Adrenal Insufficiency and Mild Rhabdomyolysis Revealing a Human Immunodeficiency Virus Infection: A Case Report

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ABSTRACT

The spectrum of human immunodeficiency virus (HIV) endocrinopathy is large. Adrenal insufficiency (AI) is common in both early and late stages of HIV syndrome, resulting in significant morbidity and mortality. However, rhabdomyolysis is a muscle disease in which striated muscle fibers disintegrate, excreting myoglobin in the urine, leading to acute kidney failure. It is currently reported that rhabdomyolysis may be a direct result of the HIV infection, especially in the advanced stages of HIV/AIDS disease. This case study reports AI and a mild rhabdomyolysis in a patient with no medical history and no medicine or toxic intake. Laboratory assessment revealed HIV infection.

Key words: Adrenal insufficiency, human immunodeficiency virus, rhabdomyolysis

INTRODUCTION

It is well-established that human immunodeficiency virus (HIV) may cause functional disturbance of almost every part of the endocrine system. In fact, the spectrum of HIV endocrinopathy is wide. Adrenal insufficiency (AI) is common in both early and late stages of HIV syndrome, resulting in significant morbidity and mortality. However, rhabdomyolysis is a muscle disease in which striated muscle fibers disintegrate, excreting myoglobin in the urine, leading to acute kidney failure.

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In this article, we report the clinical case of a 45-year-old man who presented to our hospital with AI and mild rhabdomyolysis, and was found to have primary HIV infection. The possible underlying pathophysiologic mechanisms will be discussed.

**CASE REPORT**

A 45-year-old caucasian male was admitted to the emergency department of our hospital with asthenia and muscle pain. He had no significant medical history, and denied drugs or toxic plants intake. He reported anorexia with significant weight loss in recent months. He complained of diffuse muscle pain especially in the calves that started a few days before admission and diarrhea.

Upon physical examination, the patient was asthenic, emaciated with a body mass index at of 19 kg/m² (normal range: 18.5-24.9 kg/m²) dehydrated and hypotensive (80/60 mmHg). He was febrile (38.3°C) and oliguric.

Laboratory assessment, conducted in the emergency room concomitantly with the conditioning of the patient, found renal failure: High blood urea nitrogen levels at 2.31 g/l (0.15–0.45 g/l), serum creatinine at 20 mg/l (7–10 mg/l), with severe electrolyte disorders of hyponatremia at 113 mmol/l (135–145 mmol, hyperkalemia of 7 mmol/l (3.5–5 mmol/l) without signs on the electrocardiogram as well as hypoglycemic.

Hemogram showed anemia, leukopenia (blood white cells-3200/mm³, (3,500–10,500 cells/mm³) lymphopenia: 210 mm³ (1,000–3,500 cells/mm³). Moreover, elevated muscle enzymes-creatine phosphokinase of 1900 UI/L (25–195 UI/L), high aminotransferase and low hemoglobin were noted; urine analysis was positive for myoglobin. Stool test and culture revealed *Escherichia coli*.

Plasma corticotropin (ACTH) was elevated at 150 pg/mL (10 and 60 pg/mL), while morning Cortisol was low at 4 ug/dL (5–23 ug/dL), which is consistent with the diagnosis of primary AI.

Management included supportive care and correction of volume depletion and hypoglycemia with intravenous saline and dextrose as well as the medical treatment of hyperkalemia. Hydrocortisone was administered at a dose of 100 mg intravenously every 6 h then reduced. Fluoroquinolones were administered to treat the diarrhea. We observed an initial good response and the patient stabilized. Two days later, he developed a fever with worsening diarrhea.

Retroviral serology was positive on both ELISA and Western blot, with less than 200 CD4+ T cells per cubic millimeter of blood. Highly active retroviral therapy was decided, but, unfortunately, the patient died 4 days after admission due to severe septic shock.

**DISCUSSION**

This case reports AI and a mild rhabdomyolysis in a patient with an HIV infection, which was not known. In fact, AI is not uncommon in HIV-infected patients, especially those with an advanced stage of AIDS. Nevertheless, it is seldom diagnosed in practice since most patients, as in ours, present with non-specific clinical signs such as fatigue and hyponatremia. In hospitalized AIDS patients, biochemical presentation of AI is relatively common. Critical illness in these patients often complicates the assessment of adrenal function.

The incidence of AI in critically ill HIV-infected patients lacks accuracy, partly due to the different criteria used to diagnose AI.

Human immunodeficiency virus may either directly damage the glandular function or lead to secondary endocrine perturbation through opportunistic infections and neoplasms in the advanced stage of AIDS. In fact, the interaction between HIV and endocrine system is complex. It can present as mild biological disturbance to obvious glandular insufficiency. Medications, especially antiretroviral therapy may also lead to AI. In advanced stages of AIDS, ACTH levels increase over time, suggesting impaired adrenal reserve. However, rhabdomyolysis, is a life-threatening muscle lesion in which the breakdown of striated muscle fibers leads to the release of myoglobin into the bloodstream and then excreted in the urine. In fact, Rhabdomyolysis is one of the common causes of acute renal failure and can be associated with a variety of predisposing factors. In HIV patients, rhabdomyolysis is increasingly diagnosed and represents an additional cause of morbidity and mortality.

The usual clinical presentation in rhabdomyolysis includes muscle pain, weakness, and dark urine. Nonetheless, more than half of patients may not present with muscular complaints.

It has been reported that rhabdomyolysis may be a direct result of the HIV infection. Whereas, in the advanced stages of HIV/AIDS disease, patients...
often develop rhabdomyolysis-associated infections such as pneumocystis carinii, Toxoplasma gondii, *Staphylococcus aureus*, and *Cryptococcus*. Moreover, in these patients, other factors including illicit drug use, medications (Statins and HAART), alcohol abuse can be linked to striated muscle breakdown.\[^4,12\] HIV-associated rhabdomyolysis seems to be the consequence of an immunologically mediated injury rather than the direct viral invasion of the muscle.\[^4,11\]

The authors acknowledge that in the present case, the exact cause of rhabdomyolysis is unknown. Considering that this patient was neither under the lipid lowering agents nor HAART medications, it is very likely that this case of rhabdomyolysis was entirely secondary to HIV infection alone.

**CONCLUSION**

Adrenal insufficiency and rhabdomyolysis are not rare in AIDS patients. Mechanisms by which the virus directly alters the endocrine function are still not understood. Thus, practitioners involved in the management of HIV patients should be aware of these possible causes of complications in their patients.

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There are no conflicts of interest.

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