Hematospermia Due to Schistosome Infection in Travelers

Sir—Schwartz et al. [1] highlight hematospermia as a presenting symptom of schistosomiasis and comment that this presentation seems to be rare among travelers with schistosomiasis, although it is perhaps more common than is generally perceived. Indeed, several reports other than the 2 mentioned in their article have documented this presentation in returning travelers [2–4]. It is important to note that hematospermia is only one alteration in semen quality associated with male genital schistosomiasis. Other changes include yellow (rather than red or brownish) discoloration and reduction in viscosity to a “watery” consistency [2, 5, 6], and these symptoms may be more common than is hematospermia.

Subjective change in semen quality was a relatively common presentation among a series of returned travelers with schistosomiasis in New Zealand, being present in 7 of 13 males [2, 5]. All 7 had swum in Lake Malawi in southern Africa during the preceding 12 months, were infected with Schistosoma haematobium, and responded to treatment with praziquantel. Three also described testicular ache or urethral tingling.

Interestingly, all 7 presented to a sexual health clinic with changes in their ejaculate as their principal symptom. This is an unusual presenting symptom, even in sexual health clinics, and should prompt a high suspicion of schistosomiasis for anyone who has visited an area of endemicity. Furthermore, this symptom is likely to be reported more frequently if specifically sought in males with suspected schistosomiasis.

References


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Hepatitis C Virus Persistence Suppressed by HAART

Sir—We read with great interest the review by Cooper et al. [1] on the effect of highly active antiretroviral therapy (HAART) on hepatitis C virus (HCV) RNA in human immunodeficiency virus (HIV) and HCV co-infection. The authors found that the initiation of HIV treatment might cause an initial increase of HCV RNA levels (accompanied or not by elevated transaminase levels) followed by a decrease to levels lower than those before treatment. This response might be due to the so-called “immunorestoration syndrome.” Our data agree with those of Puoti et al. [2], but we would like to report a singular case of complete and persistent suppression of HCV RNA levels and normalization of serum transaminase levels in a patient coinfected with HIV and HCV who was receiving HAART.

In May 2000, a 39-year-old homosexual man was admitted to our department with a persistent fever (temperature, >38°C), weight loss of >15% of initial weight, vomiting, diarrhea, and myalgias. We found that the patient was seropositive (which was unknown before hospitalization) and diagnosed AIDS-related wasting syndrome (category C3, according to the classification of the Center for Disease Control and Prevention [Atlanta, GA] [3]). We also detected HCV antibodies in serum samples.

At the time of diagnosis, laboratory values were as follows: CD4 T cell count, 140 cells/mm³; HIV RNA in plasma (as detected with the Amplifor HIV Monitor Test [Roche Diagnostic System]), 750,000 copies/mL; amino alanine transferase (ALT), 89 U/L; aspartate alanine transferase (AST), 120 U/L; γ-glutamyl transpeptidase, 172 IU; HCV RNA in plasma (as determined by Amplifor HIV Monitor Test), 714,000 copies/mL. The HCV genotype was 1b. Liver ultrasonography revealed medium-grade steatosis. The patient did not consent to undergo liver biopsy.

We started HAART therapy with zidovudine (600 mg daily), efavirenz (600 mg daily), and lamivudine (300 mg daily). The treatment was well tolerated by the patient, who showed a strict adherence to therapy.

The patient’s CD4 T cell count showed a progressive increase (245 cells/mm³ in

References

July 2000), while HIV RNA levels decreased to undetectable levels during the same period. Both serum transaminase and HCV RNA levels showed an initial increase (in July 2000, the AST level was 111 U/L, the ALT level was 152 U/L, and the HCV RNA level was 752,000 copies/mL) and then a progressive decrease, a finding that is in agreement with data from the literature [1, 2]. In April 2002, the patient’s CD4 T cell count was 681 cells/mm³, HIV RNA levels were still suppressed (<50 copies mL⁻¹), serum transaminase levels were normal (AST level, 24 U/L; ALT level, 20 U/L) and the HCV RNA levels were undetectable. In blood samples obtained in June 2002 and September 2002, HCV RNA levels were still suppressed and serum transaminase levels were stable. Liver ultrasonography performed in June 2002 showed low-grade steatosis.

In short, the patient showed a persistent suppression of HCV viremia by HAART. Therefore, we suggest that HAART may suppress HCV viremia, that lamivudine may be effective against the replication of HCV as well as hepatitis B virus (HBV), and that lamivudine may be effective against infection with HCV genotype 1b, which is the most difficult to treat.

We will keep our patient under continuous monitoring. The natural history of HCV RNA levels in HIV-HCV coinfected individuals needs to be fully defined, possibly through prospective studies.

### Table 1. Association between clinically significant abnormal liver function test (LFT) results, irrespective of whether baseline values were normal or abnormal.

<table>
<thead>
<tr>
<th>Laboratory parameter</th>
<th>Aspergillus</th>
<th>Esophageal candidiasis</th>
<th>Febrile neutropenia</th>
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</thead>
<tbody>
<tr>
<td>LFT value</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total bilirubin &gt;1.5× ULN</td>
<td>35/180 (19.4)</td>
<td>27/138 (19.6)</td>
<td>8/185 (4.3)</td>
</tr>
<tr>
<td>AST &gt;3× ULN</td>
<td>21/180 (11.7)</td>
<td>7/136 (5.1)</td>
<td>38/187 (20.3)</td>
</tr>
<tr>
<td>ALT &gt;3× ULN</td>
<td>34/180 (18.9)</td>
<td>24/134 (17.9)</td>
<td>20/187 (10.7)</td>
</tr>
<tr>
<td>ALP &gt;3× ULN</td>
<td>29/181 (16.0)</td>
<td>23/134 (17.2)</td>
<td>19/187 (10.2)</td>
</tr>
<tr>
<td>Duration of treatment, median days (range)</td>
<td>73 (2–288)</td>
<td>12 (1–84)</td>
<td>14 (1–45)</td>
</tr>
</tbody>
</table>

**NOTE.** Data are no. of patients with abnormality/no. of patients (%), unless otherwise indicated. ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ULN, upper limit of normal. Data are from [2–4] and on file at Pfizer.

### References

### Safety of Voriconazole and Dose Individualization

**Sir—**In the 15 November issue of *Clinical Infectious Diseases*, Potoski and Brown [1] suggested there is a need for individual voriconazole dosage modification based on plasma concentrations. Their concerns focus on the possibility of voriconazole-related liver toxicity.

More than 2000 patients, most of whom were severely immunocompromised, were treated with voriconazole during the clinical development program, which provided an extensive database for safety analysis. The data on clinically significant abnormal liver function test (LFT) results from 3 pivotal comparative studies [2–4] are presented in table 1. Although the frequency of abnormal LFT results was relatively high, the abnormalities themselves were mostly mild to moderate in severity and led to the discontinuation of voriconazole therapy only rarely (for 3% of patients). Only in patients with esophageal candidiasis was the frequency of abnormal LFT results greater among patients treated with voriconazole than among those treated with fluconazole. Whether these data show that voriconazole is potentially more hepatotoxic than other azoles, as suggested by Potoski and Brown [1], remains to be established. Patients with invasive aspergillosis or febrile neutropenia demonstrated higher frequencies of abnormal LFT results overall than did patients with esophageal candidiasis. However, no differences were found in the frequency of such abnormalities between patients treated with voriconazole and those receiving amphotericin B formulations. This observation occurred even though the duration of exposure to vor-