Fulminant Leptospirosis in a Patient with Human Immunodeficiency Virus Infection: Case Report and Review of the Literature

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We report a case of fulminant leptospirosis that was acquired in New York City by a patient with underlying infection with human immunodeficiency virus (HIV). Review of the literature on leptospirosis in HIV-infected persons showed that all patients were severely ill but responded well to treatment, which highlights the importance of recognizing this potentially life-threatening illness, especially in unusual settings.

Leptospirosis is a zoonosis caused by spirochetes from the genus *Leptospira* and is typically transmitted via contact with urine from infected animals. The clinical syndrome of leptospirosis may be mild and self-limited or fulminant with jaundice, renal failure, and hemorrhage (Weil's syndrome) [1]. Although leptospirosis is typically associated with rural settings because of exposure to potentially infected livestock, cases have been reported in urban locations [2–4]. There are few reported cases of leptospirosis in patients with HIV infection. We report a case of fulminant leptospirosis acquired in New York City in a patient with HIV infection, and we review the literature about leptospirosis in HIV-infected persons [5–7].

**Case report.** A 33-year-old homeless Latino man with AIDS (CD4 cell count, 16 cells/mm³) presented to the emergency department with multiple nonspecific complaints, including nausea, vomiting, sore throat, cough, and generalized malaise. The findings of an examination in the emergency department were thought to be unremarkable, and results of a complete blood count and chemistry panel were within normal limits. The patient was discharged with supportive care for a presumed viral syndrome.

One week later, the patient returned with persistent symptoms now complicated by decreased urine output and jaundice. The patient denied taking medications other than occasional acetaminophen. On examination, he was afebrile, his initial blood pressure was 70/50 mm Hg, and his heart rate was 74 beats per min. His blood pressure improved to 105/71 mm Hg after resuscitation with 3 L of fluid. The findings of an examination were remarkable for icteric conjunctivae without suffusion and bibasilar rales. His skin was intact, except for visible track marks on his extremities. He was neurologically intact without any meningeal signs. The following laboratory values were significant: acetaminophen level, <10 µg/mL; creatinine level, 12.8 mg/dL; platelet count, 57,000 cells/mm³; total bilirubin level, 11.3 mg/dL; prothrombin time, 11.5 s; alkaline phosphatase level, 134 U/L; and alanine aminotransferase level, 27 U/L. His chest radiograph showed mild pulmonary congestion, and an electrocardiogram revealed T wave inversions of unknown duration in V1–V3. The findings of an abdominal ultrasound were unremarkable.

A non–Q wave infarction was diagnosed and hemodialysis was begun. Two days after admission, the patient had acute decompensation, with fever (temperature to 38.8°C) and acute respiratory distress that required intubation. A second chest radiograph revealed bilateral, diffuse alveolar infiltrates that were consistent with acute respiratory distress syndrome. The patient received multiple antibiotics, including vancomycin, cefazidime, trimethoprim-sulfamethoxazole, and methylprednisolone sodium succinate, for treatment of sepsis and possible *Pneumocystis carinii* pneumonia. The patient’s condition continued to deteriorate, and he required pressor support. He developed significant bleeding from his nose and mouth despite normal coagulation parameters, which did not respond to platelet transfusion or desmopressin. Two days after his decompensation, treatment with penicillin was begun for the possibility of leptospirosis. Three days after the initiation of penicillin therapy, pressors were discontinued, the patient had defervescence, his respiratory status improved, and the bleeding abated. All other antibiotics and steroids were discontinued.

Five days after penicillin was initiated, the patient developed a presumed drug rash and his treatment was changed to doxycycline. The patient continued to improve slowly and was discharged on hospital day 38, by which time he was no longer dependent on dialysis but was left with residual renal insufficiency (creatinine level of 3.6 mg/dL). A microscopic aggluti-
Table 1. Clinical features of 5 cases of leptospirosis in HIV-infected patients.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age, years</th>
<th>CD4 count, cells/mm³</th>
<th>Jaundice</th>
<th>Organism(s) isolated</th>
<th>Symptoms</th>
<th>Outcome</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 [5]</td>
<td>22</td>
<td>NA</td>
<td>Yes</td>
<td><em>L. interrogans</em> serovar <em>copenhagenii</em>, <em>L. interrogans</em> serovar <em>icterohaemorrhagiae</em></td>
<td>Flu-like symptoms, renal insufficiency, thrombocytopenia, meningitis</td>
<td>Recovered</td>
<td>NA</td>
</tr>
<tr>
<td>3 [6]</td>
<td>NA</td>
<td>13</td>
<td>Yes</td>
<td><em>L. interrogans</em></td>
<td>Acute renal failure, myalgia, thrombocytopenia, hypertension</td>
<td>Died at 3 months (unrelated causes)</td>
<td>Tetracycline</td>
</tr>
<tr>
<td>4 [7]</td>
<td>30</td>
<td>60</td>
<td>Yes</td>
<td><em>L. interrogans</em> serovar <em>icterohaemorrhagiae</em></td>
<td>Renal failure, first-degree atrioventricular block, thrombocytopenia, hypertension</td>
<td>Recovered</td>
<td>Ceftriaxone, amoxicillin, ciprofloxacin</td>
</tr>
<tr>
<td>5 (PR)</td>
<td>33</td>
<td>16</td>
<td>Yes</td>
<td><em>L. interrogans</em> serovar <em>copenhagenii</em>, <em>L. interrogans</em> serovar <em>bratislava</em></td>
<td>Myocarditis, thrombocytopenia, acute respiratory distress syndrome, hypotension, renal failure</td>
<td>Recovered, residual renal dysfunction</td>
<td>Penicillin, doxycycline</td>
</tr>
</tbody>
</table>

**NOTE.** All patients were male. NA, not available; PR, present report.

...with the fulminant icteric form of leptospirosis. In general, 5%–10% of patients with leptospirosis present with the more severe icteric form that is known as Weil's syndrome [1]. It is not possible to say from such a small sampling whether HIV infection predisposes to more fulminant disease. Milder cases may escape diagnosis, and the tendency toward severe disease in the cases that were reviewed may reflect reporting bias. The patient in this report, in addition to having the classical biphasic course of hemorrhagic leptospirosis with hepatic and renal involvement, also presented with severe manifestations of some of the less frequently seen manifestations; these include pulmonary involvement [8–10], myocarditis, and severe bleeding due to vasculitis [1]. The mortality rate for patients with Weil's disease has been reported to be as high as 25%. However, with improvements in intensive supportive therapy, the mortality rate has decreased to 5%–10% [1]. All patients who were reviewed recovered with treatment, which underscores the importance of a high index of suspicion and prompt institution of treatment.

**References**