

## PAPER DETAILS

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## Acute HIV Induced Rhabdomyolysis: Not on Antiretroviral Therapy

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### ABSTRACT

Acute human immunodeficiency virus (HIV) infection is an underrecognized cause of acute rhabdomyolysis which leads to significant morbidity and mortality. Many cases are related to concomitant infections, or substance abuse, however acute HIV infection can also lead to rhabdomyolysis due to derangements in skeletal muscles from high viral load and electrolyte derangements. We present a case of a 26-year-old male without past medical history that presents with diffuse muscle pain and dark brown urine with CK>100,000 units/L without other confounding causes for rhabdomyolysis. *J Microbiol Infect Dis* 2020; 8(3):177-180.

**Keywords:** *Acute HIV, Rhabdomyolysis, Acute renal failure*

### INTRODUCTION

Acute human immunodeficiency virus (HIV) infection is defined by a negative HIV antibody test, with a concomitant elevated HIV RNA PCR level [1-2]. Unfortunately, acute HIV infection is often missed because it presents as a constellation of non-specific findings such as headache, nausea, fever, rash, pharyngitis, arthralgia, vomiting, myalgia and lymphadenopathy [1-5]. Rhabdomyolysis is increasingly recognized as an initial presentation of acute HIV infection, delays in aggressive treatment result in significant morbidity and mortality [1-5]. However, overall prognosis is good with supportive care [4].

### CASE REPORT

A 26-year-old male without past medical history presented to an outside hospital with complaints of dark brown urine and diffuse muscle pain for 24 hours. Social history was significant for sexual intercourse with 14 male partners over the past year, with intermittent condom use, many of whom were positive for HIV. He had several tattoos which were not professionally performed. No history of hepatitis, blood transfusions or intravenous drug use.

His initial examination was remarkable for fever 100.8°F and lymphadenopathy but otherwise unremarkable. He denied any preceding symptoms including mononucleosis-like symptoms, no heavy exercise, traumatic injury,

or travel. He denied any prescription or over-the-counter medications, herbal supplement, tobacco, alcohol, or illicit drug use. His laboratory revealed WBC 4.4, creatinine 1.1, AST 288 and ALT 55, total bilirubin 2.2 and creatine kinase (CK) >100,000units/L. UA was consistent with rhabdomyolysis with large amount of blood but no notable RBCs in the urine, (Table 1). His abdominal ultrasound was reported as normal, with a common biliary duct measuring 7 mm diameter and 3 hyperechoic structures within the liver measuring up to 15 mm in diameter which were thought to be benign hemangiomas. Pertinent negative labs and imaging findings included an acute hepatitis panel, HIV antibody, anti-nuclear antibody, chest X-ray, computed tomography scan of the abdomen and pelvis.

He was started on aggressive intravenous fluid resuscitation without significant improvement of his CK levels (trends noted on Table 2) at an outside hospital and continued to have persistent fevers therefore was transferred to a tertiary care hospital for further evaluation (Table 2).

During his hospital stay, he developed intermittent fevers ( $T_{max}$  102.3 °F), chills, and continued myalgias during his hospitalization. His infectious work up was negative and his fevers were attributed to viral myositis. With further aggressive intravenous fluid resuscitation, his CK improved. A repeat HIV antibody test was positive with an inconclusive

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HIV 1&2 confirmation and differentiation assay; his HIV RNA PCR had over 10 million copies of HIV virus, and absolute CD4 count with 265 cells. He was started on Bictegravir, Emtricitabine, and Tenofovir alafenamide with a significant decrease in his HIV RNA PCR of 5660 copies/ml within 2 weeks of starting cART, and ultimately viral suppression and rise in absolute CD4 in 750 range. After initiation of anti-retroviral therapy, he followed up in HIV Table 1. Urinalysis trends.

Clinic in one-month, his quantitative HIV decreased from >10 million copies to 5,660 copies, absolute CD4 count improved from 269 to 1389, CK improved from 69,473 to 158. At his four-month follow-up, he was found to be virally suppressed (quantitative HIV <20), with absolute CD4 count >800, and CK <200. He continues to be compliant with treatment and follow-up in HIV Clinic.

Parameters	HD 1	HD 2	HD 3	Reference
Color	Brown	Yellow	Yellow	
Appearance	Cloudy	Clear	Clear	
pH	6.5	6.5	6.5	[5.0-8.0]
Spec. Grav.	1.02	<1.1005	1.02	[1.000-1.030]
Glucose	Negative	Negative	Negative	[Negative]
Bilirubin	Negative	Negative	Negative	[Negative]
Ketones	15	Trace	Trace	[Negative]
Blood	Large	Large	Large	[Negative]
Protein	>300	30	30	[Negative]
Nitrite	Positive	Negative	Negative	[Negative]
Leukocyte Esterase	Trace	Negative	Negative	[Negative]
Urobilinogen	1	1	>8.0	[0.1-1.0]
WBC	15-25	5-Feb	2-Jan	
RBC	10-May	10-May	2-Jan	
Bacteria	Moderate	Few	Few	
Epithelial	Many	Trace	Trace	

Table 2. CPK, Kidney, and Liver Function Trends

Parameters	HD 1	HD 2	HD 3	HD 4	HD 5	HD 6	HD 7	HD 8	HD 9
CPK (Units/L)	50972	52868	>100,000	>100,000	161,360	148,510	143,075	111,196	69,473
AST (IU/L)	288	272	1430	645	1203	1328	1017	1044	731
ALT (IU/L)	55	53	605	141	554	621	478	485	393
T. Blr. (mg/dL)	2.2	2	1.7	1.8	2	2.1	1.8	1.9	1.5
ALP (IU/L)	42	36	55	40	66	76	63	72	62
Creatinine (mg/dL)	1.1	1	0.7	0.7	0.74	0.72	0.75	0.75	0.71

## DISCUSSION

Acute HIV infection often presents with non-specific symptoms now termed "acute retroviral syndrome", which appears 2-6 weeks after initial infection, making the diagnosis difficult and often missed [1-5]. Laboratory findings are often non-specific as well with leukopenia, thrombocytopenia, elevated transaminase levels and elevated erythrocyte sedimentation rate being the most common [1-2,4]. Acute HIV infection is diagnosed when a high viral RNA level is detected in conjunction with a negative HIV-1 antibody test [1]. Many manifestations of renal disease are seen in late stage HIV or acquired immunodeficiency syndrome (AIDS) such as focal segmental glomerulosclerosis, minimal change disease, and membranous nephropathy have been reported [3-4]. Several viral infections including "influenzas A and B, Coxsackie virus B5, echoviruses 6 and 9, adenovirus 21, herpes simplex, and Epstein-Barr virus" have reported complications of rhabdomyolysis [4].

HIV associated rhabdomyolysis has been reported in literature, but often associated with other concomitant disease states that precipitate muscle breakdown, in a small case series of 20 HIV+ patients, 7 had acute HIV associated rhabdomyolysis, 6 had drug induced rhabdomyolysis secondary to prescription medications such as antiretroviral therapy, or antibiotics, and the remaining 7 had secondary infections known to cause rhabdomyolysis [6]. In another case series of 7 patients, all had other confounding variables such as viral, bacterial or fungal infection, initiation of antiretroviral medications, intravenous drug abuse, or alcohol abuse which are all known precipitants for rhabdomyolysis [1,3,6-7]. Isolated rhabdomyolysis as the presenting symptom of acute HIV infection remains a relatively rare occurrence without concomitant secondary causes of rhabdomyolysis [5,8].

A retrospective study performed by Koubar et al. analyzed the John Hopkins HIV clinical registry with 7079 patients (both newly diagnosed and known infections) with 362 identified as having rhabdomyolysis each with a confounding cause for rhabdomyolysis beyond isolated HIV infection [9]. It was also noted that regardless of the cause of rhabdomyolysis this resulted in a

1.5-4-fold increase in the rate of death compared to the general population [9]. Further, the significant healthcare expenditure associated with HIV associated rhabdomyolysis is significant with the average length of hospitalization approaching 20 days [6].

The mechanism of HIV associated rhabdomyolysis (without other confounding variables) may be associated with HIV myopathy [6]. The involvement of HIV in skeletal muscle is pervasive, with a small study indicating approximately 2/3 (64 of 92) cases had changes in histopathology of the skeletal muscle [6].

Due to the severe morbidity and mortality associated with rhabdomyolysis (severe electrolyte imbalance, and acute renal failure) in the HIV population, prompt recognition is important to expedite and initiate treatment [10]. Increasingly recognized, rhabdomyolysis in an otherwise healthy individual with high risk sexual behaviors may warrant further investigation of acute HIV infection as the cause of their rhabdomyolysis.

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## REFERENCES

1. McDonagh CA, Holman RP. Primary human immunodeficiency virus type 1 infection in a patient with acute rhabdomyolysis. *South Med J* 2003; 96(10):1027-30.
2. Douvoyiannis M, Litman N. Acute encephalopathy and multi-organ involvement with rhabdomyolysis during primary HIV infection. *Int J Infect Dis* 2009; 13(5):e299-304.
3. Prabahar MR, Jain M, Chandrasekaran V, Indhumathi E, Soundararajan P. Primary HIV infection presenting as non-traumatic rhabdomyolysis with acute renal failure. *Saudi J Kidney Dis Transpl* 2008; 19(4):636-42.
4. Del Rio C, Soffer O, Widell JL, Judd RL, Slade BA. Acute human immunodeficiency virus infection temporally associated with rhabdomyolysis, acute renal failure, and nephrosis. *Rev Infect Dis* 1990; 12(2):282-5.
5. Rastegar D, Claiborne C, Fleisher A, Matsumoto A. A patient with primary human immunodeficiency virus infection who presented with acute rhabdomyolysis. *Clin Infect Dis* 2001; 32(3):502-4.

6. Joshi MK, Liu HH. Acute rhabdomyolysis and renal failure in HIV-infected patients: risk factors, presentation, and pathophysiology. *AIDS Patient Care STDS* 2000; 14(10):541-8.
7. Delo D, Brett AS, Postic B. Primary HIV infection presenting with acute rhabdomyolysis. *Am J Med Sci* 2006; 332(1):46-7.
8. Same RG, Mcaleese S, Agwu AL, Arav-boger R. Acute HIV in an Adolescent Male with Fever and Rhabdomyolysis. *J Adolesc Health* 2019;65(4):567-569.
9. Koubar SH, Estrella MM, Warriar R, et al. Rhabdomyolysis in an HIV cohort: epidemiology, causes and outcomes. *BMC Nephrol* 2017; 18(1):242.
10. Noe MM, Jain AG, Shahid S, Majeed U. Severe Rhabdomyolysis as an Unusual Presentation of Primary Human Immunodeficiency Virus Infection. *Cureus* 2018; 10(7):e3041.