Acute Lung Disease After Exposure to Fly Ash*

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A 48-year-old man with no history of pulmonary disease developed acute lung disease after the intensive exposure of fly ash. He subsequently had progressive worsening of shortness of breath and hypoxemia to the point of requiring mechanical ventilation. Fly ash is a compound consisting of silicon dioxide and various other substances and is used in industrial settings to generate electricity. Exposure to fly ash may cause irritation to the mucous membrane of the respiratory tract and even pulmonary fibrosis in humans. To our knowledge, this is the first case report described in the medical literature of acute lung disease developing after fly ash exposure.

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Acute lung disease may occur in persons exposed to heavy concentrations of respirable silica present in fly ash. Fly ash is a compound consisting of silicon dioxide and other mineral substances (Table 1) that are end products of cogeneration plants. Fly ash is used in thermal power stations to generate electricity. It has been associated with a different pattern of tissue injury, including development of silicotic granuloma in lungs after exposure to coal fly ash in experimental animal studies. We report the unusual case of a 48-year-old man who was exposed to fly ash and developed acute lung disease within 2 weeks of the exposure. To our knowledge, this is the first case of human fly ash exposure as an occupational hazard reported in the medical literature.

CASE REPORT

A 48-year-old man was admitted to the hospital for progressive worsening of cough and shortness of breath for 2 weeks, after he was exposed to a massive dose of fly ash. He has a 50-pack-year smoking history and had an occasional cough due to the long-term smoking. However, after he was exposed to the fly ash heavily in an industrial accident, he developed a persistent and paroxysmal dry cough with worsening dyspnea. He denied fever,

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Table 1—Chemical (%) and Physical Characteristics of Fly Ash

<table>
<thead>
<tr>
<th>Chemical compositions</th>
<th>%</th>
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<tbody>
<tr>
<td>Silicon dioxide</td>
<td>36.00</td>
</tr>
<tr>
<td>Aluminum oxide</td>
<td>23.28</td>
</tr>
<tr>
<td>Iron oxide</td>
<td>4.80</td>
</tr>
<tr>
<td>Calcium oxide</td>
<td>26.12</td>
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<tr>
<td>Magnesium oxide</td>
<td>3.36</td>
</tr>
<tr>
<td>Sulfur trioxide</td>
<td>6.31</td>
</tr>
<tr>
<td>Moisture content</td>
<td>0.12</td>
</tr>
<tr>
<td>Loss on ignition</td>
<td>2.97</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical properties</th>
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<tbody>
<tr>
<td>pH</td>
<td>12.15</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>2.70</td>
</tr>
</tbody>
</table>

References

3 Vidyarthi SC. Diffuse miliary granulomatosis of the lungs due to aspirated vegetable cells. Arch Pathol 1967; 83:215-18
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chill, sweating, nausea, vomiting, abdominal pain, or any other constitutional symptoms.

Medical history was noncontributory except for an appendectomy. Patient denied intravenous drug abuse, homosexual activity, or blood transfusions. He was a truck driver and was responsible for trucking loads of fly ash to dump sites occasionally for 2 years. However, he denies any prior exposure to fly ash or dust. Family history revealed no history of asthma or any other respiratory disease.

Physical examination revealed only mild, diffuse inspiratory crackles with some expiratory wheezing throughout the lung fields. There was neither lymphadenopathy nor cutaneous lesions. The BP was 130/70 mm Hg and the heart rate was regular at the rate of 108 beats/min. The respiratory rate was 40 breaths/min. Results of the remainder of the physical examination were normal. After the patient was admitted to the ICU, his symptoms continued to worsen with severe dyspnea, tachypnea, and hypoxemia, requiring endotracheal intubation and mechanical ventilation.

Laboratory findings, including serum chemistry studies and complement levels, were all nondiagnostic. Total blood cell counts revealed mild leukocytosis with 5 percent bands. Blood gas revealed a pH of 7.45, Pco2 of 50 mm Hg, Po2 of 53 mm Hg, and HCO3 of 34, with 89 percent oxygen saturation at room air.

Studies for Legionella, Mycoplasma, Coccidioides, Histoplasma, Brucella, and other fungal infections were negative, as was a tuberculin skin test.

The chest radiograph revealed diffuse infiltrates with small nodular densities or rounded opacities of p and q size (p: 0 to 1.5 mm, q: 1.5 to 3.0 mm) and right hilar lymphadenopathy (Fig 1). In addition, the base of the right lung contained small pleural effusion. Chest computed tomographic scan findings revealed small rounded opacities (less than 1.5 mm in diameter) and patchy air-space opacification (Fig 2). The patient underwent bronchoscopy with transbronchial lung biopsy.

Microscopic examination of the biopsy specimens revealed nonspecific interstitial inflammation and fibrosis without granulomas. Routine microbiologic studies, acid-fast studies, and fungal studies of bronchoalveolar lavage failed to disclose any infecting microorganisms. The patient initially refused an open lung biopsy; however, a few weeks later, he underwent a diagnostic open lung biopsy; these biopsy specimens showed classic silicotic hyaline nodule associated with adjoining parenchymal scarring and limited lymphoid exudate (Fig 3).

The patient was started on a regimen of high-dose corticosteroids intravenously. He also received intravenous antibiotics empirically, including erythromycin. On these regimens, the patient improved and was extubated successfully. His PaO2 on room air returned to 82 mm Hg. He was discharged home after 1 month of hospitalization.

DISCUSSION

Fly ash is considered as one of the common environmental pollutants, where coal is burnt in thermal power stations to generate electricity. It consists of silicon dioxide and various other substances, and can be obtained from electrostatic precipitator of a thermal power station in which fly ash contains more than 90 percent of particles less than 5 μm in diameter.

The primary routes of fly ash entry to the human body are through the respiratory system (inhalation), eyes, and skin. Short-term exposure may result in irritation to eyes,
skin, or the mucous membrane of the respiratory tract. Persistent exposure to airborne dust may cause chronic bronchitis and pulmonary fibrosis.

Exposure to fly ash and dusts rich in free silica may occur in and around a number of industrial settings in many countries—among workers handling substances containing high concentrations of free silica such as cement, concrete, and sand for use in road and building construction. A small dose of fly ash is readily deposited in terminal airways and alveoli, engulfed by alveolar macrophages, and retained in the lungs for long periods. It is well suited to penetrate and reach even the peripheral portions of airways due to its fine size of dust particles. The experimental study of Kaw and associates has shown that pretreatment of rats with fly ash produces the development of silicotic granuloma with reticulin and collagen fiber formation in lungs, and it demonstrated that fly ash exposure can significantly modify the development of a silicotic pulmonary reaction.

Development of silicosis is well known to be a serious health hazard whose parameters have been reasonably well established in men who were exposed to silica dust by engaging in a variety of occupations involving generation of airborne quartz particles encountered in the workplace. Sporadic outbreaks still occur, most frequently in sandblasters and tunnel high-power drillers of tunnel rock. It occurs within 5 years of the onset of exposure. It can also occur as short as 6 to 8 months. The disease is often rapidly progressive, with death caused by respiratory failure.

Our patient was exposed to fly ash and dusts rich in free silica only about 2 weeks ago prior to hospital admission. His clinical course was marked by a progressive dyspnea, hypoxemia, and paroxysmal cough without constitutional symptoms. However, he did respond well to the corticosteroid therapy and was discharged home subsequently.

It was initially difficult to ascertain only from this patient’s history, physical examination, and noninvasive studies whether he had acute silicosis before he underwent open lung biopsy a few weeks later. Our patient had a relatively unusual presentation, because the time from exposure to the onset is against the diagnosis of acute silicosis, which usually occurs within a few months. Also, there is no previously reported case documenting acute silicosis after fly ash exposure to humans. Although there is, in contrast, some disagreement about making a diagnosis of silicosis, it appears that our patient had an acute silicosis on the basis of findings on clinical course, sequence of events, radiographic presentations, and the histologic findings of the silicotic hyaline granuloma in the lungs.

There is no specific treatment available at present for this disease against the exposure of fly ash. In the interim, supportive care remains the basis of treatment for this disease with a trial of corticosteroid therapy. Efforts should be focused on protecting the workers from exposure to fly ash by using protective devices. Also, the information of health hazards should cover the possible complications of acute lung disease, leading to hypoxemia and a clinical picture consistent with acute silicosis.

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A New Complication Related to Laser Bronchoscopy in a Single Lung Transplant Recipient*


We report a nearly complete obstruction of the left mainstem bronchus by a fibrinomyxoid plaque about 12 h after laser resection of scar/granulation tissue at a left bronchial anastomosis 27 days after a left single lung transplant. The formation of this plaque was associated with respiratory failure. The plaque was removed by grasping the plaque with biopsy forceps inserted through a fiberoptic bronchoscope that was placed into the left mainstem bronchus via an endotracheal tube while the patient was receiving manual ventilation under general anesthesia. The respiratory failure resolved with removal of the plaque. To our knowledge, this is a complication that has not been reported previously.

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