Professional article

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ALLERGIC RHINITIS - POSSIBLE OCCUPATIONAL DISEASE - CRITERIA SUGGESTION

SUMMARY

After the skin, the respiratory tract is the most commonly affected organ system in workers exposed to ever increasing number of different respiratory agents. Allergic rhinitis could be caused by occupational allergens which characterises rhinitis as a occupational disease. Our country does not recognize allergic rhinitis as an occupational disease, although a lot of countries do. It has been suggested, in accordance with European Union Commission recommendations from 2003, to add allergic rhinitis to the occupational disease list, provided that the necessary suggested criteria are met. Regarding allergic rhinitis as an occupational disease will present important prevention measure regarding the occurrence of occupational asthma and disability in exposed workers. It has been suggested to take into account the following criteria in order to classisfy the allergic rhinits as a occupational disease: positive work history, a worker is a non-smoker, there was no nasal allergic disease before obtaining a job, ORL examination result (specific physical examination result), proof of specific sensibilisation existence (exposure test, prick skin testing, IgE, nasal specific provoking test etc.) **Compulsory elements for establishing the diagnose would be the following: medical history, physical examination (ORL examination), in vivo skin test, determining total and specific IgE in serum, measuring maximum nasal airflow, non-specific nasal provoking test, cytological examination of nasal lavage sample, mucociliary cleaning test. Olfaction ability test, computed tomography of paranasal cavities and pulmonary function test.**

Key words: **occupational rhinitis, diagnose, criteria**

INTRODUCTION

After the skin, the respiratory tract is the most commonly affected organ system in workers exposed to ever increasing number of different respiratory pathogenic agents. Respiratory tract mucous membrane with its limited defense mechanisms is often shock prone tissue for numerous toxic, cancer and antigene agents. Today greater significance is ascribed to interdependency of mucous membrane reaction of whole respiratory tract regardless of physiological conditions (normal or disease). Having this in mind, sensitisation to antigene stimulants in exposed workers can occur with different allergic manifestation such as allergic rhinitis and sinusitis, nose polyps, laryngitis, bronchioloalveolitis, angioneurotic syndrome and asthma.

Upper respiratory tract, as initial passage for entrance of all airborne agents is the first defensive mechanism but also the first target for respiratory pathogenic agents. Allergic rhinitis is a disease which every doctor faces on a daily basis. It can oc-

cur as isolated disease or as a disease which follows disease conditions of the lower respiratory tract (2-5). However, generally considering, this disease is not taken seriously enough, although it is well known that it affects immensly the workers' quality of life, causes increased number of sick leave days and decreases working days productivity. Nasal obstruction in allergic rhinitis, if persists for a prolonged period, could lead to various complication such as sinusitis and otitis. Also, since breathing is mostly done through mouth (thus nasal mucous membrane protective role being avoided), lower respiratory tract diseases can occur, e.g., asthma which is preceded by allergic rhinitis (6,7).

Allergic rhinitis could occur due to the exposure to different allergens in the working environment, which characterises allergic rhinitis as an occupational disease. All the chemicals which can cause occupational asthma can cause occupational allergic rhinitis as well.

According to our laws, allergic rhinitis is not recognised as an occupational disease, i.e., it is not included in the Occupational Disease List. European Commission gave the recommendations regarding the European Occupational Disease List in Brussels in September 2003. Annex I of the above stated document comprises the list of occupational diseases which should be included in the official occupational disease lists in European Union countries by the end of 2006. Allergic rhinitis is registered as item 304.07 in the above mentioned list, since it is caused by being exposed to known and recognised allergens in working environment. European Commisssion suggested that each country should adjust the criteria for verifying particular disease as an occupational one in accordance with the legislation of that particular country (8).

This paper treats allergic rhinitis as an occupational disease with the final aim to have this disease recognised as an occupational disease in our country as it is in most developed countries of the world.

DEFINITION

Occupational rhinitis is the episodic, work-related occurrence of sneezing, nasal discharge, and nasal obstruction. Many substances are known to cause occupational rhinitis.

EPIDEMIOLOGY

According to the research, 15% of workers in the world suffer from occupational rhinitis which actually amounts to 4% of all respiratory diseases. These data should not be taken for granted since occupational rhinitis may not often be reported or may not be connected to the occupation. The industrial workers suffer the most from this disease (48%) followed by administration workers (29%) and the workers in production (16%). High risk occupations include farmers, pharmaceutical workers, carpenters or wood workers, food processing workers and health care workers. Occupational rhinitis can occur as a consequence of wearing protection masks or while using ventilation and cooling devices (10).

The workers with personal or family medical history of allergic diseases are more prone to occupational rhinitis. Another risk factor is smoking. With the increase of allergen concentration in environment and exposure period, the risk of obtaining the occupational rhinits increases as well.

ETIOPATHOGENESIS

The agents which could cause the allergic occupational rhinitis as well as agents causing occupational asthma are numerous and they are increasing on a daily basis (11) (table 1).

Table 1. Agents associated with occupational allergic rhinitis

High molecular weight compounds (proteins)
Animal antigens (animal handlers, farmers, veterinarians) Green coffee bean and castor bean (dock workers) Proteolytic enzymes (detergent workers, cosmetologist) Grains/contaminants (bakers, farmers, grain handlers) Insect antigens (various occupations) Gum arabic/gum acacia (printers) Psyllium (health care workers) Latex (health care workers)
Low molecular weight compounds
Diisocyanates (polyurethanes -painters, boat builders, etc) Acid anhydrides (plastics- painters, fabricators, etc) Colophony (rosin core solder- electronics workers) Plicatic acid (western red cedar- saw mill workers) Antibiotics (health care workers)

Occupational rhinitis symptoms occur during repeated exposure to offending allergen. Several months, even several years can pass between first exposure to allergen and the occurrence of symptoms. More diagnoses of occupational rhinitis are being made because we now recognize that substances of low molecular weight can act as hapten that become allergenic after inhalation and conjugation to body proteins such as human serum albumin. Allergic rinitis is a type I hypersensitivty reaction, which is an IgE-mediated response that consist of 3 phases: sensitization, early-phase and late phase response. During the first phase of the allergic reaction, allergens from environment comes into contact

with mucous epithelium of nasal membrane and initiates antigenes presentation to immune cells. Antigene-presenting cells such as macrofags and dentritic cells ingest and degrade allergens into small peptides which are then presented to ThO lymphocytes originating from thymus and they are called "naive" lymphocytes T. Besides antigene presentation, there is the process of antigene recognition involving class IIMHC molecules (major histocompatibility complexes). Polymorphism of class II MHC molecules expressed by each individual and molecule affinity for specific antigene peptides have a significant part in immune system "deciding" whether to respond to specific protein.

T lymphocytes recognize antigene peptides in the context of class II MHC molecules which initiates their differentiation into Thl and Th2 and realising of inflamatory factor. Allergic rhinitis like asthma is determined by Th2 lymphocyte participation.

Th2 lymphocytes secrete cytokines such as interleukines IL3, IL4,IL5,IL9,IL10 and IL13.

Interleukines IL4, IL10 and IL13 promote B lymphocytes proliferation and IgE antibody production. Antigene peptides and IgE antibodies have central part in allergic response early phase.

Circulating IgE connects to high affinity receptor on the surface of mast cells and basophils which causes degranulation. Mast cells degranulation is a critical moment in initiating early phase of allergy inflamation process leading to the release of histamine, prostaglandines, triptases, leukotrienes and other chemotactic factors which stimulate the inflamatory cells such as eosinophils, basophils and neutrophils. Mediators released by the mast cell realise its effect on blood vessels (vasodilatation, mucose thickening, increased permeability, watery rhinorrea), on glands (gland exocitose and secrete creation) on nerves (itching, sneezing, parasymphatic reflexes).

Late phase of allergic response in rhinitis occurs 4 to 12 hours after allergen exposure and it is characterised by the presence of inflamatory cells such as eosinophils, basophils and neutrophils in nasal mucose. The inflamatory cells (particularly eosinophils) release mediators which causes disfunction of the nasal epithelium which, as a consequence, causes nasal congestion, heavy breathing, sleep disorder, snoring, etc.

Under the conditions od extended exposure to occupational allergens, local inflamation is increased by continued secretion of different cytokines and alpha tumor necrosis factors (TNF) produced by mastocytes, macrophages, eosinophils and lymphocytes which stimulate sensory nerves in nasal mucose. This neural stimulation causes hyperreaction and hypersensitivity of the nasal mucose even to non-allergic stumuli such as cold air and cigarette smoke which provides an explanation for workers with chronic occupational rhinitis who experience the symphtoms outside the working environment while they are exposed to any respiratory irritants (10,12,13).

The occupational rhinitis can very rarely develope according to type IV of the allergic reactions, when sensitised to hard metals such as chrome andnickle (10).

PATHOPHYSIOLOGY

Since nasal epithelium and epithelium of the lower respiratory tract are similar, and since even the allergic manifestation are alike, rhinitis could be taken as a marker for what is happening in lower respiratory tract. Allergens bigger than 5 microns stay in upper respiratory tract and they could cause rhinitis, while smaller particles pass the nasal filter and reach lower respiratory tract where they could cause asthma or hypersensitive pneumonitis. However, while breathing through mouth, even bigger particles can reach lower breathing airways causing asthma and rhinitis simultaneously. In most cases the allergic manifestations occur on nasal mucuos membrane since it is the first the allergen encounter.

NASAL SYMPTOMS

Simptoms of occupational rhinitis are the same as those associated with other types of rhinitis and include sneezing, itching, clear rhinorrhea, nasal congestion, and nonpurulent, postnasal discharge. Occupational rhinitis is characterised by time and space connection of the symptoms with the working environment of the patient where offending allergen could be found. If the patient is exposed to these allergens continuously, symptoms can persist throuhout the workday. These allercic reactions also involve a late-phase response that occurs approximately 4 to 8 hours after the initial allergen exposure. The primary symptom of a late-phase reaction congestion rather than sneezing, itching, and rhinorrhea.

A sensitised worker who has a single exposure to an allergen on arrival at work may experience symptoms immediately, only to be relatively symptom-free (provided he is working away from the stimulus) until 6 hours later, when symptoms recur. Similary, a single exposure to an allergen at the end of the shift may produce immediate symptoms, followed by spontaneous relief and recurence of symptoms 6 hours later, after the patient has returned home.

The challenge in these situations is to determine whether the recureht symptoms represent a

late-phase response to an early exposure or an acute phase of a subsequent exposure. Futhermore, a worker may have an isolated early-phase response, an isolated late-phase response, or both. These reactions may vary within the worker, depending on the exposure and the allergen. A weekday worker who is asymptomatic on weekends, only to become symptomatic when resuming work on Monday, probably has occupational rhinitis.

DIAGNOSIS

History

For prompt diagnosis of occupational rhinitis, it is important maintain a keen sense of how the worker presents. The *symptom history* is the first and often the most important aspect of presentation. Symptom history of the patient should be established in details, with special attention paid to the links between the symptom occurence in working environment and their withdrawal during the vacation period and weekends. Medical Questionnaires should be used (general health care questionnaire SF-36: Medical Outcomes Survey Short Form and health care questionnaire for rhinitis: rhinitis QoL questionnaire- JuinerEF). The questionnaires should be completed related to the period when the worker is working and when he/she is on vacation and then do the comparative analyses (12,14,15). The worker should be questioned in details, particularly regarding the medicines he/she uses.

Occupational history. The circumstances of the exposure must be thoroughly investigated. Manufacturing data on the compounds should also be obtained from the manufacturers to note their safety and side effect. The nature, duration and frequency of exposure should be documented. In addition, the working conditions at the site of the allerged exposure must be ascertained in order that the effectiveness of extraction /ventilation can be assessed. Chemicals, which cause severe irritation, will quickly become intolerable to the work force and the exposure will be limited as a result. The substance or substances may be a gas, vapor or particulate. Sometimes the conditions will result in a mixture of compounds. Particle size is important, since larger or heavier particles will not stay in the air for long. The nature of the material has a direct relationship to the sites of toxicity in the body. Substances with mild irritant properties will only produce effects at very high levels of exposure and, under normal circumstances, may not be expected to have any detrimental or long term effect on the worker. The conditions of exposure are just as important as the chemical nature of the substances themselves.

Family history (presence of allergic diseases in family) should be established.

Personal history, with special attention paid to occurrence of respiratory disease in childhood and to the possibility of worker being atopic, should be established. Details of any diseases, particularly cardio-vascular and central nervous system problems, should be documented along with a detail of all medication received.

Social and epidemiological history (smoking, alcohol, hobbies and other free time activities, pets) should be taken into consideration as well.

Physical examination

Workers with occupational rhinitis have clear, watery discharge; swollen turbinate. Nasal mucosa being typical: pale, pink, hyperemic or boggy. The examination is done by routine method (rhino scopy) or by flexible or rigid endoscopic examination which enables nasal, pharynx and glottis structure to be seen easily. Also nasal polyps can be determined. Pus secretion from nasal sinus opening can occur, also lymphoid hyperplasia, neoplasm, changes in vocal cords are all important for diagnose differentiation. Other physical stigmata of allergic rhino conjunctivitis, such as injected conjunctivae or sclera, chemosis, or allergic shiners, may be present.

In vivo skin test, with standard inhalation allergens and suspect allergens of the working environment, is very useful for diagnosing. Prick or interdermal (ID) is used.

Determining total and specific serum IgE. The measurement of total serum IgE is barely predictive for allergy screening in rhinitis and should no longer be used as a diagnostic tool. In contrast to low predictive value of total serum IgE measurements in the diagnosis of immediate type allergy, the measurement of allergen-specific IgE in serum is of importance. The demonstration of specific IgE antibody (RAST, ELISA) is dependent upon preparation of an extract of the relevant allergen or, in the case of low molecular weigh chemicals, the preparation of hapten-protein conjugates suitable for immunological testing.

Measuring nasal inspiratory or expiratory peak flow. The diary of maximum nasal airflow (together with symptom gradation) is kept before, during and after working shift. It is desirable to carry out measurements during one working week including weekends. The interpretation of thus obtained data is similar to interpretation of maximum inspiratory flow in diagnosing occupational asthma.

Non-specific nasal provocation test. Nasal provocation with non-specific stimuli (histamine, metacholine, cold dry air, etc.) is not relevant for daily clinical practice and diagnosis but can be used in research.

Nasal-specific provocation test is the most characteristic test. The method implies measurement of the flow and resistance of the air through nasal cavities by rhinomanometry, before and after exposing the nasal mucous membrane to suspect allergen. The test is considered to be positive if, after the exposure, the nasal airflow is decreased for more than 40%, i.e., if the nasal resistance is increased for more than 60%. Test interpretation is done by comparative following of the symptoms and measuring quality and weight of the nasal secretion before the test, during and after the test. Acoustic rhinomanometry, rhinostereometry, head-out body pletismography and oscilometry could be used for this purpose. Acoustic rhinometry, a simple, easily reproducible, and less invasive procedure than the anterior or posterior rhinomanometry, has been reported to be useful in diagnosing occupational rhinitis.

Cytological examination of nasal lavage sample (eosinophils) and determining eosinophil cationic protein (ECP) before the test, 30 minutes, 4 and 24 hours after the provocation test (16-18).

Mucociliary clearance test: (working environment factors decrease mucociliary clearing which is significant for microbiological defense)

O If action ability test (quantity and quality smell test for smell identification ability).

Computerised tomography ofparanasal cavities, sometimes magnetic resonance imaging, are of importance for differential diagnose.

Pulmonary function test. Occupational rhinitis can be likened to occupational asthma with which there are many parallels and frequently co-exist. Asthma is often preceded by rhinitis.

SUGGESTIONS

Criteria for evaluating rhinitis as occupational disease

- positive work history
- a worker is a non-smoker
- there was no nasal allergic disease before obtaining a job,
- ORL examination result (specific physical examination result)

proof of specific sensibilisation existence (exposure test, prick skin testing, IgE, nasal provoking test, nasal lavage sampling, etc.).

WORKING ABILITY EVALUATION

Worker with established occupational disease (occupational allergic rhinitis) is not able to perform jobs where he/she would be exposed to either the chemicals which he/she is proven to be hypersensitive or to respiratory pathogenic agents of any origin.

PREVENTION

Prevention includes:

- allergen avoidance
- removal of the allergen in cases when that does not harm production or any other process in the working environment
- change of the working place by removing the worker from the allergen present zone
-control of the working conditions
-ventilation improvement
-usage of personal protection devices
-occupational selection and orientation
-education of workers
-health care protection educational measures

CONCLUSION

Allergic rhinitis does not endanger the life of the patient. It does not require hospitalization or urgent interventions and that is often the reason why health care workers do not often view the allergic rhinitis as serious disease. However, the allergic rhinitis symptoms decrease the patient's quality of life. The disease leads to the frustration of the patient, decreases his or her social activities, causes sleep disorder which as a consequence leads to chronic tiredness, loss of concentration, decrease in production rates, frequent sick leave days and increased injuries at work.

Allergic rhinitis often represents the initial phase of a more serious disease such as asthma. It is not rare case that allergic rhinitis and asthma appear simultaneously. Allergic rhinitis could be caused by occupational disease which gives it the nature of the occupational disease. It is not in the list of the occupational diseases in our country although other countries recognize it as such. It has been susaested.

in accordance with European Commission recommendations from 2003 and in accordance with the suggested criteria for occupational rhinitis, that occupational allergic rhinitis should be included in occupational disease list which will present significant preventive measure in occupational asthma occurrence and exposed worker disability.

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ALERGIJSKI RINITIS - MOGUĆE PROFESIONALNO OBOLJENJE - PREDLOG KRITERIJUMA

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SAŽETAK

Posle kože respiratorni sistem je najčešće ugrožen organ kod radnika u uslovima izloženosti sve većem broju različitih respiratornih noksi. Alergijski rinitis može biti izazvan alergenima prisutnim na radnom mestu što mu daje karakter profesionalnog oboljenja. U našoj zcmlji se ne nalazi na listi profesionalnih bolesti mada se u mnogim zemljama priznaje kao takvo. Predlažemo, a u skladu sa preporukama Komisije Evropske Zajednice iz 2003 godine, da se pod uslovom ispunjenih navedenih kriterijuma ovo oboljenje uvrsti u listu profesionalnih bolesti što bi značilo bitnu preventivnu meru za pojavljivanje profesionalne astme i invalidnosti kod eksponovanih radnika. Predlog je da kod proglašavanja ovog oboljenja za profesionalno budu sledeći kriterijumi: pozitivna radna anamneza, da radnik nije pušač, da pre zaposlenja

nije postojalo alergijsko oboljenje nosa, karakterističan ORL nalaz fizikalnim pregledom i dokaz postojanja specifične senzibilizacije (test ekspozicije, kožni test, IgE, nazoprovokacioni test, nazalni lavat i dr.). Obavezni elementi pregleda u dijagnostičkom postupku bill bi: anamneza, fizicki nalaz (ORL), in vivo kožni test, određivanje ukupnog i specifičnog IgE u serumu, merenje maksimalnog nosnog protoka, nespecifičan nazoprovokacioni test, nazoprovokacioni test, citološko ispitivanje nazalnog lavata, test mukocilijarnog čišćenja, testovi za ispitivanje olfaktivne sposobnosti, komjuterizovana tomografija paranazalnih supljina i ispitivanje pluc'ne funkcije.

Ključne reči: profesionalni rinitis, dijagnoza, kriterijumi