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

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minimal exertion, wheezing, a cough productive of clear white sputum, and a pressurelike 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 lymphadenopathy. There was dullness and decreased fremitus and breath sounds at the right lung base with egophony at the superior margin of the area of dullness. There were no adventitious sounds heard. Cardiac examination showed slight enlargement on percussion, but findings were otherwise normal. The abdomen was obese with striae gravidarum and normoactive bowel sounds. There was no clubbing, cyanosis, pedal edema, or calf swelling or tenderness.

The chest radiograph showed cardiomegaly and a large right pleural effusion (Fig 1). Pelvic ultrasound revealed a gestational sac consistent with a 4-week intrauterine pregnancy, markedly enlarged ovaries, and fluid in the cul-de-sac. The left ovary and right ovary measured 125 mm × 70 mm and 162 mm × 93 mm, respectively, with multiple large ovarian follicles. Abdominal ultrasound showed multiple gallbladder calculi. The kidneys were of normal size. The CBC count and routine blood chemistry studies were normal. Arterial blood gas analysis was not performed. The chorionic gonadotropin level was 1,212 mIU/ml. The diagnosis of grade IV severe OHSS was thus confirmed.

Upon admission the patient was put on complete bed rest with relief of pain by intramuscular meperidine hydrochloride and low-dose diuretic therapy. Frequent vital signs, daily body weights, intake and output, CBC count, and serum chemistry were closely monitored. With this conservative approach, she lose 2.3 kg, with resolution of her symptoms. A repeat chest radiograph showed a minimal residual right pleural effusion and minor atelectasis. After a 5-day hospital stay, she was discharged home in an asymptomatic state. Follow-up chest radiographs revealed complete resolution of pleural effusion.

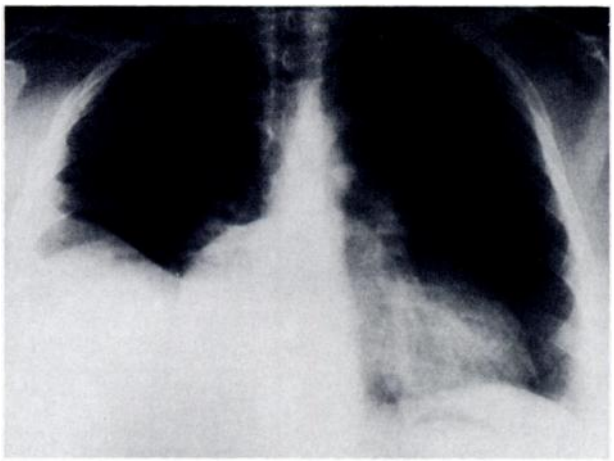

FIGURE 1. Chest radiograph demonstrating a large right pleural effusion and cardiomegaly.

This patient presented with features of severe OHSS secondary to ovulation induction. The two major components of OHSS are massive bilateral ovarian enlargement secondary to multiple cysts with stromal edema and acute fluid shifts due to the sequestration of fluid from the intravascular space to the third space.[2] The clinical course is self-limited in nonpregnant women, but the duration may be longer with a more severe expression in pregnancy since the ovaries are restimulated by endogenous chorionic gonadotropin production.[2] Thoracentesis is usually not necessary unless respiratory embarrassment is present. Knowledge of this syndrome is important to avoid unnecessary diagnostic maneuvers. Awareness of life-threatening complications, including thrombotic stroke, venous thrombosis, massive pericardial effusion, and adult respiratory distress syndrome, is necessary in dealing with OHSS.

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Response of Pulmonary Nocardiosis to Ceftriaxone in a Patient With AIDS

To the Editor:

The infrequency of Nocardia infection in cases of AIDS (not above 0.3 percent)[1] is surprising, and it is exceptional for Nocardia infection to be the form of presentation of AIDS. We recently had the opportunity to treat a heterosexual man who had no history of intravenous drug abuse and who was a carrier of HIV antibodies. He presented with pulmonary infection by Nocardia, which responded excellently to ceftriaxone.

The patient was a 35-year-old man with a history of frequent contact with prostitutes. He reported having had a constitutional syndrome, fever, and hemoptysis 3 months before admission. Examination showed a temperature of 39°C, moderate malnutrition, multiple buccopharyngeal whitish plaques, and a crepitant stertor in the right pulmonary base. A chest radiograph taken on admission showed multiple-cavity infiltration in the right lung. Treatment with erythromycin, 4 g/d, was initiated. On the seventh day after admission, ceftriaxone, 2 g/d, was added to the treatment because of a general worsening of the clinical picture. Forty-eight hours after initiation of treatment with ceftriaxone, the patient had no fever, and his clinical condition had clearly improved.

Transthoracic needle aspiration of the right lung revealed the presence of necrotizing granulomas; acid-fast and Löwenstein smears were negative. Culture isolated *Nocardia asteroides*. Enzyme-linked immunosorbent assay and Western-blot technique showed HIV-positive antibodies. Study of lymphocytes in peripheral blood using flow cytometry showed the following values: CD4, 58×10^6/L; CD8, 219×10^6/L; CD4-CD8 ratio, 0.26.

The patient left the hospital 25 days after admission, and ceftriaxone was replaced by trimethoprim and sulfamethoxazole, since they can be administered orally. Three months after leaving the hospital the patient was asymptomatic, and thoracic radiography was normal.

Nocardia infection is usually introduced via the lungs. Consolidation with cavitation is the most frequent radiologic pattern.[2] This

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*,[3] its clinical efficacy has been demonstrated only in isolated cases.[4] 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 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.[1] We were most interested in his recent Society of Critical Care Medicine (SCCM) conference,[2] and we read with enthusiasm in the June 1992 issue of *Chest* the recent ACCP-SCCM Consensus Conference article,[3] 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 $PaCO_2$ 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 (585 mm Hg in our city) and so to relatively lower PAO_2 and PaO_2, 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.[4-6]

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 ($PaCO_2$) criterion of 28 mm Hg or less in the arterial blood gas sample, and propose that the $PaCO_2$ 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[1] and has been strongly linked to *Pneumocystis carinii* pneumonia and aerosol pentamidine prophylaxis.[2] 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