Toxoplasma gondii-Associated Guillain-Barré Syndrome in an Immunocompetent Patient

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We report a case of Guillain-Barré syndrome associated with an acute, disseminated toxoplasmosis due to a new and unknown zymodeme (zymodeme 12) in an immunocompetent patient. The patient’s condition improved with pyrimethamine and sulfadiazine. Only the standard treatment for toxoplasmosis was effective, whereas intravenous immunoglobulins were ineffective.

Guillain-Barré syndrome seems to be the result of an immunologic process activated by a variety of infectious agents or certain live vaccines (4, 5). Among infectious agents, Campylobacter jejuni is the most frequently identified cause of Guillain-Barré syndrome (3). Only a few cases of acute polyradiculoneuritis have been reported in patients with increasing levels of immunoglobulin G (IgG) and IgM antibodies directed against Toxoplasma gondii (1). We report a case of Guillain-Barré syndrome associated with an acute, disseminated toxoplasmosis due to a new and unknown zymodeme.

A previously healthy 21-year-old man was transferred from French Guyana to our Paris hospital in September 1997 with fever, confusion, polyradiculoneuritis, and diffuse lymphadenopathy. His symptoms had begun 2 months before (15 days after eating undercooked meat of warthog and doe during a 3-day tour in the tropical forest of French Guyana) and included fever, headache, maculopapular rash, dry cough, and diarrhea. A month later, he was admitted to the local hospital because of the persistent symptoms and a 15-kg weight loss. His body temperature was 39°C. Examination revealed diffuse lymphadenopathy and mild hepatosplenomegaly. Laboratory tests were normal, and blood cultures were negative. Other laboratory tests revealed mild hepatitis and pancreatitis (Table 1). Other laboratory tests were normal, and blood cultures were negative. Seven days later, a limb weakness that predominated in his legs and a facial diplegia appeared. Examination of the retina showed an acute left retinochoroiditis. Serologic tests for Toxoplasma gondii confirmed the acute infection by showing the presence of IgG, IgM, IgA, and IgE with high titers of IgG (Table 1). After intraperitoneal injection of blood, mice died of acute infection within 3 days. PCR of Toxoplasma gondii was not detected.

As T. gondii serology was positive with increasing titers of IgG and IgM, the patient was treated with spiramycin (6 MU/day). However, the patient’s neurological conditions were worsening, and he was given intravenous Ig. Due to the inefficacy of this last treatment, he was referred to our hospital. He presented with fever, diffuse lymphadenopathy (cervical, occipital, axillary, and inguinal), mild confusion, and lower-limb weakness associated with a moderate facial diplegia but without other cranial nerve abnormality or pulmonary manifestation. The abnormal results of laboratory tests are reported in Table 1. The blood culture also remained negative. Antibodies to Campylobacter jejuni, Treponema sp., Rickettsia conorii, Borrelia burgdorferi, Leptospira sp., Brucella sp., Chlamydia sp., Salmonella sp., Cryptococcus neoformans, hepatitis C and E viruses, and human immunodeficiency virus were absent. Serologic tests revealed antibodies to hepatitis A and to hepatitis B surface antigen referring to past immunity. Computed tomography of the abdomen showed mild hepatosplenomegaly. Results of a magnetic resonance imaging study of the brain were normal. Examination of the retina showed a new left retinochoroiditis. Serologic tests for T. gondii confirmed the acute infection by showing the presence of IgG, IgM, IgA, and IgE with high titers of IgG (Table 2). After intraperitoneal injection of blood, mice died of acute infection within 3 days and presented parasite-rich ascites. PCR of T. gondii was negative in the blood.

Genetic study of the strain of T. gondii by isoenzyme analysis yielded a new and unknown zymodeme (zymodeme 12). The patient’s condition improved with pyrimethamine (50 mg/day), sulfadiazine (4 g/day), and folinic acid (25 mg/day). Fever disappeared within 5 days, lymph node disorders within 10 days,

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### TABLE 1. Laboratory test results

<table>
<thead>
<tr>
<th>Date of measurement (1997)</th>
<th>Aspartate aminotransferase (20–32 U/liter)</th>
<th>Alanine aminotransferase (16–35 U/liter)</th>
<th>Amylase (&lt;200 U/liter)</th>
<th>Lipase (7–60 U/liter)</th>
<th>Protein (g/liter)</th>
<th>Leukocyte count (% lymphocytosis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>August</td>
<td>152</td>
<td>175</td>
<td>752</td>
<td>466</td>
<td>2.07</td>
<td>30 (80)</td>
</tr>
<tr>
<td>September</td>
<td>76</td>
<td>94</td>
<td>128</td>
<td></td>
<td>3.56</td>
<td>27 (88)</td>
</tr>
</tbody>
</table>

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and neurologic disorders and retinochoroiditis within 15 days. The treatment was stopped after 6 weeks. Ten months later, the patient was fully recovered.

Cases of severe toxoplasmosis have been reported involving immunocompetent patients living in French Guyana, where oocysts are found in river water and wild animals (2). A poor host adaptation to the uncommon highly virulent tropical strains of *T. gondii* can explain these unusual clinical presentations. In our case, Guillain-Barré syndrome was observed in an immunocompetent patient during a disseminated infection with a new strain of *T. gondii*. This strain was highly virulent, as confirmed by the rapid death of the mice (within 3 days). Moreover, this strain was not altered by a 10-day treatment with spiramycin (which is ineffective in toxoplasmosis with central nervous system symptoms), and parasitemia remained after this therapy.

The combination of Guillain-Barré syndrome, massive alteration of the patient’s condition, and skin, pulmonary, liver, pancreatic, and ocular involvement is unique for toxoplasmosis. Only the standard treatment for toxoplasmosis was effective, whereas spiramycin and intravenous Igs were ineffective.

### REFERENCES


### TABLE 2. *T. gondii* serologic test results

<table>
<thead>
<tr>
<th>Date of testing (1997)</th>
<th>Enzyme-linked immunosorbent assay</th>
<th>Immunofluorescence</th>
<th>Immunosorbent agglutination assay (index)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>IgG (U/ml)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>IgM (index)</td>
<td>IgG (IU/ml)</td>
</tr>
<tr>
<td>July</td>
<td>&gt;240</td>
<td>24.5</td>
<td>200</td>
</tr>
<tr>
<td>August</td>
<td>&gt;240</td>
<td>28.1</td>
<td>9,000</td>
</tr>
<tr>
<td>September</td>
<td>&gt;240</td>
<td>27.8</td>
<td>&gt;9,000</td>
</tr>
<tr>
<td>October</td>
<td>&gt;240</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Assay was performed with a Platelia Toxo kit (Sanofi Diagnostics Pasteur).

<sup>b</sup> 1/last positive dilution.

<sup>c</sup> Assay was performed with a Toxo ISAGA kit (Biomérieux).