A Patient with Primary Human Immunodeficiency Virus Infection Who Presented with Acute Rhabdomyolysis

Darius A. Rastegar, Claudia V. Claiborne, Adam S. Fleisher, and Alan K. Matsumoto
Johns Hopkins Bayview Medical Center and Johns Hopkins School of Medicine, Baltimore

Primary human immunodeficiency virus (HIV) infection is usually symptomatic, and infected patients can present with a variety of symptoms. We describe a 51-year-old man who presented at our hospital with acute self-limited rhabdomyolysis and who was found to have primary HIV infection. Our case and other reports suggest that a diagnosis of primary HIV infection needs to be considered for patients who present with acute rhabdomyolysis.

Primary HIV infection, or “acute retroviral syndrome,” is increasingly recognized as a clinical syndrome with a wide variety of presentations. Symptoms that are commonly reported include fever, fatigue, sore throat, myalgias, headache, rash, and lymphadenopathy [1]. We report an unusual case of a patient with primary HIV infection who presented with acute rhabdomyolysis.

A 51-year-old man presented to our emergency department. He complained of persistent leg pain. One week prior to admission, he began to experience sharp, stabbing pains in the posterior muscle groups of both thighs. The pains radiated down to his calves and persisted as a dull, aching sensation that worsened with activity. There was no history of trauma or heavy physical exertion. He also complained of emesis and an occasional productive cough that started 3 days prior to presentation. He denied having fatigue, weakness, or weight loss, and the remainder of his verbal review of systems was unremarkable.

The patient’s past medical history was significant for 2 myocardial infarctions, the last of which happened 5 years prior to admission. He was taking neither prescribed nor over-the-counter medications. He denied that he smoked tobacco, but did inform us that he consumed approximately 72 ounces of beer per week. He reported that his last sexual contact occurred 2 years prior to presentation. He denied having any history of high-risk sexual behavior or injection drug use. He worked at a cardboard box factory; there was no known inhaled or ingested toxin exposure.

Physical examination revealed the following values: temperature, 38.4°C; heart rate, 90 bpm; and blood pressure, 129/91 mm Hg. Shoddy bilateral inguinal lymphadenopathy was noted, but the results of his physical examination were otherwise unremarkable. Of note, there was no muscle tenderness, and strength was 5/5 in all his extremities. There were no neurologic deficits.

Laboratory investigations revealed the following values: creatine phosphokinase, 32,720 IU/L with an MB fraction of 67 ng/mL; hemoglobin, 14.9 g/dL; platelet count, 81,000 cells/mm³; and total WBC count, 2200 cells/mm³ with a differential of 28% lymphocytes, 6% monocytes, 36% neutrophils, and 30% bands. The result of a urine toxicology screen was negative. His serum creatinine level was 1.6 mg/dL and his serum electrolyte level was normal. His urine dipstick result showed large hemoglobin pigments and grade 4+ protein; microscopic analysis revealed 25–35 WBCs/high power field, numerous granular casts, no epithelial cells, and no RBCs. However, a test for urine myoglobin had a negative result (we felt that this was probably a false-negative result). Liver function tests were remarkable for an aspartate aminotransferase level of 875 U/L and alanine aminotransferase (ALT) of 236 U/L; total bilirubin, alkaline phosphatase, total protein, and albumin levels were all within normal limits. C3 complement was 68 mg/dL (low), C4 complement was 23 mg/dL (normal), and the erythrocyte sedimentation rate was 7 mm/hr. The result of an antiplatelet antibody IgG screen was positive, but the results of antinuclear antibody and anti-JO 1 antibody tests were negative. Cultures of blood samples tested negative, and a purified protein derivative skin test was nonreactive. A chest x-ray film, lower extremity x-ray films, and an electrocardiogram did not reveal any abnormalities. Nerve conduction and electromyographic studies showed no evidence of muscle pathology. An HIV ELISA was nonreactive and the plasma HIV RNA level was positive at >750,000 copies/mL. The patient was not tested for other viral illnesses.

During the 3 days after admission, the patient’s leg pain
resolved and his creatine phosphokinase level fell to 2700 IU/L. His serum creatinine level normalized with hydration (0.7 mg/dL). The result of a repeat ELISA for HIV antibody performed 1 month after discharge was positive, as was the result of a Western blot assay; the plasma HIV RNA level at that time was 2628 copies/mL, and the CD4 count was 515 cells/mm³.

The presence of hypocomplementemia in our patient is unknown, but it is likely due to an immune-mediated process including in¯uenza, Coxsackie viruses and Epstein-Barr virus [9]. The role of HIV and other viruses in rhabdomyolysis is associated with a number of other acute viral infections, including in¯uenza, Coxsackie viruses and Epstein-Barr virus [10]. The role of HIV and other viruses in rhabdomyolysis is unknown, but it is likely due to an immune-mediated process [9]. The presence of hypocomplementemia in our patient is consistent with this.

In summary, our case and others suggest that patients with primary HIV infection can present with acute rhabdomyolysis, and that a diagnosis of primary HIV infection should be considered when a patient presents with rhabdomyolysis, even if there are no apparent risk factors.

Our case is unique in that our patient presented with acute rhabdomyolysis in the absence of other symptoms that would suggest primary HIV infection. The patients in the previous reports had other symptoms, including sore throat [4, 5, 7, 8], abdominal pain [4, 8], and diarrhea [6, 8]. In addition, the diagnosis was established in these other case studies through detection of HIV p24 antigen, whereas we established the diagnosis by measurement of the HIV RNA level by means of PCR analysis.

Acute rhabdomyolysis has also been described in patients who are in the later stages of HIV disease [9], and it has been associated with a number of other acute viral infections, including in¯uenza, Coxsackie viruses and Epstein-Barr virus [10]. The role of HIV and other viruses in rhabdomyolysis is unknown, but it is likely due to an immune-mediated process [9]. The presence of hypocomplementemia in our patient is consistent with this.

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References


