Communications

clinically picture is similar to that produced by other organisms, such as mycobacteria, although Nocardia is also capable of inducing a granulomatous reaction in tissue. This gives rise to the possibility of making an erroneous diagnosis of tuberculosis, above all in areas such as Spain where this disease is relatively frequent.

Although ceftriaxone has been shown to be active in vitro, its clinical efficacy has been demonstrated only in isolated cases. A clear clinical and radiologic initial response was obtained in the present case, which was maintained over time. Other studies on the clinical utility of ceftriaxone in cases of Nocardia infection should be carried out.

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Ventilatory Criteria for Systemic Inflammatory Response Syndrome

To the Editor:

Concerned as we are about ARDS and its high mortality, we have followed for a number of years Dr. Bone’s concept of ARDS diagnosis as a logical means of decreasing its morbidity and mortality, which later resulted in his description of the septic syndrome. However, since Mexico City’s metropolitan area, with its 23 million inhabitants, is at high altitude (2,240 m above sea level), we are obliged to use different ventilatory parameters, because the proposed PaCO2 level of 32 mm Hg or less for diagnosis of hyperventilation (as appropriate at sea level) is fairly normal for our patients, which creates the possibility of overdiasing SIRS.

For a long time it has been recognized that people living at high altitude, being exposed to lower barometric pressure (585 mm Hg in our city) and so to relatively lower PaO2 and PaCO2 tend to hyperventilate as an automatic mechanism of compensation. The intrinsic physiopathologic mechanism involved in this regulatory pattern is poorly understood, but it is supposed to be mediated through the peripheral chemoreceptors, causing changes in blood and cerebrospinal fluid bicarbonate concentrations, which return pH to normal unless other factors account for ventilatory acclimatization.

We are currently conducting a prospective trial correlating SIRS mortality with the simplified acute physiologic score and the complete septic shock score, using a hyperventilation (PaCO2) criterion of 28 mm Hg or less in the arterial blood gas sample, and propose that the PaCO2 level should be adjusted to altitude in order to inadvertently include some non-SIRS patients.

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Pneumothorax During Pulmonary Tuberculosis in an HIV-Infected Patient

To the Editor:

Pneumothorax is becoming an increasingly important problem in HIV-infected patients. It has been reported in 2 percent of hospitalized HIV-infected patients1 and has been strongly linked to Pneumocystis carinii pneumonia and aerosol pentamidine prophylaxis.4 This report describes an unusual case of spontaneous pneumothorax during the course of pulmonary tuberculosis in a patient with HIV infection.

A 42-year-old HIV-seropositive former intravenous drug abuser was admitted to the hospital with a 3-week history of left-sided chest pain and weight loss. There was no cough, fever, or night sweats. He had no previous opportunistic infections and was not receiving any treatment. His vital signs were stable, and the physical examination findings were unremarkable. The chest roentgenograms showed alveolar infiltrate in the lingular segment of the left upper lobe. The purified protein derivative test was positive, and sputum could not be obtained.

On the fifth hospital day the patient felt a pleuritic left-sided chest pain. A chest roentgenogram revealed a left pneumothorax with persistent lingular infiltrate (Fig. 1). A chest tube was placed, and the left lung reexpanded. Consequently, fiberoptic bronchoscopy was performed. The bronchoalveolar lavage fluid showed acid-fast bacilli, which on subsequent culture grew Mycobacterium tuberculosis. There was no evidence of P carinii, viruses, fungi, or malignancy. The patient was started on a regimen of isoniazid (300 mg daily), rifampin (600 mg daily), ethambutol (1,500 mg daily), and pyrazinamide (1,500 mg daily). The chest tube was removed 12 days later, and the patient was discharged on antituberculosis medications.

This is the first reported case in the English language literature, to my knowledge, to describe spontaneous pneumothorax in an HIV-infected patient with pulmonary tuberculosis. It suggests that we should consider conditions other than P carinii pneumonia when...
a patient with HIV infection presents with spontaneous pneumothorax.

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Recurrent Interstitial Pneumonitis and Dexfenfluramine

To the Editor:

Although fenfluramine (Pondimin, A. H. Robins, Richmond, Va; Pondéral Longue Action, Laboratoires Biopharma, Neuilly, France) and dexfenfluramine (Isoméride, Laboratoires Ardis, Orléans, France) are very widely prescribed as adjuvant therapy in obesity, we know of no reports of interstitial pneumonitis induced by these drugs (racemic and dextrogyre forms). We have observed one such case in a patient given dexfenfluramine.

A 39-year-old man was admitted for the first time to the pneumology department on April 10, 1989, complaining of progressively worsening dyspnea and a dry, nonproductive cough. The patient's otherwise unremarkable medical history included obesity (weight, 110 kg; height, 188 cm) and tobacco use evaluated at 36 packs per year. In addition, he reported one previous episode of dyspnea and coughing, which improved after antibiotic therapy, in November 1988. On admission the patient was afebrile. Physical examination revealed bilateral basilar rales and a right basal pleural rub. Arterial blood gas analysis showed hypoxemia and hypocapnia (pH, 7.40; PCO₂, 22 mm Hg; Po₂, 57 mm Hg). Chest radiography showed a bilateral micronodular interstitial infiltrate and a small right pleural effusion. The vital capacity was decreased by 30 percent, and the steady-state carbon monoxide diffusion capacity was decreased by 20 percent. Laboratory findings included hyper-eosinophilia (780/mm³) and an erythrocyte sedimentation rate (ESR) of 64 mm after the first hour. Pleural tap yielded an exudate (protein, 53 g/L) with predominating eosinophils (42 percent). There was no evidence of neoplasia. The findings from fiberoptic bronchoscopy, biopsy, and aspiration specimen analysis were normal. Electrocardiographic and echocardiographic findings were also normal. Normal lung perfusion scan and plethysmography eliminated thromboembolism. Abdominal ultrasonography ruled out subdiaphragmatic disease. Serologic studies for the usual helminthic parasites and stool analysis were negative. The clinical course was immediately favorable, and the patient was discharged on April 14, 1989.

On July 26 the patient was readmitted with a 15-day history of progressively worsening exertional dyspnea, dry cough, and left chest pain. Physical signs included bilateral basilar rales and pleural rubs. Chest radiography demonstrated recurrent bilateral basilar interstitial infiltrates as well as bilateral pleural effusion. Chest computed tomography confirmed the diffuse interstitial syndrome, especially at the level of the peribronchovascular sheaths. Bronchial wash was performed in the middle lobe. The aspirate was hypercellular (110.000 cells/mm³) with 13 percent lymphocytes and 9 percent neutrophils. Eosinophils were absent. The pleural tap fluid was exudative, and cytologic study showed predominantly lymphocytic cells (68 percent). The patient was unable to undergo functional respiratory tests because of the intensity of his dyspnea. Bacteriologic and mycologic investigations were negative.

The blood cell count was normal, and the ESR was 65 mm after the first hour. Upon further questioning, the patient recalled that each of the three episodes of dyspnea (November 1988, April 1989, July 1989) occurred while taking dexfenfluramine prescribed to help him lose weight. The drug was initially prescribed at a dose of 15 mg twice daily in October 1988. Treatment was interrupted after 6 weeks because of the "flu-like" syndrome he experienced in November 1988. The drug was then prescribed from February to April 1989, interrupted only during his first hospital stay. The last prescription began in July 1989 and was discontinued during his second hospitalization.

The patient was discharged in good health on August 1, 1989. Routine clinical and radiographic follow-ups in July 1991 were normal.

This case strongly suggests a drug-induced lung disease because of the sequence of events, the absence of pulmonary history, the exclusion of cancerous or infectious pulmonary pathology, and the spontaneous regression of the interstitial pneumonitis, without corticosteroid or antibiotic therapy (in April and July 1989).

The responsibility of dexfenfluramine in this case of recurrent interstitial pneumonitis is quite likely. This possible complication widens the spectrum of pulmonary toxicity of this drug, to which several cases of severe primary pulmonary hypertension have already been ascribed.1,2

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