anaphylaxis to walnuts and pine nuts induced by ACE

WE report a case of severe anaphylactic shock from walnuts and pine nuts which was unquestionably induced by the daily intake of an angiotensin-converting enzyme (ACE) inhibitor.

An 87-year-old woman had never been ill until April 1996, when hypertension was diagnosed. She was given Ramipril 2.5 mg/day. She displayed widespread pruritis with edema of the palms of the hands in June, and then three times in October, immediately after eating unspecified food items. These attacks were treated with antihistamines. On 6 January 1997, she suffered severe anaphylactic shock, beginning with edema of the lips and tongue, vomiting, collapse, and loss of consciousness. After a telephone call, the cake she had just eaten was stored in the freezer, and immunoallergic examination was performed on 29 January with four parts of the cake: cake mixture, dark cream, light cream, and dried fruit decoration. Parallel prick tests were performed for all the dried fruits and 32 standard food allergens, as well as latex and 12 standard aeroallergens. On a skin normally positive

to codeine (3 mm), the prick test was clearly positive to the light cream (3 mm), walnuts (4 mm), and pine nuts (10 mm). The CAST indicated a huge release of leukotriene C₄ from the leukocytes (2250 to 3100 pg/ml) ($N < 50$ pg/ml), and the basophil activation test (identification of CD63⁺ cells by flow cytometry) confirmed basophilic activation with respect to the walnuts. The RAST was negative to the walnuts and pine nuts. No oral provocation test was performed because of the severity of the shock.

This case clearly demonstrates the advantage of having the incriminated food available for skin and *in vitro* testing, in order to detect masked allergens. We later had confirmation of the presence of walnuts in the cream. On the other hand, the subject's total absence of allergic reaction until the age of 87 clearly indicates the role of the converting enzyme inhibitor in stimulating this reaction. The anaphylactic reaction is linked to the release of chemical mediators inducing the vasopermeability and vasodilation responsible for the collapse. It may be thought that angiotensin II, which is a powerful vasoconstrictor, opposes the risk of collapse, as seems to be indicated by the increased doses of angiotensin I and II at the time of anaphylaxis (1). Herrmann & Ring have also shown that patients displaying anaphylaxis to Hymenoptera venom have significantly reduced levels of angiotensin I and II compared with normal subjects (2).

Did an ACE inhibitor trigger anaphylaxis to pine nut masked allergens?

Converting enzyme inhibitors block the conversion of angiotensin I into angiotensin II. They may increase the severity of anaphylactic reaction during LDL apheresis or hemodia-

lysis (3). ACE inhibitors could also be responsible for the occurrence of allergic reactions or anaphylaxis to an allergen during specific immunotherapy (4). We suggest that food anaphylaxis should be systematically suspected in a patient displaying repeated allergic reactions with ACE inhibitors before concluding that these drugs are directly responsible (5).

Key words: angiotensin-converting enzyme inhibitor; pine nut anaphylaxis.

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Chronic "idiopathic" urticaria and hydatid disease

PARASITIC diseases, mainly those caused by helminths, can induce urticaria. Hydatid disease, or echinococcosis, is an infestation by cestode larvae belonging to three *Echinococcus* species (*E. granulosus*, *E. multilocularis*, and *E. vogeli*). Liver and lung are the most frequently involved organs. Skin involvement is rare; furthermore, nonspecific cutaneous manifestations (pruritus, urticaria) associated with hydatid disease have been very rarely reported in the literature.

We describe a case of hydatid cyst of the pancreas associated with chronic urticaria.

■ A 49-year-old man from Puglia (southern Italy) was admitted to hospital in November 1996 with a diagnosis of chronic idiopathic urticaria, the disorder having appeared in May of the same year. The patient had been previously treated at other centers, with slight remissions of cutaneous lesions and pruritus, first with cetirizine (10 mg/day for 2 months) and subsequently with loratadine (10 mg/day for 1 month). He stated also that in the past he had been affected by hydatid disease due to *E. granulosus*. In 1958, multiple liver hydatid cysts, which were removed by surgery, as well as splenic hydatid cysts, for which the patient underwent splenectomy, were diagnosed. The patient enjoyed good health until 1969, when multiple new liver cysts and one cyst in the right lung were found: the patient was subjected to liver segmentectomy and surgical removal of the pulmonary cyst. In 1975, the appearance of new cysts in the right lung necessitated the inferior lobectomy. The patient was in good health until 1996.

At the time of admission, the patient had not been taking any drugs.

Dermatologic examination revealed the presence of wheals which were widespread particularly on the trunk and were accompanied by pruritus. General physical examination did not show anything pathologic, with the exception of the already mentioned lung, liver, and splenic features.

Laboratory examinations showed increase in total IgE (1.012 kUA/l) and γ -gt (249 U/l). Specific anti-*E. granulosus* IgE were positive (1.5 kUA/l [normal values: <0.7 kUA/l; Pharmacia® Cap RAST method]). All the other laboratory tests were within normal ranges. Chest radiography revealed a retraction of the right

Skin manifestations, including urticaria, associated with hydatid disease have been very rarely reported in the literature.

hemithorax and a thickening of the costal and mediastinal pleurae, as well as the diaphragm. Abdominal echography revealed, at the head of the pancreas, a cyst measuring 7 × 4.7 × 7.5 cm. It appeared to be circumscribed by a thick fibrous capsule.

The presence of the cyst was confirmed by CT scan, which also allowed us to exclude other extrapancreatic localizations of the hydatid disease.

The patient was successfully subjected to surgical removal of the cyst. Histopathologic examination confirmed the diagnosis of hydatid cyst. The urticaria regressed in about 3 weeks without any therapy. During follow-up (21 months), no recurrences were observed of either the hydatid disease or the urticaria.

■ Cutaneous manifestations of hydatid disease may be divided into "specific" and "nonspecific" (1). The former are rare and characterized by the development of fistulae from liver cysts to the skin (2, 3), and by "metastatic" cutaneous localizations. Bresson-Hadni et al. (3) described two patients with hydatid disease due to *E. multilocularis*, initially localized to the left liver lobe, who developed lesions on the abdominal skin surface. These lesions presented as epigastric and supraumbilical nodules: they were clinically polymorphous (some subcutaneous, some inflammatory, some ulcerative). The authors hypothesized that the larvae may have penetrated into the skin through the falciform ligament, which appeared to be thickened in the CT scan.

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