onset of their symptoms; therefore, we cannot rule out the possibility that desquamation of type II cells could have occurred during the early stages of the disease process.

The finding of large numbers of alveolar macrophages with normal in vitro antibacterial activity suggests there is a persistent stimulus for the migration of large numbers of macrophages into the alveolar spaces and/or a marked degree of proliferation of macrophages entering the alveoli. If DIP is a stage in the development of chronic diffuse interstitial fibrosis as has been suggested by some investigators,13 consideration of a stimulus for macrophage migration and/or proliferation might advance our understanding of the pathogenesis of interstitial pneumonitis and subsequent chronic fibrosis.

REFERENCES


Acute Hydrogen Selenide Inhalation*

A. Schecter, M.D.; W. Shanske; A. Stenzler; H. Quintilikan; and H. Steinberg, M.D.

Acute exposure to selenium hydrochloride resulted in severe dyspnea and a pneumomediastinum in a young healthy man. Pulmonary function tests revealed restrictive and obstructive airways disease. Although the patient's pulmonary function slowly improved, subsequent studies revealed persistent impairment. Therefore, exposure to selenium gas may result acutely in severe coughing and wheezing and may lead to irreversible obstructive lung disease.

Selenium is a nonmetallic element widely distributed throughout nature. Compounds of selenium have been used as glass decolorizers, in the production of red glass and glazes, in the manufacture of photoelectric cells, as an alloy of stainless steel and copper base alloys, and in the manufacture of paints, insecticides, and shampoo. Its potential as an industrial hazard was recognized by Hamilton1 and Dudley2 who recorded the symptoms of industrial selenosis to be gastrointestinal disturbances, garlicky odor of the breath, irritation of upper airway, metallic taste in the mouth, and pruritis. Our patient developed acute pulmonary symptoms after exposure to hydrogen selenide.

CASE REPORT

A 24-year-old white man accidentally inhaled hydrogen selenide while transferring this gas from one cylinder to another. He immediately experienced burning in his eyes and throat followed by coughing and wheezing. He was given oxygen and improved over a period of two hours. Eighteen hours later, however, because of recurrent cough and progressive dyspnea, he was hospitalized. His past history was negative for smoking, previous lung disease, allergies, or asthma. He had been employed for two years in the same chemical plant where he had been working with hydrogen selenide and arsine gases.

On physical examination, he was pale, diaphoretic, and in moderate respiratory distress. His vital signs were normal except for a respiratory rate of 40. No mucosal lesions were seen. Crepitation was felt over the right axilla and right anterior chest wall denoting subcutaneous emphysema. Expiratory wheezes were heard over both lung fields.

Laboratory data demonstrated a hemoglobin value of 17.4, and WBC of 18,500/μm with a normal differential. The remainder of the laboratory examination results were within normal limits. Arterial blood gases drawn on room air revealed a pH of 7.41, PCO2 of 39, PaO2 of 69 mm Hg, and a saturation of 92 percent. Chest x-ray film failed to demonstrate any pulmonary parenchymal infiltrates but did disclose a pneumomediastinum and subcutaneous emphysema (Fig

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CHEST, 77: 4, APRIL, 1980
Acute exposure to selenium compounds rarely results in respiratory symptoms. Sore throats, coryza, bronchitis, and pulmonary edema have been reported after exposure to two selenium gases, methyl selenide and selenium dioxide. Exposure to hydrogen selenide, as occurred in this case, has been reported infrequently. Buchan described principally gastrointestinal symptoms in five workers exposed to hydrogen selenide accidentally generated from etching ink. In guinea pigs exposed to hydrogen selenide, Dudley noted respiratory symptoms including coughing and difficulty breathing. Pathologic examination of these animals revealed fatty metamorphosis of the liver as well as diffuse pneumonitis.

Inhalation of hydrogen selenide by this patient resulted in acute bronchial irritation manifested by cough and reduction in expiratory flow rates. The pneumomediastinum that occurred was most likely a result of alveolar rupture following a sudden rise in intraalveolar pressure caused by the violent coughing and bronchospasm. The rapid improvement in pulmonary function demonstrates that the airways disease is somewhat reversible. However, persistent abnormalities in flows at 50 and 25 percent of the vital capacity even up to three years after exposure indicates that there may be an irreversible component to the obstructive airways disease. This phenomenon has been observed after exposure to other irritant gases such as ammonia and chlorine as well as to a gas analogous to hydrogen selenide, hydrogen sulfide.

Since pulmonary function studies had not been performed on this patient prior to the hydrogen selenide exposure, it would be difficult to exclude the possibility of prior airways disease. However, this seems unlikely in view of the absence of previous pulmonary disease and absence of a smoking history. Additionally, we have observed a further reduction in this patient’s mid- and terminal-expiratory flow rates when we compared the study performed three years after the exposure to that performed 30 days after the exposure. This may reflect the fact that he has continued to work with hydrogen selenide in addition to other irritant gases. Since selenium has many industrial uses, further studies are indicated in order to determine if chronic exposure to selenium gases results in obstructive airways disease.

References

3 Motley HL, Ellis MM, Ellis MD: Acute sore throats following exposure to selenium. JAMA 1718, 1937
5 Dudley HC: Effects of subacute exposure to hydrogen selenide. J Indust Hyg 1941; 23:470

Discussion

TABLE 1—Pulmonary Function Tests

<table>
<thead>
<tr>
<th></th>
<th>Predicted</th>
<th>Day 1*</th>
<th>Day 5</th>
<th>Day 30</th>
<th>Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forced vital capacity, L</td>
<td>4.65</td>
<td>2.00</td>
<td>2.50</td>
<td>3.80</td>
<td>4.20</td>
</tr>
<tr>
<td>Forced expiratory volume in one second, L</td>
<td>3.86</td>
<td>0.90</td>
<td>1.50</td>
<td>3.20</td>
<td>3.20</td>
</tr>
<tr>
<td>Forced expiratory volume in one second/forced vital capacity, percent</td>
<td>78</td>
<td>45</td>
<td>60</td>
<td>84</td>
<td>76</td>
</tr>
<tr>
<td>Peak flow, L/sec</td>
<td>9.15</td>
<td>2.00</td>
<td>5.20</td>
<td>8.20</td>
<td>8.50</td>
</tr>
<tr>
<td>V at 50 percent vital capacity, L/sec</td>
<td>5.98</td>
<td>0.40</td>
<td>1.00</td>
<td>3.80</td>
<td>3.00</td>
</tr>
<tr>
<td>V at 25 percent vital capacity, L/sec</td>
<td>3.35</td>
<td>0.20</td>
<td>0.40</td>
<td>1.80</td>
<td>1.20</td>
</tr>
<tr>
<td>Residual volume, L</td>
<td>1.49</td>
<td>1.33</td>
<td>. . .</td>
<td>1.20</td>
<td>1.14</td>
</tr>
<tr>
<td>Total lung capacity, L</td>
<td>6.14</td>
<td>3.33</td>
<td>. . .</td>
<td>4.91</td>
<td>5.04</td>
</tr>
</tbody>
</table>

*Pulmonary function unchanged after bronchodilators