Acute Human Immunodeficiency Virus Syndrome in an Adolescent

Mridula Aggarwal, MD, and Jeffrey Rein, MD

ABSTRACT. Acute human immunodeficiency virus (HIV) seroconversion illness is a difficult diagnosis to make because of its nonspecific and protean manifestations. We present such a case in an adolescent. A 15-year-old boy presented with a 5-day history of fever, sore throat, vomiting, and diarrhea. The patient also reported a nonproductive cough, corza, and fatigue. The patient’s only risk factor for HIV infection was a history of unprotected intercourse with 5 girls. Physical examination was significant for fever, exudative tonsillopharyngitis, shotty cervical lymphadenopathy, and palpable purpura on both feet. Laboratory studies demonstrated lymphopenia and mild thrombocytopenia. Hemoglobin, serum creatine, and urinalysis were normal. The following day, the patient remained febrile. Physical examination revealed oral ulcerations, conjunctivitis, and erythematous papules on the thorax; the purpura was unchanged. Serologies for hepatitis B, syphilis, HIV, and Epstein-Barr virus were negative. Bacterial cultures of blood and stool and viral cultures of throat and conjunctiva showed no pathogens. Coagulation profile and liver enzymes were normal. Within 1 week, all symptoms had resolved. The platelet count normalized. Repeat HIV serology was positive, as was HIV RNA polymerase chain reaction. Subsequent HIV viral load was 350,000, and the CD4 lymphocyte count was 351/mm3. HIV is the seventh reaction. Subsequent HIV viral load was 350,000, and the CD4 lymphocyte count was 351/mm3. HIV is the seventh reaction. Subsequent HIV viral load was 350,000, and the CD4 lymphocyte count was 351/mm3.

CASE REPORT

A 15-year-old boy without significant medical history presented with a 5-day history of fever, sore throat, vomiting, and diarrhea. The patient also reported a nonproductive nocturnal cough, corza, and fatigue. His mother noted a pedal rash for 1 day. He denied recent travel, night sweats, arthralgias, dysuria and penile discharge, and sick contacts. On physical examination, the patient was a thin, well-appearing boy with a fever of 38.6°C, an ornamental staph found in an otherwise normal tongue, exudative tonsillopharyngitis, a supple neck, and shotty cervical lymphadenopathy. The cardiac, pulmonary, and abdominal examinations were normal except for guaiac positive brown stool with mild perianal excoriation. The patient had linear palpable purpura tracing the plantar-volar junction of both feet. Laboratory values included a leukocyte count of 3500 cells/mm3 with 63% neutrophils, 25% lymphocytes with rare atypia, 11% monocytes, and a platelet count of 100,000/mm3. Hemoglobin, serum creatine, and urinalysis were normal.

Although unaccompanied by his mother, the patient denied intravenous drug use, homosexual contact, and sex with prostitutes. He reported a lifetime history of unprotected intercourse with 5 girls.

At follow-up examination the next day, the patient remained febrile, with persistent sore throat. The diarrhea had resolved. Physical examination revealed resolution of tonsillopharyngitis, presence of several 2-mm ulcerations on the hard and soft palate, bilateral conjunctivitis, scattered erythematous, and blanching 5-mm papules on the thorax; the purpura was unchanged. Additional laboratory studies revealed negative serologies for hepatitis A and B, syphilis, HIV, and Epstein-Barr virus; HIV DNA polymerase chain reaction (PCR) and hepatitis C serology were unavailable. Bacterial cultures of blood and stool and viral cultures of throat and conjunctiva showed no pathogens. Throat culture demonstrated β-hemolytic nongroup A streptococci, for which the patient was treated. Coagulation profile and liver enzymes were normal. Biopsy of a papule revealed lichenoid dermatitis, consistent with a viral exanthem. Blood counts were unchanged but for a platelet count of 83,000/mm3. The erythrocyte sedimentation rate was 17 mm/h.

During the following week, the patient’s fever, purpura, and papular rash resolved. The platelet count returned to normal. Repeat HIV serology was positive, as was HIV RNA PCR. Subsequent HIV viral load was 350,000, and the CD4 lymphocyte count was 351/mm3.

DISCUSSION

HIV is the seventh leading cause of death among people aged 15 to 24 in the United States.1 Although some HIV-related sexual risk behaviors among high school students are decreasing,2 up to half of all new infections occur in adolescents.3 This patient presented with a febrile, multisystem...
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syndrome with a polymorphous eruption. In the appropriate setting, clinicians should always consider acute HIV seroconversion. Our patient presented with many of the typical signs and symptoms of acute HIV infection: fever, fatigue, pharyngitis, lymphadenopathy, oral ulcers, nausea, emesis, and diarrhea. Although the rash of HIV seroconversion is classically described as a macular or morbilliform eruption predominantly on the trunk, cutaneous vasculitis has been described. Histopathology is consistent with a viral exanthem, as in this case. The 10-day time course of the patient’s illness is within the typical range of several days up to 10 weeks. Other symptoms commonly reported include headache, myalgias, arthralgias, aseptic meningitis, peripheral neuropathy, thrush, weight loss, night sweats, and genital ulcers. Mucocutaneous ulceration is highly suggestive of acute HIV infection. These symptoms are similar to those of other illnesses such as infectious mononucleosis, acute hepatitis, roseola and other viral illnesses, secondary syphilis, and toxoplasmosis. Of note in one study, 2% of heterophil antibody-positive blood samples were HIV RNA positive, with half of these representing acute HIV infection. The common seroconversion laboratory findings of leukopenia and thrombocytopenia were present in our patient. Another common abnormality, absent in this case, is elevation of hepatic enzymes. Our finding of atypical lymphocytes on peripheral smear, although reported, is less common.

The suspicion of acute HIV illness should prompt virologic and serologic analysis. Initial serology is usually negative, with seroconversion later in the course, as observed here. Diagnosis therefore depends on direct detection of the virus. Viral load (HIV RNA) and DNA PCR, as well as p24 antigen assay, have been used for this purpose. Both false-positive and false-negative results for these assays have been reported, further complicating early diagnosis.

Pediatricians should play an active role in identifying HIV-infected patients. Both the American Academy of Pediatrics and the Centers for Disease Control and Prevention encourage HIV testing for those at risk, such as sexually active adolescents. Recognition of acute HIV syndrome may be especially important. Early initiation of appropriate antiretroviral therapy improves surrogate markers of disease progression and should be considered for all patients, optimally in a clinical trial.

CONCLUSIONS

The nonspecific, mononucleosis-like symptoms of acute HIV infection make it an easy diagnosis to miss. Data suggest that early diagnosis may affect morbidity and mortality. It is imperative that we maintain a high index of suspicion as physicians for adolescents who present with a viral syndrome and appropriate risk factors.

REFERENCES

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