Building-Related Asthma in Denver Office Workers

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ABSTRACT

Objectives. Reported cases of building-related asthma and hypersensitivity pneumonitis among workers in a Denver office building prompted an epidemiologic investigation.

Methods. A cross-sectional, self-administered survey of employees in the office building of the reported cases was compared with that of employees in a comparison building.

Results. A significant excess of respiratory disease existed among 512 Denver workers (case building) compared with 281 office workers in a suburban agency (control building). Denver employees had a higher prevalence of respiratory symptoms, and the prevalence of physician-diagnosed asthma with onset or exacerbation since building occupancy was 4.9 times greater for Denver employees than for suburban employees. Asthma was not associated with any particular ventilation system in the building. Water incursion from a below-grade wall may have contributed to the problem, but the etiology is unknown.

Conclusions. This investigation provided evidence of office building–related asthma. Individual cases may be sentinel events for other cases of work-related asthma or hypersensitivity pneumonitis and may indicate a need for public health investigation of remediable causes. (*Am J Public Health.* 1993;83:89–93)

Introduction

Building-related asthma has been infrequently reported. Three case reports of office building–related asthma with a clear work-related pattern exist, and all were related to humidifiers or the biocides used in humidifiers.[1–2] Epidemic asthma occurred in a printing factory in association with a contaminated humidifier.[3] Reports of another building-related allergic disease, hypersensitivity pneumonitis, were related to bioaerosols disseminated from ventilation systems or from water-damaged furnishings.[4–8]

This paper describes two sentinel cases of office building-related lung disease that triggered a public health investigation. We report our findings regarding building-related respiratory disease that was not associated with a humidification system.

Sentinel Case Reports

Case 1

A 48-year-old social services eligibility technician began working in a Denver office building in October 1986. She had an insidious onset of dry cough in January 1987, which was diagnosed as asthma in March. Skin prick tests were negative to aeroallergens, and she was referred to a tertiary occupational medicine clinic.

She performed peak flow measurements with a mini-Wright peak flow meter; the results indicated reproducible, striking airflow limitation shortly after she entered the office building, with partial recovery on lunch breaks outside the building and full recovery on weekends (Figure 1). After relocating to another building in February 1988, she had resolution of her symptoms, of her need for asthma medications, and of her work-related airflow limitation as documented by peak flow measurements.

Case 2

A 37-year-old administrator began working in the same Denver office building in April 1985. In February 1987, she developed exertional dyspnea but sought no medical attention. In April 1987, an elective surgery was canceled on the basis of a preoperative chest x-ray showing bilateral interstitial infiltrates with some alveolar component. Her presumed pneumonia did not respond to courses of two antibiotics, and she was referred to a pulmonary consultant. Her forced expiratory volume in 1 second was 49% of predicted, and her forced vital capacity was 42% of predicted. She began taking 60 mg per day of prednisone.

When she discontinued steroids in August, she had a recurrence of her symptoms. Her progressive symptoms and 20-lb weight loss over 6 months led to an open lung biopsy in October 1987. The pathology report diagnosed usual interstitial pneumonitis with focal bronchiolitis obliterans and extensive honeycombing.

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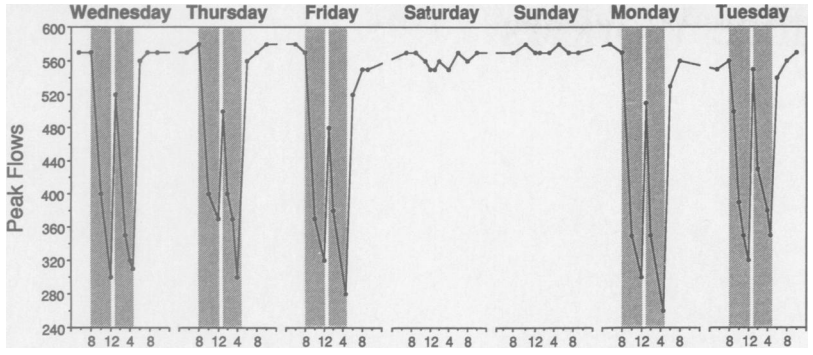

FIGURE 1—Representative peak flow measurements for Case 1 in relation to hour of day and day of week, 1987 to 1988.

Recovering from surgery for a month at home, she became much better, with resolution of her dyspnea on exertion and cough.

After she returned to work, she had recurrence of fatigue and shortness of breath associated with a flulike illness characterized by cough, pleuritic chest discomfort, shaking chills, and sweats. Her respiratory and constitutional symptoms improved over weekends but became much worse during the next 2 work weeks. Her restrictive pulmonary functions deteriorated from preoperative values.

She was referred to the same tertiary occupational medicine clinic as the patient in Case 1, where her clinical course was felt to be consistent with hypersensitivity pneumonitis. Again, she had symptomatic improvement on 60 mg per day of prednisone and medical restriction from the Denver building.

Background

Both sentinel cases of building-related lung disease were reported to the Occupational Epidemiology Program of the Colorado Department of Health, and by August 1988 the department had received reports of five additional cases of respiratory illnesses potentially related to working in the building. Because of these reports, an epidemiologic survey was designed to determine whether there was an excess of building-related respiratory disease in people working there.

The single-story, 108 000-ft^2 office building was occupied by the Denver county agency on April 21, 1985. The building was mechanically ventilated, without operable windows or a humidification system. Twenty-seven fan coil units with drip pans above the false ceiling provided cooling and heating of air, with intakes on the north wall. Water damage was visible in ceiling tiles, and employees reported recurrent water damage to the carpet from overflow of a janitor's sink and sewage backup. The south wall of the building was built into an earthen bank below street level.

In the spring of 1988, carbon dioxide levels were documented by National Institute for Occupational Safety and Health industrial hygienists as increasing from 0.04% to 0.06% in the morning to 0.11% to 0.15% in the afternoon, suggesting insufficient fresh air intake during the hours that the building was occupied. The employer hired a private consultant to conduct bioaerosol sampling. The results revealed low counts of viable airborne bacteria and fungi, both inside and outside the building, with 10 of 20 outdoor samples surprisingly showing no growth. Counts of colony-forming units showed little variation across specific areas within the building and between two sampling days, with the exception of a higher afternoon fungal count near the south entrance. The rank order of fungal species inside differed from the rank order outside the building. Aspergillus was the only organism found inside the building near the south entrance; no aspergillus grew from outdoor samples. No sampling for nonviable organisms or antigens was performed.

Methods

In late 1988, we performed cross-sectional surveys of employees in the Denver building (case building) and a comparison building (control building) housing the same type of agency in Adams County, in suburban Denver. The two sentinel cases were not survey respondents because they were not employed at the time of the survey.

The comparison building was first occupied on June 1, 1980, and the office workers were known to have indoor air-quality complaints. The six-story building was air conditioned and had inoperable windows. We hypothesized that no differences existed between building groups in the prevalence of respiratory symptoms and physician-diagnosed asthma or hypersensitivity pneumonitis.

We distributed self-administered questionnaires consisting of standardized respiratory questions[9] supplemented by questions about additional symptoms, the relationship of symptoms to the work environment, physician diagnoses, and medication use. Self-reported diagnoses of asthma or hypersensitivity pneumonitis were verified by the participant's physician. We ascertained the dates of symptom onset and diagnosis, objective supportive data, and history of exacerbation since building occupancy. Four physicians, blind to employment building, then categorized cases independently and by consensus as one of the following: insufficient evidence of the condition, preexisting disease, preexisting disease with exacerbation since beginning work in the building, and disease with onset postoccupancy.

Because the maximum interval from Denver building occupancy to the date of the survey of Denver employees was 3.6 years, cases for both Adams and Denver employees were counted only if they were diagnosed within the first 3.6 years of employment.

We divided the Adams population into two groups: group A, consisting of persons who began work in the building between June 1, 1980, and April 20, 1985, and group B, consisting of persons who began work in the building on or after April 21, 1985 (the date Denver workers occupied their building). We compared the Denver employees with all Adams employees and with each Adams subgroup. Analyses of Adams group A and Denver permitted comparison of persons who newly occupied an office building; analyses of Adams group B and Denver permitted comparison of persons diagnosed during the same time period, thereby controlling for temporal differences in medical practice.

We classified Denver building employees into four work areas demarcated by floor-to-ceiling partitions and different sets of ventilation units. Prevalence rates were compared in the four areas.

We performed univariate analyses of demographic variables and symptom and disease prevalences using Fisher's exact, χ^2, or t tests on PC SAS.

Results

Of 671 Denver employees, 512 returned questionnaires (76% response rate); 281 of 357 Adams employees returned questionnaires (79% response rate). The only statistically significant difference in demographic characteristics among the two employee groups was a greater proportion of Black and Hispanic employees in Denver than in the Adams building. Mean age, gender distribution, educational level, mean hours worked per week, and smoking status did not differ between groups.

Seventy percent of Denver employees thought there was a problem with their building, compared with 75% of Adams employees. Prevalences of nonspecific symptoms attributed to the work environment by respondents were high and comparable in both buildings (Table 1). The mean number of sick days taken per year was significantly greater for Denver employees than for Adams employees (10 days vs 8 days, $P < .05$).

Statistically significant excesses existed among Denver building occupants for shortness of breath, shortness of breath while at rest, and chest tightness (Table 2). The differences in shortness of breath at rest and chest tightness remained significant when analyses were limited to nonsmokers.

There were 32 asthma cases in Denver workers, compared with 11 in Adams employees; no cases of hypersensitivity pneumonitis were reported. Of the asthma cases, 28.1% in Denver and 18.2% in Adams County were diagnosed with abnormal pulmonary function tests; the primary method of diagnosis for both groups was response to bronchodilating agents. No single attending physician diagnosed more than one case.

The median interval from beginning work in the building until date of asthma diagnosis for the 10 Denver cases with onset of asthma postoccupancy (Table 3) was 1.5 to 2.0 years; the shortest interval was 1 year. Two of the four postoccupancy Adams cases had an interval greater than 3.6 years. Eight Denver employees had exacerbation of preexisting asthma in their building, and no Adams employees reported such exacerbation.

Table 3 presents the prevalence of asthma in Denver employees compared with all Adams employees, Adams group A employees, and Adams group B employees. For every comparison, the Denver prevalence rate was greater than the Adams rate and was significantly different for the comparison of Denver with all Adams employees and with Adams group A employees for the total number of cases with either exacerbation or postoccupancy asthma. No statistically significant differences in prevalence rates of asthma existed between Adams group A and group B employees.

Because the distribution of race/ethnic groups differed in the two buildings, we examined whether race/ethnicity could be a confounding factor. No statistically significant association was found

TABLE 1—Prevalence of Symptoms Attributed to the Work Environment by Denver and Adams County Employees

Symptom, %	Denver (n = 512)	Adams County (n = 281)	P
Headaches	57	61	.3
Throat irritation	27	35	.02
Eye irritation	45	41	.3
Runny nose	38	43	.1
Dry skin	22	22	.8
Itchy skin	22	13	.001
Fatigue	35	33	.6
Mean no. of symptoms	4.8	4.8	.9

TABLE 2—Prevalence of Respiratory and Systemic Symptoms among Denver and Adams County Employees

Symptom	Denver, % (n = 512)	Adams County, % (n = 281)	P
Cough	24	19	.1
Shortness of breath	34	27	.03
Shortness of breath at rest	20	13	.009
Wheeze with a cold	53	56	.3
Wheeze apart from colds	30	27	.4
Wheeze with shortness of breath	20	18	.3
Wheeze with exertion	22	23	.7
Chest tightness	40	31	.02
Feverishness	18	16	.5
Achiness	25	25	.9

TABLE 3—Prevalence Rates of Asthma in Denver and Adams County Employees, Grouped by Dates of Building Occupancy

Type of Asthma	Group	No. of Cases	No. of Noncases	Rate, %	P
Postoccupancy onset of asthma	Denver	10	434	2.3	
	Adams total	2	243	0.8	.23
	Adams A	0	141	0.0	.13
	Adams B	2	102	1.9	1.00
Exacerbation of existing asthma	Denver	8	14	36.4	
	Adams total	0	7	0.0	.14
	Adams A	0	3	0.0	.53
	Adams B	0	4	0.0	.28
Postoccupancy and exacerbation	Denver	18	448	3.9	
	Adams total	2	250	0.8	.02
	Adams A	0	144	0.0	.01
	Adams B	2	106	1.9	.40

Note. People began work between June 1, 1980, and April 21, 1985, in the Adams A building and on or after April 21, 1985, in the Adams B building.

between race/ethnic group and exacerbation or postoccupancy asthma: the asthma rate was 3.6% in Anglos, 2.7% in Hispanics, and 2.4% in Blacks. There was no evidence of effect modification by cigarette smoking when analyses were stratified by smoking history.

The prevalence of respiratory symptoms and physician-diagnosed asthma among Denver employees did not differ in the four work areas. However, asthma cases appeared to cluster along the south side of the building, irrespective of floor-to-ceiling partitions.

Discussion

The two sentinel cases of building-related allergic respiratory disease led to a public health investigation that suggested that they were neither unique nor the first reported cases. Case 1 met the criteria for occupational asthma of the surveillance case definition disseminated by the National Institute for Occupational Safety and Health.[10] For case 2, the temporal relation of respiratory and constitutional symptoms to work in the Denver building pointed to a diagnosis of occupational hypersensitivity pneumonitis; the pathologic diagnosis was compatible with hypersensitivity pneumonitis, in which all cases have interstitial pneumonitis and at least 50% have bronchiolitis obliterans.[11,12] Although asthma and hypersensitivity pneumonitis are distinct clinical entities, there may be airway hyperreactivity in both; the clinical expression depends, in part, on the location of antigen deposition and the host response. They are both seen in relation to microbial bioaerosol exposure, isocyanates, phthalic anhydride, animal antigens, detergents, and coffee dust.[13,14]

Clinicians frequently fail to recognize hypersensitivity pneumonitis when airway symptoms common to both diagnoses suggest asthma. We are unable to determine whether cases of hypersensitivity pneumonitis were diagnosed as asthma among Denver employees. We are aware of an additional case of interstitial lung disease in a former Denver employee. Her building-related symptoms began within 4 months of building occupancy, and she died with a diagnosis of bronchiolitis obliterans within 4 years. In retrospect, her pulmonary physician felt that hypersensitivity pneumonitis was the probable diagnosis.

Sentinel cases of occupational asthma and hypersensitivity pneumonitis should provide impetus for epidemiologic or industrial hygiene studies, which can lead to preventive recommendations.[15–17] We found a 4.9-fold increase of physician-diagnosed asthma arising or exacerbated since building occupancy among employees working in the Denver office building compared with employees working in a similar agency in Adams County. In addition to the excess of diagnosed disease, increased prevalences were observed in several respiratory symptoms suggestive of asthma.

In buildings plagued with indoor air-quality complaints, overreporting of symptoms is expected.[5,18] Denver employees may have had increased health concerns because of an April 1985 outbreak of complaints attributed to mass psychogenic illness by management, previous indoor air-quality investigations, and newspaper publicity. Nevertheless, the observed differences between the Denver and Adams buildings were probably not attributable to reporting bias because a higher proportion of Adams County workers thought there was a problem with their office building and the response rates of the two groups were high and nearly the same. Furthermore, even if we assume that nonresponders from both groups had the same prevalence rate as the Adams County responders, the Denver rate of asthma would still be 2.1 times greater than the Adams County rate.

Our survey probably underestimated the rate of respiratory disease in Denver employees. A cross-sectional study may reflect a healthy-worker effect by not identifying persons with disease who terminated or were absent from work as a result of illness, such as the two sentinel cases. The choice of comparison group was conservative, since the Adams group had registered indoor air-quality complaints with their county health agency and there was no reason to suspect that the out-migration of sick workers would be greater in Adams County than in Denver.

Allergic respiratory disease associated with office buildings is usually caused by dissemination of antigenic material from a microbial source in ventilation systems or humidifiers. No evidence to support a ventilation-related etiology surfaced in the epidemiologic investigation. The possible clustering of cases along the south wall, which was built into an earthen bank, suggests that moisture incursion may have led to microbial amplification and dissemination. Bioaerosol sampling documented a low level of aspergillus in this area that was not present in outdoor air, suggestive of an indoor source. However, technical difficulties in the sampling, which resulted in low viability, make environmental characterization of the building incomplete.

This investigation leaves many questions pertinent to prevention unanswered. Nevertheless, we report our findings to alert clinicians, indoor-air-quality investigators, and public health epidemiologists to the phenomenon of office building-related asthma. Individual cases may be sentinel events and indicate a need for public health investigation of remediable causes. In this respect, occupational public health differs little from traditional communicable disease control activities. Early recognition of causal association and removal from exposure may result in better prognosis or cure of occupational asthma.[19] Public health surveillance and further investigation of these types of outbreaks will provide better understanding of risk factors for office building-related respiratory disease. □

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