Articles

A Case Series of 71 Patients Referred to a Hospital-Based Occupational and Environmental Medicine Clinic for Occupational Asthma

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In a ten-year period at the Occupational and Environmental Medicine Program (OEMP) of the University of Washington in Seattle, 71 patients were determined by attending physicians to have work-related asthma. In this cross-sectional descriptive study, we describe these patients. Data were obtained from a database maintained by the OEMP and from chart reviews. We found that the three most common specific agents causing asthma were isocyanates, red cedar, and crabs. At least one pulmonary function study was available for all patients and was positive in 56 patients (79%). Among the 71 asthma cases reported in this article, 18 (25%) were attributed to reactive airways dysfunction syndrome (RADS); 19 (27%) to exacerbation of pre-existing asthma; 27 (38%) to sensitization; and 7 (10%) had undetermined causes. We conclude that occupational asthma presents as a result of diverse exposures in multiple work settings and with an array of characteristics. Prevention efforts need to recognize this diversity.

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Occupational asthma is one of the most common work-related diseases.[1] Although the overall prevalence of occupational asthma is unknown, it is estimated that up to 15% of all adult-onset asthma can be attributed to occupational exposures.[2] As the number of agents (particularly synthetic chemicals) introduced to the workplace rises, the number of occupational asthma cases is also likely to rise. Although during the last few decades considerable advances have been made in its diagnosis, occupational asthma remains underrecognized. At the same time, the number of hospital-based, academically affiliated occupational medicine clinics providing services to patients with work-related diseases has risen.[1]

Although there have been surveillance studies of occupational respiratory diseases and occupational asthma,[3,4] clinical series of patients affected by occupational asthma have largely been confined to groups of workers reacting to single specified agents.[5] We report here a cross-sectional descriptive study of 71 patients with occupational asthma seen in the University of Washington OEMP over a ten-year period.

Methods

The potential study population was identified from information contained in a database maintained by the OEMP at the University of Washington. The OEMP has used a computerized data base since the clinic began in July 1981. This database contains routine demographic data and coded information regarding a patient's referral source, occupation and industry, employer name, exposures, and work-related diagnoses. When applicable, each diagnosis is associated with a specific exposure and contains a physician-derived determination of how likely it was that the diagnosis was work-related ("none," "possible," "probable," and "definite"). Conditions believed to be readily attributable to nonoccupational causes were generally excluded from the database.[1] All coding of clinical patient information was done by attending physicians. The clinic industrial hygienist often assisted with job exposure data.

Between July 1981 and July 1991, 1540 people were seen in the diagnostic OEMP clinic, and 126 were identified as possibly having occupational asthma. Of these, 71 had asthma determined to be "definitely" or "probably" work-related. These 71 individuals form the study group on which we report herein.

For the purposes of this study, we define occupational asthma as a condition that meets the following criteria: a patient has asthma and an association between symptoms and the workplace, either documented with specific test-

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ABBREVIATIONS USED IN TEXT

RADS = Reactive Airways Disease Syndrome
OEMP = Occupational and Environmental Medicine Program
FEV_1 = Forced expiratory volume in 1 second
IgE = Immunoglobulin E

ing or by clinical history; there has been a workplace exposure, with an association between asthma symptoms and exposure to some process, substance, or environment at work; there are work-related changes in spirometry or peak flow results; or there is a positive response to bronchial provocation testing with the agent to which the patient was exposed at work. A positive methacholine challenge test was used to support a diagnosis of asthma, but was not a necessary criterion for its presence.

Demographic data, as recorded by clinic attending physicians, were obtained from information coded in the database and from job description and exposure data. Additional clinical data were derived from patient chart reviews and included information regarding the patient's smoking history, workers' compensation status, medication use, and symptoms, as well as the relationship of the symptoms to workplace exposures, the agent suspected of causing the asthma, the presumed pathogenic mechanism (sensitization, irritation, or exacerbation of preexisting asthma), and the exposure parameters (type, onset, duration). Information regarding the presence of atopy was also obtained, with atopy considered to exist if the patient's history included one or more of the following: childhood asthma, childhood eczema, seasonal rhinitis, or skin tests positive for common environmental allergens. A standard allergy panel of skin tests was not used in the clinic at that time, nor were more specific allergens similar to workplace allergens available.

The association between symptom patterns and work-relatedness was considered positive if the patient reported one or more of the following: symptoms occurred at work only; symptoms worsened on Monday mornings; symptoms improved on weekends or vacations; symptoms only occurred in the evenings of workdays; symptoms worsened during the course of each work week; or symptoms resolved after a change in the work environment. The presumed pathogenic mechanism (such as sensitization based on an immunologic exposure or reaction to an allergen) was determined on a clinical basis by the examining physician; this information was taken from the patient's chart.

Spirometry data and the degree of bronchodilator responsiveness were recorded for all patients. Spirometry data were included only if American Thoracic Society criteria for reproducibility and reliability were met.[6] Results of a methacholine challenge test were considered positive if the concentration that induced a fall of 20 percent (PC20) in the results of a FEV_1 (forced expiratory volume in 1 second) test was 8 mg per ml or less.[8]

Specific provocation testing was performed in an environmental-challenge chamber. Each challenge procedure included a control period of exposure to filtered air, followed by exposure to progressively increasing doses that sought to re-create the workplace exposures. Spirometry levels were measured according to American Thoracic Society standards before the test was performed and at periodic intervals throughout the test. Provocation tests in which the FEV_1 level decreased by more than 10% from baseline were considered to be positive for workplace related asthma. Serial peak flow measurements were required to show a more than 20% variation in relation to work (decreasing across a work shift or improving during days away from work) to be considered positive.

Results

Results of the various tests administered to the 71 patients are shown in Tables 1–8. The first tables outline information regarding the patients' age, race, gender, and occupations, as well as the presence of atopy. Important to note are the many different workplace causative agents listed in Table 4.

Table 5 outlines the mechanisms involved with occupational asthma and the accompanying patient characteristics. Overall, the age, race, and gender distribution among the subgroups of each mechanism was similar, except for the exacerbation of preexisting asthma, which was more common in women. Smokers were evenly distributed among all subgroups except the undetermined group, which was overrepresented by smokers.

Information also was collected on whether the attending physician believed the causative agent to be acting as either an irritant or a sensitizer. Overall, in 31 patients (44%) the agent was felt to be a sensitizer; in 38 patients (53%) it was believed to have been an irritant; and in 2 patients (3%) it was not classifiable due to a lack of data in the patient's chart. Table 6 reveals the most frequently associated sensitization agents.

Dyspnea, wheeze, cough, and chest tightness were the symptoms extracted from the patients' charts. Five patients (7%) were symptom-free at the time of evaluation but had experienced symptoms in the past, and 54 (76%) were currently on medication—34 (63%) on inhaled bronchodilators; 9 (17%) on inhaled steroids; and 8 (15%) on systemic corticosteroids. Table 7 outlines in further detail the patterns of these symptoms.

Exposure and Time from Exposure to Symptoms

Information about the time elapsed from the first suspected causative agent exposure to the to the onset of symptoms was available for 68 patients; among these the range was from immediate onset in 8 patients (11%), all of whom were diagnosed with RADS, to 28 years, reported by 1 patient (1%). The elapsed time was reported as being five years or less in 52 (77%) of the patients. Eight patients (11%) reported only a single exposure to the agent in question. The time elapsed from the onset of

TABLE 1.—*Characteristics Of Patients With Occupational Asthma (n = 71)*

Characteristic	Number of Patients (%)
Sex	
Male	53 (74.6)
Female	18 (25.4)
Race	
White	57 (80.3)
Black	6 (8.5)
Hispanic	2 (2.8)
Asian/Pacific Islander	2 (2.8)
Native American	4 (5.6)
Smoking	
Never-Smoker	23 (32.4)
Ex-Smoker	32 (45.1)
# Years Quit (Mean ± SD)	9 ± 7
Pack Years (Mean ± SD)	23 ± 22
Current Smoker	16 (22.5)
Pack Years (Mean ± SD)	22 ± 21
Atopy	24 (33.8)
Age (Mean ± SD)	44 ± 13

symptoms to a clinic evaluation was available for 67 patients. On average, 4.5 years had elapsed, although 3 patients (4.5%) were evaluated within one month of symptom onset, and 1 patient had been experiencing symptoms for 22 years. Only 14 patients (21%) were evaluated within 6 months of the onset of symptoms.

At the time of first clinic evaluation, 34% of the patients were still exposed to the offending agent in the workplace. Of the patients no longer exposed, 17 patients (36%) were on sick leave or disability, 16 (34%) had moved to a different job, 10 had quit (21%), and 7 (15%) had been fired. In 2 cases (4%) the agent in question had been eliminated, and in 2 cases (4%) protective equipment was introduced and used.

Pulmonary Function and Diagnostic Testing

Assessment of the patients' pulmonary functions was most often performed using spirometry with bronchodilator responsiveness (Table 8). The 4 patients with negative methacholine challenge test results were all found to have evidence of asthma on another objective test. Six patients (9%) underwent a specific agent challenge test; 5 (7%) tests were positive, meaning that they displayed a 10% or greater decline in FEV_1. The agents used in these challenge tests were polyurethane foam (n = 2), and red cedar dust, fir dust, carbonless copy paper, and epoxy (each represented once). Thirty-six patients (51%) had a spirometry test on at least two occasions; 33 of the 36 (92%) had an FEV_1 variation of at least 10% between tests. Five patients (7%) underwent measurement of serial peak expiratory flow rates over the course of a workday; among those patients, 3 (60%) showed at least a 20% work-related variability. Overall, 56 patients (79%) had at least one abnormal pulmonary test.

Discussion

The distribution of our patients by job and industry is similar to that reported for all patients seen in the occupational medicine clinic.[1] Additionally, the agents implicated in causing or aggravating asthma are similar to those described elsewhere. They reflect the diverse economic base in the Pacific Northwest, which includes the timber, maritime and fishing, and manufacturing and aerospace industries.[3,4]

The specific agents that are irritant substances are most often associated with RADS. Known sensitizers, such as isocyanates and red cedar dust, are most often associated with sensitization asthma. Both sensitizers and irritants were related to an exacerbation of preexisting asthma. It should be noted that some crossover was found with certain agents thought to cause asthma by more than one mechanism. Isocyanates, a common denominator in both RADS and sensitization asthma, provide an example of this crossover. These findings are similar to data reported elsewhere.[7,9] Not all cases were associated with a specific agent; the inability to identify a specific agent, however, should not preclude attributing asthma to an occupational environment. Cases in which the causative agent is not easily identified emphasize the need for extra focus on other measures to deal with work-related asthma—through cross-shift spirometry, for instance.

The role of atopy in asthma varies according to agents and mechanisms.[7,10] Atopy apparently is an important risk factor both in cases of occupational asthma caused by high molecular weight compounds[8] and in cases of asthma caused by low molecular weight agents that act as haptens via IgE hypersensitivity (such as platinum salts). The risk of occupational asthma caused by

TABLE 2.—*Most Common Occupations Of Occupational Asthma Patients*

Occupation	Number of Patients (%)
Painter (Shipyard, Auto)	7 (9.9)
Plumber, Pipefitter	6 (8.5)
Assembler, Fabricator	6 (8.5)
Janitor, Cleaner	4 (5.6)
Machine Operator, Machinist	4 (5.6)
Machine Operator, Misc.	4 (5.6)
Mechanic, Industrial	3 (4.2)
Construction, Laborer	3 (4.2)
Manager, Engineer	2 (2.8)
Sales Occupations	2 (2.8)
Supervisors, Business	2 (2.8)
Food Service, Cook	2 (2.8)
Forestry Worker	2 (2.8)
Mechanic, Auto	2 (2.8)
Carpenter	2 (2.8)
Shipfitter	2 (2.8)
Shipscaler	2 (2.8)
Other	16 (22.5)
Total	55 (77.5)

TABLE 3.—*Common Industries Employing Occupational Asthma Patients*

Industry	Number of Patients (%)
Construction	8 (11.3)
Shipbuilding	8 (11.3)
Automotive/Painting	6 (8.5)
Airplane Manufacturing	6 (8.5)
Fabrication/Metal	5 (7.0)
Pulp and Paper Manufacturing	4 (5.6)
Lumber/Building Material	4 (5.6)
Electronics Manufacturing	3 (4.2)
Grocery Store/Misc.	3 (4.2)
Federal/State Government	3 (4.2)
Other	21 (29.6)
Total	71 (100)

such low molecular weight compounds as isocyanates[8] and red cedar,[11] however, does not appear to be increased because of atopy.

As we expected, a large number of atopic patients (41, or 58%) showed exacerbation of preexisting asthma as the primary mechanism of their occupational asthma. Unlike Tarlo and Broder,[9] we did not find atopic patients overrepresented in other categories such as sensitization. (Tarlo and Broder found a significant increase in the number of atopic patients among those with occupational asthma attributed to a specific sensitizer.) The difference in results could be due to a different patient classification systems: Tarlo and Broder did not include a patient group with asthma exacerbation. The difference may also reflect our caution in attributing new sensitization to our patients who had preexisting asthma.

The role of smoking also appears to vary depending on the agent and the mechanism. For instance, smoking is considered to be a risk factor for occupational asthma caused by high molecular weight compounds and those mediated by IgE mechanisms, but not by red cedar and toluene diisocyanate.[10] Overall, in our study, the number of smokers was fairly evenly distributed across the different mechanism groups; but of the 4 mechanism groups, the "undetermined" group contained the highest percentage of active smokers. It is likely that the diagnostic uncertainty in this subgroup is due to difficulties in attributing mechanisms to current or former smokers, since smoking may contribute to preexisting airway disease. Other studies have demonstrated a low proportion of smokers in patients with sensitization asthma[9,11] and a high proportion of smokers in patients with RADS.[9,12] The patients in our study had a smoking history similar to the patients in the other studies (49, or 69%, were past or current smokers); however, the number of current smokers was relatively low for sensitization asthma (13, or 18%), RADS (13, or 18%), and asthma exacerbation (15, or 21%).

The study by Tarlo and Broder[9] found a similar proportion of patients with sensitizer-induced occupational asthma as did we (33% and 39%, respectively). They reported a smaller number of patients with RADS, however; it appeared to be the mechanism at work in only 6% of their patients, compared to the 24% (17 patients) that we observed in our series.

Over half of our patients (38, or 53%) reported that symptoms improved on weekends and/or that symptoms resolved after time away from work (24, or 34%), occurred only at work (14, or 20%), or were worse at work (7, or 10%). Our case series cannot address the likelihood that those with such symptom patterns do have occupational asthma. Malo and colleagues,[13] however, have shown that these particular symptom patterns do not have a very high positive predictive value of sensitizer-induced asthma. Only 63% of patients in their study with these patterns had occupational asthma, according to objective testing.

There was a wide range in the reported elapsed time between first exposure to the offending agent and the onset of asthma. The eight patients reporting an immediate onset of symptoms were all determined to have RADS, consistent with the suspected mechanism of acute mucosal injury triggering nonspecific bronchial hyperresponsiveness. The remaining patients reported periods ranging from several weeks to 28 years, although most (55, or 77%) experienced the onset of asthma within five years of their first exposure to the agent. This degree of latency is consistent with other reports.[2,8] Most patients waited more than six months from the onset of symptoms to be evaluated at the clinic. This lapse of time made it difficult to evaluate the relevance of the patients' symptoms that existed at that

TABLE 4.—*Agents Associated With Patients With Occupational Asthma*

Agents	Number of Patients (%)
Fumes, General	9 (12.7)
Isocyanates	8 (11.3)
Solvents/Hydrocarbons	7 (9.9)
Red Cedar	6 (8.5)
Corrosive Agents	5 (7.0)
Crabs	3 (4.2)
Formaldehyde	3 (4.2)
Polyaromatic Hydrocarbons	3 (4.2)
Welding Fumes	3 (4.2)
Inorganic Dusts	3 (4.2)
Irritant Gases	3 (4.2)
Asbestos	3 (4.2)
Organic Dusts	2 (2.8)
Asphyxiant Gases	2 (2.8)
Paint Vapors	2 (2.8)
Epoxies	2 (2.8)
Plastic Vapors	1 (1.4)
Other Wood	1 (1.4)
Unknown	5 (7.0)
Total	71 (100.0)

TABLE 5.—*Patient Characteristics By Mechanism*

Characteristic	Total (n = 71)	RADS (n = 18) (25%)	Exacerbation (n = 19) (27%)	Sensitization (n = 27) (38%)	Undetermined (n=7) (10%)
Sex					
Male	53 (74.6)	12 (66.7)	11 (57.9)	25 (92.6)	5 (71.4)
Female	18 (25.4)	6 (33.3)	8 (42.1)	2 (7.4)	2 (28.6)
Race					
White	57 (80.3)	15 (83.3)	16 (84.2)	20 (74.1)	6 (85.7)
Black	6 (8.5)	1 (5.6)	1 (5.3)	3 (11.1)	1 (14.3)
Hispanic	2 (2.8)	1 (5.6)	0 (0.0)	1 (3.7)	0 (0.0)
Asian/Pacific Islander	2 (2.8)	0 (0.0)	0 (0.0)	2 (7.4)	0 (0.0)
Native American	4 (5.6)	1 (5.6)	2 (10.5)	1 (3.7)	0 (0.0)
Smoking					
Never-Smoker	23 (32.4)	9 (50.0)	5 (26.3)	9 (33.3)	0 (0.0)
Ex-Smoker	32 (45.1)	6 (33.3)	10 (52.6)	13 (48.1)	3 (42.9)
Current Smoker	16 (22.5)	3 (16.7)	4 (21.1)	5 (18.5)	4 (57.1)
ATOPY	24 (33.8)	2 (11.1)	14 (73.7)	6 (22.2)	2 (28.6)
AGE (mean ± SD)	44 ± 13	42 ± 12	48 ±	45 ± 11	42 ± 15

n = Total number of patients affected by specific mechanism.

point. In many cases, the patient had already left the work setting associated with the asthma, obscuring the work-relatedness of the asthma and affecting the type and usefulness of available diagnostic tests.

Only 19 (27%) of the patients who were diagnosed as having occupational asthma by clinic physicians in our study were receiving workers' compensation. Of those not receiving workers' compensation, 11% had received compensation benefits at some time in the past. (We did not evaluate the number of patients receiving compensation after diagnosis of occupational asthma at this clinic). These findings are consistent with others, and they demonstrate the large underestimation of the number of patients with occupational asthma when using data from compensation records only.[1,4] The inability to provide objective data on many patients exists for several reasons: they have left work; specific bronchoprovocation testing is unavailable; or their work-related asthma is not currently present. This may result in the misclassification of some patients as having occupational asthma or lead to the mistaken denial of insurance benefits.

We recorded a higher percentage of tests to assess change in pre- and post-bronchodilator FEV_1 than did Klees and colleagues [5] in another series of clinic patients (92% received spirometry in our study, versus their 67%). Our percentage of abnormal test results was also higher (65% in our study, compared to 44% in their study). In addition, the use of a 10% post-bronchodilator increase in FEV_1 as a criterion for asthma may be too sensitive and insufficiently specific, which may, in turn, result in an overestimation of the number of cases. This criterion has, however, been used by others in Klees's study.[5] We performed methacholine challenge and serial measurements of PEFR less often than Klees and colleagues, but we had a higher percentage of positive test results: 60% for both methacholine and PEFR values, versus 31% and 18%, respectively, reported by Klees and colleagues. The fewer number of PEFR measurements in our series may be explained either by differences in the time of evaluation of the patient relative to the onset of

TABLE 6.— *Most Common Agents By Mechanism Mechanisms*

Agents	Number of Patients (%)
RADS (n = 18)	
Fumes, General	4 (5.6)
Solvents/Hydrocarbons	2 (2.8)
Corrosive Agents	3 (4.2)
Formaldehyde	3 (4.2)
Irritant Gases	2 (2.8)
Other	4 (5.6)
Exacerbation (n = 19)	
Fumes, General	4 (5.6)
Solvents/Hydrocarbons	2 (2.8)
Corrosive Agents	2 (2.8)
Polyaromatic Hydrocarbons	2 (2.8)
Other	9 (12.7)
Sensitization (n = 27)	
Isocyanates	8 (11.3)
Solvents/Hydrocarbons	2 (2.8)
Red Cedar	6 (8.4)
Crab	3 (4.2)
Other	8 (11.3)
Undetermined (n = 7)	
Fumes, General	1 (1.4)
Solvents/Hydrocarbons	1 (1.4)
Dusts, General	2 (2.8)
Welding Fumes	1 (1.4)
Asbestos Dust	1 (1.4)
Plastic Vapors	1 (1.4)

TABLE 7.—*Symptoms In Patient With Occupational Asthma*

	Number of Patients (%)
Symptoms	
Dyspnea	63 (88.7)
Wheeze	44 (62.0)
Cough	42 (59.2)
Chest Tightness	21 (29.6)
Symptom(s) in Past	5 (7.0)
Symptom Pattern	
Occurring at Work Only	14 (19.7)
Worse on Monday	5 (7.0)
Improved on Weekday	38 (53.5)
Occurring only in the evening	5 (7.0)
Worse during work week	7 (9.9)
Resolves while away from work	24 (33.8)
Time Between Initial Exposure And Symptom Onset	
Immediate onset	8 (11.3)
< 1 month	22 (31.0)
≤ 1 year	38 (53.5)
≤ 5 years	55 (77.5)
> 5 years	16 (22.5)

TABLE 8.—*Pulmonary Function Testing (n = 71)*

Test	Test Performed	Test Positive
Bronchodilator Responsiveness	65 (91.5)	42 (64.6)
Methacholine Challenge	10 (14.1)	6 (60.0)
Specific Challenge	6 (8.5)	5 (83.3)
Spirometry Variability	36 (50.7)	33 (91.7)
Serial PEFRs	5 (7.0)	3 (60.0)
At Least One Objective Test	71 (100.0)	56 (78.9)

symptoms or by considering whether the patient was still exposed. These results, associated with a high rate of positivity, raise the question of whether some modalities such as PEFR testing and specific bronchoprovocation are underutilized in general clinical practice. Only 6 specific challenge tests were administered, and 4 (67%) were positive. So few tests were administered most likely because of the complexity of such testing.

Fifteen (21%) of our patients were not found to have at least one positive objective test. This fact reflects, in part, that the clinical evaluation of many patients took place a number of years after the exposure to the suspected agent had ended. Sixty-nine patients (97%) had symptoms that correlated with exposures at work. When the exposure in question occurred long before the evaluation, a symptom pattern was the evidence for work-relatedness most often available. This limitation occurs as the result of the lack of accessibility to different workplaces to evaluate specific agents or processes.

It is also possible that some of the patients were misclassified. Although there are many difficulties in obtaining objective data, the high proportion of cases without objective results—associating changes in lung function with exposure—argues for the more active pursuit of information such as spirometry, methacholine challenge, and serial peak flow monitoring. An alternative would be to develop highly specialized centers for the objective evaluation of occupational asthma; these would include staff and facilities available for specific bronchoprovocation. This option, however, is very expensive. Given the reliance on nonobjective measures in our series, we believe that an increased reliance on objective measures for diagnosis and attribution, when available and feasible, is warranted.

The definition of occupational asthma generates considerable controversy. Some exclude, in their definitions, causes of variable airflow limitation not due to sensitization, such as RADS. The definition of occupational asthma we used in our study includes both sensitization and RADS; we used both to establish the presence or absence of variable airflow limitation and its relation to workplace exposures, regardless of the pathophysiologic mechanism.

There are some limitations of the study presented here. One limitation is selection respondent bias: patients were included based on a physician's clinical assessment of the *likelihood* of the presence (either probable or definite) of occupational asthma. Additionally, the data we collected depended on the extent of inclusion of those data in the medical chart and relied on clinical impressions for some risk factors such as atopy, the association of symptoms and work, and likely mechanisms. Regarding the exacerbation of preexisting asthma, it is not possible to tell if asthma was present at the time of exposure or reappeared subsequently. Also, the OEMP at the University of Washington is a referral clinic; thus, the patient population does not represent workers in the community as they first present to a physician.

Despite the limitations of a descriptive study of a selected series of patients, the data we present do provide an overview of the wide range of agents, jobs, and industries associated with occupational asthma. The problem of occupational asthma is admittedly widespread and scattered across many disparate work settings. Data such as those presented here can educate physicians, employers, and employees about local and regional risks; they can provide a type of surveillance system, targeted at preventing future cases.

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