
Clinical characteristics of patients with mustard hypersensitivity

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Background: Although mustard is frequently consumed in Spain and elsewhere, only isolated case reports of mustard allergy have been reported. No large series of case studies have been published.

Objectives: We sought to describe demographic, clinical, and immunologic characteristics of patients with mustard hypersensitivity and to determine whether any significant differences exist in age, sex, atopic family history, total immunoglobulin E (IgE) level, and specific IgE to mustard ratio among patients with differing characteristics.

Methods: Twenty-nine patients with a history of mustard allergy underwent skin prick tests with mustard, determination of total IgE, and evaluation of specific IgE to mustard. Skin prick tests with *Lolium perenne*, *Olea europaea*, and *Artemisia vulgaris* were done in patients with symptoms of pollinosis. The aforementioned variables were compared between various subgroups of patients for systemic versus local reactions to mustard, association versus no association with allergies to other vegetable foods, and association versus no association with pollinosis.

Results: The male:female ratio was 10:19. Skin prick tests to mustard were positive in all patients. The total IgE geometric mean was 189.3 kU/L. The specific IgE to mustard was positive in all patients (0.7 to >100 kU/L). Of the 29 patients, 19 (66%) had a systemic reaction after consumption of mustard, and 10 (34%) had a local reaction; 14 (48%) had anaphylaxis. Fifteen patients (52%) had symptoms after ingestion of other vegetable foods, and 15 also had typical symptoms of pollinosis. No significant differences were found in age, sex, atopic family history, total IgE, and specific IgE to mustard between the various subgroups studied.

Conclusions: Most study patients with mustard hypersensitivity had a systemic reaction and had associated pollinosis or allergy to other vegetable foods. Mustard sensitivity should be routinely tested in patients with idiopathic anaphylaxis.

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INTRODUCTION

Mustard is an herbaceous plant of the Cruciferae family. The mustard seed is increasingly being used in Spanish cooking as a condiment. In the past, it was also used as a popular remedy for gastric ulcers, constipation, bronchitis, pharyngitis, and hoarseness, either by ingestion or by application as a household liniment.

Mustard can be ingested as is, in mustard sauce, and in other sauces (such as mayonnaise, curry, and ketchup sauces). Mustard powder is usually a mixture of black and white mustard (*Brassica nigra* and *Sinapis alba*). Typically, mustard sauce is composed of mustard seed, salt, vinegar, wheat flour, and other spices and additives.

Although mustard is a spice that is frequently consumed in Spain and the rest of the world, relatively few articles on mustard as an allergen (some case reports, but no extensive series) have been published. Contact dermatitis,^{1,2} contact urticaria,³ and anaphylaxis^{4–11} caused by contact with or ingestion of mustard have been described. A case of acute pancreatitis as a complication of mustard anaphylaxis has

also been published.¹² Further, mustard has been implicated as a cause of occupational asthma.¹³

Mustard allergens, however, are well known and have been characterized by Spanish investigative groups. Sin a I, the major allergen of yellow mustard seed (*S. alba* L.), has been isolated, sequenced,^{14–17} cloned, and expressed.¹⁸ It was the first food allergen to be cloned and expressed by molecular biology techniques.¹⁸

The objectives of this study of mustard allergy were to describe the demographic and clinical features of patients with mustard allergy and to ascertain whether significant differences exist relative to sex, age, atopic family history, total IgE, and specific IgE to mustard between various subgroups of patients. We studied subgroups with the following characteristics: systemic versus local reactions to mustard, association versus no association with other vegetable food allergies, and association versus no association with pollinosis.

PATIENTS AND METHODS

Study Cohort

From 1989 to 1996, 19 female and 10 male patients between 15 and 58 years of age (mean age, 27.3 ± 10.0) underwent evaluation at our outpatient clinic because of mustard allergy. All the patients had symptoms suggestive of mustard allergy less than an hour after its intake.

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Detailed Anamnesis

A detailed history was recorded with respect to symptoms associated with ingestion of mustard, other food-related symptoms, symptoms suggestive of pollinosis, other allergies, and family history of allergy. Patients were categorized into subgroups on the basis of the following features: 1) systemic versus local reactions to mustard; 2) association versus no association with other vegetable food allergies; and 3) association versus no association with pollinosis.

Definitions

A reaction was considered as local when the symptoms affected body regions in direct contact with mustard (for example, the mouth or pharynx) and as systemic when they involved parts of the body not in direct contact with mustard. When the symptoms involved two organs, the reaction was considered to be anaphylaxis.

Skin Prick Tests

Skin prick tests were performed on the volar aspect of the forearm with use of a standardized prick lancet (DHS, Leverkusen, Germany), in accordance with the Subcommittee on Skin Tests of the European Academy of Allergology and Clinical Immunology.¹⁹ Histamine chlorhydrate (10 mg/mL) and physiologic saline solution were used as positive and negative controls, respectively. The maximal wheal diameter and its orthogonal perpendicular were measured 15 minutes after the prick, and the mean of both diameters was recorded as the test size. A mean wheal diameter at least 3 mm larger than the one produced by the negative control was considered a positive reaction.¹⁹

A mustard extract of *B. nigra* at 1:10 weight to volume (Bencard-Bayer, Leverkusen, Germany) was purchased and tested in all patients. If the reported reaction was severe, a skin prick test was initiated with a 1/100 dilution of the purchased solution; if the reaction was negative, skin prick tests with a 1/10 dilution and a pure solution were performed.

The patients underwent skin tests with other food or inhalant extracts on the basis of a history of other symptoms. If the patient demonstrated typical pollinosis symptoms, skin prick tests with *Lolium perenne*, *Olea europaea*, and *Artemisia vulgaris* (ALK-Abelló, Madrid, Spain) were performed. A control group of 373 consecutive patients with pollinosis was used to compare the prevalence of each pollen sensitization in the subgroup of patients with pollinosis. The control group were patients 14 years of age or older who were treated in our outpatient clinic.

Total Serum IgE

Total serum IgE was measured by the microparticle enzyme immunoassay IMx system (Abbott Diagnostics, Abbott Park, IL), following the manufacturer's instructions.

Serum-Specific IgE

Specific IgE to mustard (*Brassica/Sinapis* species) was measured by CAP fluorescence enzyme immunoassay (Pharmacia Diagnostics, Uppsala, Sweden), following the manufac-

turer's instructions. A CAP level equal to or greater than class 1 (0.35 kU/L) was considered a positive reaction.

Oral provocation tests with mustard were not performed because mustard is a nonessential food and because of the high risk of precipitating anaphylaxis with exposure to even small amounts of mustard.

Statistical Analysis

For statistical analysis, IgE values were converted into decimal logarithmic values. The geometric means of total IgE and specific IgE to mustard were calculated.

Data were analyzed with the statistical program RSIGMA (Horus Hardware, 1990, Madrid, Spain). Statistical significance was tested with the Student *t* test, the χ^2 test, or the Fisher exact test, as appropriate.

RESULTS

The demographic characteristics (age and sex), family background of allergy, results of allergologic study, and other characteristics of the entire study group and of the subgroup of patients with anaphylaxis are summarized in Table 1.

Of the 29 patients, 10 (34%) had local symptoms, whereas 19 (66%) had systemic symptoms. Of the 19 systemic reactions, 14 (74%) could be considered anaphylaxis.

The symptoms reported by patients after ingestion of mustard are outlined in Table 2. The most frequent symptoms were angioedema (in 16 patients), dyspnea (in 11), general urticaria (in 10), and oral allergy syndrome (in 8).

Skin prick tests with mustard were positive in all study patients. Total IgE ranged from 18 to 1,567 kU/L, and the geometric mean was 189.3 kU/L. Specific IgE to mustard was also positive in all patients and ranged from 0.7 to >100 kU/L. If an arbitrary value of 100 kU/L is assigned to CAP 6, the geometric mean would be 6.3 kU/L (standard deviation, 1.5 to 27.2; Table 1).

More than 50% of the patients had symptoms suggestive of allergy to other vegetable foods, 17% had allergies to other nonvegetable foods, 52% of patients had symptoms suggestive of pollinosis, and 52% had other allergies (Table 1). The allergies to other vegetable foods are specified in detail in Figure 1. The vegetable foods most often involved were nuts, followed by Rosaceae, Leguminosae, and Compositae. Only two patients had sensitivity exclusively to mustard.

Of the 15 patients with symptoms of pollinosis, all complained of rhinitis and conjunctivitis, and 7 (47%) had asthma. Sensitization to pollens in patients with symptoms of pollinosis and those in a control group with pollinosis is shown in Table 3. Gramineae sensitization was the most frequent (93%), followed by mugwort (73%) and olive sensitization (67%). Mugwort sensitization was significantly more frequent in these patients than in a population of patients with pollinosis ($P < 0.0001$).

No significant differences were found with respect to age, sex, atopic family history, total IgE, and specific IgE between the various subgroups of study patients (Table 4).

Table 1. Epidemiologic and Allergologic Characteristics of Study Patients*

Factor	Entire study group (n = 29)	Subgroup with anaphylaxis (n = 14)
Mean age (range) (yr)	27.3 ± 10.0 (15–58)	27.6 ± 9.2
Sex (M:F)	10:19	4:10
Atopic family history (yes:no)	16:13	8:6
Positive mustard skin prick test	100%	100%
Positive mustard IgE	100%	100%
Total IgE (kU/L)		
Range	18–1567	18–1567
Geometric mean (SD)	189.3 (63.1–568.2)	138.0 (88.6–215.0)
Specific mustard IgE (kU/L)		
Range	0.7–>100	0.7–>100
Geometric mean (SD)†	6.3 (1.5–27.2)	7.0 (1.6–31.3)
Pollinosis symptoms	15 of 29	7 of 14
Positive pollen skin prick tests		
Olive tree	10 of 15	3 of 7
Grass	14 of 15	6 of 7
Mugwort	11 of 15	4 of 7
Allergy to other vegetable foods	15 of 29	6 of 14
Allergy to other nonvegetable foods	5 of 29	2 of 14
Other allergies	15 of 29	7 of 14
No other allergies	2 of 29	2 of 14

* SD = standard deviation.

† If CAP 6 = 100 kU/L.

M, male; F, female; IgE, immunoglobulin E.

Table 2. Clinical Symptoms After Intake of Mustard

Clinical symptom	No. of patients
Angioedema	16
Dyspnea	11
General urticaria	10
Oral allergy syndrome	8
Gastric symptoms	5
Dysphonia	5
Conjunctivitis	4
Rhinitis	3
Loss of consciousness	2
Dysphagia	2
General pruritus	2

Other ingredients of mustard sauce, such as vinegar and pepper, were tolerated by all the patients later in their routine diet. Ingestion of mustard, however, precipitated a reaction.

DISCUSSION

We present a series of 29 patients who had an allergic reaction after consumption of mustard and whose specific IgE to mustard was evaluated by means of skin prick tests or measurement of serum specific IgE, or both. Despite the antigenic potency of mustard, a frequently consumed spice, few cases of hypersensitivity from ingestion have been described^{1–13}; the largest previous series involved three patients.¹⁰ To our knowledge, our current study group is the largest published series of patients with mustard allergy.

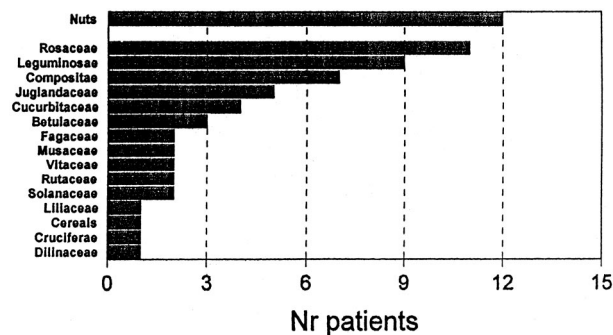


Figure 1. Allergy to other vegetable foods, stratified by botanical families, on the basis of anamnesis. Nuts were considered as part of their corresponding family (almond, Rosaceae; peanut, Leguminosae; sunflower seed, Compositae; walnut, Juglandaceae; hazelnut, Betulaceae; and chestnut, Fagaceae) and also as a collective group (“nuts”).

More than 65% of the mustard-allergic patients in this study experienced systemic symptoms after ingestion of mustard, and nearly half of the entire group had anaphylaxis. This finding correlates with the occurrence of anaphylaxis found in most previously published case reports of mustard allergy.^{4–12} Further, mustard had been implicated previously as a potential cause of anaphylaxis in a series of cases of food allergy-induced anaphylactic shock²⁰ or idiopathic anaphylaxis.²¹ Angioedema was the most frequent symptom reported by our study patients.

Skin prick tests with mustard were positive in 100% of our patients. Notwithstanding the previous description of a sys-

Table 3. Sensitization to Various Pollens in the Subgroup of Patients With Symptoms of Pollinosis (Group A) and in a Control Group with Pollinosis (Group B)*

Pollen	Group A (N = 15)		Group B (N = 373)		P
	No.	%	No.	%	
<i>Lolium perenne</i>	14	93	299	80	NS
<i>Olea europaea</i>	10	67	286	77	NS
<i>Artemisia vulgaris</i>	11	73	77	21	< 0.0001

* NS = no significant difference.

temic reaction to a mustard skin test⁴ and the presence of anaphylaxis in 48% of our patients, skin tests precipitated no systemic reactions in our study group. Only one patient had an extensive local reaction some hours later. The absence of systemic reactions could be the result of the prick test technique, which is safer than the intradermal test²² used by Panconesi et al.⁴ Nevertheless, severe reactions with the prick test have also been described with other foods such as kiwi or fish.²³ Therefore, as a preventive measure, the prick test was performed with commercial diluted mustard extract in some patients who had experienced severe reactions.

Allergy to other members of the Cruciferae family was not routinely tested in all study patients. Only one patient had mild symptoms after ingestion of cabbage and had a positive reaction to a skin prick test for cauliflower and cabbage. In Spain, the consumption of other types of Cruciferae (such as cauliflower and cabbage) is common. Most patients with mustard allergy did not have symptoms after intake of such vegetables and claimed to tolerate them. Some patients had positive skin prick test reactions to cauliflower and cabbage but tolerated them in their diet. This could be because mustard is a seed with allergens resistant to digestion and high temperatures, whereas the other Cruciferae are leafy vegetables, and their allergens are more labile to digestion and high temperatures. This explanation is supported by the publication of only anecdotal reports of allergy to other Cruciferae foods. We do not forbid the consumption of other Cruciferae foods in a patient with mustard hypersensitivity, unless the

patient reports related symptoms. Skin tests and oral provocation tests would then be indicated.

In this series of patients, mustard allergy was frequently associated with pollinosis and with allergy to other vegetable foods by anamnesis. Leanizbarrutia et al²⁴ demonstrated a high prevalence of positive results of intradermal skin tests to mustard in patients with pollinosis, but few of these patients had symptoms after intake of mustard. This finding could be attributable, in part, to a low specificity of the intradermal test (used instead of the skin prick test) or to possible cross-reactivity between the pollen and mustard extracts. If we consider the type of pollen, we found a significant association between mustard allergy and sensitization to mugwort pollen in this study. In addition, we had previously found a significant association between mustard sensitization and Compositae pollen sensitization in children,²⁵ which could be explained by the presence of common allergens. Further studies should be done to confirm this hypothesis. Other investigators have recorded allergens common to other spices, especially from the Apiaceae family, and mugwort pollen.²⁶⁻²⁹ For example, cross-reactivity between coriander and mugwort pollen was demonstrated by radioallergosorbent test inhibition,²⁶ and common allergens were demonstrated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis immunoblotting inhibition between mugwort pollen and Apiaceae spices (anise, fennel, coriander, and cumin)²⁷ as well as between mugwort pollen and pepper (Piperaceae) and paprika (Solanaceae).²⁸

In our evaluation of allergies to other vegetable foods, nuts were the most frequently implicated. Castillo et al²⁹ also found a significant association between allergies to nuts and spices. This relationship could be because both nuts and mustard are seeds and can have common allergens with similar functions. In fact, different seeds (mustard, cottonseed, castor bean, Brazil walnut, and English walnut) share 2S storage albumins or their precursors, which have been recognized as major allergens.³⁰ In addition, hypersensitivity to nuts has been shown to be associated with mugwort pollen sensitization,²⁵ and in a previous article, cross-reactivity between one particular nut (hazelnut) and mugwort pollen was demonstrated by radioallergosorbent test inhibition and so-

Table 4. Comparison of Various Subgroups of Patients*

Factor	Mustard-related symptoms		Symptoms from other vegetable foods		Pollinosis symptoms	
	Systemic (N = 19)	Local (N = 10)	Yes (N = 16)	No (N = 13)	Yes (N = 15)	No (N = 14)
Age (mean ± SD) (yr)	27.9 ± 11.3	26.0 ± 6.9	24.1 ± 7.0	31.2 ± 11.8	24.3 ± 8.3	30.5 ± 10.8
Sex (male:female)	6:13	4:6	5:11	5:8	5:10	5:9
Atopic family history	10 of 19	6 of 10	9 of 16	7 of 13	9 of 15	7 of 14
Log total IgE	2.22 ± 0.54	2.39 ± 0.36	2.42 ± 0.29	2.10 ± 0.62	2.35 ± 0.53	2.19 ± 0.43
Log specific IgE	0.78 ± 0.60	0.83 ± 0.76	0.65 ± 0.64	0.99 ± 0.63	0.69 ± 0.62	0.91 ± 0.68

* SD = standard deviation. For all analyzed factors, no significant differences were found between the various subgroups of patients. IgE, immunoglobulin E.

dium dodecyl sulfate-polyacrylamide gel electrophoresis immunoblotting inhibition techniques.³¹

The factors we studied (sex, age, atopic family history, total IgE, and specific IgE to mustard) did not differ significantly between the various subgroups analyzed. Patients with other associated allergies, such as pollinosis or allergy to other vegetable foods, tended to be younger than patients without them; however, this difference was not statistically significant, perhaps because our sample was small. Fernández-Rivas et al,³² who compared age and total IgE in patients with Rosaceae allergy with or without pollinosis, found total IgE to be significantly higher in the subgroup of patients with pollinosis. They also found that patients without pollinosis were older than those with pollinosis.

Oral provocation tests with mustard were not performed, in accordance with the guidelines used by other investigators,^{4,6,10,11} because mustard is not an indispensable food and because anaphylaxis could develop with exposure to even small quantities of mustard. Further, we noted a clear relationship between symptoms and the ingestion of mustard and the positive results of skin prick tests and specific IgE to mustard. Although some investigators suggest the labial food challenge as a prior step in the oral food challenges,³³ it is controversial because of its low sensitivity in weak reactions and the possibility of false-positive results.

As previously mentioned, mustard is a potent food allergen that causes severe reactions. Sin a I, the major allergen in mustard seeds, interacts with acid phospholipid vesicles, a process that can influence the allergenicity of mustard.³⁴ The pronounced resistance of mustard allergens to high temperatures^{6,35} could also be the reason for the augmented allergenicity of mustard in contrast to other vegetable foods whose allergens are destroyed during cooking.

We can not reach any conclusion about the prevalence or incidence of mustard allergy in our geographic area because this study was not designed to evaluate this factor. Some studies on the prevalence of food allergy have been conducted in Spain. Castillo et al²⁹ found that mustard allergy constituted 7% of food allergy consultations (8 of 120) in the Canary Islands. Joral et al³⁶ reported that 10% of food allergies (3 of 30 cases) were caused by mustard in San Sebastián (northern Spain). Similar reports have also been published in other countries; André et al³⁷ found mustard to be responsible for 11% of food allergy reactions and 3% of the anaphylactic reactions attributable to food allergy. According to these authors, the frequency of mustard sensitization is increasing in comparison to previous studies, a pattern they ascribed to increased consumption and more attentive clinical research. In a study of labial food challenges in 142 children, Rance and Dutau³³ described 23 cases of mustard allergy (16%). Moneret-Vautrin and Kanny²⁰ studied 81 patients with food-induced anaphylactic shock and found that mustard allergy triggered it in two cases. In a cohort of 102 patients with suspected idiopathic anaphylaxis, Stricker et al²¹ performed skin tests with 79 food allergens. They found that 31% (32 of 102 patients) had

at least one positive skin test result to one of the food allergens, and mustard was responsible for the reaction in two cases.

Most of our patients had more than one reaction after consumption of mustard (data not shown). This finding could be related to the masking of mustard in numerous foods (specially processed and restaurant foods) and to the high probability of contamination during food preparation, which makes it difficult to avoid mustard.

Avoidance of the triggering substance is the key treatment for allergen-induced anaphylaxis. Because of the high probability of mustard contamination in some foods, we advised all our patients with anaphylaxis to carry epinephrine for auto-injection as a rescue medication and instructed them in its appropriate use for anaphylactic reactions.³⁸ We also prescribed an orally administered corticosteroid and antihistamine.

CONCLUSION

Mustard is a potent food allergen that frequently causes systemic or even anaphylactic reactions. It is often associated with pollinosis or allergy to other vegetable foods. We believe that anamnesis plus skin prick tests with mustard and determination of serum specific IgE to mustard is a good approach for the diagnosis of mustard hypersensitivity.

Anaphylaxis is a severe and life-threatening condition, and every effort should be made to determine its cause. We propose the use of a skin test battery with selected allergens most frequently involved in anaphylaxis, which includes mustard. Mustard can be present but masked in many foods and has been shown to produce anaphylaxis. Further, mustard should be included in the skin test battery for patients treated for urticaria and angioedema. Labeling of processed and fast foods should be improved to avoid the risk of exposure in patients with known allergies to substances that can be found in trace amounts in these types of food.

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