Response of pulmonary nocardiosi to ceftriaxone in a patient with AIDS.

J I Garcia del Palacio and I Martin Pérez

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minimal exertion, wheezing, a cough productive of clear white sputum, and a pressure-like sensation under the right rib cage and midsternal area. There was no history of fever, chills, night sweats, or hemoptysis. She denied recent travel, prior pneumonias, or exposure to tuberculosis. She had a history of chronic sinusitis with postnasal drip but was otherwise in good health. A workup for infertility 4 years previously revealed polycystic ovarian disease. Ovarian stimulation was being given in the form of clomiphene citrate (Clomid) and human menopausal gonadotropin (Pergonal). Other medications included amitriptyline, astemizole, and amoxicillin/clavulanate potassium for recent urinary tract infection. She was a nonsmoker and denied any history of alcohol abuse.

The physical examination revealed a heavyset woman with hirsutism, in mild distress with dyspnea and abdominal pain. The oral temperature was 36.5°C; the respiratory rate was 22 breaths per minute; the blood pressure was 130/80 mm Hg; and the pulse was 90 beats per minute. Her weight was 288 lb. Jugular venous distension was absent, and the trachea was central. There was no clubbing, cyanosis, pedal edema, or sputum, and a pressure-like sensation under the rib cage and right upper rib. Minimal exertion, wheezing, a cough productive of clear white sputum, and a pressure-like sensation under the rib cage and midsternal area. There was no history of fever, chills, night sweats, or hemoptysis. She denied recent travel, prior pneumonias, or exposure to tuberculosis. She had a history of chronic sinusitis with postnasal drip but was otherwise in good health. A workup for infertility 4 years previously revealed polycystic ovarian disease. Ovarian stimulation was being given in the form of clomiphene citrate (Clomid) and human menopausal gonadotropin (Pergonal). Other medications included amitriptyline, astemizole, and amoxicillin/clavulanate potassium for recent urinary tract infection. She was a nonsmoker and denied any history of alcohol abuse.

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clinical 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.

To the Editor:

Concerned as we are about ARDS and its high mortality, we have followed for a number of years Dr. Bone’s concepts about early ARDS diagnosis as a logical means of decreasing its morbidity and mortality, which later resulted in his description of the septic syndrome. We were most interested in his recent Society of Critical Care Medicine (SCCM) conference, and we read with enthusiasm in the June 1992 issue of Chest the recent ACCP/SCCM Consensus Conference article, in which a notable group of colleagues describe systemic inflammatory response syndrome (SIRS) and offer an important body of practical definitions.

In essence, we fully support these definitions and believe that they will improve our way of practicing medicine in the septic patient. 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 overdiagnosing SIRS.

For a long time it has been recognized that people living at high altitude, being exposed to lower barometric pressure (885 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 not 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 patients and has been strongly linked to Pneumocystis carinii pneumonia and aerosol pentamidine prophylaxis. This report describes an unusual case of spontaneous pneumothorax in an HIV-infected patient with pulmonary 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

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