Occupational Airways

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Occupational Rhinitis

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Rhinitis may be caused or exacerbated by exposures in the workplace similar to some skin and lung diseases. In Connecticut, occupational rhinitis accounts for 4% of the respiratory diseases and disorders reported to the Departments of Labor and Public Health. Workers in service industries account for almost half of these reports (48%), followed by workers in public administration (29%) and manufacturing (16%).[1] Occupational rhinitis is generally not considered a reason for removal from the workplace, and is often ignored or overlooked as an occupational disease. However, the symptoms associated with rhinitis may affect a worker's quality of life, contribute to lost time away from work and result in decreased productivity.[2,3] More serious consideration should be given to this disease condition.

The nose is the first defense of the respiratory tract. It warms and humidifies inhaled air, filters many allergens and other large particles, absorbs water-soluble gases such as sulfur dioxide, and filters some less soluble gases such as ozone.[4] Nasal congestion, rhinorrhea, sneezing, itching, and sometimes postnasal drainage characterize rhinitis.[2] Swelling of the nasal turbinates can lead to anosmia, obstruction of the sinus ostea with secondary sinusitis, or obstruction of the eustachian tubes with secondary otitis. Obstruction of the nose leads to mouth breathing, thereby bypassing this filtering and humidification mechanism and resulting in increased risk of diseases of the lower respiratory tract. Asthma is often preceded by rhinitis. [2,3,5]

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Table 1[3,7]
Some occupational irritants and allergens that may induce occupational rhinitis

Irritants	Allergens
Ammonia	Acid anhydrides
Benzene	Animal proteins
Chlorine	Colophony
Detergents	Cotton fibers
Formaldehyde	Flour dust
Ozone	Green tea
Paint vapors	Latex
Sulfur dioxide	Isocyanates
Tobacco dust	Permanent wave solution
Toluene	Psyllium
Xylene	Western red cedar

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Irritant vs. Allergic Rhinitis

The major occupational risk factors of rhinitis can be classified as irritant or immunologic induced (See Table 1). Rhinitis due to irritation tends to clear soon after exposure stops. Symptoms from irritation can be difficult to distinguish from an allergic or immunological response. Clinically, the symptoms consist of nasal burning or itching, rhinorrhea, and congestion. The distinction can be made either on the basis of identifying the offending agent or obtaining a smear of the nasal secretions, a nasal mucosal brushing or a biopsy. Polymono-nuclear neutrophils (PMNs) have experimentally and clinically been shown to be associated with irritation, while eosinophils are associated with a type I, allergic response. Lymphocytes are seen with a type IV or delayed hypersensitivity response or non-specific conditions. Graham et al. demonstrated neutrophilia in nasal lavage from experimental exposure to 0.5 ppm ozone for 4 hours,[7] and Frischer et al. confirmed this in children exposed to ambient ozone.[8] Clapp et al. have shown a similar PMN response in both the upper and lower respiratory tract to grain dusts and endotoxin.[9]

Frequently, chronic or recurrent rhinitis is due to allergy or type I response. Occasionally, a type IV or delayed hypersensitivity response can be seen, particularly in association with sensitizing heavy metals such as chromium and nickel. The type I or allergic response can be sub-classified into acute and late phase response. The acute phase begins within 15 minutes of exposure to an allergenic substance. Mediators are released from the mast cells in the nasal mucosa giving rise to itching, rhinorrhea and sneezing, mucosal turbinate swelling and congestion. If the exposure ceases, the reaction usually subsides in a few hours. With prolonged or high exposures, a late phase response may ensue. This response is characterized by more marked mucosal edema and nasal obstruction. This reaction typically begins about 4 hours after the initial exposure and is associated with recruited inflammatory cells (eosinophils) following the acute phase response. This reaction may take 72 hours or longer to subside after exposure stops. Thus, in the occupational setting, one may not find the improvement of symptoms on weekends that one expects with work related conditions. Longer time frames such as a week's vacation may be necessary to appreciate the work association. Individuals with allergic rhinitis usually react to multiple allergens, some at home, some at work and some outdoors. This adds to the difficulty in making a work association.

Pathophysiology

Since the epithelia in the nose and lower respiratory tract are similar, the types of reactions seen are similar, and rhinitis may be a marker of what is occurring in the lower respiratory tract. The size of the particles may influence the nature and severity of the reaction at each site. Larger particles, those greater than 5 microns, tend to be trapped in the upper respiratory tract and are more prone to cause rhinitis, whereas, smaller particles bypass this filtration and tend to trigger more asthma or

hypersensitivity pneumonitis. However, with high degrees of sensitization and because of turbulent air flow, this distinction may become blurred. Desrosiers et al. experimentally exposed eight subjects to a high molecular weight agent (flour and guar gum) and seven subjects to a low molecular weight substance (isocyanate) on two occasions in a random fashion. On one occasion the subjects inhaled through the nose, on the other occasion through the mouth. Inhalation through either the nose or the mouth gave both nasal obstruction and asthmatic responses.[10] Since the nose is exposed at higher concentrations, it is the first area of the respiratory tract to react.

Other nasal effects can occur based on toxic mechanisms. Nasal septal ulceration or perforation may be caused by arsenic, calcium oxide, chromic acid, or copper dust. Rhinorrhea associated with miosis, lacrimation and salivation may occur from pesticides such as carbaryl, malathion, parathion, phosdrin, or pyrethrum. A blue-gray nasal septum may be caused from silver. Numb mucous membranes may occur from DDT or rotenone.[6]

"Treatment of occupational rhinitis should be approached similarly to that of other occupational diseases and not ignored. Chronic rhinitis should be considered as a precursor to lower respiratory disease …"

Treatment

Treatment of occupational rhinitis should be approached similarly to that of other occupational diseases and not ignored. Chronic rhinitis should be considered as a precursor to lower respiratory disease for the reasons mentioned above. Individuals with this condition should be evaluated and followed carefully for evidence of asthma or hypersensitivity pneumonitis. The precipitating cause should be identified and exposure limited, preferably through engineering or administrative means. Respirators can be helpful, but are seldom utilized for rhinitis alone.

Medications need to be based on the specific diagnosis. Nasal decongestants should be avoided in chronic rhinitis because of their propensity to cause rhinitis medicamentosa. Antihistamines are helpful for the sneezing and rhinorrhea of allergic rhinitis, but are less effective on nasal congestion and will have little effect on other types of rhinitis. Because of the sedative effect, cautions need to be taken in prescribing them. Even the so-called non-sedating antihistamines can induce some slowing of reflexes in certain individuals. Cromolyn nasal spray is a good antihistamine substitute for individuals with the early phase allergic response. Steroid nasal sprays are very effective in decreasing the swollen turbinates from most types of chronic rhinitis with congestion. Individuals with a history of long-term use should be followed carefully with nasal exams since atrophic rhinitis and nasal septal perforation have been reported. Although other steroidal side effects are rare, cataracts, glaucoma, and mild adrenal suppression have been reported with high doses and prolonged use of inhaled steroids, and also may be associated with intranasal steroids.

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Summary of number of reported cases of selected respiratory diseases in CT by year*
CT DPH Occupational Disease Surveillance Data

	1997	1998	1999	2000	ODSS Total**
Asthma	27	19	24	27	223
RADS* **	4	7	5	9	41
Silicosis	1	3	1	0	13
Asbestosis	3	7	6	7	117
Asbestos-related pleural diseases	2	10	9	4	122
TOTAL	37	46	45	47	516

* As of May 1, 2001. Data subject to change.
** Occupational Disease Surveillance System (ODSS) total since 1/1990
*** Reactive Airways Dysfunction Syndrome

Web-Sources

- ***Environmental Health in Family Medicine***

A free online curriculum for teaching and learning environmental health is available. The target audience includes residents in family medicine, pediatrics, and ob/gyn, practicing physicians, and other health professionals such as nurses, nurse practitioners and midwives. The case-based modules cover six areas: lead, indoor air quality, outdoor air quality, pesticides, clusters-human health and water quality, and persistent organic pollutants.

www.ijc.org/boards/hptf/modules/content.html

- ***Diagnosis and Management of Rhinitis: Complete Guidelines of the Joint Task Force on Practice Parameters in Allergy, Asthma and Immunology***

These comprehensive guidelines include information on the clinical characteristics and diagnosis of the various types of rhinitis, and recommendations on patient evaluation and disease management. The document also stresses the importance of managing rhinitis when other disease conditions such as asthma, sinusitis and otitis media are present.

www.jcaai.org/Param/Rhinitis/Default.htm