MacConkey’s agar. The organisms test positive for oxidase, catalase, and indole; are nonpigmented; and produce urease [2]. The organisms are a component of the normal oral flora of dogs and other animals, and most clinical isolates are recovered from bite wounds [3]. Although few cases of *B. zoohelcum* bacteremia have been reported in the literature, most cases have been have been associated with leg abscesses [4], septicemia [5], tenosynovitis [6], meningitis [7], and pneumonia [8]. The species is susceptible to β-lactam antibiotics, fluoroquinolones, and chloramphenicol, and it has variable susceptibility to trimethoprim-sulfamethoxazole and tetracycline.

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**References**


**Opportunistic Parasitic Infections of the Intestinal Tract in the Era of Highly Active Antiretroviral Therapy: Is the CD4+ Count So Important?**

SIR—Opportunistic parasitic infections of the gastrointestinal tract frequently occur in patients with AIDS. In previous reports [1–3], we have described the epidemiology and clinical characteristics of cryptosporidiosis, microsporidiosis, and isosporiasis among patients with AIDS in our geographic region. However, during the last 3 years, the incidences of these diseases have sharply decreased, mainly because of the introduction of highly active antiretroviral therapy (HAART). Immediately after the introduction of this potent combination therapy, we observed its positive effect on the clearance of parasites in patients with cryptosporidiosis or microsporidiosis [4] who were being treated with antiprotozoal drugs. Currently, these diseases are rarely observed in our center. We describe 5 cases of opportunistic enteric infection (1 case of isosporiasis and 4 cases of cryptosporidiosis) in patients who demonstrated a poor virological and immunological response to HAART.

The 5 patients were 3 men and 2 women aged 32–61 years. Three patients were injection drug addicts, and 2 were sexual partners of HIV-infected patients. All 5 patients had late-stage HIV infection (stage B3 or C3) with very low CD4+ counts (range, 14–95 cells/mm³) and virus loads of 100,000–540,000 copies/mm³ (as determined by use of nucleic acid sequence–based amplification).

With regard to treatment, all patients had already received extensive antiretroviral therapy and were experiencing therapy failure (table 1). After the detection of parasites in stool samples obtained from the patients, antiprotozoal therapy was initiated. This treatment resulted in the clinical and microbiological resolution of the infection in all patients except patient 2 (table 1). Patient 2 had a relapse of symptoms after 10 days of treatment; however, the patient admitted to an irregular intake of paromomycin. When paromomycin was administered again, a stable remission of symptoms was observed. All patients were followed up for at least 6 months, and, until now, no relapses have been observed.

Cryptosporidiosis and isosporiasis are infections associated with HIV infection that occur in patients with a low CD4+ lymphocyte count. These opportunistic infections can lead to chronic diarrhea that responds poorly to antimicrobial therapy, and failures or relapses occur frequently. As shown in table 1, the 5 cases we describe all occurred in patients who had experienced multiple failures of drug therapy and had a marked immune deficiency and a very high virus load. Nevertheless, in spite of the severe condition of these patients, antimicrobial therapy resulted in a rapid and definitive clearance of protozoa from their stool.

Therefore, we hypothesize that antiretroviral therapy, even if poorly effective against HIV infection, can exert a certain degree of protection against parasitic diseases. In fact, previous studies [5–6] have demonstrated that microsporidiosis and cryptosporidiosis improve after the initiation of HAART. In addition, a recent report documents remission of microsporidial keratoconjunctivitis in a patient with AIDS who was treated with antiretrovirals, even in the absence of a specific antiprotozoal therapy [7]. The mechanism of this response is not clear, but it might involve IFN-γ, which has been demonstrated to be the major inhibitor of *Cryptosporidium parvum* infection [8, 9]. Therefore, because protease inhibitors increase the production of IFN-γ and IL-2, they could, theoretically, also exert a
Table 1. The effect of highly active antiretroviral therapy on opportunistic isosporiasis and cryptosporidiosis in 5 patients infected with HIV.

<table>
<thead>
<tr>
<th>Patient</th>
<th>CD4⁺ count, cells/mm³</th>
<th>Regimens</th>
<th>Duration, months</th>
<th>Reason for change of therapy</th>
<th>Disease</th>
<th>Symptom(s)</th>
<th>Antiprotozoal therapy</th>
<th>Duration, months, by diagnostic criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>73</td>
<td>AZT, 3TC, d4T, Ind, Rtv 3TC, d4T, Nvp</td>
<td>11</td>
<td>Therapy failure</td>
<td>Isosporiasis</td>
<td>Diarrhea, nausea, vomiting</td>
<td>Cotrimoxazole</td>
<td>4 4</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>AZT, d4T, ddl, Rtv, Sqv AZT, 3TC, Sqv AZT, 3TC, Ind AZT, 3TC, Ind, Rtv</td>
<td>12, 3, 2, 5</td>
<td>Poor compliance</td>
<td>Cryptosporidiosis</td>
<td>Diarrhea</td>
<td>Paromomycin</td>
<td>5 6</td>
</tr>
<tr>
<td>3</td>
<td>19</td>
<td>3TC, d4T, Sqv AZT, 3TC, Nfv d4T, Nvp, Ind, Rtv</td>
<td>24, 12, Ongoing</td>
<td>Therapy failure</td>
<td>Cryptosporidiosis</td>
<td>Diarrhea, cramps</td>
<td>Azithromycin</td>
<td>4 5</td>
</tr>
<tr>
<td>4</td>
<td>14</td>
<td>ddl d4T, 3TC, Sqv</td>
<td>24</td>
<td>Therapy failure</td>
<td>Cryptosporidiosis</td>
<td>Diarrhea, fever</td>
<td>Azithromycin</td>
<td>2 3</td>
</tr>
<tr>
<td>5</td>
<td>95</td>
<td>AZT AZT, 3TC d4T, ddl, Rtv d4T, ddl, Ind d4T, Nvp, Nfv</td>
<td>15, 12, 7, 10, Ongoing</td>
<td>Therapy failure</td>
<td>Cryptosporidiosis</td>
<td>Diarrhea</td>
<td>Paromomycin</td>
<td>4 4</td>
</tr>
</tbody>
</table>

NOTE. AZT, azidothymidine; ddl, didanosine; Ind, indinavir; 3TC, lamivudine; Nvp, nevirapine; Nfv, nelfinavir; Rtv, ritonavir; Sqv, saquinavir; d4T, stavudine.

* Dosages were as follows: cotrimoxazole, 1 tablet q.i.d. for 10 days then 1 tablet b.i.d. for 3 weeks; paromomycin, 1 g b.i.d. for 4 weeks; azithromycin, 600 mg per day for 4 weeks.
positive effect on opportunistic microsporidiosis and cryptosporidiosis [10]. If these observations are confirmed in a larger patient cohort, the combination of HAART and appropriate antimicrobial treatment might be able to resolve such opportunistic infections, independent of the CD4+ lymphocyte cell count.

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References

Desmopressin Treatment for a Case of Dengue Hemorrhagic Fever/Dengue Shock Syndrome

Str—Dengue fever presents as a benign viral illness or may progress to dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS). Since 1988 we have endured 4 epidemics of dengue in French Polynesia that have mainly affected children—the so-called “Southeast Asian” epidemiological pattern. Ten children who were admitted to critical care units for treatment of uncontrolled DHF/DSS died during the 1989–1990 outbreak. Since then, we have had no more deaths to deplore. In cases of DHF/DSS, thrombocytopenia is a major concern because of the risk of hemorrhage. Another risk is hypovolemic shock due to increased vascular permeability and massive plasma leakage requiring intensive support [1, 2]. Hypovolemic shock develops in 24 h approximately 5–8 days after the onset of fever; it lasts up to 3 days, a duration that is predictable [3]. If the vital functions of the patient can be sustained during this period and there are no complications, the patient will recover.

For all of our patients, we had to infuse large amounts of fluid for hemodynamic assistance and to maintain diuresis. The infused fluids leaked from the vascular beds and induced generalized edema, causing weight gain that was up to 25%–30% greater than initial body weight; the infused fluids also produced major serious effusions and impaired respiratory function. Therefore, our aim was to restore a functional vascular barrier to avoid these complications and to reduce the risk of thrombocytopenia.

A 7-year-old boy with grade III DHF/DSS was admitted to the hospital on the fourth day after the onset of symptoms. He was afibrile and had abdominal pain, oral intolerance, and pleural effusion. Laboratory data were as follows: pulse, 130–140 beats/min; blood pressure, 110/90 mm Hg; platelet count, 21 × 109 cells/L, with purpura; hematocrit, 52%; albumin, 31 g/L; sodium, 126 mEq/L; and urea, 11.2 mM/L. In addition to administering initial infusions (50 mL/kg over 2 h), we introduced desmopressin (1-deamino-8–d-arginine vasopressin, or DDAVP; 0.3 μg/kg over 30 min). Three hours later, the pulse rate slowed to 80–90 beats/min and the blood pressure differential increased to 100/50 mm Hg and remained normal thereafter. Desmopressin was administered for 3 days (once daily at the same dosage: 0.3 μg/kg), and we monitored the level of sodium and the fluid balance. Daily fluid and caloric maintenance was done with iso-osmotic infusions. Diuresis increased from 500 mL on the fourth day of symptoms (the day of admission) to 1600 mL on the fifth day and 3000 mL on the sixth day. On the eighth day of symptoms, the platelet count was 90 × 109 cells/L, the pleural effusion had decreased, and there was no bleeding.

Since we treated this child, 3 more children with DHF/DSS have been successfully treated in the same manner. One of the children had a digestive tract hemorrhage that stopped after infusion of desmopressin.

It has been proposed that blood products should be infused for bleeding associated with DHF/DSS, but little attention has been paid to alternative treatments or prevention. Because of its ability to prevent hemorrhages and allow surgical procedures, desmopressin is used to treat some hereditary bleeding disorders, and it also may be used to treat acquired impaired hemostatic conditions [4, 5]. Desmopressin shortens prolonged bleeding times, releases endothelial hemostatic factors, and, in vitro, promotes the adhesion of platelets to the vascular sub-