

We report two cases of immediate reactions after local administration of rifamycin, probably provoked by systemic absorption of the drug through the conjunctivae and the surgical wound. In both cases, previous local instillation had occurred, favoring sensitization to rifamycin. The clinical data and positive skin prick testing suggested an IgE-mediated immunologic mechanism.

Allergy to rifamycin is rare. A few cases of contact dermatitis have been reported (1–5). Topical rifamycin is also an uncommon but well-known cause of anaphylactic reactions (6–10); all those immediate reactions were caused by cleaning wounds with this drug. However, to the best of our knowledge, this is the first report of systemic reaction after application of rifamycin in eye-drops. We also demonstrated cross-sensitivity between rifamycin and rifampicin by skin testing.

*Sección de Alergología
Hospital General Yagüe
Avda del Cid, 96
09005, Burgos
Spain

Accepted for publication 19 January 1999
Copyright © Munksgaard 1999
ISSN 0105-4538

References

- Melchior EJ, Samsoen M, Fousereau J, Grosshans E. Quels sont les médicaments topiques les plus sensibilisants de la pharmacopée française?. *Rev Praticien* 1979;**29**:3535–3537.
- Riboldi A, Pigatto PD, Morelli M, Altomare GF, Polenghi MM. Allergy to Mercurochrome and rifamycin. *Contact Dermatitis* 1985;**12**:180.
- Milpied B, van Wassenhove L, Larousse C, Barriere H. Contact dermatitis from rifamycin. *Contact Dermatitis* 1986;**14**:252–253.
- Balato N, Lembo G, Patruno G, Ayala F. Allergic contact dermatitis from rifamycin. *Contact Dermatitis* 1988;**19**:310.
- Guerra L, Adamo F, Venturo N, Tardio M. Contact dermatitis due to rifamycin. *Contact Dermatitis* 1991;**25**:328.
- Piazza I. Anaphylactic reaction to local administration of rifamycin [Abstract]. *Allergologie* 1989;**12**(Suppl):96.

- Mancuso G, Masara N. Contact urticaria and severe anaphylaxis from rifamycin SV. *Contact Dermatitis* 1992;**27**:124–125.
- Cardot E, Tillie-Leblond I, Jeannin P, et al. Anaphylactic reaction to local administration of rifamycin SV. *J Allergy Clin Immunol* 1995;**95**(1 Pt 1):1–7.
- Laxenaire MC, Mouton C, Frederic A, Viry-Babel F, Bouchon Y. Chocs anaphylactiques après levée de garrot en chirurgie orthopédique [Abstract]. *Ann Fr Anesth Reanim* 1996;**15**:179–184.
- Erel F, Karaayvaz M, Devenci M, Ozangic N. Severe anaphylaxis from rifamycin SV. *Ann Allergy Asthma Immunol* 1998;**81**:257–260.

Histamine content of peanuts

S. Frémont*, D.A. Moneret-Vautrin, N. Zitouni, G. Kanny, J.P. Nicolas

Key words: histamine content; peanut; quality control.

• THE severity of food allergy to peanuts has been well established, and since reactive doses are sometimes very minute (1–3), the search for potentiating factors seems to be justified. Peanuts are usually consumed roasted, and we are given no information

about the length of time between their harvesting and their roasting, nor any indication of the storage time before their consumption. The fermentation processes are likely to generate a large quantity of histamine. We have evaluated the protein concentration by the method of Bradford (Biorad, Hercules, CA, USA), and the histamine concentration by radioimmunoassay (Immunotech, Marseille, France) on 16 batches of roasted peanuts, all of different brands, and on four batches of raw, unshelled peanuts.

The protein concentration is higher in 100 g of raw peanuts (16.6 ± 3.4 g) than in 100 g of roasted peanuts (2.6 ± 2.2 g) (Table 1). However, the range of histamine concentration is 0.08–0.56 nmol for 100 g of raw peanuts, reaching 35–150 nmol for 100 g of roasted peanuts. This high histamine concentration in some peanut batches seems to confirm that histamine appears or increases

The storage and roasting of peanuts increase the histamine content, possibly promoting concomitant allergy symptoms.

Table 1. Histamine content in different batches of peanut

Roasted peanuts				Raw peanuts		
Trademark	Processing	Protein (g/100 g)	Histamine (nmol/100 g)	Country of origin	Protein (g/100 g)	Histamine (nmol/100 g)
Benenuts	Dry roasted	1.5	105	Senegal	14.8	4.7
Benenuts	Roasted	4.2	78	Senegal	12.8	7.2
Bahlsen	Roasted	0.8	78	Africa	18.4	4.4
Bahlsen	Dry roasted	1.4	85	Thailand	20.3	1.7
G et A	Roasted	4.2	80			
G et A	Dry roasted	1.5	144			
Menguy's	Dry roasted	9.6	72			
Golden Club	Dry roasted	1.0	150			
Reddi Snack	Roasted	1.7	73			
Rencontre	Dry roasted	3.9	41			
Ancel	Roasted	1.4	90			
Nutsco	Roasted	1.2	113			
Tokapi	Dry roasted	0.9	117			
Trinidad (giant)	Roasted	2.3	60			
Trinidad	Roasted	2.4	35			
Trinidad	Dry roasted	4.3	59			

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.

*INSERM U 308
Faculty of Medicine
54505 Vandœuvre les Nancy
France

Accepted for publication 22 January 1999
Copyright © Munksgaard 1999
ISSN 0105-4538

References

1. Foucard T, Edberg U, Malmheden Yman I. Fatal and severe food hypersensitivity. Peanut and soya underestimated allergens. *Åkartidningen* 1997;**94**:2635-2638.
2. Yunginger JW, et al. Fatal anaphylactic reactions induced by peanuts. *Allergy Proc* 1989;**10**:249-253.
3. Hourihane J, Kilburn S, Nordlee J, et al. An evaluation of the sensitivity of subjects with peanut allergy to very low doses of peanut protein: a randomized, double-blind, placebo-controlled food challenge study. *J Allergy Clin Immunol* 1997;**100**:596-600.
4. Moneret-Vautrin DA, Kanny G. Pseudo-allergic reactions to food: non-immunological food intolerance. In: Schlierf, et al., editors. *Recent advances in clinical nutrition*. Smith Gordon, 1993:145-151.
5. Kanny G, Moneret-Vautrin DA, Schohn H, Feldman L, Mallié JP, Guéant JL. Abnormalities in histamine pharmacodynamics in chronic urticaria. *Clin Exp Allergy* 1993;**23**:1015-1020.

Failure of montelukast to prevent anaphylaxis to diclofenac

E. Enrique, P. García-Ortega*, P. Gaig, M.M. San Miguel

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).

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

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

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.

*Allergy Unit
Hospital Universitari Joan XXIII
Universitat Rovira i Virgili
Avda Dr Mallafré Guasc, 4
43007 Tarragona
Spain

Accepted for publication 25 January 1999
Copyright © Munksgaard 1999
ISSN 0105-4538