Occupational Rhinitis

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Kristin Claire Sokol and Daniel L. Hamilos

Case Presentation 1

A 50-year-old male woodworker presents to the allergy clinic with complaints of nasal stinging and burning, watery nose, nasal congestion, and frequent sinus headaches. These symptoms have been ongoing for years. He has noticed that when he took a few months off from work due to a back injury, his nasal symptoms improved somewhat. He denies a history of seasonal allergies, asthma, or eczema. Other past medical history includes hypertension and hyperlipidemia. He reports a history of about one sinus infection requiring antibiotics every 2 or 3 years. He denies any other recurrent infections. He is taking a thiazide diuretic for his high blood pressure and a statin for his high cholesterol. He does not take any medications on a regular basis for his nasal complaints. He does take ibuprofen or a decongestant spray as needed for acute symptoms. He is a former smoker; he quit 15 years ago. He does not drink alcohol. On physical examination, his inferior nasal turbinates are erythematous and boggy. His oropharynx is somewhat erythematous, but has no exudate or visible drainage. He has no facial tenderness on palpation. The rest of his HEENT examination is normal. CBC does not reveal eosinophilia or any other abnormalities. Nasal smear shows a predominance of neutrophils. CT sinus reveals small mucus retention cysts in his bilateral maxillary sinuses, but is otherwise normal. He is instructed to start daily saline nasal lavage, in addition to an intranasal corticosteroid once a day prior to going to work. He comes in for a follow-up visit 3 months later with moderate improvement in his symptoms.

Case Presentation 2

A 28-year-old female with a history of intermittent asthma, allergic rhinitis, and eczema presents to the allergy clinic with a 6-month history of worsening symptoms of watery nose, sneezing, watery/itchy eyes, and cough. She recently graduated from a school of pharmacy and started a new job about 1.5 years ago working in a hospital pharmacy with the main task of compounding antibiotics and other drugs. She is currently taking an oral antihistamine intermittently, and uses an intranasal corticosteroid daily without much relief. She does not smoke cigarettes or drink alcohol. She is not taking any additional medications. She does note that her symptoms improve

K. C. Sokol (✉)
National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD
e-mail: kristin.sokol@nih.gov

D. L. Hamilos
Department of Rheumatology, Allergy & Immunology, Harvard Medical School, Massachusetts General Hospital, Boston, MA, USA
e-mail: dhamilos@partners.org

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somewhat on weekends and they improved significantly when she took a vacation to the Caribbean 4 months ago. Physical examination reveals pale boggy inferior nasal turbinates, an absence of nasal polyps, cobblestoning in the oropharynx, and mild conjunctival erythema and watery eye drainage. Her lung exam reveals no wheezes, rhonchi, or rales. Specific IgE via skin prick testing reveals sensitization to several environmental allergens including dust mite, trees, and grasses. Spirometry reveals an FEV1 of 75% predicted with significant reversibility after an inhaled short-acting beta-agonist. After a detailed work history, it is determined that she is exposed to many different antibiotics, but also highly exposed to lactase, a disaccharide enzyme produced by *Aspergillus oryzae* and *A. niger*, which is used extensively in the food and drug industries [1]. Specifically, skin prick testing of several antibiotics and lactase only reveals sensitization to lactase. Nasal smear identifies a predominance of eosinophils. Her workplace is provided with this information and asked to reduce her exposure to lactase. She is also instructed to wear personal protective equipment when compounding any drug. She starts an oral antihistamine on a daily basis, and increases her nasal corticosteroid dose to two sprays in each nostril twice a day with proper technique. She comes in for follow-up 6 months later with markedly improved symptoms.

Introduction

Occupational rhinitis is defined as inflammation of the nasal mucosa that causes symptoms of rhinitis, such as nasal congestion, rhinorrhea, sneezing, and itching, due to causes associated with a particular work environment. This must be distinguished from work-exacerbated rhinitis where there is a preexisting history of rhinitis and symptoms worsen at work [2–4]. In 2009, the European Academy of Allergy and Clinical Immunology (EAACI) published a consensus paper that classified occupational rhinitis into two general types, allergic and nonallergic [5]. Allergic occupational rhinitis is characterized by a latency period of months to years. It is attributed to an immune-mediated hypersensitivity reaction to a particular workplace exposure. The term "allergic occupational rhinitis" has traditionally been used to encompass occupational agents that can be either IgE mediated or non-IgE mediated. Nonallergic occupational rhinitis does not have a known underlying immunologic basis for disease.

Allergic Occupational Rhinitis

There are now over 200 agents that have been associated with occupational rhinitis; thus a review of each substance would be beyond the scope of this chapter. Occupational agents capable of causing allergic occupational rhinitis can be classified as either high-molecular-weight (HMW) (>5 kDa) or low-molecular-weight (LMW) (<5 kDa) agents [2]. The agents include the same high- and low-molecular-weight sensitizers that are known to cause occupational asthma.

High-Molecular-Weight Agents

HMW agents are organic biological substances derived from plants or animals, such as flour, grain dust, latex, mites, mold spores, laboratory animals, enzymes, and other sources. It is noteworthy that the prevalence of latex sensitization in healthcare workers (HCW) was found to be strongly related to the level of airborne latex allergen exposure [6]. Furthermore, an intervention designed to reduce airborne latex allergen exposure (use of powder-free latex gloves) was associated with a 16-fold reduction in the latex sensitization rate [6]. The profound reduction of latex sensitization among healthcare workers and susceptible patients is a testimony to how effective environmental control to prevent exposure and subsequent sensitization can be.

Common occupations associated with HMW agents include bakers, laboratory workers, veterinarians, seafood packagers and processers, farm workers, healthcare workers, and detergent industry workers [7]. High-molecular-weight

agents can cause upper airway inflammation via an IgE-mediated immune response leading to Th2-driven inflammation. In healthcare workers, recently implicated causes of occupational rhinitis include ethylenediamine tetraacetic acid (EDTA)-containing detergent enzymes used for cleaning medical instruments and aliphatic or alicyclic amines used in cleaning products [8, 9]. In these reports, tetrasodium EDTA and certain of the amines were found to elicit positive nasal provocation testing in some of the affected healthcare workers.

Low-Molecular-Weight Agents

In contrast to HMW agents, low-molecular-weight (LMW) agents are mostly inorganic compounds and include synthetic chemicals, such as diisocyanates, persulfate salts, acid anhydrides, aldehydes, and drugs, as well as metallic agents and chemicals derived from wood dust. Common occupations associated with these agents include chemical workers, epoxy resin production workers, carpenters, furniture makers, painters, hairdressers, and textile workers [7]. Only a small number of LMW compounds have elicited an IgE-dependent mechanism [10], with many eliciting allergic disease through other immune mechanisms that remain to be fully characterized [11].

Nonallergic (Irritant-Induced) Occupational Rhinitis

Nonallergic occupational rhinitis, also known as irritant-induced occupational rhinitis, is caused by agents capable of producing mucosal inflammation without evidence of a latency phase or immunologic sensitization (Table 6.1). The mechanisms by which irritants can induce airway inflammation are far less known [2], but mechanisms involving epithelial damage and neurokinin release from nociceptive nerve fibers are thought to play a significant role [12]. It is known that sensory nerve fibers exist underneath the airway epithelium that express chemoreceptors

Table 6.1 Examples of agents implicated in occupational rhinitis

Allergic	Nonallergic	
High-molecular-weight agents	Low-molecular-weight agents	Irritants
Natural rubber latex	Anhydrides	Ammonia
Psyllium	Diisocyanates	Cigarette smoke
Grain dust, flour dust, alpha-amylase	Abeitic acid/ colophony	Formaldehyde
Mold spores	Plicatic acid	Chlorine
Seafood proteins	Persulfates	Diesel exhaust
Pollens	Quaternary ammonium disinfectants	Wood dust
Animal proteins (urine, saliva, dander)	Cyanoacrylates	Solvent vapors
Insect antigens and mites	Wood dust	Sulfur dioxide
Proteolytic enzymes		Asphalt vapors
Lactase		

(i.e., transient response potential receptors or TRPs). When these chemoreceptors are activated by irritants and osmotic and mechanical stimuli, there is a local release of neuropeptides resulting in activation of their selective receptors located on mucosal blood vessels, submucosal glands, and inflammatory cells. The release of neuropeptides and signal transduction via nociceptive fibers through the central nervous system can cause increased parasympathetic activation and/or dampening of sympathetic responses resulting in increased blood vessel dilatation and oversecretion of mucus manifesting as upper respiratory symptoms such as rhinorrhea, nasal congestion, and sneezing [2].

Certain particulates, such as cigarette smoke, and certain water-soluble irritants, such as ammonia or sulfur dioxide vapors, organic acids, aldehydes, and chlorine, that readily dissolve in mucous membrane water, provoke these immediate irritant ocular and nasal responses [12]. Nonallergic occupational rhinitis, or irritant rhinitis, can be seen in a number of industries and professions including woodworkers, pulp mill

workers, spice grinders, animal laboratory workers, antibiotic manufacturers, firefighters, health professionals, and cleaning workers [13, 14]. Occupational rhinitis is associated with strong irritants including ammonia, chlorine gas, solvent vapors, bleach, hydrochloric acid, nitrogen dioxide, hydrogen sulfide, and certain drugs [4, 14]. Reactive upper airway dysfunction syndrome (RUDS) is a type of nonallergic occupational rhinitis that can develop following a single exposure to a very high concentration of an irritant gas, vapor, or smoke. Biopsies of the nasal mucosa among these individuals have shown epithelial desquamation, defective epithelial junctions, and increased number of nerve fibers [13, 14]. Unlike reactive airway dysfunction syndrome (RADS) which is now an established clinical entity, RUDS is still a rather vague condition with unknown incidence and prevalence [2]. However, just like work-related rhinitis can be a precursor to and often coexist with work-related asthma, RUDS and RADS can occur in the same patient.

The risk factors associated with occupational rhinitis include exposure level, length of exposure, atopy status, and smoking history [7]. The risk of IgE-mediated sensitization to HMW agents is directly related to the level and duration of exposure in certain workers, especially detergent workers, bakers, and those that work with lab animals. These workers are at greater risk not only for sensitization but also for the development of rhinitis symptoms. Underlying atopy is also a risk factor for sensitization to HMW agents such as flour, lab animals, and latex [7]. The association between smoking and risk of occupational rhinitis remains unclear, as some studies revealed an enhanced risk of sensitization in smokers, whereas others failed to demonstrate this relationship [3, 7, 15].

It is also worth noting that work-related rhinitis may precede the development of work-related asthma [16], and, therefore, work-related rhinitis should be considered a potential risk factor of work-related asthma [11]. The prevalence of occupational rhinitis in patients with occupational asthma has been estimated to be between 76 and 92% of workers [10].

Scope of the Problem

It is difficult to assess the overall incidence and prevalence of occupational rhinitis, as the epidemiology is not well investigated mainly because it is not considered a serious disease. Occupational rhinitis does tend to be about 2–4 times more prevalent than occupational asthma [3, 10]. It has been estimated to affect anywhere from 2 to 87% of workers exposed to occupational allergic or irritant agents, depending on the industrial setting [10]. Studies have shown that the prevalence of occupational rhinitis ranged from 3 to 87% in various industries (Table 6.2) [10]. Two recent studies have revealed a prevalence of rhinitis ranging from 42 to 62% in hairdressers exposed to persulfates and ammonia [19, 20]. In one study, it was shown that pharmaceutical workers are exposed to lactase during the manufacturing of digestive aid products for individuals with lactose intolerance and this can lead to symptoms of rhinitis [1]. However, the true prevalence is difficult to determine as the diagnosis of occupational rhinitis is challenging. In one study, patients underwent specific inhalational challenge (SIC) tests for confirmation of both occupational asthma and occupational rhinitis. A positive nasal challenge was observed in 25 SIC tests and a positive bronchial challenge was observed in 17 SIC tests. In 13 cases, both the nasal and bronchial challenges were positive, and these concordant responses were more commonly seen when HMW agents were tested [21].

Diagnosis/Assessment

Occupational upper airway disorders, including occupational rhinitis, are diagnosed based on history and exposure at work, physical examination, and for some, specialized diagnostic tests. A careful exposure history is essential for recognition and diagnosis. A history of prior allergic disorders must be asked. The timing of the onset, worsening, and improvement of symptoms is important, especially noting if there is improvement away from the work environment. Also, a history of a high prevalence rate of symptoms

Table 6.2 Estimated prevalence of occupational rhinitis in various industries [2, 10, 13, 15, 17, 18]

Occupation	Agent	How exposure occurs	Prevalence	Exposure evaluation	Duration of exposure	Confirmation of allergy?
Laboratory animal workers	Rat, mouse, guinea pig, rabbit	Animal handling; urinary aeroallergens	9–42%	Total dust; rat urinary aeroallergens; hours per week	NA	Specific skin test and specific serum IgE
Swine confinement workers	Pig	Animal handling	8–23%	NA	NA	Not done
Farm workers/cattle farmers	Storage mite	Grain bins, animal feed	2–60%	NA	NA	Specific skin test and specific serum IgE
Grain elevator workers	Grain dust	Grain growing, handling, processing	9–64%	NA	NA	Specific skin test
Bakers	Flour, alpha amylase	Bakers and packers exposed to flour proteins	18–29%	Total dust; flour aeroallergens	NA	Specific skin test
Detergent manufacturing	Proteolytic enzymes, lactase, papain	Cleaning medical instruments	3–87%	NA	3–6 years	Specific serum IgE
Seafood industry	Trout, crustacea, fish food	Filleting of fish; clam, crab, and shrimp processing; aquarists	5–24%	Amount of *Chironomus thummi* larvae used per month	NA	Specific skin test and specific serum IgE
Chemical workers	Reactive dyes, anhydrides	Reactive dye products; textile dyeing	10–48%	Dust concentration, duration of employment	NA	Specific skin test and specific serum IgE
Carpentry/furniture making	Wood dust, plicatic acid	Mansonia, western red cedar	10–36%	Total dust concentration	NA	Not done
Hairdressers	Persulfates, ammonia, paraphenylene-diamine (PPD)	Persulfate products	27%	NA	5 years	Specific skin test, specific inhalational challenge
Pharmaceutical workers	Spiramycin, lactase, psyllium	Drug compounding, packaging	9–40%	NA	NA	Specific skin test and specific serum IgE
Nondomestic cleaners, healthcare workers	Ethylenediamene tetraacetic acid (EDTA)	Aerosols	35%	NA	NA	Not done
Healthcare and glove manufacturing workers	Latex	Inhalation, latex thread contact	0.12–20%	NA	10 years	Specific skin test and specific serum IgE
Auto body workers/ boat builders	Diisocyanates	NA	NA	Concentration of selected isocyanates	NA	Specific serum IgE negative
Pepper mill workers	Capsaicin	Inhalation	100% ($n = 1$)	NA	4 years	Skin prick tests negative
Domestic waste collectors	Organic dust (bioaerosols)	Loading, driving	29–39%	NA	10 years	NA

NA information not available

among coworkers can support a diagnosis of irritant-induced occupational rhinitis. Another method to aid with history is the "work removal-work resumption" test where the patient is assessed after a period of a few weeks away from the suspected exposure and is reassessed again a few weeks after resumption of work [2]. There are specialized questionnaires including the Rhinoconjunctivitis Quality of Life Questionnaire (RQLQ) and the Sinonasal Outcome Test (SNOT) which can be used to assess symptoms and quality-of-life impairment [12], although questionnaires have a low specificity for diagnosing occupational rhinitis.

Physical examination, including anterior rhinoscopy and percussion of the maxillary and frontal sinuses, can help in diagnosis of rhinitis, but often does not delineate between allergic or irritant occupational rhinitis and other forms of rhinitis. Rhinolaryngoscopy and flexible rhinolaryngoscopy enable the physician to evaluate for nasal polyposis and vocal cord dysfunction, respectively. These tools can also be used to exclude other common causes of rhinitis such as structural factors like septal deviations and nasal valve dysfunction [2].

Beyond a detailed history and physical examination, specialized diagnostic tests can help with the diagnosis of occupational rhinitis. Allergy skin or serologic testing with documented reactivity to indigenous aeroallergens is important for determining the patient's allergic (atopic) status. It is also useful for testing a suspected occupational allergen which can confirm sensitization. However, a major limitation is the lack of standardized occupational allergens that can be used for testing. The immunological evaluation is more significant in high-molecular-weight (HMW) agents (i.e., animal or plant proteins, enzymes) and a few low-molecular-weight (LMW) agents (i.e., trimellitic anhydride, hexamethylene diisocyanate, platinum salts) in which IgE can be detected by skin prick testing and/or measurement of serum-specific IgE [22]. Often, symptoms of irritant-induced occupational rhinitis will mimic those of allergic rhinitis; however, it is usually difficult to determine responsible etiologic agents. In these situations, material data safety sheets (MSDS) may be helpful for providing clues to which agent(s) might be responsible for triggering rhinitis symptoms. A laboratory workup reveals a lack of systemic eosinophilia and a predominance of neutrophils on nasal smear in irritant rhinitis. Nasal cytology has been used as a tool for diagnosing occupational rhinitis in certain workers. In a recent study, woodworkers were found to have more neutrophils in nasal smears than controls. It was also found that woodworkers exposed to wood dust for a longer period of time had more lymphocytes in their nasal smears [23]. Sinus computed tomography scans can rule out the presence of acute or chronic sinusitis, fungal sinusitis, and other structural or infectious abnormalities, but is not recommended in the initial evaluation.

Nasal peak flow measurements, although not used in clinical practice with much frequency, can be used to document the response to allergen or irritant exposures that the patient may be exposed to in the workplace. A causal relationship between exposure to a specific occupational agent and rhinitis can be established by specific nasal provocation testing (NPT) with the suspected agent. The European Academy of Allergy, Asthma, and Immunology Task Force on Occupational Rhinitis states that "in the presence of work-related rhinitis symptoms, objective assessment using nasal provocation challenges in the laboratory or at the work- place should be strongly recommended" [5]. This diagnostic test has been studied much more with high-molecular-weight agents than low-molecular-weight agents [5]. In addition, NPT is only utilized by a limited number of clinical centers, especially in the United States, and remains poorly standardized [2].

Management/Outcome

The management of occupational rhinitis is threefold. Since occupational rhinitis is a preventable condition, avoidance is the first step in management. Prevention of exposure to hazardous materials can, in many cases, prevent incident cases of occupational rhinitis [6]. Secondary

prevention includes early detection of the symptoms and interruption of disease progression. Reduced exposure can be accomplished by improving ventilation systems, wearing appropriate protective clothing and masks, and, if possible, relocation of the patient to another job location [2]. For established occupational rhinitis, tertiary prevention usually implicates treatment which involves reducing exposure to the known or suspected allergen or irritant, supportive measures such as nasal saline lavage, and medications either used alone or in combination such as topical corticosteroids, topical antihistamines, and topical cholinergic blockers. There is very little evidence for any beneficial effects of specific allergic immunotherapy in occupational upper airway disease [2]. Although there are no published studies supporting the use of immunotherapy as a treatment option for IgE-mediated occupational rhinitis [24], immunotherapy may be beneficial in certain clinical settings, such as in laboratory animal workers and veterinarians who are sensitized to animal dander.

In addition to preventing or reducing nasal symptoms, the management of occupational rhinitis should also be aimed at decreasing the risk of occupational asthma onset [5]. The relationship between occupational rhinitis and occupational asthma has been examined and the frequency of association was higher for HMW compared with LMW agents [22]. Close follow-up with awareness for the progression of lower airway symptoms, including lung function testing, is required [2].

If persistence of exposure to an agent causing occupational rhinitis occurs, as stated above, occupational asthma can develop. Because of this, the European Academy of Allergy, Asthma, and Immunology (EAACI) Task Force on occupational rhinitis has proposed that patients with occupational rhinitis be considered impaired on a permanent basis for the job that caused the condition as well as for jobs with similar exposures. Although some countries offer compensation of occupational rhinitis, available data has shown that financial compensation does not adequately offset the socioeconomic consequences of the disease. Compensation systems should be directed at offering the worker an alternative job within the same company without the possibility of exposure to the offending agent [3].

If avoidance of the causative agent can be achieved, the prognosis of the patients with occupational rhinitis is generally good [15]. In a prospective study of 20 individuals with allergic or nonallergic occupational rhinitis, when suspected exposures were eliminated, the individuals noted both decreased nasal symptoms and improved quality of life [21]. Studies have not addressed the prevention of onset of development of asthma [21].

Clinical Pearls and Pitfalls

- There are two forms of work-related rhinitis: occupational rhinitis, which is defined as rhinitis symptoms due to causes associated with a particular work environment, and work-exacerbated rhinitis, in which the individual had preexisting rhinitis made worse by exposures in the workplace.
- Occupational rhinitis is often underestimated and underdiagnosed.
- More than 300 substances have been identified as possible agents producing occupational rhinitis [22].
- Allergic occupational rhinitis can be caused by high- or low-molecular-weight agents.
- Nonallergic occupational rhinitis can occur with one high-level exposure to an irritant, and this disorder is termed RUDS, or reactive upper airway dysfunction syndrome.
- The distinction between irritant occupational rhinitis from allergic occupational rhinitis in clinical practice is often difficult but might be distinguished by the predominance of irritant symptoms (rather than itching and sneezing), a high prevalence of symptoms among coworkers, a negative laboratory workup, the predominance of neutrophils on nasal smear, and when applicable a lack of in vivo or in vitro reactivity to identifiable workplace allergens.
- A detailed history is essential when evaluating a patient suspected of having occupational

rhinitis, including documented improvement away from the workplace.

- There are three forms of prevention of occupational rhinitis: primary, secondary, and tertiary prevention.
- Exposure prevention is the most practical and effective method for the primary prevention of occupational rhinitis. Early symptom identification and exposure reduction are the most practical and effective methods for the secondary prevention of occupational rhinitis. Tertiary prevention involves treatment of symptoms in the form of supportive care and/or medications.
- Early diagnosis is critical in the prevention of progression into occupational asthma or possibly rhinosinusitis.
- The prognosis of occupational rhinitis seems to be good with significant reduction or avoidance of the offending exposure.

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