

Occupational Reactions to Foods

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The spectrum of occupational diseases most commonly seen in the food industry includes occupational asthma, rhinitis, conjunctivitis, dermatitis, and hypersensitivity pneumonitis. Occupational asthma represents between 3% and 20% of all asthma cases and is the most common form of occupational lung disease. Occupational skin diseases may represent between 10% and 15% of all occupational diseases, and they have significant economic impact. Hypersensitivity pneumonitis affects the food industry, with farmer's lung representing a common form of the disease. Each of these diseases may have serious and potentially irreversible effects on the health of a farmer, food processor, or food preparer, even after removal of the offending exposure.

Introduction

The food industry employs probably the largest number of workers who are exposed to numerous allergens/antigens that are capable of inducing immunologic reactions leading to occupationally related diseases. Such reactions may occur at every level of the industry, from growing and harvesting of crops or animals to storage of grains to processing and cooking of food substances. Food allergy, by IgE- and non-IgE-mediated mechanisms, is known to affect the skin, gastrointestinal tract, and respiratory system. Occupational reactions to food allergens/antigens may be divided into three primary categories: occupational asthma, hypersensitivity pneumonitis, and occupational dermatitis.

In order to make a diagnosis of an occupational disease, existence of the disorder prior to employment in the current position must be ruled out. In addition, an agent known to cause the occupational disease must be identified and isolated from the work site. In the case of occupational dermatitis, the skin should clear up after removing the patient from the workplace. In occupational lung diseases, the symptoms may be slow to resolve or remain unchanged long after removal from the workplace.

This article discusses each of these types of reactions in greater detail and gives several examples for each type of reaction.

Occupational Asthma

A generally agreed upon definition of occupational asthma is variable air flow limitation and airway hyperresponsiveness due to causes and conditions that are attributable to a particular occupational environment and not to stimuli encountered outside the workplace [1•]. Another pulmonary disease that is significant with respect to occupational lung disorders is reactive airways dysfunction syndrome (RADS) [2]. The difference between these two diseases is that RADS occurs after a single high-intensity exposure to irritant materials, while occupational asthma results from previous sensitization to a substance and, therefore, has a latent period. There is debate as to whether RADS is a true entity or merely a non-immunologic form of occupational asthma that does not require a latent period. The latency period for occupational asthma is highly variable, depending on the specific agent and the amount and duration of exposure.

Sensitizing materials that cause occupational asthma often are viewed as high molecular weight (HMW) or low molecular weight (LMW) agents. HMW (>2 kD) allergens frequently cause occupational asthma through an IgE-dependent mechanism. LMW (<1 kD) allergens act as haptens that must be conjugated to a carrier protein to be allergenic. LMW allergens cause disease through largely unknown mechanisms, though non-IgE-mediated and cell-dependent immunologic mechanisms appear to be important. A variety of materials known to cause occupational asthma or rhinitis in food or food-related industries are summarized in Table 1. A more extensive list of airway sensitizing agents may be found in a recent review [3••].

Epidemiology

Occupational asthma is the most common form of occupational lung disease. Asthma in workers represents 3% to 20% of all asthma patients. The variability in the percentage of all asthma most likely is due to the difficulty in obtaining data in a disease with an incidence of less than 1% annually. In the Finnish Registry of Occupational Diseases, it was determined that the mean annual incidence rate of occupational asthma was 17.4 cases per 100,000 employed workers [4]. In studying occupational asthma in California over a 36-month period from 1993 to 1996, the average annual reporting rate for work-related

Table 1. Materials used in food or food-related industries that induce occupational asthma or rhinitis

Agent	Occupation
Animal Products	
Sea animals	
Prawn; crab; king crab; snow crab; lobster; oyster; clams	Seafood processors
Shrimp meal	Aquaculture workers
Fish meal; fish flour	Factory workers
Mother of pearl	Button factory workers
Sea squirt	Oyster shuckers
Seashells	Shell grinders
Trout	
Farm products	
Cows	Dairy farmers
Hogs; swine food	Hog farmers
Poultry	Poultry workers
Pheasants; quail; doves; eggs	Breeders
Egg lysozyme	Egg processors; bakery workers
Insects	
Poultry mites (<i>Ornithonyssus sylvium</i>)	Poultry workers
Grain storage mites (<i>Glycyphagus destructor</i>)	Grain workers
Honeybees	Beekeepers; honey processors
Bee moth	Fish bait breeders
Rice flower beetle	Rice flower workers
Enzymes	
Pepsin; trypsin; pancreatic enzymes	Pharmaceutical workers
Miscellaneous	
Spiramycin	Chick breeders
Pyrolysis products of polyvinyl chloride or label adhesives	Meat wrappers
Plants/fungi	
Grains/flours	
Flour (wheat, rye)	Bakers; millers
Buckwheat; carob bean flour	Food workers
Rice	Rice millers
Soybeans/soybean lecithin	Agricultural workers
Grain dust	Grain handlers
Spices/herbs	
Garlic	Factory workers; farmers
Coriander; mace; ginger; paprika	Factory workers
Cinnamon	Spice workers
Paprika plants	Greenhouse workers
Vegetables	
Green beans; okra	Homemakers
Enzymes	
Bromelain; papain	Factory workers
Miscellaneous	
Coffee	Coffee factory workers
Castor	Factory workers; dock workers
Tea; herbal tea	Tea factory workers; tea garden workers
Pollen	Sugar beet workers
Pectin	Grape workers
Alkaline hydrolysis derivative of gluten	Bakers
<i>Alternaria/Aspergillus</i> ; colophony	Poultry vendors
Hops	Brewery chemists
Devil's tongue (<i>Amorphophallus konjac</i>)	Food workers
Mushrooms	Soup manufacturers; growers
Fungal amylase	Bakers
<i>Verticillium albo-atrum</i>	Greenhouse workers

asthma in California was 25 per million workers. Reinisch *et al.* [5] estimated that the actual rate may be closer to 78 per million because of underreporting.

Host factors

Approximately 5% of workers exposed to sensitizing agents develop occupational asthma. Therefore, individual host factors likely affect an individual's development of this disease. Atopy and smoking are important risk factors for reactivity to HMW allergens. They are not significant risk factors for non-IgE-mediated occupational asthma. The epidemiology of RADS has not been studied extensively. Atopy does not appear to be a significant risk factor in RADS [6]. The amount of irritant, duration of exposure, and proximity to irritant are the significant risk factors for RADS.

Bakers' asthma

Bakers' asthma is one of the most common causes of occupational asthma worldwide. This specific form of occupational asthma is part of a spectrum of allergy that is encountered in bakers that ranges from rhinitis to conjunctivitis to asthma [7]. The number of reported cases continues to rise annually based on occupational disease registries. As with other IgE-mediated diseases, atopy is an important risk factor for development of disease. The latency period was examined by several studies, but results have been highly variable. The amount of allergen exposure is also a significant risk factor.

Bakers' asthma results from an IgE-mediated mechanism following sensitization to wheat flour (*Triticum aestivum*), rye flour (*Secale cereale*), and barley flour (*Hordeum vulgare*). Following sensitization and re-exposure to the flour, workers have an immediate-type reaction and may also have a late reaction [8].

The allergens in wheat flour may be divided into the water-soluble proteins (albumins and globulins) and the water-insoluble proteins (gliadins and glutenins). When testing these proteins for specific IgE-binding activity using sera from sensitized bakers, the water-soluble fraction demonstrated a 70% to 80% IgE-binding activity [9]. Of the approximately 70 proteins isolated by immunoblotting, some that have been more specifically analyzed include the α -amylase/trypsin inhibitor family (10 to 15 kD), 27-kD acyl-CoA oxidase variants of rice and barley, and 39-kD fructose-bisphosphate-aldolase in maize and rice [10,11]. A glycoprotein with similarity to a barley seed-specific peroxidase is the 36-kD wheat glycoprotein with peroxidase activity [12]. Analysis of the proteins in the water-soluble group revealed a group of proteins in the 10 to 15-kD family that showed significant reactivity when tested with sera from sensitized bakers. This group of proteins is part of the α -amylase/trypsin family [13]. Additions to dough in the form of enzymes from aspergillus species show reactivity in the absence of reaction to the cereal allergens. In most of these cases, such reactivity

results from the fungal α -amylase (1,4- α -D-glucan glucanohydrolase). This enzyme comes from *Aspergillus oryzae* and is added to flour to decrease the baking time and to improve the quality of the bread.

The importance of storage mites, which can contaminate flour used in bakeries, is as yet unclear. Several studies showed increased rates of sensitization to storage mites. However, because of the cross-reactivity with house dust mites and studies that showed reactivity to storage mites in individuals without known exposure, it is possible that the reactivity is more an indicator of allergy rather than being a marker of an occupational exposure.

Snow-crab processing

The ever-increasing demand for fish and shellfish has resulted in a continuing increase in processing and production of seafood. United States per capita consumption increased dramatically from 11.8 lb in 1970 to 14.6 to 15 lb in the 1990s. Occupational respiratory reactions occur as a result of aerosols generated when cutting, scrubbing, cleaning, cooking, boiling, and drying the seafood. Additionally, poor ventilation has been implicated in the development of respiratory symptoms. Specific examples of exposure include lobster "tailing," crab "cracking," prawn "blowing," oyster "shucking," and fish "heading" [14••]. The spectrum of pulmonary diseases seen in exposed individuals ranges from rhinitis, asthma, and hypersensitivity pneumonitis all the way to anaphylaxis. Of the major phyla to which seafood belongs, arthropods, specifically the class Crustacea, have been more commonly reported as an exposure risk for developing occupational asthma symptoms. The estimated prevalence of disease is 16% in eastern Canadian snow-crab processing plants. In a study by Cartier *et al.* [15] of 303 crab workers, 18% had rhinitis/conjunctivitis symptoms and one third had asthma symptoms. In following crab-processing workers 1, 2, and 5 years after leaving the job, lung function (FEV₁/FVC) was shown to reach a plateau after 1 year without any further exposure. However, bronchial hyperreactivity still was abnormal in 77% to 84% of patients at the 2- and 5-year follow-up exams. The mean half-life for specific IgE to crab boiling water extrapolated from a sample of 25 patients was 20 months. Twelve of the 25 had significant increases in specific IgE at the 1-year follow-up [16].

Diagnostic evaluation

In order to make a definitive diagnosis of occupational asthma, it is crucial to establish a temporal relationship between exposure to an occupational sensitizing agent and clinical symptoms of asthma. Demonstration of antibodies against a particular sensitizer does not imply causality, it merely suggests that there was exposure. Asthma symptoms or objective data, such as peak flows, that improve when outside the workplace also are suggestive of occupational asthma. Peak flow should be monitored serially several times a day and a log kept of symptoms and

use of medications. If possible, peak flow, symptoms, and medication use also should be followed after the patient is removed from the workplace for 1 to 2 weeks. Because of possible secondary gain issues and other factors, it is important to realize that peak flow is extremely effort dependent [17].

Repeatable spirometric or bronchial hyperresponsiveness (eg, methacholine challenge) data that shows improvement following removal from the workplace has good positive predictive value. Such improvement, if obtained, would be necessary to identify a cause-and-effect relationship and exclude preexisting (before starting work) abnormalities. However, its negative predictive value is not as good since airway hyperresponsiveness may have a long lag time for improvement; the absence of improvement does not rule out occupational asthma as a diagnosis. In addition, a normal hyperresponsiveness challenge within 24 hours of a symptomatic episode would make a diagnosis of occupational asthma unlikely [18•]. If a definitive diagnosis still remains elusive, an additional method of evaluation is to expose the patient to the agent in question in a controlled environment. Challenge testing in a chamber should be performed in a setting that is prepared to deal with a potential asthmatic episode and at doses that are not irritating.

Treatment

Once sensitized, workers may react to exquisitely small amounts of the agent. Therefore, it is imperative that treatment begins with removal of the worker from the work environment. Pharmacologic treatment for these patients should follow the National Heart, Lung, and Blood Institute asthma management guidelines [19,20]. Even after removal from further exposure, workers may not fully recover. The likelihood of persistence is based on severity of asthma at the time of diagnosis as well as the duration of exposure and symptoms prior to removal from the workplace [21].

Hypersensitivity Pneumonitis

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is an immune response to an environmental antigen resulting in an inflammatory parenchymal lung disease. The presentation varies from the acute to the chronic form of the disease. The acute form may show patchy or diffuse infiltrates on chest radiograph, with symptoms resolving within 24 hours after antigen removal. The chronic form may demonstrate granulomas and fibrosis. Hypersensitivity pneumonitis is primarily an occupational disease; however, individuals may be sensitized to culprit agents from heating and air conditioning systems. Hypersensitivity pneumonitis has a characteristic bronchoalveolar lavage (BAL) that shows neutrophilia within the first 48 hours and then a lymphocytosis.

An interesting aspect of this disease is that smoking actually has a protective effect. Farmers' lung is a common form of hypersensitivity pneumonitis and is caused by exposure to thermophilic actinomycetes growing in moldy hay. In a study of farmers' lung in central Wisconsin, antibodies to *Micropolyspora faeni* were more prevalent among nonsmokers. The disease prevalence in this study was 4.2 per 1000 farmers [22,23]. Other studies noted prevalence between 2.5 and 153.1 per 1000 farmers.

Mushroom workers' lung

Mushroom workers' lung has been linked to a variety of antigens derived from microorganisms and mushroom spores. The term "mushroom workers' lung" was first used in 1967 [24]. Thermophilic actinomycetes are released during the mixing of compost, grain, growth medium, and mushroom spores. Species that have been noted include *Thermomonospora* sp, *Streptomyces* sp, *Thermoactinomyces vulgaris*, and *Faenia rectivirgula*. Perhaps because knowledge of the disease has been disseminated, there have not been many recent reports on MEDLINE. In addition to thermophilic actinomycetes, mushroom spores also may cause hypersensitivity pneumonitis. For example, *Hypsizygus marmoreus* (Bunashimeji) is a common edible mushroom in Japan that releases its spores well before harvest. Workers may be exposed to the spores during the processing of these mushrooms [25].

In addition to hypersensitivity pneumonitis, occupational asthma also has been reported in mushroom growers and soup processors [26]. Hypersensitivity pneumonitis has been reported in the turkey-raising industry as well as from oyster shell dust [27,28]. Table 2 lists the specific agents that have been implicated in hypersensitivity pneumonitis and the diseases they cause in food-related industries.

Testing for hypersensitivity pneumonitis

If a patient is suspected of having hypersensitivity pneumonitis, the work-up should include testing for specific IgG antibody to the putative agent by immunoprecipitation. There is no single test that is pathognomonic for hypersensitivity pneumonitis. Finding precipitating antibody is highly suggestive of disease, although there may be false positive reactions, since this is indicative of exposure to high levels of antigen. It is important to have the antigen and antibody in equivalence when testing for precipitants. As with other forms of occupational disease, a careful history and physical examination are required. In addition, a routine work-up should include chest radiograph and pulmonary function testing (PFT). PFT is restrictive acutely but becomes mixed obstructive and restrictive with a more chronic presentation. In high-resolution computed tomography (CT) scans obtained from farmers' lung patients, there was a predominance of ground-glass appearance in the lower lobes [29].

Table 2. Etiology of hypersensitivity pneumonitis occurring in food and food-related industries

Agent	Source	Disorder
Thermophilic actinomycetes		
<i>Faenia rectivirgula</i>	Moldy hay	Farmers' lung
<i>Micropolyspora faeni</i>	Moldy compost	Mushroom workers' lung
<i>Thermoactinomyces sacchari</i>	Moldy sugar cane	Bagassosis
<i>T. vulgaris</i>	Moldy compost	Mushroom workers' lung
	Moldy hay	Farmers' lung
<i>T. viridis</i>	Vineyards	Vineyard sprayers' lung
Fungi		
<i>Aspergillus clavatus</i>	Moldy barley/malt	Malt workers' lung
	Moldy cheese	Cheese workers' lung
<i>A. flavus</i>	Moldy corn	Farmers' lung
<i>A. fumigatus</i>	Vegetable compost	
<i>A. oryzae</i>	Soy sauce brewing	
<i>Cladosporium</i>	Moldy hay	Farmers' lung
<i>Mucor stolonifer</i>	Moldy paprika pods	Paprika slicers' disease
<i>Penicillium</i> sp	Moldy hay	Farmers' lung
<i>P. caseii</i> ; <i>P. roqueforti</i>	Cheese	Cheese workers' lung
<i>Botrytis cinerea</i>	Moldy grapes	Wine growers' lung
Insects		
Grain weevil (<i>Sitophilus grainarius</i>)	Infested wheat	Millers' lung
Cheese mites (<i>Acarus siro</i>)	Cheese	Cheese workers' lung
Animal products		
Duck proteins	Feathers	Duck fever
Chicken proteins	Chicken products	Feather pluckers' disease
	Hen litter	
Turkey proteins	Turkey products	Turkey handlers' disease
Goose proteins	Feathers	
Bird proteins	Fishermen	
Fish meal	Fish meal workers	
Plant products		
Miscellaneous		
Mushrooms	Spores	Mushroom workers' disease
<i>Erwina herbicola</i> (<i>Enterobacter agglomerans</i>)	Contaminated grain	Grain workers' lung
Tea plants		Tea growers' lung
Oyster shells	Oyster shell dust	

Treatment

Because antigen exposure is the underlying promoter of disease, avoidance of the inciting agent is the first treatment measure. Corticosteroids have a role in the treatment of hypersensitivity pneumonitis, except in the case of chronic disease in which the fibrotic changes are irreversible.

Occupational Dermatitis

Epidemiology

Occupational dermatoses are divided into irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD). ICD is a nonimmunologic inflammatory skin disorder that does not require previous exposure to sensitize the patient. The amount of the agent and the duration of exposure are important in ICD. ACD may present as (Gell and Coombs type I) urticarial reaction or as a (type IV) delayed hypersensitivity reaction. ACD presents as an eczematous skin reaction to a previously sensitized agent. Both ACD and

ICD may present in an acute and chronic form. Table 3 reviews the various industries, exposures, and resulting disease processes resulting in dermatitis in food processing and food service workers.

Definition

Contact dermatitis is an inflammatory skin response to exposure to an allergen or irritant. Based on epidemiologic studies, contact dermatitis represents 90% to 95% of all occupational dermatoses. Further, of the two major forms, allergic and irritant, allergic represents 20% of the cases and irritant represents 80%. The most common anatomic location for occupational contact dermatitis to occur is the hands. Clinically, it may present with erythema, pruritus, edema, and a papulovesicular eruption. In chronic cases, there is minimal pruritus; however, there is fissuring, scaling, desquamation, postinflammatory hyperpigmentation, and lichenification. In the case of ACD, the reaction is a Gell and Coombs type IV delayed-type hypersensitivity

Table 3. Dermatitis in food processing and food service workers

Industry/occupation	Exposure	Diagnosis
Agriculture		
Milk controllers; milk recorders; milkers	Bronopol; Kathon CG (Rohm & Haas, Philadelphia)	Dermatitis
Milk testers	Chrome; dichromate	
Milk analyzers	Dichromate	Allergic contact dermatitis
Ewe milkers		Dermatitis
Celery harvesters	Celery fungus (<i>Sclerotinia sclerotiorum</i>)	Phototoxic dermatitis
Apple packers	Apples sprayed with ethoxyquin	Allergic contact dermatitis
Orange pickers	Omite-CR (Uniroyal Chemical Co., Geismar, LA)	Dermatitis
Grocery workers	Celery (furocoumarins)	
Food preparation		
Fish factory workers	Fish; mustard	Dermatitis; contact urticaria
Cooks	Mustard; rape	Eczema
	Garlic; onions	Eczema
	Paprika; curry	Contact dermatitis
Salad makers	Mustard	Dermatitis
Food workers	Cashews (cardol)	Dermatitis
	Lettuce	Dermatitis; contact dermatitis
	Chicory; endive	Contact dermatitis
Sandwich makers	Codfish; plaice; chicken; onion; garlic	Dermatitis
Bakers	Sodium metabisulfite; persulfate; cinnamon	Dermatitis
	Sorbic acid	Allergic contact dermatitis
	Propyl gallate; dodecyl gallate	Eczema
	Chromium	Dermatitis
	Flour mite; sugar mite	Dermatitis
	Karaya gum; flour	Contact urticaria
Meat/poultry processing		
Butchers	Rubber boots	Allergic contact dermatitis
	Knife handle	Dermatitis
	Povidone-iodine	Allergic contact dermatitis
	Calf liver; pig gut; beef	Urticaria
Slaughterhouse workers	Blood (cow and pig); gut casings	Contact urticaria; eczema
Poultry workers	Various	Irritant allergic dermatitis; eczema
Chicken vaccinators	Antibiotics	Contact dermatitis
Seafood		
Fish market workers	Shrimp	Allergic contact urticaria
Caterers	Shrimp	Contact urticaria
	Fish	Dermatitis
Seafood processors	Prawns; crabs	Asthma; dermatitis
Oyster shuckers	Oysters	Dermatitis
Mussel processors	Mussels	Dermatitis
Food handlers	Fish; shellfish	Contact dermatitis
Fishermen	Fish	Skin diseases
	Rubber boots	Dermatitis
Fish workers	Fish	Contact urticaria
Cooks	Fish	Contact urticaria
Trawlermen	Bryozoa	Dermatitis, eczema
Fishnet repairers	Fishnets	Dermatitis
Miscellaneous		
Snack bar workers	Penicillin residues from meat products	Dermatitis
Spice workers	Turmeric; cinnamon; cinnamic aldehyde	Allergic contact dermatitis
Margarine manufacturers, workers	Octyl gallate	Eczema; dermatitis
Peanut butter manufacturers	Octyl gallate	Dermatitis
Food workers	Sesame oil	Contact sensitivity
	Artichokes	Eczema
Confectioners	Cardamom	Allergic contact dermatitis
Cookie workers	"Thin mint" cookies	Eczema
Beekeepers	Propolis	Dermatitis
	Beeswax (poplar resin)	Dermatitis
Coconut climber	Coconut trees/coconuts	Dermatitis
Bartender	Citrus peel, geraniol citral	Allergic contact dermatitis

reaction in which there is recruitment of previously sensitized, antigen-specific T lymphocytes into the exposed area of the skin [30]; whereas in ICD, it generally is a non-immunologic reaction with a high concentration of agent. Morphologically, the borders of an allergic reaction are limited to the area of contact, whereas in ICD, the borders are indistinct.

Cutaneous reactions to foods generally result from handling or ingestion of the food products. Unlike occupational lung disease, there is a paucity of information on occupational dermatitis. Where information is available, there is significant discrepancy regarding the incidence of disease. The hygiene requirements of the food industry leads to repeated hand washing by workers, which potentially may damage the skin [31]. In a study by Smith [32] assessing the incidence of skin conditions in the food industry by questionnaires, the mean annual incidence in the food manufacturing industry was noted to be 2103 per million employees per year and 1414 per million employees per year in the retail/catering industry. In a cross-sectional study of 1052 food industry workers in Finland by Peltonen *et al.* [33], 17% reported a current skin disease. A diagnosis of occupational eczema was made in 8.5% of the 541 female employees studied and was generally twice as common among those fish, meat, or vegetable handlers or workers making "instant" food as among those making confectionery. Of the 196 workers handling food, hand eczema was present in 15%. Hand eczema also was noted in 8.5% of the 259 confectioners and in 6% of the 86 office employees. These numbers are much higher than estimates from the EpiDerm and OPRA (Occupational Physicians Reporting Activity) surveillance plan, which have collected incident cases for occupational skin disease since February 1993 in the United Kingdom, as reported by dermatologists and occupational physicians. They report an annual incidence of occupational contact dermatitis of 12.9 per 100,000 [34]. In a report of epidemiologic data on occupational skin diseases in northern Bavaria between 1990 and 1999 [35], it was noted that 3097 (83%) occurred in 24 occupational groups. The incidence was 6.7 per 10,000 workers per year, with the second highest incidence rates among bakers.

Host factors

Significant host factors that predispose an individual to occupational dermatitis include anatomic site, history of atopy, pigmented skin, aged skin, and immunosuppression. Important environmental factors include humidity or temperature extremes, fissured skin, and occlusion of the skin. For ICD, black skin may be more resistant than white [36]. Indeed, there may be genetic factors involved in the development of ICD [37]. Atopic dermatitis also may predispose workers to development of ICD; however, it does not appear to predispose to ACD. In ACD, any skin breakdown resulting from ICD may be a predisposing factor to ACD. Therefore, a given worker may have both

ICD and ACD [38•]. Epidemiologic data does show more women affected by hand eczema than men. However, these cases occur in occupations in which there is a high percentage of women who are involved in wet work. Experimental studies have not shown gender differences [39].

Occupational contact urticaria

Occupational contact urticaria falls within the category of occupational skin diseases and may be of importance in the food industry. This rash is the classic erythematous, papular, pruritic type seen in hives; however, in this case, the rash is associated with a specific occupational exposure. Significant risk factors include a history of atopy as well as a breakdown of the skin barrier. Although there is no significant data on occupational contact urticaria, epidemiologic data have shown that cooks, bakers, caterers, and food handlers are at increased risk. The potential allergens in these individuals include apple, bean, beer, caraway seed, carrot, egg, endive, fish, garlic, kiwi fruit, lettuce, meat (beef, chicken, lamb, liver, pork, and turkey), milk, peach, potato, rice, shellfish, spices, and strawberries [40–42]. Bakers have additional exposure from cereal flours and flour enzymes, resulting in contact urticaria.

Diagnostic evaluation

As with all occupational disorders, a careful medical and occupational history and physical examination must be performed in order to make a temporal association between exposure to a potential agent and onset of disease. Furthermore, nonoccupational exposures, such as those during leisure activities, should be excluded. This is particularly important in cases in which the dermatitis does not improve with time away from work. In occupational contact urticaria, specific reactivity to potential allergens identified in the history may be tested with skin prick or radioallergosorbent test (RAST). As with other forms of urticaria, it should be noted that individual urticarial lesions resolve within 24 hours without residual pigmentation. If this is not the case, alternative diagnosis should be considered. In cases of ICD, there is no specific testing that may be done for confirmation. In ACD, patch testing may be performed for confirmation of a suspected agent. To test for ACD, a dilute form of the allergen is placed in a vehicle and then attached to the upper back after the dermatitis has resolved. The patient is not allowed to bathe or undergo any strenuous activity. The agent is removed after 48 hours; the back is examined at that time and again at the end of 4 to 5 days. The second examination is performed both to look for a delayed reaction and to determine if any irritant reactions have cleared. The sensitivity and specificity of patch testing is about 70% [43].

Treatment

Because of the social and economic impact of occupational skin diseases, a skin disease prevention program was set up

in Germany in 1999. In that study, each subject had a detailed exposure analysis (both in and out of work) as well as careful documentation of skin findings. The history included pre-occupational skin diseases, history of atopy, and leisure activities. Of the 23 patients who completed the 1-year analysis, the majority suffered from ICD. Unsuitable skin cleaning and skin protection/care were the causes of hand dermatitis in many cases. A high frequency of hand washing, use of hot water, and use of industrial cleaning products instead of hand cleaning products were identified specifically. Protective skin products were applied by 7% of employees, while 59% claimed to use them. Relapse in cases of allergic contact dermatitis resulted from failure to avoid the etiologic agent. Effective secondary prevention of occupational hand dermatitis was demonstrated with this multidisciplinary approach. A control group must be studied to confirm the specificity of the results [44].

Regardless of whether a worker suffers from ACD or ICD, it is critical that the allergen or irritant be identified. Treatment begins with avoidance or substitution of the potential agent. This includes not only changes in the particular exposure an individual has, but also use of appropriate clothing and gloves. The affected worker should receive hands-on instructions on use of protective equipment, such as gloves and clothing. Educating patients is critical if they are going to practice proper avoidance. In addition, proper occupational hygiene must be emphasized. If these efforts do not improve or resolve the dermatitis, the worker should be advised to switch professions.

The general approach to treatment of ACD is to use topical steroids when less than 10% of the skin is involved. If there is greater skin involvement, oral steroids are used. Treatment should be undertaken over 2 to 3 weeks as premature cessation may cause a rebound exacerbation of skin symptoms.

In the case of occupational contact urticaria, pharmacologic intervention should begin with use of second-generation nonsedating antihistamines since the first generation sedating antihistamines may involve occupational safety issues. Additionally, H₂ blockers or combination H₁ and H₂ blockers may be used. The most common combination H₁ and H₂ blocker used for treating urticaria is the tricyclic antidepressant doxepin. However, doxepin may be extremely sedating and should be titrated starting at low doses. The most efficacious treatment for urticaria is oral corticosteroids, such as prednisone. However, long-term oral steroid use has many well-known complications and, therefore, is not recommended [45].

Conclusions

The examples illustrated in this paper are but a few from the wide array of food allergen-associated occupational reactions. An important aspect of making a diagnosis of occupational reaction is to take a careful and thorough

history, looking for a temporal association between the reaction and the exposure. New causative agents are continually being reported. With globalization of world markets and a continuing increase of individuals employed in the food industry, it will be important for the practicing clinician to continually keep abreast of these new reactions when approaching a new or unusual occupational reaction.

As new foods are developed, it is possible that new occupational reactions may occur. Of particular interest is the development of genetically modified crops that may contain novel proteins to which there is no prior human exposure. Although most efforts at food safety analysis are directed at ingestion of foods by consumers, it is possible that such new proteins might cause occupationally related allergic reactions. Although this is unlikely because of the low levels of expression of such proteins, such a possibility should be considered whenever occupational reactions occur in industries growing/processing such foods.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

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