Curr Allergy Asthma Rep (2015) 15:487
DOI 10.1007/s11882-014-0487-8

Occupational Rhinitis: an Update

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Published online: 28 November 2014
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Abstract Occupational rhinitis is characterized by nasal congestion, rhinorrhea, nasal itching, and/or sneezing that occur secondary to exposures in the workplace. This disease can be classified into allergic or nonallergic subgroups based upon the underlying disease pathogenesis as well as the type of causative agent. While the true prevalence of occupational rhinitis is unknown, there are certain professions and occupational exposures that place workers at a higher risk for developing the disease. Additionally, occupational rhinitis can be associated with occupational asthma and upper airway symptoms may precede those of the lower respiratory tract. Taken together, occupational rhinitis is an important disease for study given its medical as well as socioeconomic implications. This review will focus on the classification of occupational rhinitis as well the prevalence, diagnosis, and treatment strategies.

Keywords Occupational rhinitis · Allergic occupational rhinitis · Nonallergic occupational rhinitis · Rhinitis · Occupational asthma · Nasal challenge · High molecular weight · Low molecular weight · Skin test · Specific IgE · Laboratory animals · Flour · Isocyanates · Acid anhydrides · Bakers · Prevention · Occupational disease

This article is part of the Topical Collection on *Occupational Allergies*

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Introduction

There are a variety of medical conditions that can occur as a result of occupational exposures. In particular, occupational rhinitis is associated with nasal congestion, rhinorrhea, sneezing, and/or itching secondary to exposures in the workplace. There are a multitude of causative agents associated with this disease and numerous occupations at increased risk. This review will focus on the classification of occupational rhinitis as well the prevalence, diagnosis, and treatment of the disease.

Definition and Classification

Rhinitis is defined as the presence of nasal congestion, rhinorrhea, sneezing, and/or itching and can be classified by symptom etiology [1]. Work-related rhinitis is one particular form of rhinitis that encompasses symptoms that occur in the workplace. Work-related rhinitis, in turn, can be further divided into two subgroups based upon a symptom history. In occupational rhinitis, symptoms develop in a previously unaffected individual as a result of an exposure in the workplace. For example, a laboratory animal handler with no prior history of rhinitis develops nasal congestion and rhinorrhea when working with rodents. This is in contrast to work-exacerbated rhinitis where there is a preexisting history of rhinitis and symptoms worsen during exposures at work. For example, a veterinarian with a known dog allergy develops worsening nasal congestion when evaluating dogs in clinic. Another important distinguishing feature between work-exacerbated rhinitis and occupational rhinitis is that symptoms will occur outside of the workplace in the former but not latter condition.

In 2009, the European Academy of Allergy and Clinical Immunology proposed a consensus definition of occupational rhinitis as being "an inflammatory disease of the nose, which

is characterized by intermittent or persistent symptoms (*i.e.*, nasal congestion, sneezing, rhinorrhea, itching), and/or variable nasal airflow limitation and/or hypersecretion due to causes and conditions attributable to a particular work environment and not to stimuli encountered outside the workplace" [2]. From this, occupational rhinitis can be classified into two general types, allergic and nonallergic, in accordance with their underlying disease mechanisms. Allergic occupational rhinitis is characterized by a latency period, while nonallergic rhinitis can be induced by a single high-level exposure.

Pathogenesis

Allergic occupational rhinitis is secondary to an immune-mediated hypersensitivity reaction to a particular agent. There is a defined latency period of months to years during which time sensitization to the causal agent occurs. Then, upon re-exposure to the sensitized agent, an immunologic response often mediated by specific IgE antibodies ensues. In further support of a TH2 immune response occurring in allergic occupational rhinitis, an influx of eosinophils was detected in the nasal lavage fluid of occupational rhinitis patients who were challenged to their respective sensitizing agent [3]. Importantly, however, not all causative agents in allergic occupational rhinitis are associated with a specific IgE-predominant immune response as some responses may be mediated by IgG antibodies or other components of the adaptive immune response with further studies needed to better define such mechanisms [4, 5•].

Nonallergic occupational rhinitis, in contrast, does not have an underlying immunologic basis for disease. Instead, mechanisms involving epithelial damage, neurokinin release, and nociceptors may play a role in pathogenesis [6–8]. There is no latency period or sensitization required, and symptoms typically develop following exposure to an irritant compound. In particular, reactive upper airway dysfunction syndrome (RUDS) is a type of nonallergic occupational rhinitis that develops following a single exposure to very high concentrations of an irritant. Additionally, the most severe form of nonallergic occupational rhinitis, corrosive rhinitis, can occur when a significant irritant exposure leads to the development of severe symptoms with permanent inflammation of the nasal mucosa.

Etiologic Agents

There are over 200 agents that have been associated with occupational rhinitis, and an extensive description of each substance would be beyond the scope of this review. However, causative agents for the disease can be broadly classified based upon its molecular weight. High molecular weight (HMW) agents are more than 10 kDA, while low molecular weight (LMW) agents are less.

High molecular weight agents tend to be organic and thus are derived from plants (e.g., natural rubber latex, flour proteins, grain dust), microorganisms (e.g., molds, bacterial enzymes), or animals (e.g., animal dander, fish proteins). Examples of occupational rhinitis occurring from grains include a pizzeria worker exposed to rice flour [9], a farmer to rice grain [10], and another farmer to maize pollen [11, 12]. Other exposures leading to the development of occupational rhinitis include lupin, a type of legume, in industry food processers [13, 14], milk protein in bakers and dairy workers [15], and hydrolyzed wheat protein in hairdressers [16]. More recently, a case report described a 33-year-old male who worked for 5 years processing squids for frozen meals and developed occupational rhinitis secondary to the cephalopod exposure [17]. While most cases involve sensitization to a single agent, there is a report of an individual with occupational rhinitis who was dually sensitized to both wheat and guar gum with both agents thought to be simultaneously contributing to symptoms [18]. Finally, biologic enzymes can also be causative agents as reported in a study of a 31-year-old hospital nurse who developed occupational rhinitis following inhalation exposure to porcine pancreatic extract powder [19].

As opposed to HMW agents, LMW agents are mostly inorganic compounds including diisocyanates, anhydrides, metals, and certain medications. For example, trimellitic anhydride and hexahydrophthalic anhydride are both associated with occupational rhinitis in industry workers [4, 20]. Hairdressers and other beauty care professionals can develop occupational rhinitis after exposures to bleaching agents containing persulfate, oxidative hair dyes with paraphenylene diamine or toluene-2,5-diamine sulfate, and glues containing ethyl cyanoacrylate used to apply eyelash extensions [21–23]. In regards to metals, a 27-year-old male factory operator in an electroplating plant was reported to have developed occupational rhinitis secondary to his exposure to rhodium salts [24]. Finally, occupational rhinitis has been described in a nurse exposed to the hospital disinfectant chlorhexidine and separately in a pharmaceutical worker who was exposed to the medication sodium alendronate during packaging [25, 26].

Relative to agents that can cause nonallergic occupational rhinitis, they have the common property of being strong irritants. Among the agents described to cause nonallergic occupational rhinitis include ammonia, chlorine gas, solvent vapors, bleach, hydrochloric acid, nitrogen dioxide, and hydrogen sulfide [27].

Following the terrorist attacks on the World Trade Center (WTC) on September 11, 2001, a tremendous amount of particulate matter was released into the surrounding environment. Air samples collected locally within days after the attacks contained a variety of organic as well as inorganic

compounds including cement, lead, asbestos, polycyclic aromatic hydrocarbons, and pesticides [28]. A large majority of the WTC responders developed upper airway symptoms such as pharyngitis, nasal congestion, and rhinorrhea within weeks to months following the exposure [29, 30]. This form of nonallergic irritant occupational rhinitis secondary to a complex particulate matter exposure remains to be a continued focus of investigation.

Epidemiology

In an analysis of US data from 2007, the total number of occupational injuries and illnesses reported was about nine million [31]. Assuming a total number of employed people in the USA of 120 million, the prevalence is about 7.5 %. Because the number is likely an underestimation, the prevalence is probably higher. However, the overall prevalence of occupational rhinitis specifically remains unclear in part due to the lack of a uniform standard for diagnosis. Despite this, numerous studies have been published that investigate the prevalence and incidence of occupational rhinitis in certain professions.

A Finish study completed in the 1990s reported that the risk of developing occupational rhinitis was highest among furriers, bakers, livestock breeders, food-processing workers, veterinarians, farmers, electronic product assemblers, and boat builders [32]. More recently, the prevalence of occupational rhinitis among bakers in Norway was estimated to be between 23 and 50 % [33]. A review of laboratory animal handlers found the overall prevalence of occupational rhinitis to range from 10 to 42 % with the variation dependent on what criteria were used to confirm the disease [34]. Likewise, sensitization to various laboratory animals, as determined by skin prick testing, was higher among animal handlers (16 %) than non-animal handlers (3 %) in a South American study on occupational disease prevalence in laboratory workers [35].

Besides occupation, the prevalence of occupational rhinitis can also be classified by causative agent [5•, 36–38, 39•]. Such rates can range from 2 to 87 % or from 3 to 48 % upon exposure to HMW or LMW agents, respectively [2]. Overall, sensitization to laboratory animal dander and flour appears to be two of the most common agents associated with occupational rhinitis [40].

Relationship with Occupational Asthma

Occupational rhinitis does not necessarily occur in isolation and should be considered when evaluating for occupational asthma or, more broadly, cough occurring in the workplace [41•]. A Finnish study from the 1980s to 1990s reported that the relative risk of asthma was 4.8 % in workers with occupational rhinitis [42]. Conversely, as many as 90 % of patients evaluated with occupational asthma reported having work-related rhinitis symptoms [43, 44]. In particular, 88 % of patients with occupational asthma secondary to trimellitic anhydride reported rhinitis symptoms [45]. Overall, in a separate study, occupational rhinitis was found in 76 % of workers with confirmed occupational asthma due to a variety of agents [46]. Taken together, this data suggests a relationship between the two diseases and should prompt the consideration of occupational rhinitis when diagnosing occupational asthma and vice versa.

Mechanisms explaining the observed correlations between occupational rhinitis and occupational asthma continue to be actively investigated. One prevalent hypothesis involves the unified airway model in which both diseases could be secondary to the same inflammatory responses occurring throughout the upper and lower respiratory tract. In support of this, Castano investigated if there was a correlation between occupational rhinitis and asthma among individuals with a history of work-related asthma symptoms by performing challenges with either a HMW or LMW agent depending on a reported occupational exposure [46]. Of the 43 patients evaluated, the authors found a positive nasal and bronchial challenge in 13 (30.2 %) patients equating to a relative risk of 1.7 (p=0.04) [46]. Of note, however, reactions were more frequently observed among HMW than LMW agents, suggesting that the type of causative agent may also impact disease pathogenesis [46, 47].

Not only is there a potential association between occupational rhinitis and occupational asthma, but the presence of occupational rhinitis may serve as a predictor for the future development of occupational asthma. Estimates suggest that upper respiratory symptoms precede those of the lower respiratory tract in 20–78 % of workers with occupational rhinitis who later develop occupational asthma [2]. In separate studies, nasal symptoms were present prior to asthma diagnosis in 43 and 77 % of occupational asthma cases [44, 45]. In summary, these observations suggest that the identification of occupational rhinitis is an important surveillance strategy for occupational asthma.

Diagnosis and Prognosis

The evaluation of occupational rhinitis should begin with obtaining a detailed medical history. Symptom characteristics and duration should be described, and any exacerbating or alleviating factors should be identified. The occupational history should also be extensively evaluated focusing on the nature of the occupation, how long the individual was at work prior to symptoms developing, what agents the individual was exposed to while at work, and if symptoms improved when the patient was away from the work environment. Material

safety data sheets for any suspected causal agent may be obtained to determine whether respiratory sensitization or irritation is known to be an adverse effect of a given agent.

As part of any evaluation for occupational rhinitis, a physical exam should also be performed. A nasal exam may reveal nasal turbinate hypertrophy, pallor, edema, or discharge, but these findings are nonspecific for rhinitis and should be interpreted within the clinical context. Alternative etiologies for rhinitis symptoms such as nasal polyps, septal deviation, or septal perforation can also be assessed by physical exam. Finally, given the association of occupational rhinitis with other occupational lung diseases, auscultation of the chest should be performed evaluating for the presence of wheezes, rhonchi, or rales.

Unfortunately, clinical history alone has a low specificity in confirming the diagnosis of occupational rhinitis and an integrated approach is instead recommended [2, 48]. There are several diagnostic tests that can be used to assist in confirming the clinical suspicion of occupational rhinitis. For example, immunological testing either by allergen skin prick or in vitro quantification of specific IgE levels can evaluate for sensitization to a particular agent. While a negative immunologic test makes the diagnosis of occupational rhinitis unlikely, it should be noted that LMW agents should not be excluded as causal agents upon negative testing since they typically do not induce an IgE-mediated response. Also, the presence of a positive skin prick test does not necessarily provide confirmation of the disease and should always be correlated with clinical history and physical exam findings. Other limitations of immunological testing can include the lack of commercially available or standardized reagents.

Nasal provocation tests, also referred to as specific inhalation challenges, are considered to be the gold standard for diagnosing occupational rhinitis [2]. These tests are typically performed in a clinical setting but can be done in the workplace if necessary. In this test, individuals undergo intranasal challenges with either the suspected casual agent or the negative control at separate times. Subjective changes in nasal symptoms can be assessed using patient reports and questionnaires. Objective changes in nasal symptoms can be detected by examining nasal secretions for inflammatory cells or by measuring nasal patency through rhinometry, acoustic rhinometry, or peak nasal inspiratory flow [49•]. Benefits of nasal provocation testing include the ability to directly observe a causal reaction between a particular agent and the development of nasal symptoms. However, limitations of this study include the accessibility of reagents used for challenges and the lack of a universal diagnostic standard by which to define a positive reaction.

Having a suggestive clinical history and physical exam, an individual sensitized to a casual agent as shown by immunologic testing, and a positive nasal provocation test to the sensitized agent, the diagnosis of occupational rhinitis can be established. However, in the cases with unclear or equivocal results but a suggestive history, further testing of nasal patency and cytology can be completed in the workplace environment.

Prevention and Treatment

Primary prevention strategies are aimed at preventing the disease from developing. For this, it is important to reduce or eliminate the exposures to known sensitizing agents in the workplace. Improving ventilation, using less hazardous chemicals, wearing protective clothing and masks, and relocating to lower exposure areas in the workplace can all help to achieve this goal.

Studies have shown that teenagers who work in high-risk occupational environments have at greater risk of developing new onset rhinitis than unemployed controls and that symptoms can develop within the first 9 months of starting work [50]. As such, secondary prevention strategies are important to help prevent the progression of symptoms in sensitized individuals. Enhanced surveillance including questionnaires to monitor symptoms, immunological assays to evaluate for sensitization, and early referrals of symptomatic workers to a physician are all potential strategies that can be used [2, 51, 52]. It is especially important to monitor patients with potential occupational rhinitis to prevent the development of occupational asthma.

The main goal of tertiary prevention in occupational rhinitis remains to be avoidance. In a study of bell pepper greenhouse workers with occupational rhinitis, those that left their jobs reported a significantly improved quality of life compared to those workers with continued exposure [53]. Likewise, a more recent prospective study examining 20 subjects with various forms of allergic and nonallergic occupational rhinitis noted an improved quality of life and reduced nasal symptoms after the causative agent was avoided [54•]. Interestingly, even after 2 years of avoidance, patients with occupational rhinitis can still be sensitized and have a positive specific inhalation test to the prior causative agent [55]. This supports the recommendation that avoidance should be a long-standing measure in the management of occupational rhinitis.

In some individuals, avoidance strategies alone are not sufficient in controlling symptoms. As a result, pharmacological therapies can be initiated based on clinical symptoms and patient preference. While there are currently no known studies evaluating specific medical therapies in occupational rhinitis, treatment decisions can be based upon the current guidelines for the management of rhinitis [1]. Intranasal corticosteroids are the most effective class of medication for controlling rhinitis symptoms [56–58]. Importantly, first-generation antihistamines should be avoided in the treatment of occupational

rhinitis given their association with increased sedation and anticholinergic side effects [59]. Unlike non-occupational allergic rhinitis, immunotherapy is not indicated in the treatment of occupational allergic rhinitis.

Besides impacting a patient's quality of life, the diagnosis of occupational rhinitis can have socioeconomic implications. Patients may need to relocate within an office or even find different employment because of symptoms. Affected individuals may also seek compensation for their disease with workman's compensation rights and awards varying by state jurisdiction within the USA.

Conclusions

Occupational rhinitis is a multifaceted disease that develops secondary to exposures to a variety of organic as well as inorganic compounds in the workplace. The disease can be associated with occupational asthma, but the precise mechanisms underlying its pathogenesis remain unclear. Regardless of its etiology, occupational rhinitis is important to be considered not only because of its prevalence among specific occupations but also because of its potentially profound socioeconomic implications.

Compliance with Ethics Guidelines

Conflict of Interest Leslie Grammer reports grants from the NIH, the Bazley Foundation, Food Allergy Network, and S&C Electric; is a consultant for Astellas Pharmaceuticals; has given paid lectures at AAAAI and Mount Sinai; and receives royalties from Lippincott, BMJ, and Elsevier. Whitney Stevens reports no disclosures.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by the authors.

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