fulminant pulmonary hypertension in an HIV-seropositive man. His accelerated clinical course is clearly consistent with previously reported cases of pulmonary hypertension associated with HIV. However, this patient's striking pulmonary function derangements have not been described previously and suggest in vivo parenchymal changes not confined to the vascular bed.

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Carmustine Toxicity Presenting as a Lobar Infiltrate*

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Carmustine is a chemotherapeutic agent frequently employed in the treatment of malignant brain tumors. The side effect of pulmonary fibrosis occurs in 20 to 30 percent of patients receiving this drug. Herein we report a case of presumed carmustine-induced pulmonary fibrosis occurring with an unusual lobar distribution.

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Carmustine is a nitrosourea used to treat a variety of malignant tumors. A well-documented adverse effect of this drug is pulmonary fibrosis, occurring with an incidence of 20 to 30 percent. This is traditionally characterized on chest radiography as bilateral diffuse or patchy interstitial infiltrates.1 In our search of the literature, we were unable to find any reported cases of carmustine-induced pulmonary fibrosis that presented as a lobar infiltrate. Herein we report the first documented case (to our knowledge) of carmustine-induced pulmonary fibrosis presenting as a lobar infiltrate.

CASE REPORT

A 44-year-old woman who was previously in good health presented to our hospital emergency department with severe paresthesias of the left upper extremity in March 1989. A subsequent computed tomographic examination of the brain with contrast revealed a nonenhancing mass in the right temporal parietal area. Biopsy specimen of the mass revealed a grade 3 anaplastic glioma. She was subsequently treated with a combination regimen of irradiation to the brain (6,020 cGy in 35 fractions) and chemotherapy. Her chemotherapy regimen consisted of carmustine, initially 60 mg/m² of body surface area with subsequent doses adjusted based on hematologic toxicity. The total cumulative dose was 2,039 mg/m² of body surface area given in 12 treatments from June 1989 to July 1990. Six months later, she began to experience a nonproductive cough and weakness. There was no history of recent fever, chills, night sweats, or recent exposure to tuberculosis. She had smoked cigarettes in the past but had quit ten years previously. There was no history of any preexisting lung disease and her only medication was phenytoin. Physical examination revealed an afibrile, nontachypneic woman in no distress. Her lungs were clear to auscultation and there was no cyanosis, clubbing, or edema of her extremities. Results of the rest of her examination were normal. Pulmonary function testing revealed a severe restrictive pattern (FVC = 36 percent of predicted). Her chest radiograph (Fig 1) showed a left upper lobe infiltrate with volume loss in the left upper lobe. A subsequent computed tomographic examination of her chest revealed pleural thickening and volume loss of her left upper lobe. Interestingly, a chest radiograph taken 12 months earlier (Fig 2), when the patient was involved in an automobile accident, showed only bibasilar atelectasis thought to be secondary to splinting from abdominal pain. Diagnostic evaluation included a FPD skin test, an angiotensin converting enzyme level, antinuclear antibody and rheumatoid factor titers, histoplasmosis complement fixation titer, fungal immunodiffusion, and blood cultures, all of which were negative. A fiberoptic bronchoscopy with transbronchial biopsy of the left upper lobe was done. Smears and cultures of the bronchial washings failed to demonstrate any infectious organisms. There were no malignant cells seen in the bronchial washings. Pathologic

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findings included alveolar cell dysplasia and interstitial fibrosis with no evidence of malignancy or infection.

She was subsequently started on a regimen of prednisone (40 mg/d), which is gradually being tapered. Results of her pulmonary function tests have improved minimally while receiving steroids; however, the patient has reported a modest improvement in her symptoms.

DISCUSSION

Various clinical reports have detailed the incidence of carmustine-induced pulmonary fibrosis; the figures range from 1.3 to 30 percent. The 1.3 percent figure is probably an underestimation of the risk in as much as the majority of patients in this study did not survive long enough to develop toxic reactions. Also, many of them received other chemotherapeutic drugs concomitantly and/or had previous thoracic irradiation.

The true incidence of pulmonary toxicity secondary to carmustine is probably 3.5 to 30 percent. All of these studies included only patients with primary gliomas, most of whom received carmustine exclusively and in high doses.

The total cumulative dose of carmustine is the most important factor influencing the incidence of pulmonary toxicity, with approximately 50 percent of patients developing toxicity with total cumulative doses of 1,500 mg per square meter of body surface area. Other important factors reported to influence the incidence of carmustine-induced pulmonary fibrosis include the following: history of cigarette smoking, preexisting lung disease, prior thoracic irradiation, and concomitant use of other pulmonary cytotoxic drugs.

The diagnosis of carmustine-induced pulmonary fibrosis is confirmed by a combination of findings that include prior exposure to carmustine, signs and symptoms of dry hacking cough, tachypnea and dyspnea, bilateral diffuse or patchy interstitial infiltrates on chest radiography, restrictive defects and a reduced Dco on pulmonary function testing, and the finding of interstitial fibrosis in histologic specimens. Clinically, this is a diagnosis of exclusion, and it must be differentiated from infection, tumor involving the lung, radiation pneumonitis, collagen vascular disease, sarcoidosis, and toxicity to other drugs. Our patient had neither fever nor leukocytosis, and cultures of her blood and bronchial washings failed to demonstrate an infectious cause. She had a normal angiotensin converting enzyme level, normal antinuclear antibody and rheumatoid factor titers, and no history of irradiation to her thorax or exposure to other pulmonary cytotoxic drugs.

Carmustine-induced pulmonary fibrosis is the probable diagnosis since she had received a large cumulative dose of carmustine. Except for an atypical chest radiograph, the rest of her findings (nonproductive cough, restrictive pattern on pulmonary function testing, and normal results of physical examination) are consistent with this diagnosis. Histologic findings from the biopsy specimen obtained during bronchoscopy revealed alveolar cell dysplasia and interstitial fibrosis, findings compatible with this diagnosis. We believe this patient represents a case of carmustine-induced pulmonary fibrosis presenting in an atypical manner as a lobar infiltrate on chest radiography. The appearance of carmustine-induced pulmonary fibrosis in chest radiography is variable. The chest radiograph may be normal or it may mimic other disease entities such as metastatic cancer, Wegener's granulomatosis, or adult respiratory distress syn-
drome. Physicians should be alert to the possibility of a lobar distribution for Carmustine-induced pulmonary fibrosis. Patients receiving Carmustine should be monitored with serial chest radiographs and pulmonary function studies as a potential aid in the early detection of pulmonary toxicity.

REFERENCES


Acute Gastric Dilatation Causing Respiratory Failure and “Tension Pneumothorax” in an Elderly Woman With a Diaphragmatic Hernia

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The occurrence of respiratory failure as a result of a large diaphragmatic hernia is a well-described entity in infants with congenital hernias. On reviewing the literature, the authors did not find a similar clinical presentation in the adult population. They report the case of an elderly patient with a large hiatus hernia who developed recurrent episodes of life-threatening respiratory failure and hemodynamic compromise due to recurrent gastric dilatation. Decompression with nasogastric suction resulted in dramatic and immediate relief of the respiratory distress. One should keep in mind the possibility of intrathoracic gastric dilatation as a cause of acute respiratory insufficiency in patients with hiatal hernia.

The association between diaphragmatic hernia and respiratory failure in neonates is well established.1-4 The major factor causing respiratory failure in these young infants is hypoplasia of the lung due to sustained compression in utero by the herniated abdominal contents. In cases of Bochdalek hernia, without surgical correction, 75 percent of these infants will not survive,5 and extracorporeal membrane oxygenation may be required until lung function recovers adequately.6 The association between gastric herniation and pulmonary compromise is less well described in

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Figure 1. Chest x-ray film obtained on admission shows massive gastric dilatation with rightward shift of the mediastinum.

The adult population. We would like to report here the unusual occurrence of respiratory decompensation secondary to acute gastric dilatation in a patient with preexisting diaphragmatic hernia.

CASE REPORT

An 84-year-old woman, a nonsmoker, was admitted to the intensive care unit of the Hadassah University Hospital because of acute respiratory failure. She had a large diaphragmatic hernia, which had been diagnosed 20 years previously on a routine chest x-ray film. Five years ago, she was hospitalized with unexplained respiratory failure that required mechanical ventilation. Two days prior to her present admission, she developed a mild nonproductive cough with progressive dyspnea but no fever. She had no episodes of vomiting or alteration of consciousness. On admission, she was alert and in severe respiratory distress, with a respiratory rate of 46 breaths per minute, pulse of 110/min, systolic blood pressure of 70 mm Hg, and temperature of 38.0°C. She was not cyanotic and displayed no clubbing. There was rightward deviation of the trachea and marked distension of the neck veins. Examination of the chest

Figure 2. Repeat x-ray film obtained 2 h later, after insertion of a nasogastric tube shows that the mediastinum has returned to midposition.