BRIEF REPORTS

Survival of a Liver Graft Recipient Treated for an Aspergillar Liver Abscess

Infection due to Aspergillus species is frequently encountered in immunosuppressed patients [1, 2]. Liver transplant recipients are at significantly greater risk of acquiring aspergillosis than are other transplant recipients and patients with solid malignancies treated by chemotherapy [3, 4]. Although the survival of a patient treated for an aspergillar intraabdominal abscess has been reported [5], the prognosis for aspergillosis is particularly poor; the associated mortality is close to 100% [4, 6].

We report a case in which a liver transplant recipient who was treated for an aspergillar liver abscess with a combination of medical therapy and CT-guided transcutaneous drainage was alive and well 3.5 years after diagnosis of the fungal infection. We believe that this is the first report of a long-term survivor of an aspergillar liver abscess that developed following liver transplantation.

A 55-year-old woman underwent liver transplantation in November 1989 for post--hepatitis C cirrhosis (stage C, Child-Pugh grading system). Neither the patient nor the donor had any fungal disease before the operation. Surgery lasted 9 hours, and the immediate postoperative course was uneventful. The immunosuppressive regimen consisted of cyclosporine A, azathioprine, and methylprednisolone.

On postoperative day 9, graft rejection became evident but was successfully treated with three 1-g boluses of methylprednisolone. Nine months after surgery, she presented with severe respiratory infection due to cytomegalovirus and Pneumocystis carinii and was successfully treated with cotrimoxazole and ganciclovir.

Eighteen months after liver transplantation, she presented with asthenia, generalized lymphadenopathy, and splenomegaly. Burkitt’s lymphoma was diagnosed, and she was treated by the withdrawal of immunosuppressive drugs and the administration of chemotherapy (with six 5-day cycles of mitoxantrone, cyclophosphamide, vincristine, and prednisolone). Splenectomy was performed subsequently because of a persistent lymphoma in the spleen. Following these efforts, the disease completely regressed. In March 1992 the patient presented because of persistent fever and asthenia with moderate alteration of liver transaminase levels. Abdominal ultrasonography and CT showed a fluid collection in the left hepatic lobe measuring 9 × 5.5 cm. CT-guided aspiration was performed, and microscopic examination of the fluid showed septate hyphae; Aspergillus fumigatus was isolated by culture. Therapy with itraconazole was started at a dosage of 400 mg q.i.d; the dosage was titrated to maintain the plasma level in the therapeutic range (250–2,000 ng/mL).

The patient’s general condition rapidly improved, but the hepatic fluid collection remained unchanged. For this reason a drainage tube was placed (with the aid of radiology) in the collection, and the cavity was washed daily with a 0.9%-NaCl solution. One week later ultrasonography showed that the collection had disappeared, and the drainage tube was removed. Follow-up CT performed 3 months later did not show any residual liver collection. Itraconazole therapy was then discontinued. No major side effect had occurred during treatment. The patient has been examined regularly and remains well; there has been no evidence of recurrence.

Most fungal infections occur within 1 month of liver transplantation [4], and in cases of aspergillosis the pathogen is probably acquired from the environment [7–9]. However, our patient presented with an aspergillar infection almost 2½ years after surgery. There was no evidence of contamination, and the entry site was unknown.

A number of factors have been associated with fungal infection in transplant recipients [4]; in our case the following factors may have favored development of aspergillosis: overimmunosuppression as treatment for graft rejection; a long course of antibiotics and use of an antiviral drug for pulmonary infection; and chemotherapy and splenectomy as treatment for Burkitt’s lymphoma.

The mortality rate associated with invasive aspergillosis in immunocompromised patients is very high, even with amphotericin B therapy [10]. The success of our patient’s treatment is probably due to several concomitant factors: withdrawal of immunosuppressive drugs, drainage and washing of the abscess, and monitoring of the plasma concentration of itraconazole.

This case demonstrates that aspergillar liver abscesses may be treated successfully with a combination of medical therapy and interventional radiology.

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References
7. Opal SM, Asp AA, Cannady FB Jr, Morse PL, Burton LJ, Hammer PG II. Efficacy of infection control measures during a nosocomial outbreak

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Disseminated Mycobacterium simiae Infection in a Patient with AIDS: Clinical Features and Treatment

A 46-year-old man who had traveled extensively throughout Africa was admitted to the hospital in August 1994 because of pneumonia due to Pneumocystis carinii. Antibodies to HIV were detected, and the CD4 lymphocyte count was 20/mm³. In December the patient’s clinical condition deteriorated and he became febrile; a cervical lymph node contained tuberculoid lesions with numerous acid-fast bacilli in macrophages. Cultures of blood inoculated into Löwenstein-Jensen medium revealed photochromogenic colonies after 20 days of incubation, with acid-fast bacilli that resembled Mycobacterium avium (negative for niacin production, nitrate reduction, Tween hydrolysis, and urease; weak catalase activity).

The same acid-fast bacilli were then recovered from urine and sputum (2–20 colonies per tube), but hybridization with a probe specific for M. avium complex (Gen-Probe, San Diego, CA) was negative for each specimen. The organism was identified as Mycobacterium simiae at the French National Mycobacterium Reference Laboratory (Institut Pasteur).

<table>
<thead>
<tr>
<th>Reference</th>
<th>Source of M. simiae isolate</th>
<th>Underlying disease</th>
<th>Type of coinfection</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>[2]</td>
<td>Kidney, urine, bone marrow</td>
<td>Multiple sclerosis</td>
<td>Mycobacterium kansasii</td>
<td>INH + Eth + Rif</td>
<td>Persistence of M. simiae</td>
</tr>
<tr>
<td>[4]</td>
<td>Blood, liver</td>
<td>AIDS</td>
<td>M. avium complex</td>
<td>(1) INH + Rif + PZA; (2) INH + Rif + PZA + Clof + Eth</td>
<td>No effect</td>
</tr>
<tr>
<td></td>
<td>Sputum, blood, bone marrow</td>
<td>AIDS</td>
<td>None</td>
<td>(3) Eth + Cyce</td>
<td>No effect</td>
</tr>
<tr>
<td>[5]</td>
<td>Blood</td>
<td>AIDS</td>
<td>None</td>
<td>Clof</td>
<td>Death</td>
</tr>
<tr>
<td>[PR]</td>
<td>Blood, lymph node, urine, sputum, bronchial aspirate</td>
<td>AIDS</td>
<td>None</td>
<td>Clm + Rif</td>
<td>Unknown Improvement</td>
</tr>
</tbody>
</table>

NOTE. Clm = clarithromycin; Clof = clofazimine; Cpfx = ciprofloxacin; Cyce = cycloserine; Eth = ethambutol; INH = isoniazid; PR = present report; PZA = pyrazinamide; Rif = rifampin.

* No specimens were positive following 6 months of treatment.

With use of the proportion method, this isolate was found to be susceptible in vitro to cycloserine, sparfloxacin, clarithromycin, and sulfonamides. It was resistant to isoniazid, ethambutol, rifampin, streptomycin, pyrazinamide, thiocetazone, kanamycin, amikacin, capreomycin, cefamandole, tetracycline, erythromycin, trimethoprim/sulfamethoxazole, and rifabutin.

MICs, determined by the agar dilution method on Middlebrook 7H10 medium, were 16 μg/mL for clarithromycin and 8 μg/mL for rifabutin. Therapy with clarithromycin and rifabutin was initiated in January 1995. M. simiae was recovered in February 1995 from only one specimen, a bronchial aspirate inoculated into a diphasic medium. In September 1995 the patient’s clinical condition was stable, and treatment was continued.

M. simiae, a photochromogenic acid-fast bacillus, may resemble the M. avium complex. In our case a mycolate profile was indisputable for identifying M. simiae, thus illustrating the utility of specific probes to distinguish M. avium from biochemically related species. Usually, M. simiae colonizes the upper respiratory tract without causing disease [1]. In our case, M. simiae was isolated from numerous sites.

To our knowledge, only five other cases of disseminated disease due to M. simiae have been described so far (table 1). One involved a patient with multiple sclerosis [2], in association with Mycobacterium kansasii infection. The four other cases concerned patients with AIDS. One patient was an African who lived in Congo [3], and another, originally from Puerto Rico, lived in the United States [4]; both were coinfected with M. avium complex. The other two patients lived in Israel and had disseminated infection due to only