positive blood culture, an ascending route of infection from the intestinal tract was the most probable mechanism, as previously suggested [2].

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References


Acute Respiratory Failure Associated with the Human Immunodeficiency Virus (HIV) Protease Inhibitor Indinavir in an HIV-Infected Patient

The HIV protease inhibitor indinavir has been marketed recently in several countries after a relatively short period of clinical research; during this period the drug was shown to be safe and effective in the treatment of HIV infection [1–3]. Indinavir is now widely used in HIV-infected patients. The most common adverse effects include elevated serum levels of indirect bilirubin as well as nausea, headache, abdominal pain, and nephrolithiasis [4]. We report a case of acute respiratory distress syndrome probably caused by indinavir.

A 36-year-old transsexual man was admitted to the intensive care unit (ICU) because of shock and acute respiratory failure. He was known to have AIDS, which was classified as Centers for Disease Control and Prevention clinical category C3 on the basis of cervical lymph node tuberculosis, recurrent candida stomatitis, and a low CD4 cell count (15/mm³). He had no history of pulmonary diseases or drug allergies. Nine months before admission, he had started receiving antiretroviral therapy (zidovudine, 200 mg three times daily and zalcitabine, 0.75 mg three times daily) and primary prophylaxis for Pneumocystis carinii pneumonia (PCP) (co-trimoxazole, 480 mg daily). The only other medication he was receiving was cyproterone acetate (im, 300 mg biweekly). The antiretroviral treatment was considered ineffective and was switched one day before admission to triple therapy with indinavir (800 mg three times daily), stavudine (30 mg twice daily), and lamivudine (150 mg twice daily).

Two hours after receiving the first 800-mg dose of indinavir, the patient developed high fever, generalized myalgia, and malaise and began vomiting. The next day, after the second dose, he developed shock and cyanosis and was admitted to the ICU. Physical examination revealed a temperature of 40.4°C, a blood pressure of 70/0 mm Hg, a pulse of 158/min, and respiratory failure. Supportive treatment with mechanical ventilation and inotropic agents was instituted.

Figure 1. Chest radiograph showing diffuse bilateral alveolar infiltrates in a patient with acute respiratory failure associated with indinavir therapy (the radiograph was obtained while the patient was supine).

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Repeated electrocardiograms did not show any abnormalities except for initial sinus tachycardia. A chest radiograph showed diffuse bilateral alveolar infiltrates, consistent with acute respiratory distress syndrome (figure 1). Laboratory investigations revealed a profile consistent with shock that included high levels of liver enzymes, a low albumin level, and prolonged prothrombin and activated partial thromboplastin times. The level of serum cortisol was 807 nmol/L (normal value, 200–700 nmol/L), and the level of serum lactate was slightly increased to 3.7 mmol/L (normal value, 0.6–2.4 mmol/L). In addition, the patient had mild leukocytosis (leukocyte count, 10.7 × 10^9/L; normal value, 4.0–10.0 × 10^9/L) and thrombocytopenia (platelet count, 62 × 10^9/L; normal value, 130–350 × 10^9/L), with no signs of diffuse intravascular coagulopathy. Initial measurements with a thermodilution catheter showed a septic profile (high cardiac output [17.8 L/min] and low systemic vascular resistance [229 dyn·sec·cm⁻²]) with low central venous and wedge pressures.

All cultures of blood, urine, and bronchoalveolar lavage and other body fluids were negative for viral, bacterial, mycobacterial, and fungal pathogens. The patient recuperated within 6 days and was discharged without any symptoms. Antiretroviral treatment was continued without indinavir, and there were no further problems.

Respiratory failure occurs in association with AIDS-related illnesses, such as PCP, in patients with AIDS. However, the event in the present case could not be related to a concurrent infection. Alternatively, the event may have been drug related. HIV-infected patients have been reported to be at higher risk of idiosyncratic hypersensitivity reactions to sulphonamides such as sulfamethoxazole in co-trimoxazole [5, 6]. However, we considered it unlikely that the event in our patient was related to treatment with co-trimoxazole, since he had been taking this drug for 9 months and continued taking it without any problems. No other known causes of his shock could be identified from the available data, leading to the following conclusion.

The strong temporal relationship and short time between the ingestion of indinavir and development of symptoms strongly suggest an etiological role for indinavir. We believe that the severe shock and acute respiratory distress syndrome in our patient were due to an idiosyncratic reaction to indinavir. To our knowledge, acute respiratory failure and shock associated with first-time use of indinavir have not been reported previously. HIV protease inhibitors represent a new generation of drugs for which relatively little information on rare adverse effects is available, although these drugs are being used on a large scale. Physicians should be aware of possible unpredictable adverse reactions to these new therapeutic agents.

**Mycotic Aneurysm Due to Burkholderia pseudomallei**

Meliodosis is an infection caused by *Burkholderia pseudomallei*, formerly *Pseudomonas pseudomallei*, and is endemic in areas within 20° latitude north and south of the Equator [1]. Meliodosis is known for its diverse clinical manifestations, including pulmonary infections, septicaemia, and localized suppurative infections, which may present acutely or chronically [2]. We present, to our knowledge, the first case report of meliodosis manifesting as mycotic aneurysm.

A 70-year-old man with hypertension was admitted to Veterans General Hospital – Kaohsiung, Taiwan, on 25 November 1994 for evaluation of intermittent fever of 2.5 months’ duration. He had visited Hunan province in Mainland China (30 August to 17 September 1994) 3 months before admission, and his fever had developed on 10 September 1994. Upon return to Taiwan, he had been hospitalized elsewhere for 3 weeks without a definite diagnosis. Although his fever had subsided, general malaise and poor appetite persisted. Three weeks before presenting at our hospital, he had again been admitted to a local hospital because of recurrent fever and was referred to our institution because a diagnosis could not be made.

On admission, he had a fever (temperature, 39°C); findings of the remainder of the physical examination were unremarkable. Routine blood studies revealed the following values: WBC count, 5,660/mm³ with 75% neutrophils, 9% lymphocytes, and 16% monocytes; hemoglobin, 9.7 mg/dL; and platelet count, 128,000/mm³. Urinalysis revealed microhematuria (RBCs, 2–4 per high-power field). His electrolyte levels (except for a potassium level of 2.7 mg/dL) and liver function were normal. A bone marrow biopsy showed severe hypoplasia, attributed to either the aging process or the effect of drugs. Findings on a CT scan of the abdomen, obtained on

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