

(5). In conclusion, varicella should be considered a potential cause of asthma exacerbations.

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## Acetaminophen-induced rhabdomyolysis

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**Key words:** acetaminophen; cytokines; drug reactions; oral challenge; rhabdomyolysis.

● AMONG the side-effects of acetaminophen, previous reports have included anaphylactic shock and various skin reactions (1–3). Thrombopenia and agranulocytosis have been also observed, as well as hepatitis. We

report the first case of hypersensitivity rhabdomyolysis.

A 17-year-old man was admitted for numerous iatrogenic accidents. When aged 3 months, he had presented hepatitis with agranulocytosis after acetaminophen administration. At 10 years of age, desquamative dermatitis occurred during treatment with cefaclor and acetaminophen. At 14 years of age, he developed a febrile eruption after receiving amoxicillin and acetaminophen, which intensified after paracetamol injection. Epicutaneous tests (ECT) and the intradermal test (IDT) on delayed reading with amoxicillin were distinctly positive. Delayed-type hypersensitivity to amoxicillin was considered. Prick test and ECT with acetaminophen were negative, as was the IDT with paracetamol on immediate and delayed reading and paracetamol-specific IgEs. However, the whole history led the patient's parents to incriminate acetaminophen, requesting further investigations. An oral challenge was carried out with paracetamol 400 mg. Within 5 h, he presented febrile exanthema, neutropenia, and a rise of CRP and CPK MM (Table 1) and an elevation of the seric levels of TNF- $\alpha$  (49 pg/ml; N=8–10), IL-6 (23 pg/ml; N=0–2), and IL-10 (6 pg/ml; N=nd). The lymphocytic subpopulations showed a decrease of CD4+ (16%) with an increase of CD8+ (37%). The eruption and fever lasted 36 h.

### The first report of a patient with rise of serum IL-6, TNF- $\alpha$ , and IL-10.

Hypersensitivity accidents have rarely been reported. One single case of toxic paracetamol-induced myocarditis is described (4). It can be postulated that until now rhabdomyolysis has been overlooked. The mechanisms of acetaminophen hypersensitivity have not yet been elucidated. Acetaminophen is probably involved per se when the onset of symptoms is fast, and immediate tests prove positive (1, 3). The occurrence of delayed reactions, and the negative skin tests, are more in favor of a drug metabolite. Specific IgEs might be involved in shocks, but no proof has been obtained by cutaneous tests or leukocyte histamine-release tests.

The increase of TNF- $\alpha$  and IL-6 may point to activated macrophages and lymphocytes. Among their effects, fever and muscular catabolism were reported. The severity of toxidermia after a single dose of 400 mg would have been potentially life-threatening, had the acetaminophen not been immediately stopped.

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**Table 1.** Evolution of CRP and muscular and hepatic enzymes

| Days | C-reactive protein<br><5 mg/l | ASAT<br>5–35 UI/l | Total CPK<br>25–220 UI/l | CPK MM<br>UI/l | ALAT<br>5–40 UI/l |
|------|-------------------------------|-------------------|--------------------------|----------------|-------------------|
| 1    | 7                             | 49                | 1747                     | –              | 24                |
| 2    | 34                            | 53                | 1268                     | 1236           | 35                |
| 13   | <5                            | 20                | 252                      | –              | 14                |

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## Food allergy to egg and soy lecithins

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**Key words:** double-blind challenges; food allergy; lecithins; RAST inhibition.

● EGG and soy lecithins are commonly used in the food industry as emulsifiers (E322). The possibility of residual proteins leads to suspicion of their role in allergic manifestations in subjects allergic to egg or soy. Two cases are

reported in which the double-blind, placebo-controlled oral challenge

**Two patients with DBPCOC positive to 50–100 mg of allergen.**

(DBPCOC), the “gold standard” of diagnosis, led to confirmation that lecithin had caused lesions of atopic dermatitis (AD) in patients suffering from egg and soy allergies.

E.G., a 15-month-old girl, presented with AD, progressive from the age of 2½ months, as she was fed a milk formula. There was a family history of atopy (paternal pollinosis). The SCORAD was estimated at 26/103.

Prick tests to five inhalants were negative. Positive prick tests were obtained for egg white (10 mm) (codeine 9%=3 mm) and egg lecithin (3 mm) (negative in 61 patients allergic to egg), and RAST to egg was class 2 (Coopérative Pharmaceutique Française, Paris, France).

The labial challenge test (1) for egg white was positive, leading to exacerbation of eczema a few hours later, with an increase in the DBPCOC index from 26 at the start to 38/

103. The DBPCOC to egg lecithin was positive at 50 mg, with the appearance of an erythematous rash on the neck and shoulders 1 h after ingestion.

The second patient, G.S., a 4-year-old boy, with nonatopic parents, presented with asthma and AD. AD occurred at the age of 6 weeks and wheezing at 3 months. At around 1 year, the mother reported an episode of conjunctivitis complicated by angioedema in the eyelids immediately after ingestion of a small piece of peanut. A worsening of the asthmatic symptoms was noted after the age of 2 years. Strict avoidance of peanuts did not bring about satisfactory improvement.

Prick tests were positive for *Dermatophagoides pteronyssinus*, birch and grass pollens, and peanut, with crossed sensitization to soy, but the prick test with soy lecithin was negative (Table 1), and RAST was 33.2 kU/l. The DBPCOC for peanut was positive at a total dose of 15 mg. The DBPCOC for peanut oil (6-ml dose) was also positive, causing an asthma attack.

RAST for soy was 11 kU/l. The inhibition by soy lecithin rose to 62%. The DBPCOC at 100 mg of lecithin was positive, with the appearance of an erythematous rash on the jaw 1 h after ingestion.

Protein assay of soy lecithin by the Kjeldahl method revealed a level of 3.5%, and of 11.3% in the egg lecithin.

Lecithins are included in the excipients of certain drugs and/or lipidic emulsions used for parenteral feeding. Adverse reactions have been described, due to the inhalation of a bronchodilator with soy lecithins in the excipient, or to intravenous infusions (2, 3). In the food industry, asthma from soy lecithin inhalation has been reported (6). The level of soy lecithin in bread can be 0.015% (6). Many milk formulas including hydrolysates contain soy lecithin. Porras et al.'s study (6) confirmed the presence of soy proteins in oil, margarine, and lecithin, and the allergenicity of these proteins was demonstrated in Awazuhara et al.'s study (7). RAST inhibition of 62% for soy, obtained by lecithin solution, confirmed the allergenic nature of the residual proteins (3.5%). To our knowledge, we report the first case of allergy to egg lecithin.

The presence of lecithins in formulas, as well as in cookies and other foods, may explain why avoidance diets for soy and eggs that do not exclude lecithins yield only partial results. Therefore, we recommend that labeling be more precise.

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**Table 1.** Study of cross-sensitivity of legumes by prick tests to native and cooked foods

| Legumes            | Raw (mm) | Cooked (10 min, 100°C) (mm) |
|--------------------|----------|-----------------------------|
| (9% codeine: 3 mm) |          |                             |
| Peanut             | 23       | n.d.                        |
| Soy                | 6        | 10                          |
| Soy lecithin       | 0        | n.d.                        |
| Chickpeas          | 5        | 3                           |
| Lentils            | 3        | 0                           |
| Beans              | 1        | 0                           |
| Green peas         | 2        | 0                           |
| Lima beans         | 0        | 0                           |
| Lupin flour        | 0        | 0                           |
| n.d.=not done.     |          |                             |