treatment of duodenal ulcers, gastritis, and esophagitis. However, unlike most other drugs, sucralfate tablets dissolve to form a viscous, gel-like suspension that binds with high affinity to both defective and normal mucosa. While these actions contribute to its well-documented beneficial effects of protecting gastric and esophageal mucosa, it is accompanied by a rapid increase in size (Fig 2). This helps explain the bronchoscopic observation of a markedly swollen sucralfate tablet obstructing our patient's left main-stem bronchus.

Other aspirated materials have been observed to increase in diameter, but not in the short-term setting. Vegetable matter (commonly a peanut) has been observed to gradually swell within the tracheobronchial tree, causing airway obstruction after several weeks. Interestingly, the only previous report of a foreign body causing total obstruction of an adult main-stem bronchus occurred only after a chicken bone had gone unnoticed for more than 3 years. The importance of the present case is that total main-stem obstruction occurred immediately with sudden respiratory compromise. This is particularly noteworthy in that aspiration, as in most adult cases, was not immediately suspected as the cause.

The present case demonstrates that a rapidly expanding foreign body can cause respiratory compromise by acutely obstructing a main-stem bronchus. While we expect this event to be rare in most adults, patients who may be at increased risk include those with identifiable risks for aspiration and they should use caution when taking medicines that share this physical property. If such drugs are required in these patients, prescribing them in liquid or suspension form may decrease the likelihood of acute bronchial obstruction in the event aspiration occurs.

REFERENCES
4 Mittleman RE, Wetli CV. The fatal cafe coronary: foreign-body airway obstruction. JAMA 1982; 247:1285-88

Severe Diffuse Interstitial Pneumonitis Induced by Carmustine (BCNU)*

Herré Lena, M.D.; Benoît Desrues, M.D.; Alain Le Coz, M.D.; Marie Line Quinquenel, M.D.; and Philippe Delaval, M.D., F.C.C.P.

We report a fatal case of acute interstitial pneumonitis in a patient treated with carmustine (BCNU) for a brain tumor. Bronchoalveolar lavage (BAL) revealed lymphocyte alveolitis with a low CD4/CD8 ratio (0.36), consistent with an immunological phenomenon, rather than the most often evoked toxic hypothesis.

(Chest 1994; 105:1602-03)

BCNU=carmustine

bischloroethylnitrosourea (BCNU [carmustine]) is a chemotherapeutic agent used to treat various types of tumors, cerebral in particular. The first pulmonary adverse event related to this drug was reported in 1976, and several cases of pulmonary fibrosis secondary to BCNU treatment have since been reported and published. A toxic mechanism has generally been evoked. We report a case of acute pulmonary fibrosis in a 23-year-old patient with cerebral trunk tumor treated with BCNU and vincristine, the origin of which appears to be linked to an immunological mechanism, considering the characteristics of bronchoalveolar lavage (BAL).

CASE REPORT

A 21-year-old man was hospitalized in July 1986 for headache, diplopia, and balance disorders. Cranial computed tomographic

*From the Department of Pneumology, Hôpital Pontchaillou—Centre Hospitalier Universitaire de Rennes, Rennes, France. Reprint requests: Dr. Delaval, Service de Pneumologie, Hopital Pontchaillou, CHR, 35033 Rennes Cedex, France

FIGURE 1. Thoracic radiography showing diffuse infiltrative process.
scan and encephalic magnetic resonance imaging revealed a lesion of the cerebral trunk, so localized and extended as to preclude surgical biopsy. Radiotherapy (54 Gy on the tumor site) was performed in September 1986, followed by noticeable clinical recovery. The development of balance disorders in September 1988 led to another encephalic magnetic resonance imaging that confirmed relapse. Monthly treatment with 100 mg BCNU on day 1 and day 2 and 1 mg vincristine on day 3 and day 4 was initiated in October 1988. Five courses were thus implemented until January 1989 (BCNU cumulative doses: 1 g) when rapidly aggravating dyspnea and cough led to suspension of the sixth course and transfer of the patient to the department of pneumology.

At the time of admission, the patient had no fever and was very anergic. He weighed 55 kg (8 kg lost in 4 months). He was not cyanotic when at rest, but became dyspneic after the slightest effort. The lung examination revealed bilateral basilar inspiration crackles. Findings from physical examination were otherwise normal except the known neurologic disorder. Chest radiograph revealed a diffuse interstitial process (Fig. 1). Arterial blood gases substantiated hypoxemia at rest with no hypercapnia (PaO₂ 66 mm Hg; PaCO₂ 34 mm Hg; pH 7.47). Otherwise, biologic work up revealed the following: hemoglobin = 11.5 g/dL; leukocytes = 7,500/mm³; lymphonuclear neutrophilic neutrophil = 67 percent; eosinophils = 6 percent; lymphocytes = 18 percent; fibrin = 3.35 g/L; Na = 142 mmol/L; urea = 13.5 mmol/L; creatinine = 101 μmol/L; protein total = 6.6 g/dL; AST = 45 U/L (N < 50); ALT = 41 U/L (N < 50); alkaline phosphatase = 209 U/L (N < 111); lactate dehydrogenase = 750 U/L (N < 420). Pulmonary function tests revealed a severe restrictive pattern: vital capacity 890 mL (17 percent of theoretical values), total lung capacity 1,980 mL (50 percent of theoretical values), with FEV₁/VC = 63 percent.

Fiberoptic bronchoscopy was macroscopically normal. Histologic, bacteriologic, virologic, or parasitologic analysis of all bronchial samplings were negative. All the viral and bacterial blood investigations (HIV, syncytial respiratory virus, herpes virus, Chlamydia, Mycoplasma, Legionella) as well as the usual precipitins (bird fancier's lung, farmer's lung) proved negative. Bronchoalveolar lavage performed with 200 mL physiological serum yielded 475,000 cells per milliliter, including 24 percent lymphocytes, 75 percent macrophages, and 1 percent polymorphonuclear cells. The CD4/CD8 ratio of lymphocyte subsets was 0.36.

Despite steroid therapy at the initial dose of 1 mg/kg/day for 11 days, then by intravenous bolus doses of 240 mg methylprednisolone for 3 days, the condition evolved to gradual worsening of respiratory failure which provoked death on March 18, 1989.

DISCUSSION

According to some authors, pneumonitis secondary to BCNU treatment involves up to 20 percent of treated patients.6 It can occur in an acute or subacute form in the course or after the end of treatment.2,4 The course is most often fatal despite BCNU suspension and steroid treatment, as reported herein.2,3,4 O'Driscoll et al recently reported a series of 17 patients with brain tumor treated with BCNU. Four died from pulmonary fibrosis 8 to 13 years after treatment, and those who survived present with restrictive respiratory syndrome, most often symptomatic. In these patients, BAL does not reveal any excessive lymphocytosis.

A toxic mechanism has often been evoked at the origin of pulmonary fibrosis, based on several points. An animal model was developed in rats by subcutaneous injection of BCNU6 who developed pulmonary fibrosis with granulomatosis. The reduced activity of glutathione that was observed may sensitize pulmonary tissue to oxidants.7 Pulmonary disease is mainly observed with high-dose treatments (in excess of 1,500 mg/m²; 50 percent of cases), whereas its occurrence is exceptional with doses below 500 mg/m².1,2 Pathology specimens only reveal fibrosis, with no inflammatory infiltrates.1,3 Corticosteroids appear to have no effect on the course of disease.3,4 Gaetani et al8 studied 40 patients treated with BCNU for glioma in whom sequential pulmonary function tests revealed a steady decrease in Dco for all patients.8 Lastly, Aronin et al9 reported high incidence of side effects in patients with a history of respiratory disorders (42 percent vs 10 percent).

In our patient, the result from BAL was compatible with a drug-induced pneumonitis of immunoaalergic origin with high alveolar lymphocytosis, and a low CD4/CD8 ratio of 0.36. These figures are very close to those reported by Akoun et al9,10 in various cases of drug-related pneumonitis. Resistance to corticosteroids, however, appears to be conflicting with this hypothesis.

The BAL results, however, are not sufficient to determine which mechanism governs this drug-related pneumonitis. It can be suggested that fibrotic lesions are preceded by an immunoaalergic phase, variably silent clinically, but probably more sensitive to treatment, which appears to be essential considering the most pejorative prognosis when pulmonary disease is patent and symptomatic.

REFERENCES