MRSA isolates tested were identical (68%) or closely related by pulsed-field gel electrophoresis [6]. Where prevalent, these MRSA strains complicate management of pediatric infections because they occur in unexpected circumstances [1–3]. They are less frequently associated with surgery, indwelling medical devices, underlying conditions, or any medical risk factor than are other MRSA in children [1, 2]. They are often susceptible to erythromycin, cotrimoxazole, and other antibiotics as well as clindamycin [1, 2, 5, 6]. Thus, the presence of these organisms could affect antibiotic treatment of the many types of infections in children that can be due to S. aureus.

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References

Recurrent Panniculitis in a Patient Receiving Protease Inhibitor Therapy for Human Immunodeficiency Virus Infection

Recent reports have described abnormalities in lipid storage and distribution in HIV-infected persons, particularly those receiving protease inhibitor therapy. To date, hypertriglyceridemia [1] and abnormal fat distribution, including intraabdominal fat deposition [2], buffalo hump [3], and abnormal fat deposition such as lipomatosis [4], have been described. The causal relationship with protease inhibitors has not been established, and the pathophysiological mechanism is not understood.

Panniculitis is inflammation of the subcutaneous fat that can be associated with a variety of underlying conditions such as infection, immunologic diseases, cancer, and medications [5]. To date, panniculitis has not been described in HIV-infected persons receiving protease inhibitor therapy. We report a case that is likely associated with protease inhibitor therapy.

A 49-year-old obese man who was an intravenous drug user was initially diagnosed with HIV infection in 1989; he had no history of opportunistic infections. In June 1996, his plasma level of HIV type 1 (HIV-1) RNA was 6,099 copies/mL, and his CD4 cell count was 113/μL. Therapy with zidovudine (300 mg b.i.d.), lamivudine (150 mg b.i.d.), and indinavir (800 mg t.i.d.) was started; 6 months later, because of poor virological response, treatment was changed to stavudine (40 mg b.i.d.), lamivudine (150 mg b.i.d.), and ritonavir (600 mg b.i.d.). Within 1 month, he developed dramatic bilateral leg inflammation characterized by edema, erythema, and induration. Doppler ultrasound examinations of the heart and lower extremities were unrevealing. He was treated with intravenous ceftazolin, and ritonavir was changed to nelfinavir (750 mg t.i.d.). Since then, his plasma level of HIV-1 RNA has remained <2,000 copies/mL, although his CD4 cell count has dropped to 10/μL.

In the ensuing 2 years, he developed six distinct episodes of bilateral leg inflammation. Each time, he was believed clinically to have cellulitis and was treated with intravenous ceftazolin or vancomycin. During one hospitalization, he developed right leg inflammation 7 days into treatment of left leg inflammation. In December 1998, while receiving oral cefadroxil for suppression of presumed cellulitis, he developed new left leg inflammation. A CT scan of his leg showed several areas with infiltration of subcutaneous fat. Skin punch biopsy revealed mixed septal and lobular fibrosing panniculitis with sparse inflammatory cells present; there is no vasculitis (stain, hematoxylin-eosin; original magnification, × 400).
yprednisolone (40 mg q.d.) resulted in a dramatic response within 12–24 hours. No antibiotics were administered.

Since resolution of this episode, his corticosteroid dosage has been tapered to 10 mg of prednisone daily. During this treatment regimen, he has had a flare of panniculitis treated by increasing his prednisone dosage to 40 mg daily, followed by a rapid taper to 10 mg daily.

Protease inhibitors increasingly are being associated with derangements of lipid storage and distribution [2]. The pathogenesis of these syndromes remains poorly understood. Panniculitis has been described in association with other medications like penicillins, sulfa drugs, estrogen and oral contraceptives [5], ciprofloxacin [6], and atenolol [7] but not with the protease inhibitor drugs. In this case, the clinical presentation, histopathologic findings, failure to respond to intravenous antibiotic therapy, and dramatic response to corticosteroid therapy suggest protease inhibitor–induced panniculitis. However, it is possible that panniculitis is unrelated to protease inhibitor drugs and is due to HIV infection or other medications or is idiopathic.

Although some forms of panniculitis are self-limited, treatment often depends upon eliminating the underlying cause in addition to the use of antiinflammatory and immunosuppressive agents [1]. In our case, this represents a problem, given the excellent virological response to his current antiretroviral regimen, he has had a flare of panniculitis treated by increasing his prednisone dosage to 40 mg q.d. (1,000 mL of normal saline was infused before the first dose). She tolerated the infusion of two units of packed RBCs. On day 2 of treatment, Foscarnet was infused over 2 hours via a peripherally inserted central venous catheter. Normal saline (500 mL) was administered at 40 mg/kg every 12 hours (i.e., 2,920 mg intravenously q12h). Foscarnet was administered at 40 mg/kg every 12 hours (i.e., 2,920 mg intravenously q12h). Foscarnet was infused over 2 hours via a peripherally inserted central venous catheter. Normal saline (500 mL) was infused over 1 hour before each foscarnet dose (1,000 mL of normal saline was infused before the first dose). She tolerated the fluids well.

The patient was followed up with daily determinations of the complete blood cell count and electrolyte, calcium, magnesium, and phosphorous levels. Her renal function remained normal. Throughout therapy, the patient required magnesium repletion and transfusion of two units of packed RBCs. On day 2 of treatment, during foscarnet infusion, the patient had paresthesias in her extremities, a temperature of 99.2°F (37.3°C), and shaking chills.

Foscarnet Treatment of Genital Infection Due to Acyclovir-Resistant Herpes Simplex Virus Type 2 in a Pregnant Patient with AIDS: Case Report

Foscarnet (Foscavir, Astra Pharmaceuticals, Wayne, PA) is an organic analogue of inorganic pyrophosphate that inhibits the replication of herpesvirus in vitro, including herpes simplex virus (HSV) types 1 and 2 (HSV-2) and cytomegalovirus. It does not require activation (phosphorylation) by thymidine kinase or other kinases and, thus, may be active against HSV strains resistant to acyclovir [1]. There are no adequate well-controlled studies of the use of foscarnet in pregnancy. We describe a case of genital infection due to acyclovir-resistant HSV-2 in a pregnant HIV-seropositive woman treated with foscarnet.

A 21-year-old pregnant female with a history of AIDS for 3 years, recurrent genital HSV infection, abnormal Pap smears, anemia, and noncompliance with HIV therapy presented in March 1997 with a severe episode of genital HSV-2 infection with ulceration and bleeding. She was unresponsive to treatment with high doses of oral acyclovir (800 mg every 5 hours). Susceptibility testing for HSV-2 was performed. A viral posting test revealed that the HSV-2 strain was resistant to acyclovir (median infective dose [ID50], 2.985 μg/mL) and ganciclovir (ID50, 3.124 μg/mL) and susceptible to foscarnet (ID50, 14.828 μg/mL). The patient had no other AIDS-related illnesses at that time. Her medications included...

References

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