Reactive Airway Dysfunction Syndrome in Three Police Officers following a Roadside Chemical Spill*

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The reactive airway dysfunction syndrome (RADS) is a recently described syndrome in which bronchial hyperreactivity and asthmatic symptoms develop in previously healthy individuals after a single large exposure to an irritating gas, fume, or vapor. We report a cluster of three Philadelphia police officers who developed RADS after a common exposure to toxic fumes from a roadside truck accident. Results of initial pulmonary function testing were normal in all three, and methacholine challenge was required for diagnosis in two out of the three. This syndrome needs to be recognized by physicians dealing with environmental or industrial medicine as a potential cause of loss of work or inability to perform on the job. Also, there is a potential for multiple individuals to develop this syndrome from a single incident. *(Chest 1990; 98:928-29)*

Occupational exposure to dusts, gases, vapors, or fumes may result in pulmonary symptoms resembling that of asthma. Gandevia[1] classified occupational asthma into different types according to the underlying mechanism. These include reflex bronchoconstriction, pharmacologic bronchoconstriction, allergic bronchoconstriction, and inflammatory bronchoconstriction. These causes of occupational asthma and their mechanisms, prognoses, and management were reviewed by Chan-Yeung et al[2] and others.[3-6] Brooks and associates[7] recently described a new entity called reactive airway dysfunction syndrome (RADS). Patients suffering from this syndrome have a history of exposure to a high concentration of irritant gases, which results in persistent bronchial hyperreactivity and asthmatic symptoms. Brooks and associates[7] described ten cases of RADS, two of which resulted from the same exposure (spray paint). Boulet[8] later described four more cases of RADS. We also describe herein three cases of RADS, all developing from a common accidental exposure to several toxic chemicals.

Case Reports

All of our patients were members of the Philadelphia Police Department who were present at an accident in November 1985 involving a truck carrying chemicals which included sodium hydroxide, silicon tetrachloride, and trichlorosilane used for polymerized plastic coatings. Sodium hydroxide is a strong alkali corrosive which has been known to cause severe pneumonitis when inhaled.[9] Silicon tetrachloride and trichlorosilane are both extremely reactive and can emit a highly irritant vapor as they are decomposed by hydrolysis of atmospheric water, giving off hydrochloric acid. The hydrochloric acid in turn can have an irritant effect on the respiratory tract, causing laryngitis, bronchitis, and pulmonary edema.[10] This accident resulted in a large chemical spill and fire on a major highway.

Case 1

Patient 1 was a 38-year-old white male nonsmoker who was the first officer present at the scene. He had previously been healthy and had played semiprofessional baseball. Within hours of the exposure, he started to experience persistent coughing and headache. Within a few days, the patient was complaining of exertional dyspnea and wheezing which was especially severe in the cold. His wife also noted him to wheeze nocturnally. These symptoms became chronic.

Pulmonary function tests in 1987 showed an FVC of 5.77 L (112 percent of predicted), FEV_1 of 5.0 L/s (122 percent of predicted), and FEV_1/FVC% of 87 percent. The patient showed no desaturation during an exercise test. A methacholine inhalation challenge test was positive at 5 mg/ml. The patient's symptoms were controlled with theophylline and a β-adrenergic agonist inhaler; however, he remained dyspneic with minimal exertion, requiring retirement from the police force. Repeat PFTs in 1988 showed an FVC of 4.97 L (91 percent of predicted), FEV_1 of 2.67 L/s (61 percent of predicted), and FEV_1/FVC% of 54 percent.

Case 2

Patient 2 was a 36-year-old white man with a history of smoking one-half pack of cigarettes per day for ten years. He had no history of asthma or chronic pulmonary symptoms. He had a history of allergic symptoms in spring as a child but no recent allergic symptoms. Within an hour of being at the accident scene, the patient developed headache and dyspnea. His symptoms progressed in the next few weeks to the point of shortness of breath with minimal exertion, even with sexual activity. He also became dyspneic in cold air and wheezed when he exercised. His PFTs in 1986 showed an FVC of 4.22 L (83 percent of predicted), FEV_1 of 3.50 L/s (85 percent of predicted), and an FEV_1/FVC% of 83 percent). A methacholine challenge test was positive at a concentration of 5 mg/ml. The patient's symptoms improved with a regimen consisting of theophylline, β-adrenergic agonist, and cromolyn sodium inhalers, but he was still unable to return to his previous law enforcement occupation due to exertional dyspnea and wheezing.

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CASE 3

Patient 3 was a 45-year-old white man with a smoking history of 30 pack-years who had been well up until the accident. He had had no previous history of coughing, dyspnea on exertion, or wheezing induced by exercise or by cold weather. Within one hour of the exposure at the chemical spill, he developed a headache and persistent coughing. His symptoms continued to worsen, along with increasing sputum production and shortness of breath. The results of initial PFTs in 1986 were entirely normal, and the patient was started on theophylline and β-adrenergic agonist inhaler. His symptoms improved, but he remained dyspneic with exertion. A stress test done showed no evidence of myocardial ischemia. Repeat PFTs in 1987 revealed a decrease of the $FEV_1/FVC\%$ from a previous value of 86 percent to 78 percent. The patient was scheduled for a methacholine challenge test but never returned. An effort to contact him for follow-up was unsuccessful.

DISCUSSION

The RADS initially described by Brooks et al[7] discussed the appearance of asthma-like symptoms and an increase in bronchial hyperresponsiveness after a large exposure to irritants. The irritants described are nonspecific, and this syndrome has been described with sulfuric acid, household cleaners, smokes, and other such substances. In order to define this syndrome, the patient must have the absence of previous respiratory symptoms or disease plus a high level of exposure to a gas, fume, or vapor. The onset of asthma-like symptoms (cough, wheezing, shortness of breath) usually occurs within a few hours of exposure and then may become persistent. Pulmonary function studies may or may not show airflow obstruction but always show increased responsiveness to methacholine or another such agent which induces nonspecific airway hyperreactivity. In our experience of these cases and other isolated cases, standard PFTs are frequently normal, but abnormal methacholine sensitivity diagnoses the airway hyperreactivity.

Our cases describe a group of three patients with RADS initiated by a single toxic chemical spill from a roadside truck accident. Involved were police officers responding to the accident call. Because there were multiple chemicals involved in the spill, it would be very difficult to pick out the responsible agent or agents.

Charan and others[3] described pulmonary findings in three patients involved in a papermill accident in which they were exposed to high levels of SO_2. Harkonen and others[4] described seven patients who remained dyspneic on exertion four years after a high-dose exposure to SO_2 in a pyrite dust explosion. Six of the seven also complained of breathlessness provoked by cold weather. Four of the seven had a positive challenge test at the four-year follow-up. No history of pulmonary disease or complaints was given in their report.

Our cases are similar to those described by Harkonen et al[4] and the two patients (cases 3 and 4) described in the original report of Brooks et al[7] in that the cases all resulted from the same exposure. Two of our patients had documented nonspecific bronchial hyperreactivity to methacholine, while the third had symptoms suggestive of reactive airways and a reduction in $FEV_1/FVC\%$ with time. With the documented absence of preceding pulmonary complaints, our patients fit the criteria first described for RADS.

The mechanism responsible for this syndrome is still unclear. There was evidence of bronchial inflammation in two of the subjects of Brooks et al.[7] Postmortem examination of the subjects who died in the series described by Charan and associates[3] and by Harkonen and co-workers[4] showed hemorrhagic mucosa of the bronchi and denudation of the superficial columnar epithelium. It has been suggested that damage to the airway epithelium by inhaled chemicals, pollutants, or viral infections may be the mechanism for subsequent bronchial hyperreactivity.[11] Certainly, more investigation of the mechanism and more information concerning the epidemiology, natural history, and outcome of the syndrome are needed.

In our opinion, it is important for physicians practicing occupational or industrial medicine to be aware of the syndrome, as it may become an important cause of loss of work, inability to perform one's usual job, or early retirement. It should be emphasized that provocative testing (*ie*, methacholine inhalation challenge) is necessary to rule out this syndrome, as pulmonary function studies may be normal. These cases also point out the potential for multiple cases of RADS to occur from a single occupational or toxic exposure.

REFERENCES

1 Gandevia B. Occupational asthma. Med J Aust 1970; 2:332-35
2 Chan-Yeung M, Lam S. Occupational asthma. Am Rev Respir Dis 1986; 133:686-703
3 Charan NB, Myers CG, Lakshminarayan S, Spencer TM. Pulmonary injuries associated with acute sulfur dioxide inhalation. Am Rev Respir Dis 1970; 119:555-60
4 Harkonen H, Nordman H, Korhonen O, Winblad I. Long-term effects of exposure to sulfur dioxide. Am Rev Respir Dis 1983; 128:890-93
5 Flury K, Diones DE, Rodarte JR, Rodgers R. Airway obstruction due to inhalation of ammonia. Mayo Clin Proc 1983; 58:389-93
6 Whitener DR, Witener LM, Robertson KJ, Baxter CR, Pierce AK. Pulmonary function measurements in patients with thermal injury and smoke inhalation. Am Rev Respir Dis 1980; 122:731-39
7 Brooks SM, Weiss MA, Bernstein IL. Reactive airway dysfunction syndrome. Chest 1985; 88:376-84
8 Boulet LP. Increases in airway responsiveness following acute exposure to respiratory irritants. Chest 1988; 94:476-81
9 Occupational health guidelines for chemical hazards. US Department of Health and Human Services, 1978:1-5
10 Parmeggiani L, ed. Encyclopedia of occupational health and safety, 3rd ed. Geneva, Switzerland: ILO, 1983; 1:1084-85; 2:2035
11 Empey DW, Laitinen LA, Jacobs L, Gold WM, Nadel JA. Mechanisms of bronchial hyperreactivity in normal subjects after upper respiratory tract infection. Am Rev Respir Dis 1976; 113:131-39