Table 1. Summary of culture-proven cases of gastrointestinal basidiobolomycosis.

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sex/age (y)</th>
<th>Risk factor(s)</th>
<th>Symptoms</th>
<th>Diagnostic material</th>
<th>Histopathology</th>
<th>Organism cultured</th>
<th>Treatment (dosage), duration</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[1]</td>
<td>M/4</td>
<td>None</td>
<td>Abdominal pain, fever, heavy sweats, diarrhea</td>
<td>Tissue from gastrointestinal junction and colon</td>
<td>Epithelial cell and foreign body giant cell granuloma, Splendore-Hoeppli phenomenon around hyphae</td>
<td>Basidiobolus ranarum (as Basidiobolus haptosporus)</td>
<td>None</td>
<td>Died</td>
</tr>
<tr>
<td>[2]</td>
<td>M/69</td>
<td>Anergy, diabetes</td>
<td>Fever, nausea, constipation, bilious vomiting, lower quadrant pain</td>
<td>Tissue from cecum, ascending colon, part of duodenum</td>
<td>Prominent eosinophilic infiltration with histiocytes</td>
<td>B. ranarum (as B. haptosporus)</td>
<td>Amphotericin B, 2 w</td>
<td>Died</td>
</tr>
<tr>
<td>[PR]</td>
<td>M/30</td>
<td>None</td>
<td>Rectal bleeding, constipation</td>
<td>Biopsy specimen from rectal wall</td>
<td>Marked eosinophilic infiltration, few neutrophils and lymphoplasmocytic cells</td>
<td>B. ranarum</td>
<td>Amphotericin B (700 mg) and ketoconazole (200 mg/d), 2 w (incomplete treatment)</td>
<td>Left the country</td>
</tr>
</tbody>
</table>

NOTE. PR = present report.

References


Severe Allergic Reaction After Repeated Exposure to Indinavir

To our knowledge, severe allergic reactions to indinavir with hypotension, rash, fever and elevation in liver enzyme levels (ELELs) have not been reported [1]. We describe a patient with a severe clinical picture of a drug-induced allergic reaction after exposure (repeated) to indinavir.

A 40-year-old male homosexual patient with AIDS (CD4+ lymphocyte count, 20/µm³) started receiving a triple-therapy (TT) regimen with stavudine, lamivudine, and indinavir together with oral ganciclovir, itraconazole, pentamidine, and temazepam after being hospitalized for iv treatment of cytomegalovirus (CMV) retinitis with ganciclovir. He was rehospitalized 22 days later with fever (temperature, 39°C), icterus, hepatosplenomegaly, and ELELs. Two of six blood cultures yielded Staphylococcus epidermidis, and treatment with fluconoxacillin was instituted. TT and itraconazole were stopped. Serology for CMV in the buffy coat was negative as was serology for hepatitis A and B. Mycobacterial blood cultures remained negative. Funduscopic examination showed stable disease.

After 5 days of treatment with fluconoxacillin, there was normalization of the liver enzyme levels and disappearance of the fever and icterus; TT was reinstituted at 1500 hours. Five hours later the patient developed a diffuse rash, hypotension (blood pressure, 85/50 mm Hg), fever (temperature, 40.2°C), and diarrhea. Again there were ELELs and new neutropenia (200 cells/µm³; previous values, >1,200/µm³). Treatment with fluconoxacillin was discontinued because of possible penicillin intolerance in the past and was replaced by vancomycin, amikacin, and granulocyte colony-stimulating factor (G-CSF). Because the fever persisted, TT was again discontinued 24 hours later. The fever and rash resolved after 12 hours, and the ELELs resolved 36 hours later, followed by a diffuse exfoliation. Blood cultures remained negative.

Finally, 5 days after withdrawal of the antibiotic treatment (the patient was receiving only oral ganciclovir, G-CSF, and topical

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Clinical Infectious Diseases 1998;26:523–4
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1058-4838/98/2602-0055$03.00
oral amphotericin B), he had a normal neutrophil count, normal liver enzyme levels, and no fever. We decided, after receiving informed consent, to challenge the patient with lamivudine for 2 days, add indinavir for the following 2 days, and finally add stavudine. He received lamivudine for 48 hours; there were no clinical or biochemical changes. On the third day, he received 400 mg of indinavir (2300 hours). The next morning (700 hours), there was again hypotension (blood pressure, 60/40 mm Hg), diffuse rash, diaphoresis, and fever (temperature, 39.2°C). He received iv fluid replacement, an antihistamine once, and corticoids once. The hypotension and fever resolved 12 hours later, and the ELELs resolved 72 hours later. Blood cultures remained negative.

When the patient was hospitalized 3 weeks after the institution of TT with the above-described clinical picture, we suspected TT to be the cause. We discontinued the TT but administered antibiotics for possible bacteremia due to S. epidermidis (two of six blood cultures were positive). Because the patient was still receiving flucloxacinillin at the time of his first episode of fever, hypotension, rash and ELELs, which appeared immediately after reinstatement of TT, and we noted new neutropenia at that time, we were still not certain about the etiology of his symptoms. It also was not yet clear which of the three antiviral agents comprising the TT was responsible. However, the same clinical picture was evident soon after a new exposure to indinavir when the patient had a normal neutrophil count and was not receiving antibiotic treatment. Twelve hours after the indinavir (and lamivudine) was discontinued, the symptoms again resolved. We strongly believe that the patient’s reaction was a result of the exposure to indinavir. However, we cannot exclude the possibility that an interaction between lamivudine, ganciclovir, and indinavir is necessary to induce this severe allergic reaction to indinavir.

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Reference

Meningoradiculitis with Severe Tetraparesis, an Unusual Manifestation of Infection Due to Cryptococcus neoforms variety gattii

Cryptococcus neoforms variety gattii has a propensity for causing disease in immunocompetent hosts [1]. We describe an immunocompetent patient with severe meningoradiculitis (a very uncommon manifestation of cryptococcal disease) due to C. neoforms variety gattii.

A previously healthy 36-year-old woman from Brazil presented with a 4-day history of progressive, generalized muscle weakness. She had noticed the gradual onset of occipital headaches 2 weeks earlier. She reported a 10-kg weight loss within the previous 4 months. On admission to the hospital, she was afebrile and anorexic (height, 175 cm; weight, 44 kg). At that time, neurological examination revealed moderate neck stiffness, bilateral papillaedema (2 diopters), bilateral facial nerve palsy, and flaccid tetraparesis (grade 3/5 paresis) with almost no tendon reflexes; she was unable to walk. Babinski’s sign was negative. She had no bowel or bladder dysfunction. Sensation was normal except for paresthesias of both hands.

Evaluation of CSF obtained via lumbar puncture showed a mixed pleocytosis (WBCs, 199 cells/μL; 40% neutrophils, 37% lymphocytes, 6% plasma cells, 10% monocytes, and 7% lymphoid cells), an elevated total protein level (120 mg/dL), and a reduced glucose level (23 mg/dL; serum glucose level, 120 mg/dL). Ziehl-Neelsen staining for acid-fast bacilli and cultures for mycobacteria in the CSF were negative. An India ink–stained preparation of the CSF revealed encapsulated yeast-like fungi. The Cryptococcus neoforms polysaccharide antigen titer, as measured by use of the latex agglutination test, was elevated in both serum (1:4,096) and CSF (1:4,096). C. neoforms variety gattii was isolated from the CSF. Findings on a plain chest radiograph were normal, but chest CT revealed a small pulmonary mass within the apical segment of the lower lobe of the right lung. The WBC count was 13.1 × 10⁹/L. Tests for antibodies to HIV-1 and HIV-2 were negative. The T4/T8 lymphocyte ratio was 1.4.

Nerve conduction studies revealed normal motor and sensory conduction velocities and normal distal motor latencies. However, F responses of the median, peroneal, and tibial nerves were absent bilaterally. Compound muscle action potentials were slightly reduced in amplitude. An MRI of the spine showed bilateral contrast enhancement of the nerve roots, including the dorsal root ganglia (figure 1). An MRI of the cranium revealed generalized brain edema and meningeal enhancement following injection of gadolinium (T₁ weighted), which was particularly pronounced in the interpeduncular fossa.

A 3-week treatment regimen was instituted, including iv amphotericin B (0.7 mg/[kg·d]) and fluconazole (2.5 g/t.i.d. orally) followed by oral fluconazole (200 mg/b.i.d.). Cultures of CSF for C. neoforms were negative within 1 week of treatment. Follow-up electrodiagnostic studies revealed pathological spontaneous activity (positive sharp waves) in the right quadriceps muscle. F waves of the right tibial nerve were markedly prolonged, 61.2 milliseconds, but visible in only 20% of stimulation events. Cryptococcal antigen titers in the CSF fell to 1:256. Evaluation of a CSF specimen obtained on repeated lumbar puncture revealed the following values: WBCs, 44 cells/μL; total protein level, 156 mg/dL; and glucose level, 35 mg/dL (serum glucose level, 120 mg/dL). The patient’s general condition gradually improved, and after 8 weeks she left the hospital, walking without ambulatory aids. At the time of discharge, her tendon reflexes had returned to normal.

All the clinical, electrophysiological, and radiological findings in this case were indicative of meningoradiculitis (in addition to meningoencephalitis), a very uncommon manifestation of infection.