as the storage time extends, a fact which leads to the following considerations:

1) Digestive problems or rashes could appear after the ingestion of a certain quantity of histamine-rich peanuts. A high content of histamine has already been noticed for some cooked pork meats, fermented cheese, tinned fish, etc. This reaction to biogene amines is well known and can be related to a functional deficiency of the degrading enzymes (4, 5).
2) The worsening of IgE-related allergy may be caused by this associated factor: the high quantity of histamine in some peanut batches. This could explain the differences in the intensity of the disorders occurring after the ingestion of the same quantity of peanuts.

Finally, we would like to suggest that the quality of peanuts for sale could be evaluated by their histamine content; this could also be considered a quality test.

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Failure of montelukast to prevent anaphylaxis to diclofenac

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Key words: antileukotrienes; aspirin triad; montelukast; NSAID.

• The aspirin triad, or Widal triad, includes asthma, nasal polyps, and aspirin sensitivity. In this syndrome, asthma attacks may be precipitated not only by aspirin, but also by other nonsteroidal anti-inflammatory drugs (NSAID) such as indomethacin, diclofenac, ibuprofen, fenamic acid, piroxicam, and several others. The frequency with which each of these drugs produces asthma attacks depends on several factors such as dosage, anticyclooxygenase potency, and individual sensitivity (1). The hallmark of the syndrome is the precipitation of asthma attacks by the administration of aspirin or other NSAID. Approximately 5–10% of patients with bronchial asthma are affected (1, 2). Although the mechanisms implicated in NSAID-induced asthma are unknown, release of cysteinyl-leukotrienes (Cys-LTs) may play an important role (3), as shown by the increase in baseline production of Cys-LTs and the high sensitivity to inhaled Cys-LTs in bronchial challenge (2, 4). This suggests that patients with aspirin triad should benefit from being treated with anti-LTs (2).

NSAID must be avoided by patients with aspirin triad, even when taking antileukotrienes.

Leukotrienes (LTs) are lipid mediators derived from arachidonic acid and are synthesized by several cells including mast cells, eosinophils, and macrophages. LTs induce bronchoconstriction, increase vascular permeability, cause plasma extravasation and edema, enhance mucus secretion, and elicit inflammatory cell influx (3). In view of these features, anti-LTs have been reported to provide some partial protection in NSAID bronchial challenge (5). Therefore, it has been suggested that aspirin-sensitive patients under anti-LT treatment have a lower risk of bronchospasm after inadvertent NSAID exposure (6).

A 20-year-old man suffering from aspirin triad, with a 4-year history of nasal obstruction, anosmia, rhinorrhea, nasal polyps, and nonseasonal asthma, had a life-threatening bronchospasm after aspirin ingestion. He was then told to avoid all NSAID and was placed under treatment with montelukast 10 mg once daily, salmeterol 60 μg b.i.d., and budesonide 400 μg b.i.d. One month later, he developed oral candidiasis, and budesonide was discontinued. Two months after beginning treatment with montelukast, he attended the emergency room because of back pain and was administered 75 mg of intramuscular diclofenac. Two hours later, while at home, he suffered palm itching, coughing, wheezing, and severe shortness of breath, with no improvement after several administrations of inhaled terbutaline. Finally, the patient developed respiratory arrest and was resuscitated by the emergency care services.

Menéndez et al. (7) recently reported the failure of zafirlukast to prevent ibuprofen-induced anaphylaxis. Our patient was under treatment with another anti-LT (montelukast) when he suffered a life-threatening reaction after NSAID administration. The severity of these reactions emphasizes the need to avoid NSAID in aspirin-sensitive patients, even though they are receiving anti-LT treatment.

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Allergy to human seminal fluid: a case of self-diagnosis

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Key words: chronic vulvovaginitis; human seminal fluid allergy; Papanicolaou smear; skin prick testing.

● Immediate-type hypersensitivity reaction to human seminal fluid is an infrequent but well-documented disorder; there is strong evidence that this reaction is IgE mediated (1–3). Acute systemic hypersensitivity reactions to seminal fluid are rare; chronic or recurrent local reactions are more common and may be misdiagnosed (4).

A 36-year-old woman consulted us for treatment of seasonal rhinoconjunctivitis and asthma. She had experienced symptoms since the age of 26. Her past history was also positive for recurrent vulvovaginitis. As far as she could remember, the first symptoms had occurred immediately after her marriage, in 1993. The symptoms manifested were vulvovaginal itching, a burning sensation, and swelling after sexual intercourse, and took up to a week to disappear.

In 1993, she had consulted a gynecologist: the vulvovaginal examination was normal; cervical vaginal cytology (Papanicolaou smear) showed signs of inflammation. Cervical vaginal cytology after therapy showed inflammation still present.

In 1994, multiple cervical biopsies showed endocervical cystic microglandular hyperplasia, and surface and complete intraglandular metaplasia.

In 1995, cervical vaginal cytology showed inflammation still present, and multiple cervical biopsies showed squamous metaplasia and chronic inflammation.

When she started to employ condoms as an anticontraceptive method, the symptoms disappeared immediately. Cervical vaginal cytology was normal in three successive controls. She joked that she thought she was ''allergic'' to her husband and asked us about the possibility of seminal fluid allergy.

Skin prick tests (SPT) were positive to Poaceae pollen, in accord with seasonal rhinoconjunctivitis and asthma symptoms, but were negative to other pollens, animal dander, house mite, and mold. An SPT with her husband's undiluted seminal fluid yielded a wheal and flare reaction (wheal 3 mm diameter).

SPT confirmed an atopic woman's allergy to her husband's seminal fluid.

Since the couple did not wish to conceive, and since the symptoms were mild, immunotherapy was not used. In view of the atopic status and therefore the possibility of latex sensitization (5, 6), the patient was advised to use latex-free condoms, and to avoid contact with seminal fluid.

There are no accurate data on the prevalence of allergy to seminal fluid, although it seems to be more common than previously realized (7). Acute systemic hypersensitivity reaction to seminal fluid is rare and exhibits classical features: local or generalized urticaria; vulval and generalized pruritus; and erythema, periorbital and vulval edema, dyspnea, palpitations, hypotension, and loss of consciousness. The onset of symptoms may occur during sexual intercourse or within minutes afterward. The symptoms of localized reactions are vulval pruritus, a burning sensation, erythema, and swelling, but it seems possible that mild local reaction may be misdiagnosed as infective or nonspecific vulvovaginitis, as happened in our patient, in whom misdiagnosis led to numerous clinical examinations and to unsuccessful treatment.

Intractable or recurrent vulvovaginitis with coital manifestation calls for allergologic investigation. Therefore, physicians should be familiar with the many different aspects of human seminal fluid hypersensitivity in order to expedite correct diagnosis.

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