TCI-induced lung disease may vary from a relatively mild and completely reversible interstitial lung disease (case 1) to a severe form of bronchiolitis obliterans (BO) causing prolonged and complicated respiratory failure and leaving the patient severely disabled (case 2). This varied clinical course is most probably related primarily to the concentration of inspired toxic fume and to the length of the exposure period. Bronchiolitis obliterans is an uncommon disease characterized pathologically by an obstruction of the airway lumen beyond the terminal bronchioles by a cellular infiltrate or fibrosis and is associated with varied etiologic factors. Toxic-fume inhalation is a well-recognized cause of BO, and reports of SO2 or CHI-related BO have been published. However, the development of BO following TCI exposure has not been reported previously, to our knowledge, although Ducatman et al. describe one patient who died of fulminating pulmonary edema after exposure to TCI.

The triphasic evolution of the BO noted in case 2 is typical of toxic fume inhalation in both experimental and clinical settings. Our patient was almost asymptomatic immediately after the injury, although some patients can present with pulmonary edema. The acute phase was followed by a latent, clinically asymptomatic phase of over two weeks followed by a full-blown acute respiratory failure. The finding of wheezing unresponsive to bronchodilators is another feature of bronchospasm induced by fume inhalation. An initially normal chest radiograph can be encountered (as in this case) and may be misleading since acute respiratory failure may still develop following a latent period. Subsequently, the patient's hyperinflation of the lung is frequently seen in BO, and the lung function tests (showing a combined obstructive + restrictive pattern) and ABG values (showing mild hypoxia and hypocarbia before the complications described above) are also typical. Another common finding in fume-induced BO is the very rapid evolution from the acute phase to chronic lung disease, and our patient had the same evolution. Bronchiectasis is a well-recognized complication of SO2 inhalation; however, to our knowledge, the appearance of spontaneous pneumothorax, lung bullae, and BPF have not been reported previously in fume-induced BO.

The prognosis of fume-induced BO is generally poor, although in some cases, lung function may slowly improve after two to three years. The first of our patients apparently responded well to steroid therapy, but our second patient developed severe BO and remains a respiratory cripple although he was immediately treated with steroids to minimize lung damage. Generally, steroids are less useful in fume-induced BO (except for NO2 inhalation) and steroid therapy should possibly be stopped if no improvement is seen during the first days because this treatment may increase the risk of lung infection in the presence of a denuded lung epithelium.

In conclusion, our two cases demonstrate the spectrum of TCI-induced lung injury ranging from a relatively mild and reversible interstitial disease to progressive severe respiratory failure. To our knowledge, it is also the first report of BO induced by TCI, an occupational hazard that should be better recognized in the future.

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Bronchopulmonary Baricylial Angiometasis*

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A man with prior AIDS developed acute febrile interstitial pneumonitis, hilar and paratracheal adenopathy, and bronchial polyps. The polyps were histologically typical for bacillary angiomatosis and complete symptomatic and radiographic response to oral clarithromycin was seen. The clinical presentation of bacillary angiomatosis includes pulmonary disease and in particular bronchial polyps; clarithromycin is an effective oral antibiotic.

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Since the first description in 1983 of a novel subcutaneous infection in an AIDS patient, the clinical spectrum of disease produced by the rickettsial agent of bacillary angiomatosis (BA), *Bocchulima henselae*, continues to expand. Recent reports in immunocompromised patients include subcutaneous masses with contiguous erosive bone lesions, disseminated with visceral abscess formation and bone marrow infiltration, intracerebral disease, and febrile bacteremia. In the immunocompetent patient, only febrile bacteremia is described. A patient with AIDS had interstitial lung disease, concomitant hilar adenopathy, and unusual bronchial polyps histologically demonstrating bacillary angiomatosis; he responded to clarithromycin.

**Case Report**

A 42-year-old, human immunodeficiency virus (HIV)-positive, male homosexual with prior *Pneumocystis carinii* pneumonia and extensive long-standing cutaneous Kaposi's sarcoma, with an absolute CD4 count of 8 cells per cubic millimeter, reported a 1-month history of fever accompanied by recurrent shaking chills, profound nocturnal sweating, 7-kg weight loss, intense, nonproductive cough that occasionally induced emesis, and positional right-sided chest pain that was exacerbated with lying supine; he denied dyspnea with exertion. His physical examination showed normal vital signs without respiratory distress. He had scattered lesions compatible with Kaposi's sarcoma over his face, trunk, and extremities, with a single lesion on his hard palate. The lungs were clear to auscultation, the heart tones were normal, and there was no palpable organomegaly on abdominal examination. Findings from the remainder of his physical examination were unremarkable. Aside from a hematocrit of 32.7 percent and a lactate dehydrogenase value of 1,335 U/L (upper normal, 618), results of the screening laboratory examination were normal. Chest radiograph was obtained (Fig 1) and showed prominent interstitial markings and mediastinal adenopathy. The adenopathy was confirmed by computed tomographic scan of the chest.

Bronchoscopy was performed, and a solitary plaque of presumptive Kaposi's sarcoma was seen at the right upper lobe carina. Multiple pale white friable polypoid mucosal lesions measuring 3 to 5 mm in diameter were seen in the right lower lobe bronchi and the anterior and lateral basilar segmental bronchi. Stains of bronchial washings were negative for mycobacteria, fungi, bacterial pathogens, and *P carinii*. Two days later, the patient became profoundly dyspneic, with a resting oxygen saturation of 85 percent. Repeat chest radiograph (not shown) showed no pneumothorax, stable interstitial disease, and adenopathy with new bilateral small pleural effusions and a small right lower lobe infiltrate. Treatment with clarithromycin, 500 mg orally every 12 h, and ethambutol, 15 mg/kg orally once daily, was begun pending results of mycobacteria blood cultures and bronchial wash cultures.

The polypoid lesions (Fig 2) showed histologic features typical of BA (not shown). Groups of proliferating capillary-sized vessels with plump endothelial cells were present beneath a metaplastic squamous mucosa. There was a mixed inflammatory cell infiltrate, including neutrophils. Warthin Starry stain demonstrated numerous bacillary structures associated with the tissue reaction.

One month later, he had complete resolution of all symptoms, a normal chest radiograph, and room-air oxygen saturation of 95 percent at rest. He had no adverse effects from the clarithromycin. All cultures were negative, and ethambutol therapy was discontinued; he completed a two-month course of clarithromycin. There was no regression of the cutaneous Kaposi's sarcoma lesions.

**Comment**

Interstitial lung disease is a common clinical problem in the HIV-infected patient. The infectious differential diagnosis is extremely broad and includes *P carinii*, *Cryptococcus neoformans*, and dimorphic fungi, including *Histoplasma capsulatum* and *Coccidioides immitis*, Mycobacteria species, *Toxoplasma gondii*, and, rarely, viruses or pyogenic bacteria. Hilar adenopathy generally limits the infectious considerations to fungal or mycobacterial diseases, with Kaposi's sarcoma the predominant neoplastic cause. Because of this patient's extensive cutaneous disease, we were suspicious that the hypoxia and radiographic findings were those of pulmonary Kaposi's sarcoma; the fever was thought to be secondary to disseminated *Mycobacterium avium* complex, which initiated empiric therapy with clarithromycin and ethambutol while awaiting blood culture and bronchoscopy results.

Histologic study demonstrating BA, the absence of alter-
native pathogens on bronchoalveolar lavage, as well as the complete clinical and radiographic response to directed antimicrobial therapy support the pathogenicity of R. henselae in this patient. To our knowledge, bronchial polyps and interstitial lung disease with associated hilar adenopathy have not been associated previously with BA; however, this clinical picture has been reported with pulmonary Mycobacterium avium complex in AIDS patients.

The agent of BA has been recently identified as R. henselae, a rickettsial organism. Tick bites have been epidemiologically linked to transmission of infection, yet this association remains unproved. Antibiotics reported effective for BA include erythromycin, doxycycline, rifampin, and chloramphenicol. Because of the potential for intracellular localization of R. henselae coupled with slow growth characteristics, protracted administration of antibiotics capable of intracellular penetration has been recommended. Clarithromycin, a 6-0-methyl derivative of erythromycin that demonstrates unusually high tissue and polymorphonuclear cell penetration, may be a particularly active antibiotic in the treatment of BA.

This case serves to expand the clinical presentation of BA as well as the differential diagnosis of interstitial lung disease with hilar adenopathy and bronchial polyps in HIV-infected patients. Effective antimicrobial regimens should include clarithromycin.

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Inclusion Body Myositis as a Cause of Respiratory Failure

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Inclusion body myositis (IBM) is a slowly progressive myopathy that has not been reported to affect respiratory muscles. It is often refractory to treatment and a muscle biopsy specimen is necessary for the diagnosis. This is a report of a patient with IBM who quickly progressed to respiratory muscle failure requiring intubation. (Chest 1993; 104:975-77)

IBM = inclusion body myositis

Inclusion body myositis (IBM) is a form of inflammatory myopathy that is characterized by a protracted course, involvement of distal muscles, and unresponsiveness to steroid therapy. The most severely affected muscles are those of the limbs and, to our knowledge, there are no reports of respiratory muscle involvement. Extramuscular involvement of this disease has been described infrequently and usually involves the cardiovascular and the gastrointestinal systems. We describe a patient with respiratory failure secondary to IBM.

CASE REPORT

A 69-year-old woman was admitted to the ICU with respiratory failure. She had a 10-day history of progressive shortness of breath, cough, lethargy, and weakness. There was no history of fever, sweats, or chills. The patient was hospitalized in 1987 with a similar presentation. At that time, she had complained of a 2-week history of progressive dyspnea and slowly worsening limb weakness for the preceding five years. The electromyogram (EMG) at that time was compatible with a myopathy; however, the creatine kinase (CK) and liver function test results were normal. She was found to have an elevated partial thromboplastin time (PTT) that was shown to be due to a lupus anticoagulant. Her antinuclear antibody (ANA) titer was 1:320. Her platelet count was normal as were the results of her thyroid function tests. A Tension test was negative. The chest radiograph was normal. She required mechanical ventilation and was treated as having mixed connective tissue disease and received large doses of intravenous methylprednisolone sodium succinate (Solu-Medrol). Her hospital course was complicated by multiple infections that were, in part, attributed to the large doses of steroids. As there was no clinical improvement, the steroid therapy was discontinued. The patient underwent a muscle biopsy from the left quadriiceps that showed isolated myofiber degeneration, atrophic fibers, and no inflammatory infiltrates. She was discharged from the hospital 1 year later with a diagnosis of idiopathic myopathy. She remained well until the present hospital admission without worsening of the myopathy or further respiratory problems.

The physical examination showed the patient to be in moderate respiratory distress. She had petechiae on both calves and poor air entry in the lungs, which were otherwise clear. The neurologic examination showed her to be alert and oriented, with distal muscle...

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