Toxoplasmic Chorioretinitis in the Setting of Acute Acquired Toxoplasmosis

Jose G. Montoya and Jack S. Remington

Ocular toxoplasmosis is considered to be the most commonly recognized cause of chorioretinitis in the United States. It is commonly believed that the majority of cases of acute toxoplasmic chorioretinitis involving adults in the United States are late sequelae of congenital infection and that the condition is rarely associated with acute postnatally acquired infection. We report here the clinical and serological test findings for 22 adults with acute toxoplasmic chorioretinitis that occurred in the setting of acute postnatally acquired toxoplasmosis. The initial serum specimen from each adult yielded an acute toxoplasmic serological profile, on the basis of the following positive results: 95.5%, Sabin-Feldman dye test (titer of ≥1:1,024); 95.5%, IgM ELISA; 90.9%, IgA ELISA; 77.3%, IgE ELISA; 95.5%, IgE immunosorbent agglutination assay; and 86.4%, differential agglutination (AC/HS) test (acute pattern). Detection of IgA or IgE antibodies or an acute pattern in the AC/HS test was particularly helpful in diagnosis for those patients whose ELISA IgM titers at presentation were negative or lowly positive. Thus, acute toxoplasmic chorioretinitis occurring with a recently acquired Toxoplasma gondii infection would appear to be more common in the United States than previously recognized, and a toxoplasmic serological profile is useful in diagnosing this entity.

Toxoplasma gondii is recognized as an important cause of ocular disease in both immunocompetent and immunosuppressed persons [1]. Whereas acute infection in otherwise healthy adults is most often subclinical [2, 3], toxoplasmic chorioretinitis may cause decreased vision, blindness, or glaucoma, and in some cases it may necessitate enucleation. Involvement of the retina is usually self-limited but can recur, and the destructive nature of the infection places patients at risk for decreased vision or blindness if the optic nerve or macula is affected [1].

Retinochoroidal lesions can occur in the setting of acute acquired toxoplasmosis, either as a sporadic case or in the context of an epidemic of acute toxoplasmosis, but these lesions have rarely been reported [4–11]. Only in southern Brazil have high rates of toxoplasmic chorioretinitis attributed to postnatally acquired infection with T. gondii been reported [12, 13]. Most investigators have concluded that acute toxoplasmic chorioretinitis in adults, with the exception of cases in southern Brazil, is a late manifestation of congenital rather than postnatal infection [1, 2, 14–17]. Thus, its occurrence is little appreciated, understudied, and possibly underdiagnosed.

The purpose of this study was to evaluate clinical findings in patients with toxoplasmic chorioretinitis in the setting of acute acquired toxoplasmosis and to determine the value of conventional and newer serological tests (the toxoplasmic serological profile) in this diagnosis.

Materials and Methods

Patients

From December 1983 to July 1995, serologies for toxoplasma infection were performed on 533 patients because of the possibility of toxoplasmic chorioretinitis. The sera were sent to the Toxoplasma Serology Laboratory of the Research Institute, Palo Alto Medical Foundation (Palo Alto, CA), from throughout the United States. One serum specimen was from Canada. (The Toxoplasma Serology Laboratory has been a not-for-profit referral laboratory for toxoplasma antibody testing since 1966.) Each patient's condition had been diagnosed by their referring ophthalmologist as acute chorioretinitis unequivocally consistent with toxoplasmosis. The sera were not sent to the Palo Alto Medical Foundation as part of any study.

Clinical information was obtained from the records of the Toxoplasma Serology Laboratory and from written and telephone communications with the referring physicians. Clinical diagnosis of toxoplasmic chorioretinitis is based primarily on the findings of an ophthalmologic examination. Criteria for its diagnosis are beyond the scope of this study and are reviewed elsewhere [1].

Serological Tests

Serological testing was by means of the Sabin-Feldman dye test (DT); ELISAs for IgM, IgA, and IgE; immunosorbent
agglutination assay (ISAGA) for IgE; and agglutination test [18, 19]. The AC/HS test, a differential agglutination test that compares titers obtained with formalin-fixed tachyzoites (HS antigen) with those obtained with acetone- or methanol-fixed tachyzoites (AC antigen), was performed as described by Dannemann et al. [20]. Essentially, it differentiates between the avidity of IgG antibodies during acute and more distant infection [18, 20]. Results are expressed in international units (IU).

The first serum dilution for the HS antigen was 1:2,000; a titer of 100 IU/mL was assigned to this dilution, if positive. The first serum dilution for the AC antigen was 1:100; a titer of 50 IU/mL was assigned to this dilution, if positive. High titers in the AC test are associated with recently acquired infections; high titers in the HS test are associated with chronic infections. Criteria used for interpretation of these results were those described by Dannemann et al. [20].

We emphasize that our use herein of the terms acute and nonacute refer solely to interpretation of the pattern of the test and not to whether the patient had a recently acquired infection (>1,600/3,200 is an example of an acute pattern, and <50/100 of a nonacute pattern). The tests were run and results read without prior knowledge of the clinical history of the patient. Sera from each patient were always run in parallel in each test.

The following titers were considered positive and negative, respectively, in the various tests: IgM ELISA, ≥2.0 and <1.7 (equivocal, 1.7–1.9); IgA ELISA, ≥2.1 and ≤1.4 (equivocal, 1.5–2.0); IgG ELISA, ≥1.9 and ≤1.4 (equivocal, 1.5–1.8); IgE ISAGA, ≥4 and ≤2.0 (equivocal, 3). The DT was considered positive at any titer; the starting dilution was 1:16.

Results

Patients' Characteristics

Twenty-two of the 533 consecutive patients had clinically diagnosed toxoplasmic chorioretinitis associated with an acute toxoplasmic serological profile. Clinical findings, treatment, and outcome for the 22 patients are shown in table 1. The mean age of the patients was 50.2 years (median, 51.5 years; range, 16–79 years); 19 (86.4%) were older than 30 years and 10 (45%) were ≥59 years old. The male:female ratio was 1.4:1 (13 males and nine females). One of the females (patient 6) was pregnant at the time chorioretinitis was diagnosed.

Three patients had received organ transplants: liver (patient 19), heart (patient 18), and bone marrow (patient 17). Two patients (numbers 15 and 16) were known to be HIV-positive, 8 were known to be HIV-negative, and 6 did not have any known risk factors for HIV infection. One patient (number 20) had earlier been found to have immune thrombocytopenic purpura and had received corticosteroids.

Information regarding involvement of one or both eyes was available for 19 patients. Unilateral involvement was present in 16 (84.2%) and bilateral in 3 (15.8%). Among those with unilateral involvement, chorioretinitis was present in the right eye in 12 (75%) of 16 patients. The right:left eye-involvement ratio was 3:1.

Retinal examination revealed a choroidal abscess in 1 person (patient 1), involvement of the macula in 2 (patients 17 and 3), and retinal detachment in 1 (patient 2). An old scar was observed in only one (4.5%) of the patients (patient 13). Information regarding previous episodes of chorioretinitis was available for 16 patients. These patients had no history of chorioretinitis.

Information regarding treatment was available for 19 patients. Antitoxoplasmic therapy was given to 18 (94.7%) of the 19. A variety of regimens were used. The most frequently used regimen was pyrimethamine and sulfadiazine, in 8 cases (44.4%); monotherapy was given in 3 cases (16.7%) (atovaquone to patient 20, clindamycin to patient 1, and sulfadiazine to patient 9), and no therapy was given in one case (to patient 5). Corticosteroids were added to the regimen in six cases (33.3%).

Information regarding outcome was available for 16 patients. Partial or total clinical improvement was noted in 14 of these cases (87.5%). Improvement was not observed in patients 17 and 3, in whom macular involvement was detected.

Serology

Serological data are shown in table 2. The second serum sample from one patient (number 12) was sufficient to perform only the DT and IgM ELISA; a total of 38 serum samples from the 22 patients were examined. The DT was positive for each of the 22 patients and was positive at a high titer (≥1:1,024) for 21 (95.5%) of them. Among nine patients from whom ≥2 serum samples (drawn ≥4 weeks apart) were available, one (patient 18) had a significant (8-fold) rise in DT titer. This patient's first sample was drawn 2 weeks after clinical onset of chorioretinitis.

The IgM ELISA of the first serum sample was positive for 21 (95.5%) of the patients and was negative for one (patient 17). The latter serum sample was positive in the IgA ELISA, IgG ELISA, and IgE ISAGA; had an acute AC/HS pattern; and had a markedly elevated agglutination test titer.

The IgA ELISA of the first serum sample was positive for 20 (90.9%). For the patients whose IgA ELISA was negative (patients 2 and 15), both the IgM ELISA and the IgE ISAGA were positive. The AC/HS pattern was acute for patient 15 (>1,600/3,200; first serum sample) and nonacute for patient 2 (100/800).

The IgE ELISA of the first serum sample was positive for 17 patients (77.3%), and the IgE ISAGA was positive for 21 (95.5%). Results of the AC/HS test are shown in table 2. An acute pattern was observed in the first serum sample of 19 (86.4%) of the patients.
Table 1. Clinical findings, treatment, and outcome for 22 patients with chorioretinitis associated with acute toxoplasmic serological profiles.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Patient's age (y)/sex</th>
<th>Host's status</th>
<th>Eye(s) involved</th>
<th>Eye findings</th>
<th>No. of episodes of CHR</th>
<th>Treatment agent(s)</th>
<th>Degree of clinical improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59/M HIV-</td>
<td>R CHR</td>
<td>R CHR, choroidal abscess</td>
<td>1</td>
<td>Cm</td>
<td>Partial</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>59/M HIV-</td>
<td>R CHR</td>
<td>R CHR, detached retina</td>
<td>NP</td>
<td>Pyr/Sdz</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>68/F HIV-</td>
<td>R CHR</td>
<td>R CHR involving macula</td>
<td>1</td>
<td>TMP-SMZ/Cm, Cster</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>38/F HIV-</td>
<td>R CHR</td>
<td>R CHR</td>
<td>1</td>
<td>TMP-SMZ/Cm, Cster</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>63/M HIV-</td>
<td>NP CHR</td>
<td>NP</td>
<td>NP</td>
<td>None</td>
<td>NP</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>25/F HIV-</td>
<td>R CHR</td>
<td>NP</td>
<td>1</td>
<td>Sdz/Cm</td>
<td>Partial</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>76/M HIV-</td>
<td>L CHR</td>
<td>NP</td>
<td>1</td>
<td>Pyr/Sdz</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>53/M HIV-</td>
<td>L CHR</td>
<td>NP</td>
<td>1</td>
<td>Pyr/Sdz</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>33/M No RF</td>
<td>R CHR</td>
<td>NP</td>
<td>1</td>
<td>Sdz/Cster</td>
<td>NP (lost to follow-up)</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>67/F No RF</td>
<td>R, L CHR</td>
<td>NP</td>
<td>1</td>
<td>Pyr/Cm</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>65/M No RF</td>
<td>R CHR</td>
<td>1 Pyr/Sdz</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>75/F No RF</td>
<td>L CHR</td>
<td>1 Pyr/Sdz, Cster</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>28/F No RF</td>
<td>R CHR, old scar</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>36/M No RF</td>
<td>L CHR</td>
<td>1 Cm/Sdz, Cster</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>36/M HIV+</td>
<td>R CHR</td>
<td>1 Pyr/Cm</td>
<td>Partial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>45/M HIV+</td>
<td>NP CHR</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>60/F BMT</td>
<td>R CHR</td>
<td>R CHR involving macula</td>
<td>1</td>
<td>Pyr/Cm</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>50/F CT</td>
<td>R, L CHR</td>
<td>1 Pyr/Sdz, Cster</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>16/F LT</td>
<td>R, L CHR</td>
<td>1 Pyr/Sdz</td>
<td>Partial</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>39/M ITP (Cster)</td>
<td>R CHR</td>
<td>1 Atov</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>79/M NP</td>
<td>R CHR</td>
<td>NP</td>
<td>NP</td>
<td>NP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>35/M NP</td>
<td>NP CHR</td>
<td>NP</td>
<td>NP</td>
<td>Pyr/Sdz</td>
<td>NP</td>
<td></td>
</tr>
</tbody>
</table>

NOTE. Atov = atovaquone; BMT = bone marrow transplant; CHR = chorioretinitis; Cm = clindamycin; Cster = corticosteroids; CT = cardiac transplant; ITP = immune thrombocytopenic purpura; L = left; LT = liver transplant; NP = data not provided; Pyr = pyrimethamine; R = right; RF = risk factors for HIV infection; Sdz = sulfadiazine; TMP-SMZ = trimethoprim-sulfamethoxazole.

Discussion
The data described above reveal that the patients in the present study developed toxoplasmic chorioretinitis as a manifestation of acute acquired toxoplasmosis. Our findings suggest that postnatally acquired toxoplasmic chorioretinitis in the United States might be more prevalent than has previously been appreciated. The literature is replete with statements and data that support the opinion that the vast majority of cases of toxoplasmic chorioretinitis in older children and adults are late sequelae of congenital infections [1, 2, 14-17]; toxoplasmic chorioretinitis as a manifestation of acute acquired infection has been reported infrequently [4-10].

This opinion has recently been challenged [1, 12, 13]. In a population-based household survey in southern Brazil in 1990, 184 (17.7%) of 1,042 individuals examined had ocular toxoplasmosis. Of these 184, 183 (99.5%) had IgG antibodies, compared with 140 (77.4%) of 181 age-matched control subjects (P < .001). The prevalence of ocular disease increased with age, and congenital toxoplasmosis was found to be uncommon [12].

Their results suggested to the authors that in southern Brazil, ocular toxoplasmosis is most likely the result of postnatal rather than congenital infection. However, it is still believed by some investigators that outside southern Brazil, almost all cases of ocular toxoplasmosis are caused by recrudescence of latent congenital infection [17].

The clinical presentations of the patients in our study differed from those of patients with toxoplasmic chorioretinitis that occurs as a late sequela of infection acquired in utero. A significant proportion of patients with recrudescence congenital disease are relatively young and have bilateral disease, old scars, and involvement of the macula as hallmarks of the retinal disease [21]. In those patients, toxoplasmic chorioretinitis occurs more frequently in the second and third decades of life and is rare after the age of 40 [22].

Friedmann and Knox reported that the first symptomatic recurrence in patients with recurrent active toxoplasmic chorioretinitis occurred at a mean age of 25.3 years (range, 7-57 years) [23]. By contrast, patients in the present study were older and had mostly unilateral disease, without old scars or involvement of the macula. It is interesting that of those with unilateral involvement, 75% had disease in the right eye.

Previous studies have addressed the role of serology in the diagnosis of ocular toxoplasmosis [22, 24-28]. The role of serology in clinically evident ocular toxoplasmosis has been relegated to excluding the disease. Thus, serological testing has previously been used only to confirm past exposure to T. gondii [1, 27]. For each of our patients, the serological profile was consistent with recently acquired infection [19]. The IgA
Table 2. Serological results for 22 patients with chorioretinitis whose toxoplasmic serological patterns were acute.

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Date of serology</th>
<th>Dye test, reciprocal titer</th>
<th>Titers of antibodies, as determined by indicated test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>IgM ELISA</td>
<td>IgA ELISA</td>
</tr>
<tr>
<td>1</td>
<td>10/20/94</td>
<td>8,000</td>
<td>8.1 &gt;22.4</td>
</tr>
<tr>
<td>2</td>
<td>11/7/94</td>
<td>8,000</td>
<td>8.5 &gt;22.4</td>
</tr>
<tr>
<td>3</td>
<td>3/24/94</td>
<td>1,024</td>
<td>4.7 0.7</td>
</tr>
<tr>
<td>4</td>
<td>1/4/93</td>
<td>1,024</td>
<td>5.4 12.0</td>
</tr>
<tr>
<td>5</td>
<td>2/15/91</td>
<td>32,000</td>
<td>8.8 &gt;22.4</td>
</tr>
<tr>
<td>6</td>
<td>11/3/92</td>
<td>2,048</td>
<td>5.3 12.7</td>
</tr>
<tr>
<td>7</td>
<td>6/23/93</td>
<td>1,024</td>
<td>8.6 11.9</td>
</tr>
<tr>
<td>8</td>
<td>5/5/92</td>
<td>16,000</td>
<td>8.3 &gt;22.4</td>
</tr>
<tr>
<td>9</td>
<td>9/10/92</td>
<td>4,096</td>
<td>7.0 8.4</td>
</tr>
<tr>
<td>10</td>
<td>10/23/92</td>
<td>8,000</td>
<td>3.4 3.3</td>
</tr>
<tr>
<td>11</td>
<td>12/15/92</td>
<td>8,000</td>
<td>3.6 3.8</td>
</tr>
<tr>
<td>12</td>
<td>1/4/93</td>
<td>1,024</td>
<td>2.7 14.2</td>
</tr>
<tr>
<td>13</td>
<td>12/20/93</td>
<td>4,096</td>
<td>5.9 10.5</td>
</tr>
<tr>
<td>14</td>
<td>6/20/93</td>
<td>4,096</td>
<td>5.3 5.5</td>
</tr>
<tr>
<td>15</td>
<td>10/23/86</td>
<td>16,000</td>
<td>8.8 &gt;22.4</td>
</tr>
<tr>
<td>16</td>
<td>12/11/86</td>
<td>8,000</td>
<td>5.2 NQS</td>
</tr>
<tr>
<td>17</td>
<td>2/19/87</td>
<td>2,048</td>
<td>2.7 5.1</td>
</tr>
<tr>
<td>18</td>
<td>5/3/94</td>
<td>2,048</td>
<td>0.2 0.6</td>
</tr>
<tr>
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<td>12/18/85</td>
<td>4,096</td>
<td>5.1 2.7</td>
</tr>
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<td>4,096</td>
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<td>8,000</td>
<td>3.5 1.3</td>
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<td>1/21/88</td>
<td>16,000</td>
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<td>16,000</td>
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<td>4,096</td>
<td>8.8 14.2</td>
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<td>26</td>
<td>7/20/88</td>
<td>2,048</td>
<td>4.8 3.7</td>
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<td>27</td>
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<td>32,000</td>
<td>0.4 &gt;22.4</td>
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<td>28</td>
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<td>1,024</td>
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<td>29</td>
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<td>1,024</td>
<td>6.2 &gt;22.4</td>
</tr>
<tr>
<td>30</td>
<td>7/8/86</td>
<td>8,000</td>
<td>6.2 17.7</td>
</tr>
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<td>31</td>
<td>8/15/86</td>
<td>8,000</td>
<td>5.8 11.7</td>
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<td>10.1 &gt;22.4</td>
</tr>
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<td>33</td>
<td>2/16/95</td>
<td>2,048</td>
<td>7.4 4.4</td>
</tr>
<tr>
<td>34</td>
<td>7/12/95</td>
<td>32,000</td>
<td>3.5 11.4</td>
</tr>
<tr>
<td>35</td>
<td>9/8/92</td>
<td>1,024</td>
<td>6.7 10.6</td>
</tr>
</tbody>
</table>

NOTE. A = acute; AC/HS = differential agglutination test (see Methods section); ISAGA = immunosorbent agglutination assay; NA = nonacute; NQS = serum sample not quite sufficient.

ELISA, IgE ELISA, IgE ISAGA, and AC/HS test were helpful in establishing the diagnosis as acute infection, since the presence of IgM antibody alone does not establish this diagnosis [2].

IgA and IgE antibodies appear during acute *T. gondii* infection and disappear more rapidly than IgM antibodies [29-31]. This is especially true for IgE antibodies [29]. By contrast, late manifestations of toxoplasmic chorioretinitis as a result of congenital infection are associated with *T. gondii* antibody titers that are not higher than those in persons without eye disease [16, 22, 24-28].

We do not consider the acute titers that we observed to be secondary to exacerbation of chronic (latent) infection in these patients, since most cases involving adults are associated with low DT titers and the absence of IgM antibodies [1, 2, 14-17]. Our present study demonstrates the value of a toxoplasmic serological profile to establish the diagnosis of acute postnatally acquired toxoplasmic chorioretinitis.

Between 1982 and 1995, Couvreur and Thulliez studied 49 patients in France with ocular or neurological toxoplasmosis acquired postnatally [32]. Forty-three of their 49 patients had ocular disease only; 12 (27.9%) of those 49 patients had
Toxoplasmic chorioretinitis and rising antibody titers, suggestive of concomitant acute infection. The other patients with isolated ocular disease had acute toxoplasmosis either 2-24 months or >24 months prior to presentation with chorioretinitis to the referring ophthalmologist. Most of the 43 patients had unilateral disease. There were no HIV-positive patients.

The method we used to select our patients differed from that used by Couvreur and Thulliez. Although it is difficult to compare our study with theirs, it is interesting to note that the French investigators also perceive that postnatally acquired toxoplasmic chorioretinitis may be more common than has previously been appreciated in France.

More recently, on the island of Victoria, in British Columbia, Canada, during the study of an epidemic of toxoplasmosis, 20 (17.9%) of 112 individuals with acute postnatally acquired toxoplasmosis were found to have toxoplasmic chorioretinitis (Dr. Andrew Burnett, personal communication to J.S.R.). Most of these patients had unilateral disease. It is interesting that the percentage of patients with acute toxoplasmosis in whom toxoplasmic chorioretinitis developed in this epidemic is the same as the percentage of patients who were found to have toxoplasmic chorioretinitis in the study by Silveira et al., in southern Brazil [12, 13].

Two of our patients were known to be HIV-positive, three others had undergone organ transplantation, and one had received corticosteroids for idiopathic thrombocytopenic purpura. Toxoplasmic chorioretinitis is a serious disorder in HIV-infected patients and in other immunosuppressed persons [1, 33]. Clinical findings in HIV-positive patients have suggested that most ocular lesions are the result of newly acquired disease or are caused by organisms newly disseminated to the eye from extraocular sites of disease [34–36]. In most reported cases of ocular toxoplasmosis in immunosuppressed patients, no preexisting scars have been noted [1], and the lesions in HIV-infected patients may occur adjacent to retinal blood vessels [35].

However, serological testing has not been helpful in the diagnosis of ocular toxoplasmosis in immunosuppressed patients or in distinguishing between reactivated latent disease and newly acquired disease in these persons [1]. In immunosuppressed patients, titers of IgG antibody to *Toxoplasma* species vary widely, and the presence of IgM antibodies is uncommon [1]. Serological data from three studies of HIV-infected patients with toxoplasmic chorioretinitis demonstrate that IgG antibodies were present in each of 49 patients and that IgM antibodies were present in six (12.2%) of the 49 [34–36]. The serological data from our six immunosuppressed patients are classically representative of recent, postnatally acquired *T. gondii* infection.

In 87.5% of the patients in our study for whom data were available, improvement of visual acuity was partial or complete. The two patients in whom improvement was not observed had involvement of the macula. It appears that the prognosis for visual outcome for patients with acute acquired toxoplasmic chorioretinitis is better than that for patients with recrudescent congenital disease [23].

The lesions of acute toxoplasmic chorioretinitis have the same fundoscopic characteristics, whether they result from congenital or acquired infection [1]. The criteria used to determine whether to treat are also similar [1, 14]. Is it important to establish the mode of acquisition (congenital or postnatal) for patients with toxoplasmic chorioretinitis? From the patient’s perspective, it is important in that the clinical courses of the entities differ somewhat. In general, it appears that the congenitally acquired disease is associated with a more guarded prognosis [23].

From the public health perspective, it is important in an epidemiological sense to establish whether the patient has acquired acute infection so that efforts can be initiated to identify the possible source of *T. gondii* infection. It is also important to establish whether other individuals who may be at high risk for developing severe, life-threatening disease (e.g., fetuses of serologically negative pregnant women or immunocompromised individuals) shared the same exposure as the person with acute acquired toxoplasmic chorioretinitis [4, 5].

It appears that toxoplasmic chorioretinitis as a manifestation of acute *T. gondii* infection is more common than has previously been recognized. In cases of toxoplasmic chorioretinitis, serology is useful in establishing whether the patient has been recently infected. In patients with chorioretinitis and *T. gondii*-specific IgG antibodies, additional serological tests for determining a toxoplasmic serological profile should be performed to rule out the possibility of recently acquired infection.

References


