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Occupational allergic rhinitis

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INTRODUCTION

Allergic risk has acquired a notable importance in the occupational environment: it is becoming more frequent than the toxic risk; it has been shown to have an elevated clinical and etiological diversity; it is seen to have a greater severity, especially in respiratory terms.

Industrial physicians have traditionally attached importance to occupational factors in respiratory diseases. Recently, substances acting via an immunoallergic mechanism have been superadded onto the inorganic substances responsible for pneumococionis: the risk of the former is more insidious and ubiquitous than that of the latter (Charpin, 1980; Parkes, 1982).

The allergic risk usually attributed to individual predisposition, thus posing the problem of the capacity of occupying a particular work station, is a health problem which involves both specific allergenic pollutants and non-specific chemical pollutants.

There is relatively little data concerning isolated rhinitis in the occupational environment. On the other hand, most research on occupational asthma and our own observations show the importance of these symptoms preceding and accompanying the bronchial symptoms (Landhall and Blak, 1947; Proctor, 1977).

We will thus consider the ensemble of pathophysiological and clinical data as pertaining to occupational respiratory disorders which include an allergic rhinitis. The early onset and the reversibility of this "signal" symptom lead to a discussion of its epidemiological and preventive value.

DEFINITION, GENERAL CONSIDERATIONS

Occupational allergic rhinitis results from a specific IgE sensitization acquired while working.

The role of other types of immunological reactions involving IgG-mediated complement elements or cell reactivity is possible in certain cases of granulomatosis associated with a glomerulonephritis observed in the presence of chlorinated solvent fumes.

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Recent work has shown that nasal allergy is not a simple vasomotor reaction, but rather a genuine inflammatory state. In the presence of prolonged exposure to the allergen, this results in infected locoregional lesions and the development of a cholinergic bronchial hyperreactivity, favouring the appearance of asthma (Chan-Yeung, 1982; Creticos et al., 1984; Lam et al., 1979).

Allergic rhinitis is initiated by specific allergens. Their appearance is favoured and their course of progression is accelerated by non-specific irritating pollutants.

There are two types of specifically responsible substances:

Macromolecules of animal or plant origin: any species may be causative if it is the object of a specialized manipulation; in this case, there is a predisposition in atopic subjects.

Chemical substances, whose sensitizing role depends on a particular chemical reactivity, i.e. the electrophilic character, which results in a covalent binding to proteins.

PATHOPHYSIOLOGY

The chemical intermediates released during the reaction of the antigen and IgE bearing mast cells are similar to those in asthma, with the only difference being the absence of muscles in the nasal cavities:

Preformed mediators: histamine, chemotactic factors for polynuclears.

Newly formed mediators: prostaglandins, P.A.F., leucotrienes, S.R.S.A.

These factors explain edema of the chorion, hypersecretion, vasodilation, infiltration by neutrophilic and eosinophilic polynuclears, tissue lesions of the epithelium and basement membrane, and fibrosis (Appaix et al., 1977; Austen, 1980; Samuelson, 1983).

CLINICAL PICTURE

The symptoms of occupational allergic rhinitis are well known and include sneezing, nasal pruritus, rhinorrhea and obstruction. The rhinorrhea is general not infected. These manifestations appear after several months of work in contact with the responsible agent and often several hours after starting work during the day. Obstruction is often maximal in the morning after awakening (Gervais, 1983). Improvement is often obtained after a sick leave of several days. The use of vasoconstrictors aggravates and prolongs the course of the syndrome.

The rhinitis may be either isolated or accompanied by conjunctivitis. Usual complications of prolonged rhinitis include sinusitis or even otitis. Polyps in the nasal cavities and anosmia are rare. The true menace one or two years is the appearance of an asthmatic disease. This is not a consistent complications and can be avoided if the worker is protected sufficiently early on (Chan-Yeung, 1982; Tiffeneau, 1959).

ETIOLOGICAL FORMS OF OCCUPATIONAL ALLERGIC RHINITIS

A. Rhinitis due to biological agents

This may be any plant or animal form: husbandry, manipulation, utilization of a protein (enzymes, sericine) or polysaccharide (gum) components, with atmospheric contamination by fine dust particles. As examples we may cite: flours, castor oil, unroasted coffee, wood dust, small mammals, poultry, arthropods (insects, acarians, small crustacean). These are thus urban, agriculture-food, husbandry and research settings. The disorders are generally a combination of rhinitis and conjunctivitis, with asthma appearing several years later.

Subjects affected the earliest are atopic but the proportion of affected workers may reach 50% in extremely poor working conditions. These cases of allergic rhinitis are the typical IgE type, whose etiological diagnosis is uncomplicated via clinical examination, skin tests and rhinomanometry. In addition to improve working conditions, medical treatments are also possible: cromoglycate and occasionally desensitization. When the responsible factor is wood dust, there is a risk of cancer of the nasal cavities.

B. Rhinitis due to chemical agents

These cases of rhinitis are not systematically accompanied by conjunctivitis. They are the early signs of respiratory disorders in a number of industries: chemical, pharmaceutical, metallurgical, plants using plastics, in the hairdressing profession, even in agriculture.

We may cite the following pathogenic agents for which the allergic type early onset rhinitis has been described: persulfates, penicillins, cephalosporins, phenylglycine acid chloride, piperazine, macrolides, reactive biological stains, amines, formaldehyde, phthalic and trimellitic anhydrides, organic isocyanates, furans, metals (nickel, chromium, chloroplatinates, and cobalt, tungsten, titanium and tantalum carbides). These chemical rhinitis frequently precedes the asthmatic disease.

Certain of the above agents are the precursors of pulmonary fibroses (metallic carbides), while others are associated with the risk of respiratory cancers (nickel, chromium, formaldehyde). The pathophysiology of some of these etiologies is clearly not allergic (amines, formaldehyde, isocyanates, persulfates, piperazine). The etiological diagnosis is thus relatively difficult.

Specific antibodies have been revealed by skin tests and/or the assay of IgE specific for the following substances: Penicillins, cephalosporins, macrolides, phthalic and trimellitic anhydrides, isocyanates, phenylglycine acid chloride, reactive biological stains, nickel, chromium, chloroplatinates.

DIAGNOSIS OF OCCUPATIONAL ALLERGIC RHINITIS

A. Diagnosis of allergic rhinitis

1. Rhinoscopy does not reveal specific symptoms: the mucosa may be hypertrophic, secreting, pale or hyperhemic; in chronic forms it may also exhibit an atrophied appearance. Polyps, septal perforation and even carcinoma lesions must be systematically sought.
2. Radiological examination of the sinuses often shows hypertrophy surrounding the mucosa, occasionally an air-liquid level.
3. Cytology of nasal secretions may be valuable for distinguishing allergic from irritative lesions: in the latter case, there is a rarity and deteriorations of ciliated cells, neutrophilic polynuclears and malphighian junction cells are present, whereas ciliated cells are present and normal in cases of allergy; the presence of eosinophils is relatively frequent (Lam et al., 1979).
4. Blood eosinophils and increased total IgE concentrations are preferential symptoms of an atopic state.

B. Diagnosis of the occupational etiology of a rhinitis

Anamnesis must be complemented by objective examinations.

1. Skin tests to common allergens (acarians, grass pollen) are required in order to detect the allergic terrain predisposed to occupational rhinitis due to biological macromolecules and chloroplatinates. These tests are always possible with biological allergens, giving a histamine response after 10–15 min in sensitized subjects. This is a symptom of exposure, however, which does not imply the existence of respiratory disorders. They nonetheless retain considerable epidemiological value. Only several chemicals can be investigated with carefully chosen skin tests: chloroplatinates, penicillins, persulfates, phthalic anhydride, chromium, nickel, reactive stains.
2. Radioimmunological or immunoenzymatic tests enable total IgE and specific IgE to be assayed. Total IgE concentrations are elevated primarily in the atopic state. Specific IgE species can be theoretically assayed with all biological antigens, either animal or plant. In practice, however, it is rare that the material for these assays is available in ready to use form. Concerning chemicals, the following compounds have been used for assays of specific IgE: phthalic and trimellitic anhydride, penicillins, reactive biological stains, nickel and platinum, isocyanates, phenylglycine acid chloride.

The results of these tests enable a high specificity to be attributed to them, but sensitivity is lower than that offered by direct nasal challenge. These reactions are very important for affirming the immunoallergic nature of the observed disorder.

3. Rhinomanometry in occupational allergic rhinitis.

a. Methods. Measurement of resistance: we use the technique of anterior rhinomanometry for measuring nasal resistance. The mouth is tightly closed and

the airflow and anterior pressure are determined in one nostril. Posterior pressure is measured in the previously obstructed opposite nostril. Each side is investigated sequentially. The resistance of a nasal channel is determined by the ratio P/V: the difference of nostril/choanal pressure over airflow. Total resistance (Rt) is calculated using the resistances of the right (Rr) and left (Rl) channels with the equation: $1/Rt = 1/Rr + 1/Rl$. Resistance after administration of the allergen is expressed with reference to that after administration of physiological saline.

Challenge methods: resistance is measured before spraying and then the same parameter is recorded after administering 0.1 ml of isotonic NaCl (154 mM NaCl). This value is the initial resistance (Ri). The allergen is then administered by spraying 0.1 ml of a solution into each nasal cavity with the subject in voluntary apnea. In order to avoid bronchial inhalation, a nasal forceps is used to obstruct the buccal cavity for 15 minutes. The measurement is then performed after blowing each side successively. The allergen is administered in progressive concentrations and we consider a 100% increase in resistance for a flow-rate of 150 ml sec^{-1}. This increase is obtained after either the first dose or after the accumulation of successive increasing concentrations.

b. Results. Challenges with authentic plant or animal allergens give positive results which satisfactorily correspond with clinical examinations and assays of serum IgE, which are less sensitive. These tests furnish fewer positive reactions than the skin tests, which often show a sensitization in the absence of any clinical manifestation.

Our experience with chemical allergens is still very limited and we can thus report only a few positive reactions after stimulation of the occupational gesture of hairdressers exposed to persulfates. The diagnosis between a simple or irritative vasomotor rhinitis and an allergic rhinitis related to a chemical substance can remain difficult. Rhinomanometry may be a valuable tool in this distinction: in cases of vasomotor rhinitis, nasal resistance increases slightly but appreciably after spraying physiological saline, while in cases of occupational allergic rhinitis, only the responsible allergen provokes this unambiguous reaction.

SOCIAL AND PREVENTIVE CONSIDERATIONS

Under French legislation, the most frequent occupational allergic diseases are listed in Tables of Occupational Diseases. After a declaration drafted by the physician, this enables the worker to receive free medical treatment, workmen's compensation for workdays lost, and financial indemnity in case of permanent incapacity for work, which he may retain after the occupational disease has run its course. This declaration leads to a verification of working conditions and proposals for their improvement if need be. In terms of respiratory disorders, they are primarily asthma and extrinsic allergic alveolitis. The following are respectively considered in the case of organic isocyanates, proteolytic enzymes and sintered

metal carbide dust: recurrent rhinopharyngitis, rhinitis with epistaxis and spasmodic rhinitis.

In addition, every physician is legally bound to draft a declaration to the work inspection whenever he encounters a disorder which he feels to be related to the patient's occupation. This provision is designed to update the tables of occupational diseases. A system for the indemnity of sporadic cases following evaluation is in the planning stage but has not yet been implemented. Finally, in the absence of possibilities of indemnity in the preceding cases, every salaried worker can undertake legal action against his employer, although in practice this is rarely utilized. It is clearly possible to add the notion of rhinitis to all existing tables of occupational respiratory allergic illnesses. Nevertheless, this provision should require precise diagnostic criteria because of the frequency of non-professional allergic or vasomotor rhinitis.

In order to be able to more generally insure the prevention of respiratory disorders in an occupational environment before the appearance of obstructive bronchial syndromes, epidemiological research should be carried out independently of any immediate sanction. Employees and employers are currently discouraged from drafting declaration, with excessive irregularity from one company to another, may lead to very costly measures of nuisance reduction. Epidemiological surveys, including rhinitis in the search for the prevalence of respiratory disorders in the occupational environment, would lead to the determination of elevated risk occupations and to the imposition of collective health measures involving all the companies in a given occupation, even before the appearance of obstructive bronchial syndromes.

CONCLUSION

The nasal cavities are the first filters and screens of deleterious agents in the respired environment. They constitute a mucous vasomotor, secretory and complex immunological apparatus which requires the utmost attention on the part of the epidemiologist and the clinician. Modern diagnostic methods lead to the early identification of manifestation due to occupational allergens.

On the individual level, the consideration of this symptom may lead to the adoption of necessary measures before the appearance of the asthmatic disease. On the collective level, surveys of respiratory pathology which include rhinitis may constitute a powerful means for promoting better hygiene conditions in the occupational environment.

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