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Pulmonary Disease Associated with Cafeteria Flooding

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ABSTRACT. An illness among office workers consisting of cough, fever, chills, muscle aches, and chest tightness was associated with water leaks from a cafeteria. Mean single breath carbon-monoxide diffusing capacity (DLCO) of cases differed significantly from that of non-cases. There was a significant decrease in the percent of predicted DLCO with increasing number of symptoms. Testing for precipitating antibodies to microbial agents found in the building revealed no differences between cases and non-cases. DLCO is an appropriate cross-sectional instrument for field investigations of building-associated respiratory disease.

HYPERSENSITIVITY PNEUMONITIS (HP) has been reported with increasing frequency as a work-related disease.[1] These reports have shown widely differing attack rates, inconsistent physiologic changes, and a lack of uniform diagnostic criteria. Questionnaires may be the most sensitive diagnostic tool;[2] they are also most prone to the biases of subjects, employers, and investigators. There is no accepted method to validate a "standardized" questionnaire definition, should one be developed. Recently, we have had the opportunity to study an outbreak of illness resembling HP and to use single breath carbon-monoxide diffusing capacity (DLCO) in a cross-sectional field study in an attempt to validate a questionnaire.

BACKGROUND

In March, 1982, two cases of flu-like illnesses persisting over 6 wk in one area of a large eight-story office building were reported to the National Institute for Occupational Safety and Health, Centers for Disease Control (CDC). The two cases worked in a centrally located zone on the seventh floor which had been the site of a series of floods, one of which occurred on January 27, 1982. The cafeteria kitchen on the eighth floor is directly above this office zone and its water drainage system runs through the common return air plenum above the suspended ceiling over offices occupied by the personnel experiencing illness. The plumbing for the cafeteria dishwasher had no grease traps. Consequently, grease periodically clogged drain pipes, causing water to back up and eventually flood the underlying zone. After the flood in late January, the two index cases developed cough, chest tightness, muscle aches, headaches, nausea, chills, fever, and fatigue that occurred during the work week, but disappeared by Saturday afternoon only to reoccur by Monday afternoon. Ar-

thralgias in one was so severe that she could walk only with difficulty. The illness seemed to grow progressively worse. Both individuals had seen physicians before an investigation was requested.

The Epidemiology Standardization Project strongly recommended DLCO be used in the diagnosis of interstitial lung disease.[3] As the clinical presentation of the index cases was compatible with HP, DLCO was used in the evaluation of employees. The hypothesis was that ill persons sitting close to water leaks were sensitized to an unknown agent present in the office environment and should have lower DLCOs than non-ill individuals.

MATERIALS AND METHODS

The study consisted of an industrial hygiene survey, distribution and analysis of a self-administered questionnaire, pre- and post-shift spirometry, blood-drawing for testing of serum precipitins, and chest x-rays on cases. A more detailed description of the laboratory methods can be found in the Health Hazard Evaluation Report.[4]

Medical. A self-administered questionnaire[2] elicited duration, temporal pattern, and frequency of six symptoms: headaches, muscle aches, chest tightness, fever, chills, and nausea. Illness was defined as the presence of at least two of the above symptoms occurring on consecutive workdays including the last working day (Friday) with relief on weekends. This pattern was called "periodic"; all other temporal patterns were called "sporadic." The questionnaire was distributed to three different groups of workers: Group 1 (N = 51), the group in the offices affected by the water leaks; Group 2 (N = 69), a second group of individuals in an adjacent office but employed by the same agency; and Group 3 (N = 41), a third group from a different agency on another floor. An association of illness was sought with distance from water leaks, smoking, chronic bronchitis (i.e., 3 months of cough and phlegm for at least 2 yr), a history of allergies, diarrhea, sinusitis, and arthralgias.

Spirometry meeting American Thoracic Society (ATS) criteria[5] was performed with a waterless spirometer (Ohio Medical Products model 840) before and after at least 6 hr of exposure to air within the building. The values were compared to predicted values of Knudson and coworkers[6] and analyzed as percent predicted forced expiratory volume in the first second (% Pred $FEV_{1.0}$) and of forced vital capacity (percent predicted FVC).

DLCO was measured on a Hewlett-Packard System controller (47315 DLO S.B.), with helium and carbon monoxide analyzers. The mean of two measurements within 5% of each other, and where inspiratory volume on that trial was within 10% of the FVC obtained through spirometry,[3] was compared to predicted values of Cotes[7] and expressed as percent predicted (% Pred DLCO). In two individuals these requirements were not met, and a mean of all three values was used. No more than three measurements were performed by any individual. DLCO measurements were performed in the order that persons volunteered. The intra-subject coefficient of variation in our laboratory is 5.4%.

Statistical. Grouped data were analyzed with standard statistical tests. Regression analysis was performed with PROC GLM of the Statistical Analysis System. Classes of persons were defined by the presence of none, one, two, and three or more symptoms. Analyses were performed twice each on classes with periodic and on classes with combined periodic and sporadic symptoms, adjusting either for pack-years or for smoking status.

Industrial hygiene and microbiology. Airborne microorganisms were collected by Andersen viable samplers.[8] Samples of carpets and ceiling tiles, dirt from the surfaces of drain pipes in the common plenum, and filters and dust present in air handling and fan coil units were collected for isolation of bacteria, fungi, and protozoa. A water sample for microbial analysis was also obtained from the cafeteria drainage system. Plates used for the collection and isolation of fungi, bacteria, and protozoa contained rose bengal streptomycin agar (100 μg streptomycin/ml), tryptic soy agar (50 μg cycloheximide/ml), and a non-nutrient agar coated with a suspension of *Escherichia coli*, respectively. Respirable dust and total suspended particulate were collected utilizing a 2.54-cm (1-in) cyclone sampler (Bendix Model 240, polyvinyl chloride 47-mm filter, 0.8-μm nominal pore size) operating at a flow rate of 66 L/min and a General Metal Works, 20 × 25 cm (8 × 10 in), high-volume sampler (type AE glass fiber filters) operating at a flow rate of 2 m^3/min, respectively. Short-term colorimetric indicator tubes including those for carbon dioxide, carbon tetrachloride, ozone, ammonia, nitrogen dioxide, carbon monoxide, hydrocarbons, perchloroethylene, and methanol-ethanol were utilized to test for the presence of possible contaminant gases in occupied spaces. Other possible air contaminants were collected in a large charcoal tube, desorbed with ethanol, and screened by gas chromatography.[4]

RESULTS

Environmental. The illness occurred in an eight-story primarily open-concept office building with a cafeteria located on the eighth floor. The heating, ventilation, and air-conditioning (HVAC) systems consisted of 18 central air handling units (AHUs) and over 900 perimeter fan coil units. Each AHU has a chiller bank to dehumidify and to cool ventilation air. Conditioned air from each AHU is transported to offices through ducts which terminate in slots around the periphery of ceiling light fixtures and in long slot type supply outlets at some building perimeter locations. Each AHU provides supply air to vertically superimposed zones on a number of different floors. Return air from occupied spaces passes into centrally located slots in ceiling lighting fixtures and enters the return air plenum. Return air from zones on each floor moves through this common plenum and then through shafts or risers, and is transported by return fans to main AHUs or is expelled from the building during economizer operation. Air interchange between AHUs occurs because of mixing both in common return plenums and in building risers. Thus, once an air contaminant enters a common return plenum it could be easily distributed into other AHUs and then throughout the building.

The drain pans, chilled water coils, and associated

Table 1.—Frequency of Individual Symptoms in Persons Meeting Case Definition and in Non-Cases

Symptom	Cases*		Non-Cases*	
	Number with Symptom	%	Number with Symptom	%
Headaches	10	83	20	20
Chest tightness	7	58	3	3
Fever	6	50	9	9
Myalgias	5	42	13	14
Chills	5	42	5	5
Nausea	5	42	10	10
Arthralgias†	2	17	—	—

* 12 cases; 96 non-cases.
† Presence of arthralgias was not determined by the questionnaire.

AHU surfaces were clean and well maintained. There were no HVAC system dehumidifiers, air washers, or water sumps, such as have been associated with outbreaks of HP in other buildings.[1,2]

The common return plenum located between the seventh floor office zone experiencing leaks and the floor housing the cafeteria kitchen was examined. Drainage pipes in this return plenum were coated with large amounts of debris from previous floods. Ceiling tiles were water-logged with dishwater effluent. Carpeting and upholstered partitions in the office below the common plenum were also obviously soiled.

Medical. The building housed approximately 1300 employees. Both index cases worked in one office zone. Because there was an obvious problem in that zone, we concentrated our investigation on the employees in that zone. Two control groups were selected. One control group in a different zone on the same floor belonged to the same governmental agency; their offices had air supplied from a different AHU. A second control group consisting of governmental employees of another agency was selected on the fourth floor. The offices of this control group had air supplied by the same AHU as the first control group. Each group comprised less that 5% of the total building population.

Forty-one of 51 (80%) in Group 1, 38 of 69 (55%) in Group 2, and 29 of 41 (72%) persons in Group 3, completed questionnaires. Twelve persons met the case definition in Group 1, none in Group 2, and two in Group 3. These prevalences were significantly different between Group 1 and Groups 2 and 3 combined ($P < .00001$ by Fisher's Exact Test). Table 1 shows the frequency of the individual symptoms among the 12 cases and among the 96 non-cases. Of 12 cases, 3 had had symptoms for less than 1 month; 5 for less than 2 months; 2 for less than 1 yr; and 2 for more than 1 yr. Gender-specific prevalence rates were not significantly different ($\chi^2 = 1.03$). As other investigations have not included a specific temporal pattern of symptoms, we also used a second less stringent definition: any two symptoms (at least two sporadic or periodic). By this definition disease again occurred more frequently in Group 1 (17 of 41), than in Groups 2, (4 of 38), or 3, (6 of 29)($\chi^2 = 6.36$; 2 df, $P < .05$).

The only risk factor for illness in Group 1 was sitting within 15 ft of a water leak ($P = .038$, Fisher's Exact Test). Smoking, chronic bronchitis, a history of allergies, and time spent in the office were not associated with the illness. Cases were also more likely to have had diarrhea on 5 or more days in the 3 months preceding our survey ($P < .002$, Fisher's Exact Test). Neither water leaks nor diarrhea were associated with our less stringent case definition.

A total of 52 persons, 8 of 12 cases, and 44 of the other 75 in groups 1 and 2 volunteered for lung function tests. Cases undergoing lung function tests were again not significantly different from non-cases in percentage of smokers, mean age, and average sex distribution. There was a statistically significant difference in % Pred DLCO [± standard deviation (*SD*)] between cases (86 ± 26.3) and non-cases (104 ± 16.1) ($P < .02$). This was not true for baseline % Pred FVC nor % Pred $FEV_{1.0}$. Mean changes in $FEV_{1.0}$ and FVC over a workday were not different between cases and non-cases, or between Groups 1 and 2. Nine of the 12 cases volunteered for chest radiographs, which revealed no changes suggestive of HP.

All subjects with lung function measurements were grouped by the total number of symptoms. This was done for subjects with periodic symptoms alone and again for periodic and sporadic symptoms combined. In the former analysis, persons with only sporadic symptoms were grouped into the "0" periodic symptom group. Table 2 shows mean % Pred DLCO for both sets of groups, before and after adjustment for equal proportions of smokers, exsmokers, and never-smokers. Regression analysis demonstrated a significant decline in % Pred DLCO with increasing numbers of symptoms both for periodic and for combined periodic and sporadic symptoms ($P < .05$). Regression analysis for periodic symptoms revealed no association of % Pred DLCO with pack-years of cigarette smoking ($P > .10$). Adjustment for smoking status weakened the relationship between DLCO and "periodic" symptoms though the probability value was still less than 10%. Regression analysis for combined periodic and sporadic symptoms adjusted for pack-years weakened this association ($P < .10$). Adjustment for smoking status for combined periodic and sporadic symptoms resulted in no significant associations ($P > .10$) although a trend in DLCO for combined symptoms was still observed.

Industrial hygiene and microbiology. Microorganisms were isolated from damaged ceiling tiles and carpets obtained from the seventh floor zone where illness had occurred. In addition, similar analyses were conducted on debris obtained from the outside surface of pipes in the common return air plenum above this zone. All specimens were examined for protozoa contained *Acanthamoeba polyphaga*. Other predominant microorganisms isolated were *Monosporium apiospermum*, *Rhodotorula* spp., *Aureobasidium* spp., *Colpoda* spp., and *Mastigamoeba* spp. Sampling of airborne fungi was carried out in this office on March 16 and again on May 7, 1982. On both occasions levels of

Table 2.—Percentage of Predicted Single Breath Carbon Monoxide Diffusing Capacity by Number of Symptoms Unadjusted and Adjusted* for Smoking Status

	Periodic Symptoms		Combined Symptoms	
Symptoms	Mean % PRED DLCO	Mean % PRED DLCO Adjusted for Smoking Status	Mean % PRED DLCO	Mean % PRED DLCO Adjusted for Smoking Status
0	105 (34)†	104	107 (17)	105
1	103 (10)	98	103 (19)	102
2	99 (5)	100	99 (11)	101
3	82 (3)	83	90 (5)	92
	$P < .05$	$P < .10$	$P < .05$	$P > .10$

* Adjusted for equal proportions persons by smoking status in each group.
† Numbers in parentheses refer to the number of persons in each group.

fungi were low, i.e., less than 100 colony-forming units/m³. Dust samples collected from main AHUs and fan coil units throughout the building contained *Thermoactinomyces* spp. as predominant isolates.

Serologic techniques were used to determine if there was a difference between cases and non-cases in the production of specific antibody to *Acanthamoeba*, *Aureobasidium*, *Thermoactinomyces*, and other microorganisms isolated in this building. Table 3 lists agents isolated from the building and precipitin reactions to these and to agents associated with other outbreaks. Blood was drawn from 10 of the 12 cases and from 14 of the 17 persons in Groups 1 and 2 with no symptoms. The presence of precipitating antibodies to agents cultured from the building was determined. No significant differences were found between cases and noncases in rates of precipitin reactions to individual agents. Cases as a group were not more likely than controls to have at least one precipitin reaction to any agent.

The average concentration of respirable dust collected in the office zone affected by water leaks was only 25 μg/m³. Total suspended particulate and carbon dioxide levels never exceeded 40 μg/m³ (American Society of Heating, Refrigerating, and Air-Conditioning Engineers recommended limit is 260 μg/m³)[9] and 400 ppm (NIOSH recommended limit of 10,000 ppm),[10] respectively. All tests for contaminant gases by colorimetric indicator tubes were negative. Air samples collected by charcoal tubes contained toluene, xylene, and a series of mostly branched alkanes in the C_{10}-C_{12} range, but only in trace amounts, and were log orders below applicable occupational threshold limits.

DISCUSSION

Building-associated hypersensitivity lung disease has been associated with humidifiers, spray water air washers, water sumps, sewage flooding of basements, water contamination of rugs, and even contaminated tap water.[1,11,12] We have described a localized outbreak of illness that was associated with leaks from a cafeteria drain. Our subjects demonstrated a decrease in % Pred DLCO with increasing number of symptoms that was un-

Table 3.—Microbial Isolates and Persons with Precipitating Antibodies Against Specific Antigens

Antigen	Cases (N = 10)	Non-Cases (N = 14)
Standard "HP panel," Medical College of Wisconsin		
Micropolyspora faeni	0	0
Thermoactinomyces vulgaris	1	0
T. candidus	0	3
Saccharomonospora viridis	3	2
Pigeon serum	0	1
Aspergillus fumigatus #507	0	1
A. fumigatus #515	0	1
A. fumigatus #534	0	0
Penicillium notaum	0	0
Candida albicans	1	4
Agents isolated from the building		
Aspergillus fumigatus	1	0
A. niger	0	0
Aureobasidium spp.	3	2
Bacillus spp.	0	0
Monospora spp.	0	0
Mucor spp.	0	2
Paecilomyes spp.	1	2
Penicillium spp.	1	0
Rhodotorula spp.	0	0
Streptomyces griseus	0	0
Thermoactinomyces candidus	1	2
T. vulgaris	0	2
Acanthamoeba polyphaga	1	5
Protozoa isolated from other HP outbreaks		
Naegleria gruberi	0	1
N. lovaniensis	0	0
Acanthamoeba castellanii	1	2

likely due to chance and was only partly explainable by smoking. This suggests that an environmental factor associated with water drainage caused decreased pulmonary gas exchange, an abnormality compatible with hypersensitivity lung disease.

Three arguments can be made against this explanantion: chest radiographs (CXR) showed no abnormalities, no etiology was identified by precipitin testing, and smoking might explain the results. However, normal CXRs were found in ill subjects in 9 of the 13 studies of building-associated hypersensitivity lung disease where CXRs were taken.[1] In an outbreak of HP in mushroom workers, only 25% of symptomatic individuals had abnormal CXRs.[13] There have been lung biopsies of persons with negative chest x-rays demonstrating pathology compatible with HP.[12,14] Abnormal CXRs are not a necessary manifestation of HP.

Adjustment for smoking status weakened the association between number of symptoms and % DLCO. Ogilvie[15] calculated that carbon monoxide (CO) back pressure could add 3 $ml/min^{-1} \cdot mm^{-1}$ CO maximally. Addition of 10% to % PRED DLCO to all smokers still left a significant difference between cases and noncases. We cannot exclude smoking totally, but it alone does not explain our findings.

Precipitins have not been consistently useful in investigations of this kind. Some authors feel they indicate exposure to an agent rather than illness.[16] The etiology of outbreaks in the past has included *Acanthamoeba polyphaga* and *Themoactinomyces* spp. Both were isolated in this building. Distribution of precipitins to agents isolated from the building was no different among our cases than our controls. Air sampling for viable organisms has failed to detect agents responsible for pulmonary disease in the past because spores of cysts may be nonviable or failed to grow in selected culture media.[17] We cannot exclude that air sampling techniques failed to detect the agent responsible for this outbreak. Two studies have reported positive bronchial challenges in persons without precipitins to the challenge agent.[12,18] As all cases refused further studies, such as bronchial challenge with dust from the building and lavage, we could not determine the etiology.

Because of the design of the HVAC system, it was possible that some common exposure occurred throughout the building, although at lower levels in Groups 2 and 3. Microorganisms or microbial agents causing illness in Group 1 could have become entrained into the seventh floor return air plenum. From there this unidentified microbial agent could be mixed and diluted by other building return air streams, returned to main AHUs, and transported through supply ductwork to other building zones including that occupied by Groups 2 and 3.

Response to our questionnaire was variable and somewhat low. We do not believe that cooperation adversely affected the outcome of the study. All three groups to whom questionnaires were distributed consisted of highly mobile, travel-oriented employees who spend much of their time out of the office. Because of external constraints, conduct of the questionnaire survey and of the pulmonary function testing were limited to 3 days each. We made no attempt to get more persons on a second round because of the fear of introducing a bias: persons might have heard how cases were defined and responded in that form. We have no reason to believe that persons out of the office were more sick or less sick than those in the survey but cannot prove the point.

Several investigations of HP have not shown physiologic changes corresponding to complaints. DLCO has not been used as a field tool because it is thought to be susceptible to technician variation, have a high coefficient of variation, and be unreliable in the field. It has been used successfully in challenge procedures[19] and in case series[1,20] demonstrating physiologic changes of HP. DLCO was a useful tool in this field investigation.

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