



Case Report

Occupational Allergic Rhinitis and Asthma Caused by Guar Gum

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Abstract

Background: Guar gum is commonly used as a gelling substance in the food and pharmaceutical industries as well as in the manufacturing of paper and carpets. Only a few cases of respiratory or conjunctival sensitization have been reported previously.

Objective: To describe a case of occupational rhinitis and asthma from guar gum in a 29-years old woman working in a food processing factory.

Methods: The patient underwent routine otorhinolaryngological, pulmonary and allergological examinations including skin prick tests (SPT) with 15 common aeroallergens, 4 cereals, 3 storage mites, 9 spices and herbs, 14 vegetables, latex and 17 food additives including guar gum. Radioallergosorbent test (RAST) was made for guar gum, latex and banana. Placebo controlled nasal provocation test and a specific bronchial provocation test were performed with powdered guar gum.

Results: SPTs to guar gum as well as to mugwort, latex, cereals, spices and herbs, nuts and almond, banana and kiwi fruit were positive as were also guar gum and latex RASTs. Both nasal and bronchial challenge tests evoked strong positive reactions.

Conclusions: Exposure via the airways to vegetable gums is an occupational health hazard and, therefore, workers should be made aware of the risk of sensitization. In this patient, positive provocation test results confirmed the diagnosis of guar gum induced allergic rhinitis and asthma.

Introduction

Vegetable gums are commonly used in the food processing industry and many of these materials, such as guar gum and tara gum, originate from leguminous plants. Guar gum is derived from the seeds of the plant *Cyamopsis tetragonolobus* grown in India and it belongs to the vegetable gums. These materials are high polymer carbohydrates, galactomannans, and are used as protective colloids and emulsifying agents [1]. Since guar gum (E412) is soluble in cold water along with tara gum (E417), it is commonly used in the food, pharmaceutical and cosmetic industries as a gelling and thickening substance. Furthermore, it is also used in the manufacturing of paper and carpets. During making jam, guar gum thickens the mixture of cooked berries or fruits and sugar, and workers in the jam factory handle the guar gum powder. In small factories the blending of the guar gum is performed manually

from bags in open vessels without any respiratory protection. In such a situation, inhalation of this light powder cannot be avoided. Nonetheless, only a few cases of respiratory and conjunctival sensitization to guar gum have been reported previously [2-6].

Case Report

We describe a case of occupational rhinoconjunctivitis and asthma from guar gum in a 29-year old woman, who had made jam in a food processing factory for eight years. For six years she had suffered from work-related nasal and ocular symptoms. As a child the patient had suffered from hay fever but as an adult she experienced symptoms only if she entered a hay barn. Since the age of 16, she had got tingling of the mouth and angioedema in the lips from kiwi fruit and banana and therefore she tried to avoid these fruits. Furthermore, flour dust (wheat, rye, oat and especially

barley) and cigarette smoke evoked dyspnoea and latex products provoked contact eczema.

In the jam factory, the patient had worked at first for a few months in the packing department, and at that time she experienced no direct allergen exposure and did not suffer any occupational symptoms. When she was transferred to the jam cooking kitchen, she started to handle powdered guar gum and tara gum every day as she emptied these thickening substances into the cooking jam from their storage bags. Soon after starting this work, she began to experience sneezing, nasal obstruction, rhinorrhea and itching of the eyes while she was in the workplace. Occasionally she also experienced skin flushing resembling urticaria combined with mild dyspnoea. These symptoms could be relieved by antihistamine drug therapy. The lower airway symptoms became worse and during the last four years she started to experience wheezing and severe dyspnoea when handling guar gum. Outside the workplace she did not experience rhinitis, wheezing or dyspnoea, but since beginning this work she had occasionally experienced severe hypersensitivity reactions from foodstuffs. At her own wedding she suffered such a severe attack of urticaria and dyspnoea from home-brewed beer, that she had to be rushed to the hospital. Some years later she required urgent medical help when she experienced an anaphylactic reaction after eating a fruit salad which, unbeknown to her contained bananas and canned juice from a mixture of tropical fruits.

The patient was examined in the Kuopio University hospital (allergologic, otorhinolaryngologic and routine lung function examinations) and at the Finnish Institute of Occupational Health (FIOH) (specific bronchial challenge tests). When the patient at first was referred to the Kuopio University Hospital, the occupational symptoms had lasted for six years.

Methods

Skin tests

Skin prick tests (SPT) were performed with 15 common aeroallergens (alder, birch, meadow fescue, meadow grass, timothy, dandelion, mugwort, cow, horse, cat, dog, *Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, *Alternaria alternata* and *Cladosporium herbarum*) 4 cereals (barley, oat, rye, wheat), 3 storage mites (*Acarus siro*, *Lepidoglyphus destructor*, *Tyrophagus putrescentiae*), 9 spices and herbs (caraway, cinnamon, coriander, curry, garlic, mustard, paprika, vanilla, white pepper), 14 vegetables and fruits (carrot, celery, pea, potato, soy bean, swede, sweet pepper, tomato, almond, apple, banana, hazelnut, kiwi, peanut), latex and 17 food additives (guar gum, gum arabic, tara gum, xanthan gum, agar, algarroba, anthocyanin, carmine, carrageenan, cocoa, gelatin, liquorice, lutein, patent blue, pectin, sodium benzoate, sodium metabisulphite). SPTs with common environmental airborne allergens, cereals, storage mites and

latex were performed using commercial test fluids (ALK-Abelló A/S, Hørsholm, Denmark). For the other allergens, SPTs were made with the prick-prick method using the food or product in question as fresh (vegetables and fruits), dried (spices, herbs) or in some other available form (food additives). The tests were performed and interpreted according to the recommendations of the European Academy of Allergology and Clinical Immunology [7]. The positive control was histamine hydrochloride (10 mg/mL) and negative controls were allergen diluent for the commercial allergens and physiological saline for the foodstuffs and additives. A wheal diameter of at least 3-mm and half of the histamine control was considered as positive, provided that there was no reaction from the negative control.

In vitro tests

In a complementary manner to the SPTs, RASTs were made for guar gum, latex and banana. RAST results were graded in 6 classes (class 0= 0.00-0.34, class 1= 0.35-0.69, class 2=0.70-3.4, class 3=3.5-17.4, class 4=17.5-50.0 and class 5 >50.0 IU/L). Classes 2-5 were interpreted as positive.

The diagnosis of occupational rhinitis

In a single-blinded nasal provocation test (NPT), the patient was initially challenged with the negative control, which was lactose powder. This was applied with a cotton swab on the mucosa of the inferior turbinate of one nasal cavity. Nasal reactions were observed during the following 30 minutes and recorded in rhinoscopy by an otorhinolaryngologist in our study group. Subsequently, the nasal challenge test was similarly performed on the other nasal cavity, this time with guar gum powder. The interpretation of the challenge test was based on 1.) rhinoscopy findings (hypersecretion and mucosal oedema) and symptoms (nasal obstruction, itching and sneezing) and 2.) minimum cross-sectional area (MCA) changes in acoustic rhinometry. The test was considered positive if hypersecretion (weighed) and at least one of the other objective findings or symptoms were recorded [8].

The diagnosis of asthma

The diagnosis of asthma was based on the patient history and clinical examination, in addition to objective evidence of reversible airway obstruction according to the national consensus on the criteria for the diagnosis of asthma [9]. Lung function was determined by flow-volume spirometry (Sensor Medic Vmax 22D, Sensor Medics Corporation, Yorba Linda, California, USA) and Viljanen's reference values were used [10]. Peak expiratory flow (PEF) was recorded by the Mini Wright PEF-meter (Mini Wright, Clement International, Harlow, UK) using salbutamol 200 µg (Buventol Easyhaler™, Orion Pharma, Helsinki, Finland) as the bronchodilating agent. Bronchial hyperresponsiveness was assessed first with the Dry Air Hyperventilation test [11] and later, when the patient had already used inhaled corticosteroid for a

few months, in conjunction with the histamine provocation test according to Sovijärvi [12].

The diagnosis of occupational asthma

Peak expiratory monitoring at home and at the workplace was performed according to Burge [13]. Before the provocation test, the stability of asthma was assessed by measuring diurnal FEV₁ and PEF values (no daily variation over 20 %) and by spirometry with bronchodilating test (no significant bronchodilating effect). The specific bronchial provocation tests for control and active agents were performed at the FIOH in a challenge chamber with isolated ventilation, designed for this purpose. In the control test, lactose powder was sprayed once in a minute for 30 minutes into the chamber where the patient sat being supervised from outside by a trained nurse. In the first active test, guar gum powder mixed with lactose powder in a ratio 1:10 was similarly sprayed into the chamber, and in the second active test, pure guar gum powder was sprayed. The asthmatic reaction, i.e., lower airway obstruction, was followed up by a pocket sized microspirometer (One Flow, STI MEDICAL, Saint-Romans, France), recording forced expiratory volume in one second (FEV₁) values before, during and for 24 hours after the challenge test. The expiratory manoeuvres were monitored by a nurse to ensure that the procedure was repeatable and otherwise adequately performed.

Results

SPT results were positive (a 5 x 5 – 13 x 10 mm wheal) to mugwort, latex, all the cereals, curry, white pepper, mustard, paprika, coriander, peanut, hazelnut, almond, banana and kiwi fruit. Histamine hydrochloride (positive control) evoked a 6 mm wheal whereas allergen diluent (negative control) created no wheal. The occupationally used food additives were assessed, of these guar gum provoked a 6 x 5 mm wheal but tara gum caused only a 1 mm wheal. It is noteworthy that during skin testing the patient experienced immediate but transient nasal symptoms while she was pricked with guar gum, tara gum and mugwort. RAST results for guar gum and latex were positive, class 3 for both allergens whereas banana RAST was negative.

In the NPT, placebo caused slight nasal mucosal swelling bilaterally but no other signs or symptoms. In contrast, guar gum evoked immediately almost total obstruction of that nasal cavity and profuse rhinorrhea as well as conjunctival redness on the ipsilateral eye and facial flushing.

The diagnosis of asthma was suggested by the presence of dyspnoea and wheezing, which repeatedly occurred in association with occupational exposure to guar gum. The patient's spirometry showed mild obstruction (FVC 3.07 /79%, FEV₁ 2.34/ 70% and FEV% 76/88%) [10] without reversibility after bronchodilating medication with salbutamol. During PEF monitoring when symptoms were present, repeated significant bronchodilating effects

with salbutamol (> 15 %) were recorded, which was considered to confirm the diagnosis of asthma. PEF monitoring during workdays and off-days did not reveal any significant difference between workdays and off-days or any significant day-to-day variation. No hyperresponsiveness to dry air was found (FEV₁ decrease only 4.7 %), but subsequently, the histamine provocation test did reveal mild hyperresponsiveness, with the PD₁₅ value being 0.13 mg. The bronchial provocation test with lactose did not show any significant FEV₁ decline nor did it provoke symptoms. Provocation with 10 % guar gum was also negative in terms of FEV₁ recordings, although the patient experienced sneezing and cough resembling her symptoms at work. Provocation with pure guar gum induced an immediate decrease in FEV₁: 18 % compared to the starting level and 19 % compared to the control test. During this test, the patient experienced coughing as well as nasal and ocular itching, but lung auscultation findings remained normal. The positive guar gum provocation tests confirmed the diagnoses of occupational rhinitis and asthma.

Discussion

Natural gums are high polymer carbohydrates, which originate from different parts of many plants. These materials produce mucilages when they react with water [1] and thus vegetable gums are widely used in the food processing industry. Many of these gums originate from leguminous plants. For example, guar gum is derived from the seeds of an Indian vegetable *Cyamopsis tetragonolobus* and it is commonly used in the food, confectionery, soft drink and also pharmaceutical industries [1]. Therefore, exposure to guar gum is possible even after an individual is removed from the occupational environment. Furthermore, in addition to the high degree of clinical and immunological cross-reactivity between the different legumes, there is also cross-reactivity between legumes and other plant-derived foods [14]. It seems in our patient that the severe, anaphylactic allergic reactions from fruit products could be attributable to the latex-fruit syndrome [15].

The first reported case of occupational rhinitis due to guar gum was manifested with obstructive sleep apnoea symptoms in a pet food plant employee [2]. Occupational asthma has been reported in workers exposed to guar gum in a carpet-manufacturing plant, and in the pharmaceutical industry [4,5] and occupational rhinitis has been described in employees in a power cable laboratory and a paper factory [3]. Although guar gum has been reported to cause both occupational allergic rhinitis and asthma, it is still commonly handled without sufficient, personal protective ventilation equipment. In our patient, rhinoconjunctivitis was the first symptom she experienced and this phase lasted for two years before significant lower respiratory tract symptoms appeared. It is common that the symptoms of rhinoconjunctivitis precede the onset of asthmatic symptoms, especially after exposure to

high-molecular-weight agents [6]. Although rhinitis is still often considered as a minor symptom, it should always be considered as a warning sign of a more disabling disease, asthma.

To avoid occupational rhinitis and asthma, exposure to powdered foodstuffs should be minimized. This can be achieved in many different ways. The most preferred way is to automatize the working process. If the exposure is only occasional, the employee should be equipped with an efficient respiratory protective device, preferably an air-filtering hood. If symptoms do appear, the worker should not be asked to perform that task and should be examined in an occupational health care unit. After the diagnosis of occupational rhinitis or asthma, then removal from exposure is necessary to avoid worsening of the condition. Also, the cross-reactions with other fruit products must be kept in mind and the patient should be informed of this risk.

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