Red-Brown Urine in a Patient with Chronic HIV Infection and Quadriparesis

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CASE DESCRIPTION

A 42-year-old woman with chronic HIV infection presented with sudden onset of progressive limb weakness, leading to immobility within 4 days. This was preceded by severe abdominal pain, nausea, and vomiting for 2 days and episodes of confusion and agitation.

Six weeks prior, she had commenced highly active antiretroviral therapy (HAART), consisting of efavirenz/emtricitabine/tenofovir and cotrimoxazole for opportunistic infection prophylaxis. Additional history included constipation for 4 weeks and an admission for psychiatric symptoms 1 week before starting HAART. She had declined HAART when HIV infection was diagnosed 6 years earlier, but was successfully treated for multidrug-resistant tuberculosis.

Examination revealed bilateral facial-nerve palsies, quadriparesis, global areflexia, absence of proprioception, and patchy loss of sensation below T4. MRI excluded focal lesions and progressive multifocal leukoencephalopathy. The patient was apyrexial and normotensive with a tachycardia of 133 beats per minute and diaphoresis. Cerebrospinal fluid (CSF) showed increased total protein of 1.25 g/L (reference interval 0.15– 0.45 g/L) without white blood cells and no evidence of opportunistic infection (Table 1). Complete blood count, and renal and liver function tests were unremarkable except for hypoalbuminemia of 28 g/L (reference interval 35–52 g/L). Vitamin B12 was within the reference interval and serum iron studies suggested anemia of chronic disease. Serology was negative for hepatitis A and C, indicating only past hepatitis B infection. Tests for cytomegalovirus and syphilis were negative.

Since commencing HAART, the CD4 count had risen from 79 to 109 x 10^6/L and the HIV viral load had declined from 105 224 to 235 copies/mL (decrease of 2.65 log). This prompted consideration of the Guillain-Barré syndrome (GBS) variant of neurological immune reconstitution inflammatory syndrome (IRIS). In spite of a 5-day course of intravenous immunoglobulin, the clinical condition deteriorated, culminating in respiratory distress necessitating mechanical ventilation. Antiganglioside antibodies were negative and examination of the stool excluded infection with *Campylobacter* and other pathogens (Table 1). During the patient's stay in the ward, her urine was noted to have a

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red-brown color not explained by myoglobinuria because creatine kinase was only slightly increased at 229 U/L (reference interval 20 –180 U/L). However, microscopy of catheter urine samples demonstrated high numbers of leukocytes, erythrocytes, and bacteria, consistent with urinary tract infection.

Table 1. Laboratory investigations in the diagnostic workup.

CSF protein	1.25 g/L[a]
CSF cryptococcal latex antigen and India ink stain	Negative
CSF microscopy and cultures (bacterial, fungal, mycobacterial)	Negative
CSF viral screen (enterovirus, herpes simplex 1&2 PCR)	Negative
Syphilis serology (rapid plasma reagin/*Treponemal* antibodies)	Negative
Antiganglioside antibodies	Negative
Hepatitis C and A virus serology	Negative
Cytomegalovirus viral load	Not detected
Mycobacterium tuberculosis (GeneXpert real-time PCR on tracheal aspirate)	Negative
Stool pathogens (*Salmonella*, *Shigella*, *Escherichia coli* O157: H7, *Campylobacter*)	Negative
Fecal occult blood (Actim® immunochromatographic fecal blood test)	Negative

[a] Reference interval 0.15–0.45 g/L.

QUESTIONS TO CONSIDER
• What are the biochemical causes of red-brown urine?
• Can IRIS present with acute motor axonal neuropathy and acute neurovisceral crisis?
• What condition may be precipitated by HAART and/or cotrimoxazole therapy of HIV infection?

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Final Publication and Comments

The final published version with discussion and comments from the experts will appear in the September 2016 issue of *Clinical Chemistry*. To view the case and comments online, go to http://www.clinchem.org/content/vol62/issue9 and follow the link to the Clinical Case Study and Commentaries.

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