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Occupational Laryngitis Caused by Formaldehyde: A Case Report

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Formaldehyde is commonly accepted to be an allergen and irritant. However, specifically diagnosed occupational respiratory diseases caused by formaldehyde are relatively rare. Occupational laryngitis was diagnosed in a 47-year-old dairy foreman. He had been exposed for 9 years to formaldehyde emitted from a milk-packing machine situated underneath his office. His exposure level varied considerably. Under normal process conditions, the measured formaldehyde level was 0.03 mg/m^3. The patient was examined by different specialists over 1½ years. It was concluded that that he had psychogenic dysphonia. However, a specific laryngeal provocation test with formaldehyde carried out at the Finnish Institute of Occupational Health was positive. His laryngitis was so serious that he was pensioned. During the 3 years of follow-up his condition gradually worsened. He now reacts especially to tobacco smoke and other air impurities known to contain formaldehyde.

KEY WORDS: formaldehyde, provocation testing, occupational bystander exposure, laryngitis, milk packing

INTRODUCTION

Formaldehyde is a known irritant and allergen causing occupational asthma, occupational dermatitis, and conjunctival and mucosal irritation, especially in the respiratory tract [Alexandersson and Hedentierna, 1988; Holness and Nethercott, 1989; Bruze and Almgren, 1988; Nordman et al., 1985; Uba et al., 1989; Imhof and Wutrich, 1988]. Exposure to formaldehyde has been considered to be one possible cause of the sick building syndrome [Rogers, 1987]. Formaldehyde exposure provokes pharyngeal irritation in relation to occupational [Hovarth et al., 1988; Malaka and Kodama, 1990] and environmental [Uba et al., 1989] exposure.

There are two references in the literature concerning an association between exposure to formaldehyde and laryngeal irritation. Kalimo et al. [1980] reported temporary aphonia in a 46-year-old truck driver exposed to a resin containing formaldehyde while loading fiber rolls. However, their clinical findings did not indicate laryngitis with an allergic mechanism. Kwong et al. [1983] found hoarseness and edematous vocal cords in a 32-year-old nonsmoking pathology resident. The average concentration of formaldehyde within her breathing zone was 3 ppm. Our literature search (Medline, Index Medicus, CIS) for the years 1970–1992 did not reveal any case reports of occupational laryngitis associated with formaldehyde.

CASE DESCRIPTION

The patient (VA) was born in 1941 as the seventh of nine children. His father died of laryngeal cancer at the age of 70 years. His brothers and sisters are all healthy. There are no known allergies or allergic respiratory diseases among his next of kin. VA has been healthy for all of his life, except for arterial hypertension, which was diagnosed in 1989. He was first treated with captopril for 3 months; the medication was stopped because of pharyngeal irritation and changed to diltiazem hydrochloride. He quit smoking 25 years ago, has moderate alcohol consumption, and has no economic or marital problems.

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WORK HISTORY

After completing military service in 1961, VA started work in a dairy as a maintenance man. From 1980 to 1990 he worked as a foreman in the dairy. There is no evidence that he would have been significantly exposed before 1980 to allergens or formaldehyde, either at work or during his leisure time. As a foreman, VA worked mainly in an office and carried out inspection rounds in the dairy. He was not considerably exposed to noise. Air flow measurements showed that the main stream of impurities from the milk-packing line situated at a lower level ascended directly into his office through the inflow ventilation duct. During the packing process, milk boxes (Pure-Pack TM) are closed thermally and aldehydes are emitted in low quantities. In June 1990, the following concentrations of chemicals were measured in the foreman's office during a typical morning under normal process conditions: formaldehyde 0.03 mg/m^3, acetaldehyde 0.02 mg, acrolein 0.01 mg/m^3, acetic acid 0.04 mg/m^3, and formic acid 0.10 mg/m^3. The concentrations were low but may have varied considerably depending on the phase of milk-packing in progress.

SYMPTOMS AND CLINICAL EXAMINATION

In the fall of 1989, VA had prolonged periods of hoarseness associated with chest tightness. He was examined in the internal medicine unit in his local central hospital. Coronary heart disease was excluded with an exercise stress test. He reported that the symptoms appeared immediately when he entered his workplace, especially his office. He gradually began to experience episodes of aphonia in association with the pharyngeal irritation. The symptoms disappeared when he was on sick leave. At first VA had three periods of sick leave, ranging from 1 to 2 weeks. The symptoms worsened rapidly and he was obliged to avoid his workplace. Thereafter, for all practical purposes he was absent from work during the years 1990 and 1991. During this period he was carefully examined by several specialists. When absent from work for longer periods, his laryngeal status was normal unless he was exposed to tobacco smoke or traffic exhaust gases.

Allergic tests (prick) were negative, except for dog hair and house dust. Histamine provocation was normal, the patient did not suffer from eosinophilia nor the hyperventilation syndrome, his gastroscopy and bronchoscopy were normal. The liver enzymes serum alanine aminotransferase and gamma glutamyltransferase were slightly elevated at the beginning of 1990 but were normal in a control measurement 6 months later.

Work-site provocation was carried out in July 1990. When examined before going to his office, the patient had no specific symptoms and his larynx was normal. He worked 2 hours in his office and carried out his normal duties. Within 15 min after the cessation of exposure he underwent an otorhinolaryngological (ORL) examination, which showed that he was aphonic and his laryngeal mucosa and vocal cords were swollen. He was referred to the Finnish Institute of Occupational Health (Helsinki) for further examination in August 1990. After two phoniatric examinations (August and October 1990) the conclusion was "psychogenic dysphonia." However, psychological and psychiatric consultations indicated no psychiatric disorders. He firmly believed that his symptoms were associated with his job and occupational exposure to a chemical compound. Unsatisfied with the clinical examinations because his symptoms were considered psychogenic, the patient gradually became depressive and even suicidal.

Specific provocation tests were carried out in the provocation chamber in the Institute of Occupational Health with placebo (polyol) and formaldehyde (1 ppm) as described by Newman-Taylor and Davies [1981]. The patient's ORL status was checked immediately before and after provocation, and 8 and 24 hr after the beginning of provocation. The signs of redness and swelling were rated with a four-stage scale: 0 = none; 1 = mild; 2 = moderate; 3 = abundant. The test was interpreted to be positive if both redness and swelling increased by at least one point. Voice quality was also estimated with a four-stage scale according to Hirano [1981].

VA's voice became hoarse with formaldehyde provocation. The hoarseness increased from 1 to 3 on the Hirano scale. The redness of his laryngeal mucosa increased from 1 to 2, and both the redness and edema of the vocal cords increased from 1 to 3. The changes were still apparent 8 hr after the provocation but had subsided by 24 hr after provocation. Formaldehyde (1 ppm) had no effects on the ORL symptoms or signs of five healthy controls. These provocation tests were carried out by the same nurse, and the status was checked by the same ORL specialist.

The chamber provocation test was considered sufficient for the diagnosis of laryngitis. A false-positive result could not be excluded completely. Nevertheless, we consider it unlikely because the increment in laryngeal signs was so fast, specific, and in clear association with his occupational exposure. The case was reported to the patient's insurance company, which accepted the diagnosis, and granted a 1 year early pension, and then later the standard compensation for occupational disease.

Since the diagnosis of occupational laryngitis in 1991, VA's condition has been followed by the occupational health physician of his former employer. VA's laryngeal symptoms improved during the first 6 months after he left his job. However, after 6 months he reported that several other irritants caused reactions similar to those associated with his work exposure. He claimed that especially tobacco smoke and traffic exhaust gases caused hoarseness, after

which he lost his voice for 1–2 days. In the winter, cold and humid air also provoked symptoms. After 3 years of follow-up, his symptoms have worsened. VA now reacts to several other irritants. His symptoms are under control when he avoids all known irritants but his daily life has clearly become more limited, and in 1992 he was granted a permanent disability pension. He has had no other respiratory symptoms, such as wheezing or vasomotor rhinitis during the follow-up period.

DISCUSSION

Although upper respiratory tract irritation is commonly known to be caused by formaldehyde and other aldehydes, a laryngeal reaction has not been reported. Our experience indicates that outpatients sometimes associate several throat and laryngeal symptoms with their work, but their doctors do not. Although, in many cases the cause remains unclear, it seems evident that ORL symptoms are still a largely unmapped area in the field of occupational medicine.

The chamber provocation test with formaldehyde in combination with a clinical examination was considered sufficient for the diagnosis of laryngitis. A false-positive reaction was excluded by the results with the control group that participated in the provocation test with formaldehyde. The amount of change in status was significant for the patient, from normal to Reinke's edema-like status.

The pathogenesis of formaldehyde-induced laryngitis is still unknown. Our patient had negative skin prick tests with common allergens and formaldehyde. His serum immunoglobulin E level was 12 U/ml and the radioallergosorbent test (RAST) showed no specific antibodies. We recommend that an occupational history be taken carefully in all cases with voice symptoms when patients associate their symptoms with work. If the symptoms are relieved during vacation or leisure time, the patients should especially be referred for further controlled examinations whereby a specific laryngeal reaction can be identified or excluded.

REFERENCES

Alexandersson R, Hedenstierna G (1988): Respiratory hazards with exposure to formaldehyde and solvents in acid-curing paints. Arch Environ Health 42:222–227.

Bruze M, Almgren G (1988): Occupational dermatoses in workers exposed to resin based on phenol and formaldehyde. Contact Dermatitis 19:272–277.

Hirano M (1981): "Clinical Examination of Voice." New York: Springer Verlag.

Holness DL, Nethercott JR (1989): Health status of funeral service workers exposed to formaldehyde. Arch Environ Health 44:222–228.

Hovarth EP, Andersson H, Pierce WE, Hanrahan L, Wendlick JD (1988): Effects of formaldehyde on the mucous membrane and lungs. A study of an industrial population. JAMA 259:701–707.

Imhof E, Wutrich B (1988): Formaldehyd- und phtalisches anhydrid-asthma. Nachweis spezifischer IgE-antikörper im RAST. Schweitz Med Wochenschr 118:568–1572.

Kalimo K, Saarni H, Kyttä J (1980): Immediate and delayed type reactions to formaldehyde resin in glass wool. Contact Dermatitis 16:496.

Kwong F, Kraske G, Nelson AM, Klaustermeyer WB (1983): Acute symptoms secondary to formaldehyde exposure in a pathology resident. Ann Allergy 50:326–328.

Malaka T, Kodama AM (1990): Respiratory health of plywood workers occupationally exposed to formaldehyde. Arch Environ Health 45:288–294.

Newman-Taylor AJ, Davies RJ (1981): Inhalation challenge testing. In Weil H, Turner-Warwick M (eds): "Occupational Lung Diseases." New York: Marcel Dekker, pp 143–167.

Nordman H, Keskinen H, Tuppurainen M (1985): Formaldehyde asthma—Rare or overlooked? J Allergy Clin Immunol 75:91–99.

Rogers SA (1987): Diagnosing the tight building syndrome. Environ Health Perspect 76:195–198.

Uba G, Pachorek D, Bernstein J, Garabrant DH, Balmes JR, Wright WE, Armar RB (1989): Prospective study of respiratory effects of formaldehyde among healthy and asthmatic medical students. Am J Ind Med 15:91–101.