

Case report

Food allergy to sunflower oil in a patient sensitized to mugwort pollen

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Key words: cross-reactivity; leukocyte histamine release (LHR); mugwort pollen; oral challenge test; RAST inhibition; sunflower oil.

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Cross-reactivity between mugwort (*Artemisia vulgaris*) pollen and vegetables belonging to the Umbelliferae family is well known (7). We wish to draw attention to cross-reactivity between this pollen and proteins in sunflower seeds as well as possible food allergy to traces of these proteins found in sunflower oil and margarine.

Case report

A 36-year-old woman had been desensitized to mugwort pollen 1 year previously for treatment of seasonal rhinitis. Food allergy to Umbelliferae was identified in this patient in 1990. Despite avoidance of celery, fennel, carrot, and parsley, she experienced episodes of diarrhea and pruritus. Recently, within a 2-month interval, she suffered three episodes of anaphylaxis, which started 1 h after eating a meal, and which began with abdominal pain, followed by pharyngeal pruritus and facial edema with nasal

blockage, and lastly by systemic urticaria with impaired respiration. It was established that the patient had not eaten any vegetables belonging to the Umbelliferae family. A clinical assessment was made to detect any other possible food allergy, which was suspected because of the interval before occurrence of the adverse events and the characteristic association of the symptoms. Furthermore, questioning of the patient revealed that she frequently ate sunflower oil and margarine.

Material and methods

The clinical and laboratory assessment was made in early December 1992. Prick tests were performed with mugwort pollen, sunflower pollen, and 35 common foods as well as with sunflower seed proteins obtained by grinding the seeds and commercially available sunflower oil. Prick tests to vegetables in the Umbelliferae family, known to have been posi-

tive, were not repeated. The positive control used was 9% codeine phosphate. The criterion for a positive response was a wheal diameter greater than half that of the positive control. Prick tests to sunflower oil were run in 10 control subjects.

IgE specific for mugwort and sunflower pollen was measured by the Cap RAST Phadebas test. RAST inhibition to mugwort was performed with Stallergen® extracts, mugwort pollen (138 µg/ml), and sunflower pollen (368 µg/ml) and with a solution of sunflower proteins in a 1 mg/ml concentration. A volume of 50 µl serum was incubated with 50 µl allergenic extracts for 30 min, the mixture being shaken. The mixture was then incubated with the Cap allergen support. The percent inhibition corresponded to the following ratio:

$$I = \frac{\text{cpm bound to the mixture}}{\text{cpm bound in the serum}}$$

A human basophil degranulation test (HBDT) and two tests of leukocyte histamine release (LHR) were done with proteins from sunflower seeds at five different concentrations, from 10^{-1} to 10^{-5} mg/ml, according to previously described methods (3, 5). The criteria for positivity was a percentage higher than 30% of degranulation for a total basophil count higher than $30/\text{mm}^3$.

Oral challenge tests (OCT) were performed by single-blind design at a 5-day interval for 5 ml sunflower oil, and for the proteins in three doses: 100, 500, and 1500 mg, 30 min apart. These substances were masked in 30 g of applesauce. The placebo used was olive oil.

After these tests, strict avoidance of sunflower oil and margarine was instituted.

Results

The prick test was strongly positive for mugwort pollen (12 mm) (control: 3 mm), sunflower pollen (4 mm), and sunflower proteins (4 mm), and negative for sunflower oil. Fifteen minutes later, the prick test for sunflower oil became positive (Fig. 1). It was negative in the 10 control subjects. IgE specific for mugwort was measured as 34.20 kU/l, and as 3.7 kU/l for sunflower pollen. RAST mugwort inhibition by mugwort was 94%; by sunflower pollen, 50%; and by sunflower seed proteins, 28%. There was spontaneous histamine release (57 nmol). The LHR test for sunflower seed proteins was definitively positive. The maximum effect observed was with a concentration of 0.1–1 µg/ml (Fig. 2). The HBDT was positive: 60% of maximal degranulation (total count: 35 basophils/ mm^3). We failed to perform either a RAST inhibition by sunflower oil, or an LHR test for oil, because of the nature of the product.

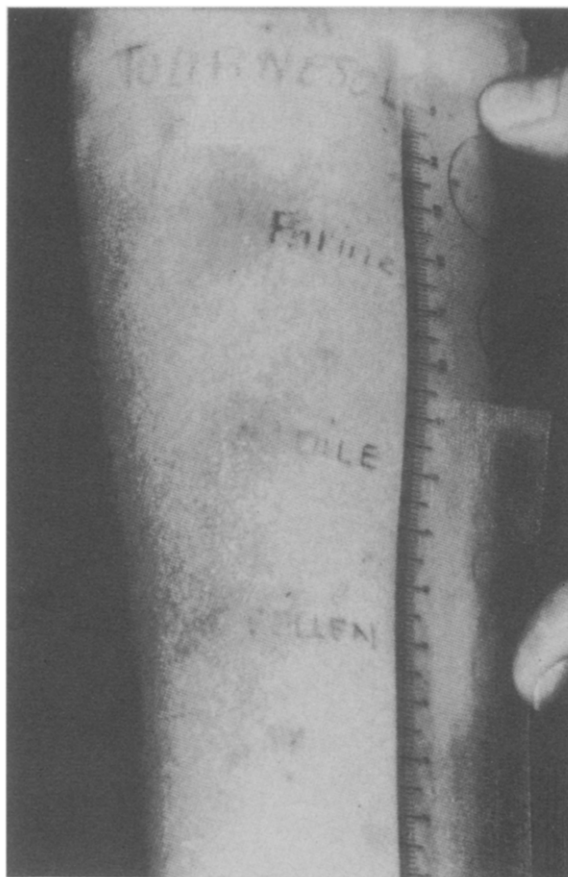


Fig. 1. Skin prick tests. Tournesol: sunflower; farine: seed proteins; huile: oil.

The OCT with sunflower oil (5 ml) did not cause any cutaneous symptoms or alterations in pulse, blood pressure, and peak expiratory flow for 2.25 h; then abdominal pain developed and mequitazine was administered. Eight hours later, the patient developed a cough, impaired respiration, and facial angioedema. An intramuscular injection of 20 mg dexamethasone was administered.

The OCT with 2 g of proteins from sunflower seeds was negative in the following hours. Twenty-four hours later, the patient manifested rhinitis and diarrhea.

Discussion

The assessment was made in December 1992, outside the pollen season. Moreover, the patient was not allergic to any perennial airborne allergen. The sunflower belongs to the Compositae family, as does the mugwort. Fifty percent RAST inhibition to mugwort by sunflower pollen confirmed the partial identity of these allergens. RAST inhibition showed a partial identity of sunflower seed proteins and mugwort pollen. The HBDT and the LHR tests con-

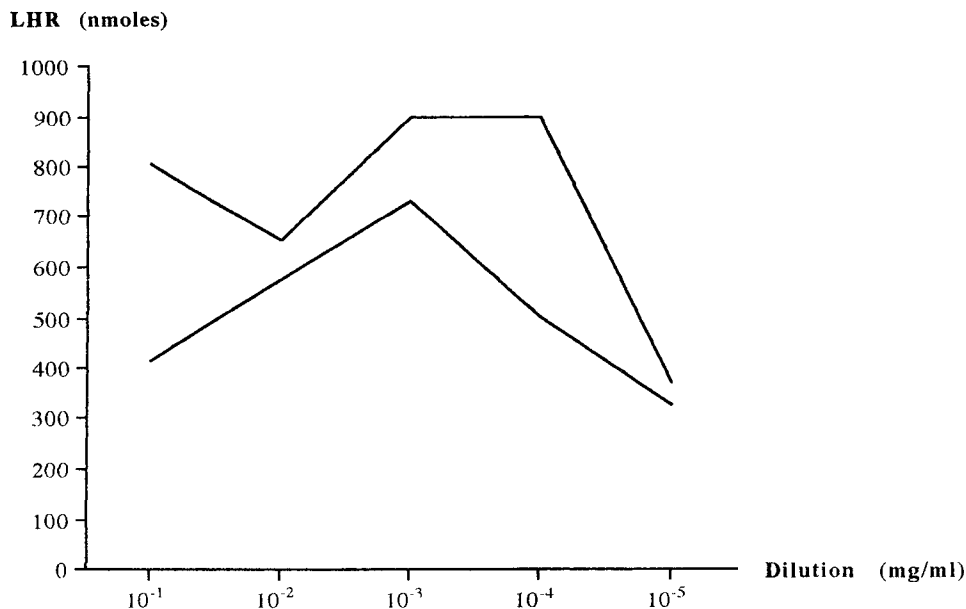


Fig 2. Leukocyte histamine release with sunflower seed proteins.

firmed the major sensitivity of this patient to these proteins.

Ingestion of sunflower seed proteins, common in the hamster, is not yet common in man. But this patient regularly ate sunflower oil and margarine. A prick test was positive for sunflower oil, supporting the hypothesis that this oil contains the proteins. One study has found low levels of residual proteins in sunflower oil, 2–8 µg/ml (4).

In addition, in a case involving anaphylaxis to sesame seed oil, a markedly positive prick test was observed with oil (2), along with skin tests with peanut oil in another case (1).

The delayed time to onset (8 h) of respiratory and cutaneous symptoms after the OCT with 5 ml of the oil may be due to the administration of mequitazine as soon as the patient developed abdominal pain, or it may reflect the partly delayed phase of IgE-dependent allergy. It is possible that heating the oil to temperatures of 150°C and higher increases its allergenicity, and that the symptoms would have been of early onset and more pronounced if a higher dose had been tested and heated oil used. However, the OCT with oil can be considered to be positive in this case. The OCT with 30 ml was negative in two of Halsey et al.'s patients (4). The result of the OCT with seed proteins is more uncertain. A higher dose might have provoked a stronger reaction.

On the basis of the clinical history, and the positive prick test and provocation test with sunflower oil, we find sunflower oil and margarine to have been involved in the anaphylactic attacks, as in the

attacks of pruritus and diarrhea reported by the patient. Another evidence in support of this involvement was the observation of spontaneous histamine release. It has been demonstrated that the latter characterizes progressive food allergies where it is related to the synthesis of a cytokine, HRF, and that it disappears when strict avoidance of the offending allergen is observed (8). Its presence thus means that there are masked allergens which are normally contained in the diet. The fact that levels of residual proteins in oil are extremely low does not rule out the possibility of a reaction in subjects who are highly sensitized: cases have been reported involving allergic reactions to peanut oil used as a vehicle for epinephrine (1), and, more recently, in infants to the peanut oil found in some milk formulas (6). Current consumption of fats in the diet normally includes sunflower oil and margarine. Thus, it now is advisable to anticipate a possible cross-reactive food allergy to sunflower proteins in subjects who are allergic to mugwort pollen. The allergenic risk of vegetable oils might be more important than now supposed.

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