while still of great benefit in preventive health care, should also be recognized as non-specific immune activators. Practitioners who are treating HIV-infected patients should be aware of such acute stimuli and the immune reconstitution syndrome.

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References


Severe Neutropenia During Therapy for Concurrent Primary Human Immunodeficiency Virus and Cytomegalovirus Infections

Treatment of primary HIV infection with antiretrovirals is based on theoretical rationale, limited clinical trial data, and the opinions of experts. We describe a patient with concurrent primary HIV-1 and cytomegalovirus (CMV) infections who had severe neutropenia while receiving antiretroviral therapy.

Serologies for antibodies to CMV and HIV and testing to determine plasma HIV RNA viral load (Amplitrac HIV Monitor Test, Roche Diagnostic Systems, Branchburg, NJ) were done with use of commercial kits. A search was performed on MEDLINE and AIDSLINE through November 1997 for concurrent acute/primary infections with HIV and CMV. Additional cases were identified by manual searches.

A previously healthy, 32-year-old woman was hospitalized (day 1) because of fever of 12 days’ duration, headache, skin rash, and enlarged cervical lymph nodes. She had been prescribed acyclovir for suspected genital herpes infection 9 days earlier. Her history revealed two episodes of condom-protected heterosexual intercourse 2 weeks before the fever. She had been HIV-negative 4 months earlier, and she denied other possible exposures to HIV thereafter. On hospital day 1, serologies for antibodies to HIV showed a distinct reaction to envelope protein gp41 and weak reactions to envelope protein gp120 and to protein p17, the plasma HIV RNA viral load was 480,000 copies/mL, and a serology for antibodies to CMV was negative. On day 3, her WBC count was 3.6 x 10^9/L (neutrophils, 1.1 x 10^9/L), hemoglobin level was 127 g/L, and her platelet count was 188 x 10^9/L. On day 5, therapy with zidovudine (600 mg/d), lamivudine (300 mg/d), and indinavir (2,400 mg/d) was instituted, and fluconazole and famotidine were started for oral thrush and retrosternal pain, respectively. On day 8, she had severe neutropenia (WBCs, 0.9 x 10^9/L; neutrophils, 0.05 x 10^9/L), an axial temperature of 39.6°C, and her condition was deteriorating. Antiretroviral therapy was discontinued, and empirical treatment for sepsis syndrome was started with ceftriaxone and tobramycin. The CD4+ lymphocyte count was 0.18 x 10^9/L on day 9. An esophagogastroduodenoscopy done on day 17 revealed a necrotic esophageal ulcer (1 cm by 10 cm). CMV and HIV were cultured from biopsy material. Neither staining nor culture showed evidence of fungal etiology. CMV inclusion bodies could not be identified. Ganciclovir was started and the retrosternal pain quickly abated.

On day 15, the patient’s WBC count was 3.5 x 10^9/L (neutrophils, 0.36 x 10^9/L) and her plasma HIV RNA viral load was 5,700 copies/mL; zidovudine (600 mg/d) was restarted. Lamivudine (300 mg/d) was added on day 17. Because of the declining neutrophil count (0.13 x 10^9/L), therapy with zidovudine was replaced by that with stavudine (80 mg/d) on day 19. The neutrophil count increased to 1.5 x 10^9/L and the CD4+ lymphocyte count to 0.607 x 10^9/L by day 25, when saquinavir (1,800 mg/d) was added. The patient tolerated stavudine, lamivudine, and saquinavir and was discharged to her home. On day 54, her WBC count was 4.9 x 10^9/L (neutrophils, 2.6 x 10^9/L), CD4+ lymphocyte count was 0.640 x 10^9/L, and her plasma HIV RNA viral load was 2,200 copies/mL. A serology for antibodies to HIV showed strong reactions to gp41 and gp120, but no reaction to p34. A serology for IgG antibodies to CMV was positive. Esophagogastroduodenoscopy findings were within normal limits.

The cause of neutropenia in our patient was probably multifactorial; antiretroviral agents, two acute viral infections, and sepsis may have contributed to neutropenia. There was no definite histological proof for CMV causing the esophageal ulcer, but positive culture from biopsy material, CMV seroconversion, and response to ganciclovir supported this diagnosis.

Ten cases of HIV and CMV primary coinfections have been described. In general, HIV infection seemed to progress rapidly, as measured by the decline in CD4 cell counts. Several patients had complications during the primary disease. One patient had CMV and toxoplasma retinitis 5 months after the primary infection [1]. He died 2 months later of lymphoma. Other complications included pancytopenia, hepatitis, and pericarditis [2], CMV colitis [3], and CMV encephalitis [4]. Only one of the
Melioidosis Brain and Lung Abscess After Travel to Sri Lanka

Melioidosis is an infection caused by the soil and water bacterium *Burkholderia pseudomallei*. Melioidosis is endemic in Southeast Asia and Northeast Australia [1]. Occasionally tourists who have traveled for a limited period in an area of endemicity, such as the northern part of Thailand, may develop melioidosis [2]. Herein, we describe a patient who had acute melioidosis, with necrotizing pneumonia and a brain abscess, that occurred after travel to Sri Lanka, a region that was considered nonendemic [1].

A 66-year-old man with a negative medical history traveled in Sri Lanka for 15 days. He returned to Europe 2 days before the onset of fever (temperature, 39°C) and headache. He was treated initially with paracetamol. Ten days after onset of symptoms, the patient was admitted to the hospital because of persisting fever and increasing stupor and disorientation. There was no nuchal rigidity. A CT scan of the head revealed a frontal lobe mass. A chest radiograph and CT scan showed an infiltrate with central necrosis in the right upper pulmonary lobe. Direct microscopy of a specimen obtained by CT-guided aspiration of the pulmonary lesion revealed polymorphonuclear WBCs; there were no bacteria evident on gram-staining of the aspirate. Culture of the aspirate yielded *B. pseudomallei*. The strain was susceptible to ceftazidime (MIC, 1.0 mg/L), imipenem (MIC, 0.09 mg/L), and ciprofloxacin (MIC, 0.5 mg/L). Serology for *B. pseudomallei* (agglutination assay) performed at the Pasteur Institute (Paris), showed a titer of 1:320. Antibiotic treatment with ceftazidime (2 g iv t.i.d.) was continued for 56 days. Oral maintenance treatment (ofloxacin, 800 mg per day) was administered for 8 months. The patient’s clinical condition improved and the radiological evolution of the pulmonary and brain lesions was favorable. The patient had no recrudescence of melioidosis during 2 years of follow-up.

This case of melioidosis is intriguing because of its clinical presentation and geographic origin. The disease was acquired by a tourist who traveled in a region that was considered nonendemic [1]. Indeed, only very few cases of melioidosis have been reported from the Indian subcontinent or Sri Lanka, despite similarities in geographic location, weather, and environmental conditions with Southeast Asian countries. Melioidosis may, however, be underdiagnosed because of difficulties in culturing the causative microorganism or may be misdiagnosed as plague [3].

The common clinical presentations of acute melioidosis are sepsis and pneumonia. Neurological involvement is infrequent [4]. Only one other case of melioidosis brain abscess acquired during travel has recently been described [5]. The patient we describe, with multiple organ involvement, was treated with ceftazidime followed by a prolonged maintenance treatment with ofloxacin. Ceftazidime has been shown to halve the mortality rate of acute melioidosis and has become first-choice antibiotic for treatment of severe melioidosis [6, 7]. Fluoroquinolones have been used for treatment of melioidosis, but recent experience indicates that primary treatment failures and relapses are more frequent than with a conventional antibiotic combination [8]. The main reasons for these failures may be the marginal in vitro activity against most isolates of *B. pseudomallei* and the emergence of resistance during treatment. The favorable response of our patient may be due to the lower MIC, the higher dose of the fluoroquinolone, and a longer primary treatment with ceftazidime compared to the experience in the literature.

In conclusion, we described a patient with acute pulmonary and neurological melioidosis acquired during travel in Sri Lanka, a region that was considered nonendemic. Ceftazidime monotherapy followed by long-term maintenance treatment with ofloxacin resulted in complete cure.