Multiple Organ Failure during Primary HIV Infection

Pierre Tattevin,1 Christophe Camus,1 Cédric Arvieux,1 Annick Ruffault,2 and Christian Michelet1

1Service de Maladies Infectieuses et Réanimation Médicale and 2Département de Virologie, Pontchaillou University Hospital, Rennes, France

The appearance of primary HIV infection ranges from an asymptomatic presentation to a symptomatic illness resembling infectious mononucleosis. Severe unusual presentations include acute myopericarditis, renal failure, and opportunistic infections such as esophageal candidiasis, cytomegalovirus infection, and Pneumocystis jirovecii pneumonia. We report a case of multiple organ failure during primary HIV infection.

A 16-year-old woman presented to a health care facility with fever of acute onset and a 4-day history of chills, myalgia, and diarrhea. Her past medical history was unremarkable. The only prescription medication that she was taking was oral contraceptive pills, and she reported only 1 regular sexual partner in the last 6 months. At admission, the patient appeared to be acutely ill, with a body temperature of 40.4°C and a blood pressure of 80/40 mm Hg. Physical examination revealed diffuse lymphadenopathy, hepatomegaly, and muscle tenderness. The patient’s WBC count was cells/L with cells/L segmented neutrophils. The patient’s aspartate aminotransferase and alanine aminotransferase levels were 4266 and 1875 IU/L, respectively (normal value for each, <40 IU/L). Her creatinine phosphokinase level was 2734 IU/L (normal value, <190 IU/L), her troponin Ic level was 2.18 ng/mL (normal value, <0.15 ng/mL), and her prothrombin index and factor V were 0.61 and 0.61, respectively (normal value for each, >70%). She was administered 1000 mL of normal saline and was admitted to the intensive care unit for severe sepsis of unknown cause. Transthoracic echocardiography revealed slightly impaired cardiac function with an ejection fraction of 55% despite adequate preload. A chest radiograph had normal findings. Abdominal CT revealed homogenous hepatomegaly. Three sets of blood sample cultures and urocultures remained sterile. The patient received intravenous amoxicillin-clavulanate and gentamicin without any improvement in her condition.

On day 3 after admission, blood sample smears revealed atypical lymphocytosis. P24 antigen level was 1460 pg/mL. Two HIV enzyme assays had discordant results: Axsym HIV1/2 (Abbott) had a negative result, and Enzygnost (Dade Behring) had a positive result. An HIV Western blot assay disclosed 1 band at p25. Plasma HIV RNA load was 5,304,804 copies/mL (6.7 log10 copies/mL). When the patient was informed of the diagnosis of HIV infection, she declared that she had unprotected sex with an occasional partner 1 month earlier who had received a retrospective diagnosis of asymptomatic HIV infection.洛匹那韦- ritonavir, didanosine, and stavudine therapies were initiated, and the patient’s condition dramatically improved. By day 7, she had normal findings on physical examination, normal liver function, a normal creatine phosphokinase level, and a normal echocardiogram (ejection fraction, 65%), and she was discharged from the hospital. At day 45 after receipt of diagnosis of HIV infection, her CD4+ T cell count was 780 cells/mm3, and her viral load was 50 copies/mL. Repeated serological testing for hepatitis A, B, and C viruses, cytomegalovirus, and syphilis had negative results. Epstein-Barr virus infection was not recently acquired (the patient was positive for IgG anti–Epstein-Barr virus nuclear antigen at admission). Three and one-half years later, she continues to receive antiretroviral treatment (efavirenz, didanosine, and lamivudine), has an undetectable viral load (<40 copies/mL) and a CD4+ T cell count of 1080 cells/mm3, and is asymptomatic.

To our knowledge, this is the first report of acute liver failure during primary HIV infection. This patient also presented with severe sepsis, myocarditis, rhabdomyolysis, and profound leukopenia, all of which are rather unusual in this situation [1–4]. The nonspecific nature of the acute symptoms of primary HIV infection often makes the diagnosis challenging [5]. In a cohort of 46 patients with primary HIV infection, although >85% sought medical attention for acute retroviral syndrome, only 25% received a correct diagnosis [3]. When patients present with unusual manifestations and do not report any significant risk factor, as in our patient, the diagnosis is frequently missed and may result in serious consequences. Although systematic initiation of antiretroviral treatment during primary HIV infection remains controversial, most physicians would treat patients presenting with life-
threatening manifestations [6, 7]. In addition to the potential clinical, immunological, and virological benefits of early therapy [8–10], there may be important public health considerations associated with the reduction of HIV transmission [11, 12]. Indeed, peak viremia during primary HIV infection is associated with concurrent, high-level genital shedding of the virus [11]. Thus, early identification of primary HIV infection could be a unique opportunity to abort rapid epidemic spread in sexual networks [7, 12].

In our observation, given the unusual severity of acute HIV infection and the very good tolerance of antiretroviral treatment, the patient and her physicians are reluctant to consider discontinuation of antiretroviral treatment. Indeed, the first report of retroviral rebound syndrome occurred in a patient who had initiated combined antiretroviral treatment very early (before seroconversion) and who developed a second acute syndrome when such treatment was discontinued 6 months after initiation of therapy [13]. Moreover, fatal outcome during retroviral rebound syndrome has been recently reported [14].

In conclusion, multiple organ failure may be added to the list of severe manifestations of acute HIV infection. Early initiation of combined antiretroviral treatment to control high-level viremia seems to be the most rational therapeutic intervention; in this case, this measure was followed by a dramatic improvement in the patient’s condition.

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References