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Allergic sinusitis and severe asthma caused by occupational exposure to locust bean gum: Case report

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Abstract

We present a case that highlights the difficulties with diagnosis and the dangers of occupational allergic sinusitis and asthma left unrecognized. We describe the case history of a man who experienced work-related symptoms 1 year after beginning work as a cheesemaker at a creamery, and whose respiratory symptoms progressively worsened over 16 years before an occupational cause of his asthma was identified. His initial discrete episodes of sinusitis and acute bronchitis evolved into persistent asthma of increasing severity with exacerbations requiring repeated emergency room treatment. The case described in our report emphasizes the importance of clinician diagnosis of OA, and subsequent removal from exposure, such that asthma severity does not progress to near-fatal or fatal asthma in the sensitized worker. As demonstrated by this case report, identification of an occupational cause of asthma relies on a high degree of suspicion and excellent detective work by the clinician.

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AUTHORS' CONTRIBUTIONS

Brie Hawley contributed to the conception of the work, interpreting the data for the work, drafting the work, final approval of the version to be published, and agrees to be accountable for all aspects of the work in ensuring that questions relating to the accuracy of any part of the work are appropriately investigated and resolved. Kristin Cummings and Rebecca Bascom contributed to the design of the work, acquiring and interpreting the data, revising the work critically for intellectual content, and final approval of the version to be published. Mohammed Mohammed and Anne Dimmock contributed to the acquisition and interpretation of the data used in the work.

ETHICS APPROVAL AND INFORMED CONSENT

The authors obtained written informed consent from the patient described in this case report prior to publication.

DISCLOSURE (AUTHORS)

The authors declare no conflicts of interest.

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DISCLAIMER

The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the National Institute for Occupational Safety and Health. Mention of product names does not imply endorsement by NIOSH/CDC.

Keywords

asthmagen; carob bean gum; locust bean gum; occupational asthma; severe asthma

1 INTRODUCTION

Occupational asthma is the most widely reported occupational lung disease. Previous studies suggest that 5–25% of adult asthma cases are caused or exacerbated by occupational exposures.[1–4] A recent review article by Tarlo and Lemiere[4] emphasized that occupational asthma "outcomes are best when the diagnosis is established early, the exposure is stopped, and the asthma is not yet severe." Because the list of sensitizing or irritant agents that initiate asthma is extensive and new asthmagens are discovered each year, a clinician's ability to identify the causal workplace asthmagen relies upon a high degree of suspicion and excellent detective work. Failure to identify a causal agent and eliminate exposure can result in increased disease severity, substantial morbidity, or even death.

We present a case that highlights the difficulties with diagnosis and the dangers of occupational allergic sinusitis and asthma left unrecognized. Although asthma from locust bean gum (LBG) exposure has been previously reported, to our knowledge, this is the first severe case.[5–7] LBG, also known as Carob bean gum, is one of several seed-derived vegetable gums and is derived from grinding the endosperms from the seeds of Carob trees, which fall under the family Fabaceae.[8] LBG is used as a thickening and gelling agent in food production,[8] and recently has also been used in drug delivery systems in pharmaceutical industries, and in tissue scaffolds in biomedical applications.[9,10] Often sold and used as a powder,[8] LBG can be aerosolized during various occupational tasks in food production, pharmaceutical, or biomedical industries. Approximately 2.9 tons of LBG were imported by the United States in 2011 alone.[11] We describe the case history of a man who began with symptoms 1 year after beginning work as a cheesemaker at a creamery, and experienced progressively severe respiratory symptoms over 16 years. His initial discrete episodes of sinusitis and acute bronchitis evolved into persistent asthma of increasing severity punctuated by exacerbations requiring repeated Emergency Room treatment despite daily controller inhaler therapy and courses of corticosteroids. His severe dyspnea, exercise limitation, and chest pain led to referral for cardiac disease, with eventual cardiac catheterization excluding coronary artery disease. Peak flow monitoring that demonstrated improved peak flow rates during a work holiday provided an impetus to refer him for evaluation of suspected occupational asthma. Specialty evaluation, including careful review of his work history and safety data sheets from his workplace, led to allergy testing which confirmed LBG as the cause of his sinusitis and asthma. A recommendation for medical removal ended his need for repeated emergency room care of his asthma although he continues with severe persistent asthma requiring daily medication and work accommodations.

The case of severe occupational sinusitis and concomitant asthma described here supports the united allergic airway hypothesis.[12] Additionally, this case highlights the importance of identifying an Occupational Sentinel Health Event early in the disease process such that

controls designed to mitigate exposure may be implemented[13] thus avoiding progression to severe disease. This case further emphasizes the value of peak flow monitoring connected to prolonged work absences as a means of screening for occupational asthma, and of obtaining a thorough work history, and pursuing information about workplace exposures to establish a diagnosis early when the airway disease is not yet severe.

2 CASE REPORT

A 35-year-old man with no significant past medical history began working at a creamery in 1986. He held several ancillary positions before becoming directly involved in cheesemaking in 1995 (Fig. 1). His tasks as a cheesemaker's assistant and later as a cheese-room processor included obtaining and measuring finely powdered locust bean gum (LBG) from bulk containers in storage areas and adding the LBG to cream cheese mixtures. In early 1996, he presented with the complaint of wheezing upon exposure to cold air. Subsequently, he was seen repeatedly over the course of 8 years for recurrent episodes of acute respiratory illness, with listed diagnoses including rhinitis, sinusitis, acute bronchitis, and cold-induced asthma. Therapy gradually intensified from brief courses of a short-acting beta-agonist, an antitussive, and antibiotics in 1996, to include oral corticosteroids, first prescribed in 1998; nasal steroids, first prescribed in 2000; a long-acting beta-agonist, first prescribed in 2001; and an inhaled steroid, first prescribed in 2003 (Fig. 1).

In 2003, chronic symptomatic sinusitis prompted functional endoscopic sinus surgery with pathology demonstrating increased eosinophils and inflammatory polyps. The surgery was complicated by post-operative hemorrhage requiring air transport to a tertiary care medical center and red cell transfusion. Hematologic evaluation revealed a disorder of platelet function which impairs hemostasis. A prolonged at-home convalescence was required to recover from these events, during which he noted complete resolution of his respiratory symptoms. He was treated with daily nasal steroids post-operatively and returned to work, but did not require inhaled corticosteroids or beta-agonists for 18 months (Fig. 1). After his return to work, his acute and chronic upper and lower respiratory symptoms gradually returned, as did the eosinophilia, peaking at 800 in 2010. Initial pulmonary consultation indicated possible mild allergic triggers, but testing for workplace allergy was not pursued. Despite ongoing therapy with multiple inhalers, courses of antibiotics and oral steroids, and addition of a leukotriene receptor antagonist, his asthma control score (ACT) of 16 (ACT scores range 5–25; score of >19 indicates control), indicated his asthma was not under control. Additionally, spirometry showed marked reversible air flow obstruction with hyperinflation and air trapping (Table 1). During this period, he had visited the Emergency Department multiple times for dyspnea, chest pain, and marked exercise limitation, leading to referral for cardiology consultation.

He was referred in 2011 to a second pulmonologist. Discussion of possible symptom triggers revealed that his eyes and skin would sometimes itch at work and he had difficulty breathing when performing tasks that involved scooping a powder and adding the powder to cheese mixtures. Consultation with a colleague with training in occupational medicine led to immediate initiation of portable peak expiratory flow rate monitoring. Incidentally, he was scheduled for a 10-day holiday work break later that month. Evaluation in January showed

that when he was away from work his peak expiratory flow improved from 200 lpm to 400 lpm. Referral to a pulmonologist with training in occupational medicine led to further discussion of his work tasks and review of Safety Data Sheets (SDS's) requested from the workplace identified the powder as LBG. A subsequent flurorescent enzyme-linked immunosorbent assay (ImmunoCAP, Thermo Fisher Scientific-Phadia AB, Uppsala, Sweden) quantified high levels (85 kU per L) of LBG specific IgE (Table 2). Consequently, the patient was removed from job duties that required the use of LBG in mid-2012. At that point he had worked for 16 years as a cheesemaker at a creamery.

His need for emergency room care ended as soon as he was removed from contact with the LBG powder. Additionally, his FEV1 increased by almost a liter and a half over the next 2 years (Table 1). Although his ACT scores improved, he continued to need daily inhaler therapy with inhaled corticosteroids and long acting beta agonists as well as the leukotriene modifier. A cardiac catheterization showed no coronary artery disease, and he returned to being able to exercise vigorously. He required inguinal hernia repair, a condition that developed during the years he was having severe coughing.

Because his tasks as a cheesemaker required the use of LBG powder, he had to be removed from work in the cheese room and was forced to bid for other jobs. His subsequent relocation, however, included job duties that required the use of cleaning chemical irritants and sensitizers. Although LBG was identified as the primary sensitizer, he had developed generalized airway reactivity to chemicals. Cleaning tasks associated with symptoms included floor stripping and surface cleaning with quaternary ammonium compounds. Subsequently, he was reassigned to custodial tasks in office areas which did not require the use of these triggers and his symptoms improved.

3 DISCUSSION

Identification of an Occupational Sentinel Health Event has important preventive implications, for both a patient and a patient's coworkers.[13] However, as occurred in this case, occupational respiratory disease that occurs outside of traditional industrial settings can be difficult to recognize, and can delay diagnosis and appropriate therapy. Previous studies suggest that occupational asthma (OA) is a common, but often unrecognized disease.[14,15] Occupational upper airway disease is often more prevalent than OA[16] and several studies suggest that rhinosinusitis may precede or occur concomitantly with asthma.[16,17] The common airway hypothesis suggests that occupational upper airway disease should alert a clinician to a risk for lower airway involvement.[16–19] Despite associations between occupational rhinosinustis and OA, occupational upper airway disease is frequently not regarded as serious.[18] In this case, the severity of the eosinophilic sinusitis led to a need for surgery with a serious post-operative complication. The patient was temporarily able to be taken off daily asthma medications during his time away from work while he recovered from surgery. Upon return to work, his acute and chronic upper and lower airway symptoms gradually returned. Nine years passed after his sinus surgery with recurrence and progression to chronic rhinosinusitis and severe persistent asthma before his occupational disease was diagnosed and he was removed from exposure. Recognition of

occupational upper airway disease may allow a clinician to recommend preventive measures when the asthma is not yet severe and avert this progression.

When OA is suspected, serial peak expiratory flow measurements taken during times at work and times away from work can be used to confirm a work-related pattern.[20–22] If OA is confirmed using this approach, the exposure associated with OA may still be difficult to readily identify. Parhar et al[14] observed that clinicians most readily recognized OA when the asthma was caused by an asthmagen with which they are already familiar. Additionally, time constraints can preclude clinicians' obtaining a thorough work history.[14,23] Clinicians can enlist the help of trained occupational health nurses or nurse practitioners who can assist in obtaining a comprehensive occupational history.[24] Clinicians should consider consulting with occupational hygienists familiar with the workplace processes and practices.[24] Workplaces where known asthmagens are used may have occupational health programs aimed at the early identification of incident cases. Referral to an occupational medicine specialist may also be warranted. The medical records for this case indicated occasional comments about his job, but no knowledge of the specific exposure. Although the process of identifying the potential causal workplace agent demands clinician awareness and time, the consequences of unrecognized asthma and a delayed diagnosis can be fatal. Furthermore, unrecognized and uncontrolled asthma results in a heavy public health burden and economical cost. In 2010, there were 1.8 million asthma-related Emergency Department visits and 439 000 asthma hospitalizations in the United States alone[25] and previous studies highlight that an estimated 16.3% of all cases of adult-onset asthma are caused by occupational exposure.[4,26]

OA is complex in that it can be caused by four mechanisms, to include (1) IgE-mediated sensitization to an allergen; (2) non-IgE-mediated sensitization; (3) irritant-induced asthma or reactive airways dysfunction syndrome; or (4) a combination of sensitization and irritant-induced mechanisms.[27,28] Of these mechanisms, specific diagnostic immunologic tests for IgE or IgG sensitization are available and can identify many, but not all, high-molecular weight asthmagens. On the other hand, few immunologic tests are available for low-molecular weight asthmagens. When specific diagnostic tests are unavailable, the ability to link an occupational exposure to asthma is only as effective as the clinician's ability to identify a potential workplace exposure that is associated with OA and to perform serial clinical assessments.

Workplace safety data sheets (SDS's) provide helpful information about chemical irritants and toxicants and can alert a clinician to potential occupational respiratory hazards.[20] However, SDS's are not always utilized by a clinician. Indeed, as noted in this case report, 16 years of work-related symptoms elapsed before a pulmonologist with occupational medicine training reviewed SDS's from his workplace for possible exposures. Furthermore, as highlighted by Bernstein, sole reliance upon the health hazard information presented in SDS's may not be sufficient.[29] We note that in this case, locust bean gum was evident on the SDS; however, in other cases chemical names and formulas may be omitted from SDS's if the manufacturer deems the information trade secret. Additionally, companies are not required to state which products may be respiratory sensitizers or share documented clinical information relevant to occupational lung disease.[29] A clinician may need to contact the

chemical manufacturer and inquire about omitted information in the SDS. Another useful resource is asthmagen information published by the Association of Occupational and Environmental Clinics (AOEC). As Bernstein emphasizes, clinicians need to be persistent when seeking this information so as to avoid an unnecessary delay in diagnosis.[29]

Delays in diagnosis can be fatal for OA. We describe here a case that resulted in severe and refractory asthma, a group that is at increased risk for fatal asthma.[30] Fatal OA has been described for workers in wide-ranging work environments to include food industries,[31] farming,[32] pharmaceutical and nutraceutical industries,[33,34] cosmetology,[35] chemical factories,[36] autobody industries,[37] metal foundries,[38] and printing industries.[39] Fatal asthma cases documented in the previous studies were described in workers that ranged from 26 to 75 years of age, and the delay between the onset of occupational asthma symptoms and death ranged from 6 months to 20 years. The previously described fatal asthma cases emphasize the grave consequences of failure to reduce or eliminate exposure in unrecognized OA.

Agents associated with previous fatal OA cases include flour dust, papain powder, shark cartilage dust, hair dye, bicycloheptadiene dibromide, acacia gum, and isocyanates. Of these agents, only isocyanates and flour dust have US occupational exposure limits, despite documented fatal outcomes. Moreover, it is important to note that even when occupational exposure limits do exist, the exposure limits may not be protective for a worker that has become sensitized.[34,40] Clinician diagnosis of OA and subsequent exposure reduction or removal from exposure are necessary protective measures to ensure that asthma severity does not progress to near-fatal or fatal asthma in the sensitized worker. However, as demonstrated by this case report, identification of an occupational cause of asthma relies on excellent detective work by the clinician.

We describe here a worker sensitized to LBG with occupational sinusitis and asthma that remained undiagnosed for the 16 years that the patient reported increasingly severe respiratory symptoms. Despite his development of sinusitis and intermittent asthma within a year of being transferred to cheese-maker duties, the cause of his OA was not identified until persistent uncontrolled asthma had developed including multiple Emergency Department visits for severe asthma. In summary, this case report emphasizes the importance that clinicians (1) have an elevated index of suspicion for occupational sinusitis and OA, especially in patients presenting with an accelerating disease history; (2) obtain a thorough occupational history (3); utilize peak expiratory flow measurements to identify a work-related pattern; (4) consult with occupational health professionals familiar with workplace processes and practices; (5) use workplace Safety Data Sheets and other information sources to identify potential asthmagens; and (6) when possible, utilize immunologic testing that includes suspected workplace asthmagens such that a diagnosis and removal from exposure may be established early when the asthma is not yet severe.

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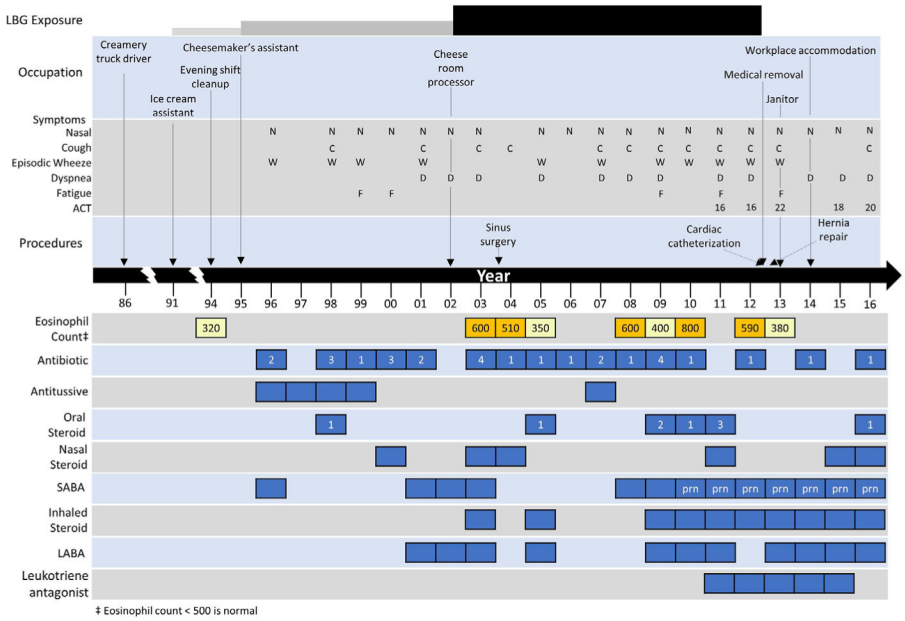

FIGURE 1.

Locust bean gum (LBG) exposure and symptoms, medications prescribed, medical procedures, and eosinophil count in a cheese-maker with unrecognized occupational etiology for sinusitis and asthma. ACT indicates asthma control score. SABA and LABA indicate short-acting beta agonist and long-acting beta agonists, respectively. Prn indicates medications that were used on an as needed basis

TABLE 1

Pulmonary function test results

Exposure timeline (year)	18 Months prior to removal from workplace exposure to LBG[a] (2011)		18 Months after removal from workplace exposure to LBG; 6 months after removal from exposure to chemical irritants (2014)		48 Months after initial testing (2015)	
Parameter	**Value**	**% Pred**	**Value**	**% Pred**	**Value**	**% Pred**
FEV1 (Pre)[b]	3.08 L	74	4.50 L	108	4.12 L	99
FEV1 (Post)[b]	4.11 L	99	4.58 L	110	4.25 L	103
% Change	33	–	2	–	3	–
FVC	5.43 L	101	7.03 L	129	6.35 L	117
FEV1/FVC%	57	–	64	–	65	
TLC (L)	9.53	129	10.08	134	8.3	110
RV (L)	4.10	189	3.05	134	1.91	83
Interpretation	Obstruction with significant bronchodilator response, hyperinflation, and air trapping		Improved FEV1 and air trapping but persistent hyperinflation.		Resolution of air trapping and hyperinflation	

[a] LBG refers to locust bean gum.

[b] Refers to pre- and post-administration of a bronchodilator.

TABLE 2

Respiratory allergen profile results from fluorescent enzyme immunoassay (Immuno-CAP® [Thermo Fisher Scientific-Phadia AB])

Allergen	Value (kU/L)	Class[a]
Horse dander	<0.35	0
Cat dander	<0.35	0
Dog dander	<0.35	0
Dermatophagoides farina	<0.35	0
Cockroach	0.89	2
Dermatophagoides petronyssinus	0.37	1
Bermuda grass	<0.35	0
Timothy grass	<0.35	0
Penicillum notatum	<0.35	0
Aspergillus fumigatus	<0.35	0
Alternaria alternata	<0.35	0
Maple (box elder)	<0.35	0
Mountain cedar	<0.35	0
Oak	<0.35	0
Birch	<0.35	0
Elm	<0.35	0
Walnut tree	<0.35	0
Sycamore	<0.35	0
Cottonwood	<0.35	0
White ashes	<0.35	0
Common ragweed	<0.35	0
Mugwort	<0.35	0
Rough pigweed	<0.35	0
Sheep sorrel	<0.35	0
Locust bean gum	85	5
Total IgE	283	–

[a]Class designations are classified by IgE kU/L. Class designations of 0–6 indicate IgE concentrations of <0.35, 0.35–0.7, 0.71–3.5, 3.51–17.5, 17.51–50, 50.01–100, or greater than 100 kU/L, respectively.