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.4 A small dose of fly ash is readily deposited in terminal airways and alveoli, engulfed by alveolar macrophages,5 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.5 The experimental study of Kaw and associates6 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 air-borne quartz particles encountered in the workplace.6-8 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.9 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.9 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,10,11 response to corticosteroid therapy,12,13 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 bronchoscopy 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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The Nd:YAG (neodymium: yttrium-aluminum-garnet) laser has been used safely and effectively at our institution to treat bronchial anastomotic stenosis due to accumulation of scar and/or granulation tissue following lung transplantation in more than 20 recipients. We describe a previously unreported complication of this procedure in a lung transplant recipient.

**CASE REPORT**

A 40-year-old woman received a left single lung transplant due to end-stage lung disease from sarcoidosis on July 21, 1991. The allograft eventually failed due to obliterative bronchiolitis and a second left single lung transplant was performed on January 31, 1990. The patient was extubated less than 24 h after the retransplant procedure. Because of left lower lobe volume loss and infiltrate on the fourth postoperative day, fiberoptic bronchoscopy was performed that revealed 30 percent narrowing of the left mainstem bronchus (LMB) anastomosis secondary to inflammation and floroscopy of the chest revealed decreased movement of the left hemidiaphragm.

On the 27th postoperative day, surveillance fiberoptic bronchoscopy performed transnasally with topical anesthesia and systemic sedation revealed an approximately 80 percent occlusion of the LMB anastomosis by scar and granulation tissue. After clearing secretions by suctioning to identify the perimeter of the stenosis, noncontact laser ablation was performed by application of 2,328 J of YAG laser energy delivered in aliquots of 60 W for a duration of 0.5 s. This increased the diameter of the bronchial lumen from approximately 20 percent to 85 percent of normal size. The duration of the procedure was 56 min and there was no bleeding or any other complications. The bronchial mucosa distal to the lasered area appeared normal and no secretions were present distally. Arterial blood gases before the laser procedure were pH of 7.38, PCO2 of 42 mm Hg, and PO2 of 116 mm Hg on oxygen supplements of 6 L/min by nasal cannula. Arterial blood gases improved after the laser procedure with a pH of 7.45, PCO2 of 39 mm Hg, and PO2 of 74 mm Hg on room air. Approximately 12 h after the laser procedure, the patient developed wheezing and shortness of breath that improved minimally with aerosol β-agonists. Arterial blood gases revealed respiratory failure and hypoxemia with a pH of 7.34, PCO2 of 55 mm Hg, and PO2 of 42 mm Hg while breathing 100 percent oxygen via a face mask. Emergency intubation and fiberoptic bronchoscopy were performed that revealed nearly complete occlusion of the LMB at the anastomosis by a plaque of gray-white material overlying the area that had been photocoagulated with the YAG laser. The plaque was fairly solid and was removed in one piece using alligator biopsy forceps inserted through the bronchoscope. Ventilation and oxygenation improved immediately allowing for extubation within an hour after this procedure.

The plaque was gray-white with a smooth, slightly irregular surface (Fig 1). Histologic examination revealed a loose fibrin mesh with myxoid appearance infiltrated by acute inflammatory cells (Fig 2). The appearance of this fibrinomyxoid plaque was uniform throughout and no areas of organization suggesting chronicity were seen.

**DISCUSSION**

The Nd:YAG laser was introduced for use in bronchoscopy in 1981 primarily as therapy to relieve obstruction of the trachea and major bronchi. Clinically significant stenosis at the bronchial anastomosis occurs in approximately 10 percent of our lung transplant recipients. More than 20 of our lung transplant recipients have been treated with Nd:YAG laser resection for bronchial stenosis, always with immediate resolution of the offending airway obstruction. This is our first case of an acute occlusion of the airway by a fibrinomyxoid plaque following laser ablation of scar tissue at a bronchial anastomosis.

Complications after laser therapy include hemorrhage, pneumothorax, perforation, and hypoxemia. The complication rate has been 9 percent with bleeding and pneumothorax being the most frequent problems. In our lung transplant recipients, recurrent stenosis due to regrowth of scar and/or granulation tissue has been the most frequent complication of these laser procedures, but this complication typically occurs 3 to 6 weeks after laser therapy.

In this case, the patient developed respiratory failure within 12 h after correction of the bronchial stenosis. Removal of a fibrinomyxoid plaque that had occluded the bronchial lumen resulted in prompt relief of the respiratory failure. This material was localized and loosely adherent to the area that had received laser therapy. Once the plaque was removed, a patent airway was visualized. No secretions were seen distal to the anastomosis and the upper and lower lobe bronchi were normal in appearance. Histopathologic evaluation of the plaque was consistent with the acute development of this process.

The pathogenetic basis for the formation of this plaque is probably multifactorial. First, the thermal laser injury to the bronchial mucosa led to the exudation of serum and inflammatory cells into the airway lumen. This reaction is...
Pericarditis as the Initial Manifestation of Malignant Thymoma*

Disappearance of Pericardial Effusion With Corticosteroid Therapy

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A patient with malignant thymoma is described in whom the initial manifestation was pericarditis; cardiac tamponade was relieved by corticosteroid therapy.

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Thymoma is the most common primary tumor of the anterosuperior mediastinum. Patients with malignant thymoma may develop the complication of pericardial metastasis and effusion through local invasion. In this report, a patient with malignant thymoma is described in whom the initial manifestation was pericarditis, and cardiac tamponade was relieved by corticosteroid therapy.

CASE REPORT

A 53-year-old man was admitted to our hospital with complaints of precordial dull pain, weight loss, and dry cough. Physical examination showed temperature up to 38°C and pericardial friction rub. His BP measured 96/62 mm Hg. Laboratory data were nonrevealing except for iron deficiency anemia and elevated inflammatory tests (erythrocyte sedimentation rate 105 mm/h, C-reactive protein 13 mg/dl, WBC count, and differentials normal). The patient's ECG showed low voltage in limb leads. Chest x-ray film (Fig 1) showed only moderate cardiomegaly, but no abnormal mediastinal shadow was noted. Moderate pericardial fluid was recognized by echocardiography. The fluid was bloody and contained 5 mg/dl of protein and 40 IU/L of adenosine deaminase. In the fluid, neither malignant cells nor mycobacteria was proved. A malignant source of the pericarditis was investigated but without results. Because of continuous fever and asthenia, empiric antituberculosis chemotherapy was started with rifampicin, isoniazid, and streptomycin, but the effusion increased. The patient became dyspneic with pulses paradoxus, and 400 ml of the fluid was evacuated to provide temporary relief. Then 125 mg of methylprednisolone (divided) was given for 4 days in addition to 40 mg of prednisolone by mouth to relieve the pericarditis; this treatment resulted in rapid relief of dyspnea and in 2 weeks the effusion was barely recognized. Prednisolone was tapered to 10 mg in 3 weeks and this dose was continued. Eight weeks after admission, a chest x-ray film revealed an upper mediastinal mass, and computed tomography revealed that the mass encased large vessels. At surgery, the tumor was seen to originate probably common to any laser thermal injury. Why this material organized itself into an occluding plaque is less certain. Because the exudated serum and inflammatory cells were able to organize into a solid mass implies that stasis of this material was allowed to occur. While mucociliary clearance is impaired in all lung transplant recipients at all times after lung transplantation, this mechanism alone cannot explain the formation of this occluding plaque because we should have observed such plaque formation in other lung transplant recipients on whom we have performed this procedure. Our best guess is that the combination of impaired mucociliary clearance, decreased cough due to decreased contractility of the left hemidiaphragm, and possibly a more exuberant or differently composed exudation of serum proteins and inflammatory cells due to the underlying sarcoidosis all conspired to the formation of this plaque. To our knowledge, this complication in laser bronchoscopy has not been reported previously. Since use of therapeutic laser bronchoscopy for bronchial stenosis has increased, clinicians should be aware of this potential complication.

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Figure 1. Chest roentgenogram at admission showed only cardiomegaly; no mediastinal mass was seen.