Toxoplasmosis, a Severe Complication in Allogeneic Hematopoietic Stem Cell Transplantation: Successful Treatment Strategies during a 5-Year Single-Center Experience

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Toxoplasmosis is a rare but often fatal complication that occurs after patients undergo allogeneic hematopoietic stem cell transplant. At our institution, toxoplasmosis was diagnosed in 8 of 301 patients who received stem cell transplants. Disseminated toxoplasmosis with a rapid fatal course was observed in 2 patients. Six patients had cerebral toxoplasmosis diagnosed on the basis of neurological signs and observation of the patients’ mental confusion, seizures, and typical lesions (which were assessed by computed tomography, magnetic resonance imaging, or both). Seroconversion of antitoxoplasma immunoglobulin and a discovery of toxoplasma deoxynucleoside acid in the cerebrospinal fluid (confirmed by use of polymerase chain reaction) were documented in all patients. Treatment consisted of clindamycin therapy (for 2 patients) and of pyrimethamine-clindamycin therapy, sulfadiazine therapy, or both (for 5 patients). Patients showed improvement after therapy, as assessed by clinical and radiological means. Three of 8 patients survive—1 without any residual neurological symptoms and 2 with minimal neurological symptoms.

PATIENTS AND METHODS

A total of 301 patients underwent allogeneic stem cell transplantation from May 1994 through April 1999
at our center (Department of Bone Marrow Transplant and Hematology/Oncology, Teaching Hospital, Johannes Gutenberg University Mainz, Idar-Oberstein, Germany). Matched unrelated donor (MUD) stem cells were transplanted in 164 patients. Transplantation of human leukocyte antigen (HLA)–identical stem cells from sibling donors was performed in 102 patients; transplantation of HLA–partially-mismatched stem cells from related donors (PMRD) was performed in 35 patients. All grafts were comprised of unmanipulated bone marrow or peripheral blood stem cells. Eight patients had cerebral or disseminated toxoplasmosis diagnosed and treated; patient characteristics are shown in table 1. The underlying disease of patients experiencing toxoplasmosis was chronic myeloid leukemia in 6 patients and acute myeloid leukemia in 2 patients. Six of the 8 patients received a MUD transplant, and 2 received a transplant from a sibling donor; 1 patient (patient 5) was not fully HLA matched. Before transplantation, all 8 patients were seropositive for antitoxoplasma IgG. No patient had specific IgM, and all donors were seronegative for IgG and IgM antibodies to *Toxoplasma gondii*.

Patients received a conditioning regimen of fractionated total body irradiation (13.5 Gy) and iv cyclophosphamide (60 mg/kg a day for 2 consecutive days). Graft-versus-host-disease (GVHD) prophylaxis consisted of cyclosporine (2 × 5 mg/kg) from day 3 before transplantation and prednisolone (0.5 to 1.0 mg/kg) beginning on day 7 after transplantation. For patients who had transplants from PMRD or MUD transplants, an additional short course of methotrexate was given on days 1, 3, and 6 after transplantation. Beginning with day 100 after transplantation, GVHD prophylaxis was slowly tapered off. Acute GVHD of grades II–IV was treated with escalating doses of prednisolone and mycophenolate mofetil, as described elsewhere [17]. For at least 30 days after transplantation, patients were kept in rooms in which the air was filtered through high-efficiency particulate air filters, and they received nonabsorbable antibiotics for gut decontamination, itraconazole as prophylaxis for fungal infection, and acyclovir for the prevention of herpesvirus infections.

Immunoglobulin prophylaxis with 10 g of polyvalent 7S immunoglobulin was given on days 1 before and 10, 20, 30, 60, and 90 after transplantation. Preemptive ganciclovir therapy was administered to prevent cytomegalovirus viremia [18, 19]. In general, prophylaxis for *Pneumocystis carinii* pneumonia and toxoplasmosis consisted of trimethoprim-sulfamethoxazole (TMP-SMZ) from day 12 to day 1 before transplantation. All patients with stable engraftment (average time of such engraftment, day 40 after transplantation) received 2 double-strength tablets of TMP-SMZ (160 mg of TMP and 800 mg of SMZ, per tablet) per day on 2 consecutive days per week [14]. Patients who had adverse allergic reactions to TMP-SMZ or who had unstable graft function received pentamidine administered via inhalation as prophylaxis. Patients who were IgG seropositive for *T. gondii* received either TMP-SMZ extended to 3 days per week [20] or, in cases of intolerance to this drug, pyrimethamine (50 mg per day) with the substitution of folic acid. All blood components were cytomegalovirus negative and were filtered and irradiated (30 Gy).

**Diagnosis of toxoplasmosis.** Cerebral or visceral toxoplasmosis was diagnosed on the basis of at least 1 of the following 3 criteria: (1) demonstration of tachyzoites and cysts in tissue sections, and presence of typical brain lesions on CT; (2) MRI with typical lesions, PCR amplification for *T. gondii* in CSF, and seroconversion of antitoxoplasma IgM; and (3) resolution of clinical symptoms (e.g., fever and neurological signs) while the brain lesions on CT scans were reduced in size with specific therapy, as shown in figures 1–3.

**Histopathologic diagnosis.** Toxoplasmosis was confirmed by use of brain biopsy or autopsy with hematoxylin or Giemsa staining methods.

**Laboratory diagnostics: serology and PCR.** Antibody titers for *T. gondii* were measured in both donor and recipient serum samples before BMT and, at periodic intervals, in recipient serum samples after BMT during follow-up visits. Specific IgG and IgM antibodies to *Toxoplasma* were quantified by use of an enzyme-linked fluorescence assay (Vidas Toxo IgG and Vidas Toxo IgM; bioMérieux) [21]. Antitoxoplasma IgG titers were expressed in international units per milliliter. Patients were considered to be seropositive if titers were >8 IU/mL. The presence of IgM antibodies was expressed as an index. Serum samples with indexes of 0.55–0.65 were considered doubtful, and those

![Figure 1](https://example.com/1.png)
Table 1. Clinical data on 8 transplant recipients with toxoplasmosis.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
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</thead>
<tbody>
<tr>
<td>Age in y, sex</td>
<td></td>
<td>28, M</td>
<td>32, F</td>
<td>27, M</td>
<td>36</td>
<td>46</td>
<td>54</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>Disease</td>
<td></td>
<td>CML</td>
<td>AML</td>
<td>CML</td>
<td>CML</td>
<td>CML</td>
<td>CML</td>
<td>AML/MDS</td>
<td>CML</td>
</tr>
<tr>
<td>Type of transplant</td>
<td></td>
<td>MUD</td>
<td>MUD</td>
<td>MUD</td>
<td>MRD</td>
<td>PMRD</td>
<td>MUD</td>
<td>MUD</td>
<td>MUD</td>
</tr>
<tr>
<td>Toxoplasmosis IgG before transplantation, IU/mL</td>
<td></td>
<td>203</td>
<td>97</td>
<td>50</td>
<td>80</td>
<td>32</td>
<td>18</td>
<td>270</td>
<td>330</td>
</tr>
<tr>
<td>GVHD</td>
<td></td>
<td>Acute, grade I; limited cGVHD</td>
<td>Extensive cGVHD</td>
<td>Acute, grade III; limited cGVHD</td>
<td>Acute, grade II; limited cGVHD</td>
<td>Extensive cGVHD</td>
<td>No</td>
<td>Acute, grades II–III; cutaneous</td>
<td></td>
</tr>
<tr>
<td>Onset of symptoms, d after transplantation</td>
<td></td>
<td>95</td>
<td>178</td>
<td>280</td>
<td>93</td>
<td>153</td>
<td>224</td>
<td>41</td>
<td>66</td>
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<tr>
<td>CD4 cell count at onset of symptoms, cells/μL</td>
<td></td>
<td>109</td>
<td>459</td>
<td>29</td>
<td>19</td>
<td>77</td>
<td>55</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td>Initial</td>
<td>Seizures</td>
<td>Fatigue, ataxia</td>
<td>Psychotic</td>
<td>Fatigue, fever, weakness</td>
<td>Fatigue, fever, weakness</td>
<td>Fever, headache</td>
<td>Fever, ataxia, fatigue</td>
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<td>Laboratory findings</td>
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<td>Imaging</td>
<td>CT+++</td>
<td>CT++</td>
<td>MRI+</td>
<td>CT+++; MRI+</td>
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<td>CT+++</td>
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<tr>
<td>Toxoplasmosis IgM</td>
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<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
<td>Neg</td>
</tr>
<tr>
<td>Liquor PCR</td>
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<td>ND</td>
<td>Neg</td>
<td>Pos</td>
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<td>Pos</td>
<td>Pos</td>
<td>Pos</td>
</tr>
<tr>
<td>Biopsy at autopsy</td>
<td></td>
<td>Not sufficient</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>Tachyzoites</td>
<td>ND</td>
<td>Generalized</td>
<td>Brain and lung</td>
</tr>
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<td>Drug therapy (duration)</td>
<td></td>
<td>Initial</td>
<td>Cm</td>
<td>Pyr, Cm (12 w)</td>
<td>Pyr, Cm (32 w)</td>
<td>Pyr, Cm (11 w)</td>
<td>Pyr, Cm, Sdz (5 wk)</td>
<td>Pyr, Cm (9 d)</td>
<td>Cm (2 d)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intermediate</td>
<td>Cm</td>
<td>Pyr, Sdz (20 w)</td>
<td>Pyr, Cm, Sdz (6 w)</td>
<td>Pyr, Cm</td>
<td>Pyr, Cm (5 wk)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Maintenance</td>
<td>Cm</td>
<td>Pyr</td>
<td>Pyr</td>
<td>Pyr</td>
<td>Cm</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Outcome</td>
<td></td>
<td>Died day 203</td>
<td>Alive day 1691</td>
<td>Survived</td>
<td>Died day 252</td>
<td>Alive day 1252</td>
<td>Died day 234</td>
<td>Died day 47</td>
<td>Died day 71</td>
</tr>
</tbody>
</table>

**NOTE.** AML, acute myeloid leukemia; cGVHD, chronic graft-versus-host-disease; Cm, clindamycin; CML, chronic myeloid leukemia; CP, chronic phase; F, female; GVHD, graft-versus-host-disease; M, male; MDS, myelodysplastic syndrome; PMRD, partially-mismatched related donor; MRD, matched related donor; MUD, matched unrelated donor; ND, not done; neg, negative; pos, positive; Pyr, pyrimethamine; resp., respiratory; Sdz, sulfadiazine; +, minimal lesions; ++, moderate changes; ++++, big lesions with cerebral edema.

* With parallel cytomegalovirus infection.

* Testing was done 2 weeks after the start of treatment.

* Results of PCR analysis of blood and CSF samples were positive.

* Only necrotic tissue was available.

* Patient died 22 months after diagnosis of toxoplasmosis due to severe cGVHD.
with indexes >0.65 were considered to denote a positive result. Lower indexes were considered to denote a negative result. A diagnostic laboratory (Seelig, Karlsruhe, Germany) performed a nested PCR specific for a sequence on the B1 gene of *T. gondii* [22] on CSF samples obtained from patients; this generated a 176-base pair fragment.

### RESULTS

Of 301 patients who received allogeneic hematopoietic stem cell transplants at our institution, 8 had toxoplasmosis diagnosed after BMT (incidence, 2.7%). Because of adverse events or unstable graft function, none of these patients received TMP-SMZ or pyrimethamine prophylactically. Infections occurred on day 41 and day 66 after transplantation, respectively, in 2 patients with disseminated toxoplasmosis (patients 7 and 8), and they occurred between days 90–280 (median, 166 days) in patients with cerebral toxoplasmosis (patients 1–6). These patients still took considerable amounts of immunosuppressive medication, either because they had recently undergone transplantation or because they had experienced acute or chronic GVHD that necessitated intensified immunosuppression with doses of prednisolone (0.5–1.0 mg/kg) and cyclosporine. The number of CD4 T cells was low in all patients but 1 (patient 2), who had simultaneous cytomegalovirus viremia.

Patients with disseminated toxoplasmosis became clinically symptomatic, with fever that did not respond to antibiotics, regardless of whether they had headaches. For patient 7, a CT scan of the brain led to a differential diagnosis, including toxoplasmosis. For this reason, therapy with clindamycin (2400 mg/day) was started. Disease progressed rapidly, and the patient had respiratory failure 8 days later. Autopsy revealed generalized toxoplasmosis with cysts in the heart, lung, brain, and bone marrow. For patient 8, a CT scan done 3 days before his death indicated no typical lesions. Autopsy revealed cerebral edema caused by excessive growth of tachyzoites (figures 4 and 5), and his lungs showed toxoplasmonic infection. Cerebral toxoplasmosis was suspected on the basis of neurological signs (hemiparesis in 2 patients, fatigue and confusion in 6 patients, ataxia in 2 patients, and seizures in 2 patients). Typical abscess lesions were detected on CT scans of the brain in all 6 patients. *T. gondii* was found in CSF samples analyzed by use of PCR analysis in 5 patients, all of whom were tested before therapy was started; PCR analysis indicated no *T. gondii* in the other patient, who started receiving pyrimethamine-clindamycin empirically 14 days before readmission to our clinic.

For 4 patients who had cerebral toxoplasmosis diagnosed, clinical and neurological symptoms resolved after administration of adequate antitoxoplasma therapy. In addition, 2 patients underwent a brain biopsy. All patients revealed seroconversion of specific antitoxoplasma IgM at the time that their first symptoms occurred. Medication regimens for individual patients are described in table 1. Pyrimethamine therapy was started at a dosage of 100 mg/day for at least 3 weeks. Clindamycin was given at an initial dosage of 1800 or 2400 mg/day iv, followed by oral medication after 2–3 weeks. Oral sulfadiazine was given...
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Figure 4. Patient 8. Extended brain lesion observed at autopsy. Hematoxylin stain; original magnification, ×100.

Figure 5. Patient 8. Extended brain lesion observed at autopsy. Hematoxylin stain; original magnification, ×400.

at a dosage of 4 g/day by mouth. Response to therapy was achieved in the 4 patients who tolerated double or triple combination therapy (1 patient received pyrimethamine-clindamycin; 3 patients received pyrimethamine-clindamycin-sulfadiazine).

We observed mild neutropenia and thrombocytopenia as side effects related to pyrimethamine therapy, and we observed minimal renal impairment with increasing creatinine levels as side effects related to clindamycin and sulfadiazine therapy. However, dose reductions were not required. Retrospectively, we found that 3 patients (patients 1, 6, and 7) did not receive effective therapy because they were in a coma and/or they were vomiting or could not tolerate the oral application of pyrimethamine and sulfadiazine. One patient (patient 8) received no antitoxoplasma therapy because diagnosis was made only at autopsy.

DISCUSSION

Toxoplasmosis has been considered a rare opportunistic infection in patients who undergo BMT [10–15, 23, 24]. In 1997, Chandrasekar et al. [16], summarizing earlier publications (case reports and reviews), reviewed 57 documented cases of clinical toxoplasmosis in the literature. In 1998 and 1999, additional reports [8, 25–28] were published that described 41 patients with generalized toxoplasmosis, 3 patients with pulmonary toxoplasmosis, and 12 patients with cerebral toxoplasmosis or combined cerebral and ocular toxoplasmosis. The patients with generalized (disseminated) toxoplasmosis who were described in our report, like those described in other reports showed signs and symptoms soon after transplantation (days 41 and 66, and 2 and 4 months after transplantation; others reported symptoms and signs at 2–4 months) [14–16]. Disease was fulminant, with episodes of high fever that were refractory to antipyretic drugs and that did not respond to empirical anti-biotics, antimycotics, and virostatics. Definitive diagnoses were made by autopsy.

Six patients with isolated cerebral toxoplasmosis who were from our institution had late onset of toxoplasmosis after transplantation (median onset, day 166), as reported. Symptoms of disease were similar to those in patients with AIDS who had cerebral toxoplasmosis [29–33]. We found some similarities with regard to clinical signs in all patients who developed toxoplasmosis after hematopoietic stem cell transplantation (HSCT): positive test results for IgG before transplantation; allogeneic BMT-PBSCT (stem cells from HLA–PMRD or from unrelated donors); grade III–IV of acute and chronic GVHD, which necessitated intensified or prolonged immune suppression; low CD4 cell count; and insufficient prophylaxis. Most of these observations have been described already elsewhere [14]. It should be noted, however, that we did not observe a single case of toxoplasmosis in the 54 patients who received grafts from matched sibling transplant donors without experiencing higher-grade GVHD, thus indicating the influence of prolonged immunosuppression on the incidence of toxoplasmosis infection.

The finding of positive antitoxoplasma IgG titers before transplantation supports the theory regarding reactivation of latent protozoal infection [15, 16, 34]. This reactivation seems to occur during a period of severe immunosuppression, depending on the type of graft and the necessity of prolonged immunosuppressive treatment. Lymphocyte counts, and CD4 cell counts in particular, may be useful as a means to monitor cellular immune function. In the patients in our study, such counts were extremely low, a finding similar to that in patients with AIDS [30, 33]. There are limited data available on CD4 cell counts in patients with toxoplasmosis after HSCT. Schlüter et al. [35] developed an experimental model for detecting toxoplasmosis encephalitis of immunocompetent and nude mice. They showed that the acute phase of the infection in immu-
nocompetent mice was accompanied by an infiltrate composed of macrophages and CD4+ and CD8+ T cells. In contrast, nude mice died 3 weeks after the infection because of the generalized toxoplasmosis predominantly involved in the brain. Both T cell subsets produced IFN-γ; however, CD4+ cells are the major IFN-γ-producing cells. It has been shown that IFN-γ is the key inducer of TNF-α in CD4+ and CD8+ T cells, thus indicating that T cells are the major cell types producing protective cytokines in experimentally induced toxoplasmosis encephalitis [36].

We wish to emphasize 2 key observations concerning diagnosis. First, until now, IgM seroconversion has not been considered as a diagnostic tool for detecting toxoplasmosis reactivation; it has yielded negative results in patients who have had severe toxoplasmosis diagnosed by means of other methods [14]. In the patients in our study, results for IgM were negative in 2 patients with disseminated toxoplasmosis and positive in all patients with isolated cerebral toxoplasmosis. This might be explained by the comparatively later occurrence of cerebral toxoplasmosis, which happens when the immune function has already been partly restored. Second, PCR amplification (of the B1 gene or the TGR1E sequence) of T. gondii DNA in biopsy material, CSF, blood, or other body fluids has been introduced as a diagnostic method in many laboratories [20, 22, 25, 28, 34, 37±40]. Sensitivity varies widely, depending on the materials tested and the dissemination of disease. We found that PCR analysis of CSF yields positive results for all patients tested before onset of therapy. In other case reports describing patients who underwent BMT, clinicians found PCR to be a useful diagnostic tool [25, 26, 28, 34, 37, 38]. However, in the differential diagnosis of focal brain lesions in patients with AIDS, the diagnostic value of PCR remains controversial. The specificity is high (95%-100%); however, the sensitivity when testing CSF samples was found to be low (50%) in some reports [41]. In some cohorts of patients, this may be due to antiprotozoal treatment started before lumbar puncture or to chemoprophylaxis for P. carinii pneumonia. It is well known that the PCR signal becomes negative few days after the start of specific treatment [21, 42]. The sensitivity in treated patients was reported to be as low as 20%, compared with 81% in untreated patients [43]. The diagnostic “gold standard” for the detection of toxoplasmic encephalitis remains either stereotactic or open brain biopsy.

In a study of 136 patients with AIDS which involved 48 biopsies, Antinori et al. [41] reported a sensitivity of 93%. However, the rate of complications was considerably high, with perioperative morbidity of 12% and mortality of 2%. We have a standard for P. carinii and toxoplasmosis prophylaxis in patients who undergo HSCT (see the Patients and Methods section). Because of the adverse effects of the drugs or because of slow engraftment before the onset of toxoplasmosis, none of the 8 patients we describe received effective prophylactic med-
ication with either TMP-SMZ or pyrimethamine. There are some reports of toxoplasmosis that occurred in spite of prophylaxis with TMP-SMZ [14, 34]; there is evidence that these infections happened because the optimal regimen for dual prophylaxis for P. carinii pneumonia and toxoplasmosis has not yet been established. Various low-dosage and high-dosage regimens are used.

Ribera et al. [44] recently published a case-control study showing evidence that a high dosage of TMP-SMZ (4 or more double-strength tablets weekly) appears to be more effective for the prevention of toxoplasmosis than does a low dosage. In addition, low dosages are more susceptible to loss of efficacy as a result of drug interactions—for example, with rifampin [44]. Other negative influences include a lack of compliance and incomplete resorption in the gut (e.g., in patients with chronic enteral GVHD). In 1997 we changed our prophylaxis strategy, and all patients who were considered at high risk of developing toxoplasmic reactivation received either TMP-SMZ or pyrimethamine, even when they demonstrated delayed engraftment. Since this new protocol was initiated in 1997, we have not observed any reactivation of toxoplasmosis in the high-risk group of patients.

The best-evaluated therapy against toxoplasmic reactivation is the combination of pyrimethamine and sulfonamides; this combination sequentially blocks folic acid metabolism. Both drugs are well absorbed in the gut, and both cross the blood-brain barrier. The combination was proven to be effective in large studies and is recommended as a first-line therapy [29, 45, 46]. However, patients cannot always tolerate these drugs because of the drugs’ adverse side effects. The combination of pyrimethamine-clindamycin was proven to be almost as effective and is widely used as second-line therapy [30, 47]. For the patients in our study, we used all 3 drugs in double or triple combination for initial therapy and as the single agent for maintenance therapy. Various other antibiotics have some inhibitory effect on T. gondii. They are considered third-line therapies, because their efficacy is not well established.

Our experience with the strategies described above indicates that 3 of 8 patients survived with either a minimum or a complete lack of residual neurological symptoms. They had cerebral and not disseminated toxoplasmosis, and therapy was started as soon as the diagnosis was suspected (by presentation of clinical signs, CT or MRI findings, and a positive risk constellation), and all tolerated their medication well, which allowed for use of double- or triple-combination therapy. One patient who survived cerebral toxoplasmosis died 22 months after the first diagnosis of toxoplasmosis of severe chronic GVHD and bronchiolitis obliterans. Earlier reports mention few patients who survived isolated toxoplasmosis of the brain or eye [9, 26–28, 34, 48, 49]. In findings similar to those that we observed in the patients we studied, diagnosis, which was
supported by sensitive radiological studies and PCR methods, was made early; this allowed therapy to be started immediately. In conclusion, it should be noted that toxoplasmosis in patients who undergo HSCT is a severe disease that requires specific preventive measures, including serological screening of donors and recipients before transplantation as well as chemoprophylaxis in patients who are seropositive for IgG before transplantation. Donors who test seropositive for IgM should be not considered for donation whenever possible. If the disease is found, early and intensive combination therapy is necessary to improve the chance of survival. Although our results regarding the treatment of cerebral toxoplasmosis are encouraging, the treatment of generalized (disseminated) form of disease remains an unsolved problem. Consequently, at the present time, prophylaxis appears to be the most important measure that can be taken to prevent this life-threatening infection.

Acknowledgments

We thank Prof. H. P. Seelig, for performing nested PCR analysis of the CSF samples, and Mrs. Schmetzer (Eufets GmbH), for monitoring CD4+ cell counts in the peripheral blood of the patients.

References


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