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Reactive Airway Disease after Chlorine Gas Exposure

To the Editor:

We read with great interest in the September 1991 issue of *Chest* the case report by Moore and Sherman.¹ Chlorine gas exposure has multiple toxic effects on the respiratory system, including immediate airflow obstruction, ARDS, and death. The long-term effects can include abnormalities in gas transfer, restriction, and airflow obstruction. These generally resolve with time to a variable degree. Additionally, the severity and resolution of these effects appear to be related to the degree of exposure, premorbid pulmonary status, and the degree of hypoxemia on initial presentation.

In 1990, we reported two cases of reactive airway dysfunction syndrome (RADS) following chlorine exposure to chlorine gas.² Both patients had an asthmatic diathesis extending over time. One individual has had persistent asthmatic symptoms for the past six years; the symptoms have ameliorated with time, although she continues to be symptomatic. She is receiving minimal medication at the present time, compared with her immediate postexposure needs. Before exposure, she was neither a smoker nor an asthmatic. The other individual was also exposed to chlorine fumes. There was no history of preexposure asthma, and he had discontinued smoking several years prior to his exposure. Not only did this individual have asthmatic symptoms, which responded to bronchodilators, but he also had persistent hypoxemia, which has resolved over the years. His exercise stress test initially accentuated his hypoxemia, and his primary symptoms were those of dyspnea on exertion. His condition has ameliorated with time.

Thus, chlorine exposure can present with asthmatic symptoms and RADS, as described by Brooks et al.³ Our experience has been that the amount of asthma (diagnosed either by symptoms or by the medication needs) ameliorates with time. Hypoxemia with poor cardiopulmonary reserve was observed in one individual, and this also resolved with time.

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REFERENCES

- 1 Moore BB, Sherman M. Chronic reactive airway disease following acute chlorine gas exposure in an asymptomatic atopic patient. Chest 1991; 100:855-56
- 2 Demeter SL, Cordasco EM. Reactive airways dysfunction syndrome: a subset of occupational asthma. J Disability 1990; 1:23-39
- 3 Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposures. Chest 1985; 88:376-84

To the Editor:

We welcome the comments of Drs Demeter and Cordasco. We agree that our patient probably does have RADS, although he does

not fulfill all of the criteria set forth by Brooks et al.¹ Clearly the cases reported by Demeter and Cordasco represent persistent hyperactive airways disease after chlorine gas exposure.² Our patient differs from theirs in that his symptoms and level of disability have *not* ameliorated with time. On the contrary, six years after the exposure this patient still requires home oxygen therapy, oral corticosteroids at high doses, frequent use of beta-agonist inhalers, and frequent injections of subcutaneous epinephrine. In a recent telephone conversation, he informed us that his symptoms have actually worsened since our report was submitted. He is barely able to perform normal activities of daily living and reported that his physicians referred him to a transplant center for possible heart-lung transplantation.

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- 1 Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposures. Chest 1985; 88:376-84
- 2 Demeter SL, Cordasco EM. Reactive airways dysfunction syndrome: a subset of occupational asthma. J Disability 1990; 1:23-39

Pulmonary Lymphangioleiomyomatosis

To the Editor:

I read the very interesting case report by Huml et al,¹ which appeared in the December 1991 issue of *Chest*. I was curious to know whether the patient described so well by the authors had sarcoidosis or a localized sarcoidal reaction. It would be helpful in making this distinction if the authors were to state whether noncaseating granulomas were observed outside the thorax in the organs examined at autopsy. It would also be of interest to know something of the patient's ethnic background.

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REFERENCE

- 1 Huml JP, Borkgren MW, Henley LB, Fahey PJ. Pulmonary lymphangioleiomyomatosis associated with pulmonary parenchymal, hilar, and mediastinal noncaseating granulomas. Chest 1991; 100:1726-28

To the Editor:

In our case report, the absence of multiorgan involvement with noncaseating granulomas speaks against systemic sarcoidosis. No evidence of noncaseating granulomas was found outside the thorax. This raises the very interesting possibility that an immunologic mechanism may play a role in the etiology or that the presence of noncaseating granulomas may represent a localized immunologic reaction to lymphangioleiomyomatosis. Further study and clinical correlation are necessary to prove this hypothesis.

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