

Case report

House-dust mite ingestion can induce allergic intestinal syndrome

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In childhood, many pathologic situations inducing chronic vomiting can be found. They are mostly related to gastrointestinal disorders such as pyloric stenosis, gastroesophageal reflux (GER), gastritis and peptic disease, or dietary intolerance. Persistent, chronic vomiting can also be ascribed to some extraintestinal causes, such as chronic infections (mainly urinary tract infections); metabolic, endocrine, and psychiatric disorders; or central nervous system diseases. Finally, there are a few cases the pathogenesis of which remains unknown.

In the case described, an intestinal allergic reaction to house-dust mites (HDM) was demonstrated.

Case report

A 5-year-old girl was observed as an outpatient for persistent vomiting. She had been suffering from this symptom since the age of 5 months. She was sick from two to five times a day. Since the age of 2 years, she had vomited regularly, mainly in the morning. The girl had become so used to vomiting that she considered this situation as her natural condition of life. She had never suffered from asthma, rhinitis, or any other respiratory disorder. A previously performed skin prick test was positive for HDM. One of her sisters was affected by allergic rhinitis caused by HDM.

At the time of the observation, the auxologic para-

meters were in the normal range (50th percentile for height and weight).

GER was investigated by 24-h pHmetria and contrast radiography with no pathologic findings. Cow's milk protein intolerance was excluded by the negativity of occult blood loss and skin prick tests, the latter performed with both fresh milk and purified allergens (whole milk and casein, Bayropharm-Allergeni, DHS). Dietary exclusion of milk and milk derivatives gave no relief of symptoms. An ultrasound abdominal scan was negative. Serologic, hematologic, and bacterial culture data were normal. Metabolic or endocrine disorders were excluded. Neurologic examination was negative.

A prick-by-prick test including a large number of fresh foods and inhalant allergens (pollens, HDM, molds, and dog and cat danders) (Bayropharm-Allergeni, DHS) was carried out in order to exclude some other kinds of allergy. All the tests gave negative results except for *Dermatophagoides pteronyssinus* (Dp) and *D. farinae* (Df), which gave a positive reaction, resulting in cutaneous wheals larger than the histamine control and scored as 4+. The presence of serum specific IgE against Dp and Df was confirmed by RAST positivity (Pharmacia CAP System, Uppsala, Sweden); RAST was negative for all the other allergens tested. Spirometric parameters (PEF, FEV₁, FEV₁/FVC) were in the normal ranges, and chest radiographic examination was negative.

In this situation, a further anamnestic investigation was required. The girl's parents were interviewed in depth. Their statements showed that she lived in a highly acarigenic environment. Her bedroom was full of plush puppets, teddy bears, and similar toys. Moreover, she slept in a bunk bed below her sister, a sleeping arrangement which is known to cause a very high HDM-exposure rate, due to the overnight settling of mite-derived particles onto the lower bed from the upper one.

The parents were immediately informed of the precautionary steps to be taken for house dust reduction, and they carefully put our suggestions into practice. Since then, the patient has not suffered from vomiting. She was not given any drug.

Five months later, a nasal provocation test with lyophilized *Dermatophagoides* extract (Cod. Allerkin Lofarma, Milan, Italy) was performed. Two hours after testing, the patient experienced vomiting again, in particular during the 4 h following the exposure. No respiratory symptoms were observed.

At present, the girl is quite well; she has not been on a diet nor has she taken any drugs.

Discussion

Dp and Df represent the main source of allergens in house dust (9). Of patients with skin reactivity to house dust, 75–90%, have serum specific IgE against mite-derived allergens, the others being positive for cat or dog epithelia or molds (4).

The main allergenic epitopes of HDM (*Der p* 1, *Der f* 1) are concentrated in the external coat, in gut endothelium, and in fecal material (12). Mite-derived particles may be of various sizes. Some of these particles are inhaled through the larynx, while others adhere to the pharyngeal mucosa, from where they are slowly conveyed into the digestive tract, by saliva deglutition and esophageal peristaltic movements. Pollen grains follow the same route.

In atopic children, the enteric mucosa has sometimes proved to be the first tissue to become sensitized to inhalant allergens (6). Nevertheless, to my knowledge, a relationship between hypersensitivity to inhalant allergens and gastrointestinal allergic diseases has not so far been described.

At intestinal level, in physiologic conditions, an immunologic tolerance develops for the heterologous macromolecules associated with foods. Many aspects of this mechanism are still being debated. In the same way, intestinal tolerance may be induced to heterologous macromolecules associated with inhalant particles.

In lymphoid tissues, T-lymphocyte regions show specialized endothelium venules, designated high endothelial venules (HEV), presenting various adhesion molecules (7, 8, 10, 11). The interactions be-

tween lymphocytes and HEV have been thoroughly investigated. Several adhesion molecules have been characterized, among which VCAM-1 and ICAM-1 link to VLA-4, LFA-1, and MAC-1, which are expressed by lymphocytes, PMN, and macrophages (2). Some other adhesion molecules have been identified. They are known to play a significant role in the immunologic response (1). Cytokines modulate adhesion molecule expression; interleukin-1, interferon-gamma, and tumor necrosis factor-alpha are known to upregulate the expression of ICAM-1 on epithelial cells *in vitro* (10).

It can be postulated that recirculating lymphocytes from gastroenteric tissue may have a "homing" effect at the bronchial mucosal level (3).

Bronchial homing of digestive tract lymphocytes may explain why the sensitization of the respiratory mucosa to inhalant allergens sometimes appears later than gastroenteric sensitization. Furthermore, this "immunologic connection" between intestinal and bronchial tissues could possibly explain food-induced asthma.

On the other hand, an intestinal immunologic tolerance to ingested aeroallergens may partly explain the controversial clinical results obtained by oral desensitization in patients affected by bronchial asthma due to allergy to mites or pollens (5).

In the case described, a rigorous reduction of the dust mites led to the disappearance of the symptoms. It can be postulated that digestive tract sensitization to *Dermatophagoides* may be responsible for gastroenteric symptoms even in the absence of evident respiratory troubles. It could be useful to consider the possible role of inhalant allergens in cases of chronic vomiting for which no clear diagnosis is available.

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