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Chronic urticaria caused by *Hymenolepis nana* in an adopted girl

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Key words: *Hymenolepis nana*; urticaria.

We observed a 12-year-old adopted Bolivian girl living in Italy since 1997. Since her arrival, she has shown recurrent episodes of generalized urticaria that have been treated with antihistamines and steroids, showing temporary relief. Since July 2006, her urticaria has stopped to respond to systemic treatments as it has worsened, and the girl has begun to show lack of attention and irritability, mainly during school activities.

She had no animals at home and presented with no history of fever, sweats, arthralgia, cough, dyspnea, or allergic diseases. According to the EA-ACI/GA2LEN/EDF guideline (1), we performed diagnostic routine tests for chronic urticaria, which revealed normal findings, but for an increase in total serum IgE (1800 IU/ml) and eosinophils (white cell count of 9000/mm³ with 400 eosinophils/mm³). Parasitologic stool sample examination showed eggs of *Hymenolepis nana*. Her adoptive parents had negative parasitologic examinations.

The infection by *H. nana* was treated with praziquantel at a single dose of 1 g per os, during a recovery regimen and it was well tolerated.

Four weeks later, we performed a follow-up visit: the cutaneous eruption

***Hymenolepis nana*: an unusual cause of chronic urticaria in an adopted Bolivian girl**

gradually disappeared and the itching decreased and rapidly cleared. Peripheral eosinophils returned to normal limits and total serum IgE level dropped to normal values in 2 months; parasitologic investigations were repeatedly negative. The patient has been symptom free for 8 months.

Hymenolepis nana is endemic in Asia, southern and eastern Europe, Central and South America, and Africa (2). In Italy, the infection disappeared many years ago (3, 4). *Hymenolepis* worms live in warm climates; the eggs of these worms are ingested by insects, and mature into a life form referred to as a 'cysticercoid' in the insect. Humans and other animals become infected by eating material contaminated by insects. In an infected person, it is possible for the worm's entire life cycle to be completed in bowels: the infection can therefore persist for years. The infection can have an epidemiologically important impact on family units, because it is the only tapeworm that can be transmitted directly from humans to humans and because internal autoinfection may occur. This parasitosis can be asymptomatic or characterized by acute or chronic gastrointestinal manifestations such as enteritis, abdominal pain, diarrhea, anorexia, meteorism and flatulence; sometimes it presents with isolated headache and dizziness, or failure to thrive. Skin manifestations are rare.

In Italy, *H. nana* infection has been reported in two adopted children: the first case was associated with episodes of night sweats and diarrhea, the second one with failure to thrive. The present case is the first Italian report of chronic recurrent

urticaria in children with this specific infection. The pathogenesis of cutaneous manifestations is not known yet; however, due to the particular life cycle of *H. nana*, which can generate an autoinfection, the persistent or permanent antigen stimulation may elicit a strong immune response and could be the cause of chronic cutaneous manifestations. Moreover, it has been demonstrated that intestinal parasite proteinases stimulates IgE synthesis and the increase in eosinophils (5).

These observations are in agreement with the fact that after having arrested the antigenic stimulation with the eradication of tenia, we have simultaneously observed both the lowering of total serum IgE and eosinophils to normal levels and the disappearance of clinical cutaneous manifestations (Fig 1).

In conclusion, we suggest that *H. nana* has to be considered in children coming from foreign endemic countries with a history of intestinal symptoms and/or allergic diseases, including chronic urticaria.

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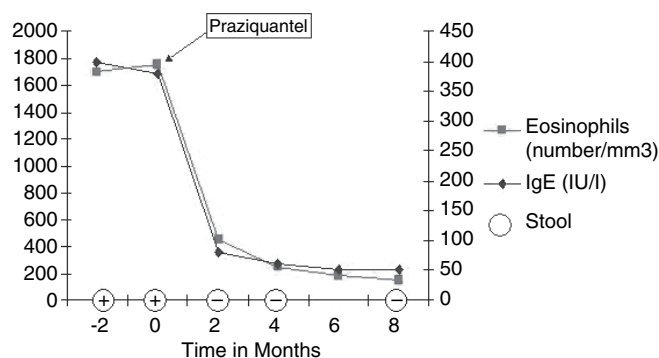


Figure 1. Total serum IgE levels, number of eosinophils and stool examination before and after treatment.

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Anaphylactic shock to guar gum (food additive E412) contained in a meal substitute

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Key words: acetylsalicylic acid guar; anaphylaxis; food allergy; gum; vegetable gum

At 7:30 AM, a 52-year-old male took 1000 mg of acetylsalicylic acid (viral rhinitis). At 2 PM he ate a meal substitute, called 'GERLINEA' which he frequently consumed for slimming. At 4:30 PM he took 500 mg of acetylsalicylic acid. Thirty minutes later he experienced generalized urticaria. At 5:30 PM he took 1000 mg of acetylsalicylic acid-vitamin C (total dose of acetylsalicylic acid 2500 mg) because of headache. A few minutes later he presented a severe anaphylactic reaction: chest and epigastric pain, sudden weakness,

We report a severe anaphylactic reaction after ingestion of a substitute meal containing guar gum.

Table 1. Results of skin prick tests and specific IgE antibodies

Skin prick tests [mean diameter of the wheal (D + d)/2)		Specific IgE levels (KU/l)
Positive control	8 mm	
Negative control	0	
Soybean	4 mm	4.72 (class III)
Gerlinea	6.5 mm	
Guar gum (E412)	12 mm	27.20 (class IV)
Carob gum (E410)	13 mm	6.42 (class III)
Arabic gum (E414)	0	<0.35 (class 0)
Tragacanth gum (E413)	0	<0.35 (class 0)

breath shortness and circulatory collapse. The treatment (shots of adrenaline and corticoids) was effective. The patient had taken acetylsalicylic acid on several previous occasions with no effects.

Skin prick tests including 25 food allergens were negative, except for soybean. Skin tests were performed with the meal substitute, with guar gum contained in the meal substitute and also with other gums of the *leguminosae* family and also radioallergosorbent tests to vegetable gums (CAP system®, Pharmacia, Uppsala, Sweden). The results were positive for the meal substitute, and for guar and carob gums with evidence of an IgE-mediated mechanism (Table 1).

An oral provocation test to acetylsalicylic acid remained negative (cumulative dose: 933 mg). Considering the severity of the reaction, oral challenge test with guar gum was not performed. We suggested an eviction of food containing guar or carob gums. Two years after the incident no relapse was observed.

Discussion

Guar gum belongs to the *leguminosae* family. It is used in paper manufacture, cosmetics, printing, carpet manufacturing, pharmaceuticals industry... It is incorporated into numerous foods (food

additive E412) as a thickener and emulsifier (1).

Guar gum has been shown to cause occupational rhinitis and/or asthma and only one subject experienced oral syndrome after eating food containing guar gum (ice cream and salad dressings) (2).

Enhancing factors are frequently involved in food-induced anaphylactic shocks: alcohol, exercise and simultaneous intake of medications, such as acetylsalicylic acid, beta-blockers and angiotensin-converting enzyme inhibitors (3).

We report the first case of exclusive food allergy to guar gum amplified by the almost concomitant ingestion of acetylsalicylic acid. On account of the growing number of masked food allergens, thorough allergological investigations are necessary to detect their roles in unexplained anaphylactic shocks.

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