MAJOR ARTICLE

Fatal Outbreak of Human Toxoplasmosis along the Maroni River: Epidemiological, Clinical, and Parasitological Aspects

Magalie Demar,[1,2] Daniel Ajzenberg,[6,7] Danielle Maubon,[1] Félix Djossou,[2] Dayanand Panchoe,[8] Widya Punwasi,[8] Nasser Valery,[4] Christian Peneau,[5] Jean-Louis Daigre,[3] Christine Aznar,[1] Benoit Cottrelle,[3] Laurence Terzan,[9] Marie-Laure Dardé,[6,7] and Bernard Carme[1]

Units of [1]Parasitology-Mycology and [2]Infectious and Tropical Diseases, Cayenne Hospital, Equipe EA 3593, and [3]Directorate of Health and Social Development, Cayenne, Units of [4]Internal Medicine and [5]Biology, Saint-Laurent-du Maroni Hospital, Saint-Laurent-du Maroni, French Guiana; [6]Laboratory of Parasitology-Mycology, EA 3174, Faculty of Medicine, University of Limoges, and [7]National Reference Center for Toxoplasmosis, Limoges University Hospital Center, Limoges, France; and [8]Epidemiology Unit, Office of Public Health and Epidemiology, and [9]Ministry for Foreign Affairs, French Surinamese Cooperation Section, Paramaribo, Suriname

Background. Well-documented outbreaks of human toxoplasmosis infection are infrequently reported. Here, we describe a community outbreak of multivisceral toxoplasmosis that occurred in Patam, a Surinamese village near the French Guianan border.

Methods. From the end of December 2003 through the middle of January 2004, 5 adult patients in Patam, including 2 pregnant women, were initially hospitalized for multivisceral toxoplasmosis. A French-Surinamese epidemiological investigation was conducted in the village; inquiries and clinical examinations were performed, and blood and environmental samples were obtained. For all serologically confirmed cases of toxoplasmosis, molecular analysis and mouse inoculations were performed for diagnosis and genetic characterization of *Toxoplasma gondii*.

Results. The hospitalized patients, who did not have any immunodeficiencies, presented with an infectious disease with multivisceral involvement. Serological examination confirmed acute toxoplasmosis. One adult died, and a neonate and a fetus with congenital toxoplasmosis also died. During the investigation, 4 additional acute cases of toxoplasmosis were diagnosed among the 33 villagers. Only 3 inhabitants had serological evidence of previous *T. gondii* infection. In total, we reported 11 cases of toxoplasmosis: 8 multivisceral cases in immunocompetent adults, resulting in 1 death; 2 cases of lethal congenital toxoplasmosis in a neonate and a fetus; and 1 symptomatic case in a child. Molecular analysis demonstrated that identical isolates of only 1 atypical strain were responsible for at least 5 of the 11 cases of toxoplasmosis in the outbreak. No epidemiological sources could be linked to this severe community-wide outbreak of toxoplasmosis.

Conclusion. This report is in agreement with the particular features of toxoplasmosis involving atypical strains that were recently described in French Guiana.

Toxoplasmosis, a ubiquitous protozoal disease caused by *Toxoplasma gondii*, can infect all warm-blooded animals, with the definite hosts being felines [1]. Therefore, there are a large variety of sources for transmission of human horizontal infection through the ingestion of infectious oocysts from the environment or tissue cysts contained in raw or undercooked meat or primary offal of different animals. Major routes of transmission are different in human populations and depend on social culture, eating habits, and/or environmental factors. The occurrence of outbreaks of *Toxoplasma* infection involving more than a single family or small group are infrequently reported [2–21], and the largest documented toxoplasmosis outbreak, which was associated with a municipal water supply, was reported in 1995

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Reprints or correspondence: Dr. Demar Magalie, Unit of Infectious Diseases and Tropical Medicine, Hospital of French Guiana, Ave. des Flamboyants, BP 6006, Cayenne 97300, French Guiana (mdemar@yahoo.com).

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in Canada [18]. We report here, to our knowledge, the first human outbreak of toxoplasmosis with isolation and characterization of the responsible strain.

MATERIALS AND METHODS

Description of the Outbreak

At the beginning of January 2004, the health services of French Guiana (Directorate of Health and Social Development) were informed by the Laboratory of Parasitology-Mycology at Hospital of Cayenne (Cayenne, French Guiana) of 5 cases of multivisceral toxoplasmosis in adults, including 1 case that resulted in death and 2 cases of lethal congenital toxoplasmosis (in 1 fetus and 1 newborn). All of the patients resided in the village of Patam. This small, rural Surinamese village, located near the French Guianan border between the Maroni River (~1 km from the village) and the Surinamese frontier town of Albina (~5 km from the village), is surrounded by the Amazonian forest. Thirty-four Maroon persons belonging to the same family were living in the village. The eldest inhabitants were 2 sisters living with their descendents. The first 4 patients (including 2 pregnant women) were hospitalized in the French hospital of Saint-Laurent-du Maroni (Saint-Laurent-du Maroni, French Guiana). A serological diagnosis of acute toxoplasmosis was made on the basis of a high level of anti-*Toxoplasma* IgG, presence of IgM, and low avidity (<30%) of IgG anti-*Toxoplasma* antibodies. At the same time, a fifth patient from Patam was admitted to the hospital in Paramaribo, Suriname. Other clinical infections were reported among the inhabitants of the village. This prompted an extensive joint epidemiological investigation in the middle of January 2004, involving the medical authorities of French Guiana (Parasitology-Mycology Unit of Cayenne Hospital and the Directorate of Health and Social Development Unit, Cayenne, French Guiana) and Surinamese health services (Bureau Openbare Gezondheidszorg, Paramaribo, Suriname).

Investigation in the Village

Detection of additional cases. After clinical examinations were performed, individuals from the village were initially categorized as being symptomatic or asymptomatic on the basis of standard clinical criteria; patients with fever or general signs (e.g., myalgia and impairment), tissue enlargement (e.g., in the liver and spleen), and/or lower respiratory symptoms were considered to be symptomatic. Symptomatic patients were treated with sulfadiazine plus pyrimethamine. Serological test findings further defined the type of infection: (1) acute toxoplasmosis, defined as positivity for *Toxoplasma*-specific IgG and IgM and a low *Toxoplasma*-specific IgG avidity in the presence or absence of symptoms; (2) chronic toxoplasmosis, defined as positivity for *Toxoplasma*-specific IgG (but not IgM) and a high *Toxoplasma*-specific IgG avidity in the presence or absence of symptoms; and (3) absence of infection, defined as negative serological test results and no clinical signs of infection. Individuals who could not be classified (i.e., they had symptoms, but they had negative serological test results or were positive only for *Toxoplasma*-specific IgM) have been subsequently tested periodically for seroconversion. In 1 case of acute infection, PCR [22] performed on a blood sample confirmed the diagnosis.

Epidemiological inquiries for risk factors. Adults of each household were routinely questioned regarding jointly consumed meals during the 2 weeks immediately preceding the beginning of the outbreak, including food practices and origin of consumed water. In addition to the description of the wild forest biotope surrounding the village (i.e., forest vegetation type, rural aspects, and type of fauna), other epidemiological data (e.g., the presence of wandering wild cats or felids, as well as traditional hunting activities) were noted.

Environmental investigations. Samples of soil and feces of wild cats were collected from 5 and 6 sites, respectively, in different zones of the village. These sampling sites were initially randomly selected, but some were selected as *Toxoplasma* risk areas on the basis of information from the inhabitants, such as soil in contact with feces from wild cats (figure 1).

Laboratory Methods

Toxoplasma *serological examination.* All serum samples were analyzed using the EIA for *Toxoplasma*-specific immunoglobulin, IgG, and IgM (Abbot Diagnostics). When results for IgM were positive, additional tests, such as the indirect

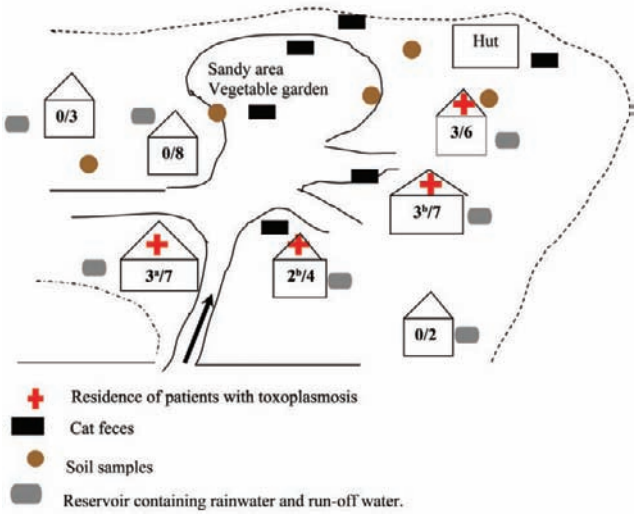

Figure 1. Diagram of Patam village, including the residences of patients with toxoplasmosis and sampling sites. Data are no. of infected patients/no. of persons in the household. [a]One patient was a visitor. [b]Includes patients with congenital cases.

immunofluorescence IgM antibody assay (IFA; bioMérieux), immunosorbent agglutination assay (Toxo-ISAGA IgM; bioMérieux), and IgG avidity test (VIDAS Toxo-IgG Avidity kit; bioMérieux), were performed to confirm the presence and specificity of IgM antibodies and, therefore, acute infection.

Retroviral investigation. For all patients (except the fetus and neonate), the HIV serological status was confirmed using 2 HIV tests (HIV [1/2] Axsym microparticule EIA system, Abbott, and Genescreen Plus HIV Ag-Ab EIA, Biorad). We used the Murex HTLV-1/2 EIA (Abbott) to screen for human T lymphotropic virus.

Detection of* Toxoplasma *oocysts. We used standard methods (e.g., sucrose flotation) for the detection of *Toxoplasma* oocysts from collected soil or feces specimens [23], followed by examination under UV excitation (excitation filter, 330–385 nm) for detection of oocyst autofluorescence [24] and real-time PCR using the B1 gene [25]. Approximately 200 g of soil were collected from each sampling site; 50 g of soil was used in a single analysis by sucrose flotation.

Toxoplasma *molecular detection and mouse inoculations.* As shown in tables 1 and 2, an attempt to detect parasite DNA and to isolate *Toxoplasma* species was performed at the Laboratory of Parasitology-Mycology at Limoges Hospital (Limoges, France) on the peripheral blood samples (preserved in EDTA) obtained from 9 patients (patients 1–4, 6, and 8–11) and on several tissue specimens (i.e., brain, liver, lung, and amniotic fluid) from the expulsed fetus (patient 7). DNA was extracted from these samples using a QIAamp DNA minikit (Qiagen) and was then subjected to a PCR-based assay for the detection of the *T. gondii* B1 gene [22, 25]. Two or 3 Swiss mice were inoculated intraperitoneally with the blood samples. Mice were not inoculated with fetal tissue samples from patient 7, because these samples had been preserved in formaldehyde. No samples were available from the neonate (patient 5), who died at 6 days of life.

Genotype analysis of* Toxoplasma *isolates. Genetic characterization of *T. gondii* isolates was performed by sequencing at 5 microsatellite markers *(TUB2*, *W35*, *TgM-A*, *B18,* and *B17)* and GeneScan analysis of length polymorphism at 3 additional polymorphic microsatellite markers *(N60608, N82375,* and *AA519150).* Reaction mixtures and amplification conditions were performed in accordance with published methods [26, 27]. All sequences at markers *TUB2, W35, TgM-A, B18,* and *B17* were submitted to GenBank (accession numbers: EF523521–EF523545).

RESULTS

Characteristics of hospitalized patients. The complete characteristics of all of the patients are summarized in table 1. The time of the beginning of symptoms implied a likely contamination during the second week of December. All of the patients initially presented with general signs, with progressive major clinical impairment. Bilateral pneumopathy and hepatic cytolysis were the most frequent conditions at presentation. Neurological disorders (loss of consciousness) and echographic cardiac abnormalities (mild dilated myocardiopathy with pericarditis) occurred in patients 1 and 4, respectively. The delivered fetus (patient 7) presented with signs consistent with a nonimmune hydrops fetalis. All of the patients expressed serological profiles consistent with acute toxoplasmosis. For patient 1, the serodiagnosis was performed post mortem, because the patient's clinical state rapidly worsened while receiving antibiotic therapy, and the patient died after global multivisceral failure, with neurological disorders, respiratory distress (oxygen saturation, 88%, with 10 L of oxygen), hepatic cytolysis, and acute renal insufficiency. Patient 5 died at 6 days of age with serological evidence of recent primary toxoplasmosis. The other microbiological investigation and retrovirus serologic test results were negative. Except for patients 1, 5, and 7, all patients were cured without any complications with sulfadiazine plus pyrimethamine treatment.

Clinical and epidemiological investigation in the village. Thirty-one individuals were examined (age, 5 months–46 years; 10 adults and 21 children). Most of them were inhabitants of the village; 4 were visitors living in the neighboring village and spending a lot of time with the inhabitants of Patam. Two residents were absent. Eight patients were symptomatic: 1 presented with symptoms of bronchiolitis, and 7 had symptoms consistent with toxoplasmosis, such as general signs, splenomegaly, or hepatomegaly (table 2). In the symptomatic patients, only 4 additional cases of acute toxoplasmosis (in 3 adults and 1 child) were confirmed serologically and by PCR (patient 11). For the latter, the diagnosis was made ~1 month after the initial outbreak. For the other 3 symptomatic patients, 2 serological tests were performed at least 3 weeks after the first serological test, and the results remained negative. Of the 24 remaining asymptomatic inhabitants, 21 had serum samples that were negative for *T. gondii,* and 3 had serological evidence of chronic toxoplasmosis. All of the patients had negative HIV 1/2 and human T lymphotropic virus 1/2 serological test results. Because all of the confirmed cases were registered, geographical mapping of acute cases was performed (figure 1). There were 7 traditional wooden houses with a central sandy area and a vegetable garden. There was no electricity or water supply, and each house had a reservoir containing rainwater and run-off water. There were wild cats roaming in the village, especially during the night, and wild felids, such as *Panthera onca,* had sometimes been seen around the dwellings. The inhabitants consumed frozen foods but also hunted. Meat-borne risk factors were not found, because undercooked meat consumption is against the Evangelist faith of the inhabitants. To date, all of

Table 1. Characteristics of the hospitalized patients in an outbreak of toxoplasmosis in an Amazonian village.

Characteristic	Patient						
	1	2	3	4[a]	5	6[a]	7[b]
Sex	F	F	F	F	F	F	Unknown
Age	56 Years	19 Years	46 Years	21 Years	6 Days	22 Years	22 Weeks gestation
Family relation	…	Daughter of patient 1	Sister of patient 1	Niece of patient 1, daughter of patient 3	Child of patient 4	Daughter-in-law of patient 1	Fetus of patient 6
Site where hospitalized	SLM	SLM	Paramaribo (Suriname)	SLM	SLM	SLM	SLM
Date of hospitalization[c]	26	29	29	30	31	29	…
Duration and type of symptoms before hospitalization	2 Weeks; fever, breathlessness, major clinical impairment	3 Weeks; fever, arthralgia, cough	18 Days; fever, headache, cough, clinical impairment	3 Weeks; fever, contractions, abdominal discomfort	At birth; fever, clinical impairment, respiratory distress	19 Days; fever, cough, respiratory distress	…
Symptoms at hospital admission	Fever, bilateral pneumopathy, alteration of consciousness	Fever, bilateral pneumopathy, myalgia, splenomegaly	Fever, bilateral pneumopathy	Fever at 35 WA, impairment, anemia, echographic cardiac abnormalities	Disseminated congenital toxoplasmosis	Fever at 24 WA, pneumopathy, headache	Disseminated congenital toxoplasmosis (24 WA)
Biological data at hospital admission							
GOT:GPT, IU/L	400:133	376:191	ND	184:52	…	214:89	ND
Creatinine level, μmol/L	200	145	ND	115	ND	110	ND
LDH level, IU/L	1944	1742	ND	ND	ND	ND	ND
CRP level, mg/L	104	5	ND	13	25	92	ND
Hemoglobin level, g/dL	ND	ND	ND	6	ND	ND	ND
Treatment	Antibiotics	Antitoxoplasmic	Antitoxoplasmic	Antitoxoplasmic	Antitoxoplasmic	Antitoxoplasmic	None
Outcome from the day of initiation of treatment	Death on day 3	Favorable	Favorable	Favorable	Death on day 6	Favorable	In utero fetal death
Toxoplasma serological test results at hospital admission							
IgG level, IU/L	1090	1027	3203	127	65[d]	256	ND
IgM EIA index[e]	8.3	>15	6.17	10.7	0[d]	>15	ND
IgG avidity	Low	Low	Low	Low	ND[d]	Low	ND
IgM Isaga:IFA	12:400	12:400	ND	12:800	0:ND[d]	12:800	ND
Result of *Toxoplasma* PCR with BI gene (sample site)	Positive (PB)	Negative (PB)	Negative (PB)	Positive (PB)	ND	Negative (PB)	Positive (fetal tissue)
Mouse inoculation result	Positive	Negative	Negative	Positive	ND	Positive	ND
Isolate	GUY-2004-ABE	…	…	GUY-2004-TER	…	GUY-2004-ANG	GUY-2004-ANG1

NOTE. CRP, C-reactive protein; GOT, glutamic oxaloacetic transaminase; GPT, glutamic pyruvic transaminase; IFA, immunoflourescence assay; LDH, lactate dehydrogenase; ND, not determined; PB, peripheral blood; SLM: Saint Laurent du Maroni (French Guiana); WA, weeks of amenorrhea.

[a] Patients 4 and 6 were pregnant.

[b] Patient 7 was a fetus.

[c] All patients presented to the hospital in December 2003.

[d] On day 6, patient 5 had an IgG level of 77 IU/L, an IgM level of 0.73 IU/L, a low avidity IgG, and positive IgM with an EIA index = 0.73 and an Isaga score = 12.

[e] An IgM index <0.6 indicates IgM negativity.

Table 2. Characteristics of the patients who received diagnoses of toxoplasmosis during the village investigation.

Characteristic	Patient 8	9	10	11
Sex	F	M	M	M
Age	23 Years	28 Years	20 Years	8 Months
Family relation	Daughter of patient 3 (inhabitant)	Boyfriend of patient 8 (inhabitant)	Boyfriend of patient 2 (visitor)	Son of patient 8 (inhabitant)
Duration of symptoms before investigation, weeks	5	~5	5–6	3
Symptoms	Fever, splenomegaly, hepatic pain	Cough, myalgia, fever, major impairment	Fever, tachyarythmia, hepatomegaly	Fever, splenomegaly
Treatment	Antitoxoplasmic	Antitoxoplasmic	Antitoxoplasmic	Antitoxoplasmic
Outcome	Favorable	Favorable	Favorable	Favorable
Serological test result				
IgG level, IU/L	1027	2732	3000	2
IgM EIA index[a]	>15	10.95	>15	>15
IgG avidity	Low	Low	Low	Not determined
Result of *Toxoplasma* PCR with B1 gene (sample site)	Negative (PB)	Negative (PB)	Negative (PB)	Positive (PB)
Mice inoculation result	Negative	Negative	Negative	Positive
Isolate	…	…	…	GUY-2004-TER1

NOTE. PB, peripheral blood.

[a] An IgM index <0.6 indicates IgM negativity.

the environmental samples (i.e., soil and feces of wild cats) have tested negative for *Toxoplasma* oocysts.

Mouse inoculations and molecular features of the isolates. As shown in tables 1 and 2, the results of PCR with *B1* gene were positive for 4 patients (patients 1, 4, 7, and 11). *Toxoplasma* species isolation in mice was successful for 4 patients (patients 1, 4, 6, and 11), including 1 patient who had negative PCR results. The *Toxoplasma* isolates were virulent (table 3), because the majority of inoculated mice died 16–30 days after inoculation.

Genetic characterization was possible for the 4 *Toxoplasma* isolates obtained in mice (GUY-2004-ABE, GUY-2004-TER, GUY-2004-ANG, and GUY-2004-TER1) and directly from an amniotic fluid sample from the fetus (GUY-2004-ANG1). Sequencing at 5 microsatellite markers *(TUB2, W35, TgM-A, B18,* and *B17)* showed identical sequences at each marker in these 5 patients (figure 2). Three additional polymorphic microsatellite markers *(N60608, N82375,* and *AA519150)* also showed identical alleles after Genescan analysis of length polymorphism (table 4). With a total of 8 markers, the description of a unique multilocus genotype in 5 patients with toxoplasmosis demonstrated that only 1 strain was responsible for the outbreak (in at least 5 of 11 patients). Compared with the 3 archetypal I, II, and III strains (figure 2), this strain presented an atypical genotype with 3 atypical alleles at *W35, TgM-A,* and *B17*; these are different from the corresponding alleles of types I, II, or III strains. The atypical alleles consisted of unique nucleotide polymorphisms in microsatellite flanking regions (at *TgM-A* and *B17*) or unusual numbers of dinucleotide repeats in microsatellite regions ($[TC]_9$ $[TG]_2$ at *W35,* $[TG]_6$ at *TgM-A,* and $[TC]_8$ at *B17).*

DISCUSSION

Outbreaks of human toxoplasmosis have previously been reported [2–21]; the largest outbreak occurred in a municipality in the western Canadian province of British Columbia [18] in 1995. The diagnostic criteria in the outbreak in Canada were based on (1) the sudden onset of symptoms consistent with toxoplasmosis, (2) the serological evidence of acute toxoplasmosis (i.e., *Toxoplasma*-specific IgG and IgM positivity), and (3) the clustering of cases in the same region at the same time. These features were present in the outbreak of toxoplasmosis reported here. In total, from the end of December 2003 through the middle of January 2004, 11 cases of toxoplasmosis occurred among 33 inhabitants of a Surinamese village, including 8 symptomatic cases of multivisceral toxoplasmosis in immunocompetent adults, resulting in 1 death; 2 cases of lethal congenital toxoplasmosis in a neonate and a fetus; and 1 symptomatic case in a child. All of the cases were serologically proven on the basis of the presence of a high level of IgM antibodies, and the parasite was detected either by mouse inoculation or by PCR for 5 cases. The genotype analysis with 8 microsatellite markers revealed that only 1 strain was responsible for at least 5 of the 11 cases of toxoplasmosis in the outbreak. This is, to our knowledge, the first time that such direct proof of a toxoplasmosis outbreak has been reported.

The atypical genotype of this strain cannot be related to the 3 known main lineages [27] and seems to be related to severe toxoplasmosis acquired by immunocompetent adults in French Guiana [28, 29]. This is in agreement with the unusual clinical features in this outbreak. Indeed, most of the adult patients who were without any immunodeficiency presented with a se-

Table 3. Diagnosis by PCR and bioassay in mice.

Variable	Patient				
	1	4	6	11	7
Sample	PB	PB	PB	PB	Fetal tissue, AF (in formaldehyde)
Result of PCR with B1 gene	Positive	Positive	Negative	Positive	Positive
Mouse inoculation result	Positive	Positive	Positive	Positive	ND
No. of mice inoculated	5	4	2	2	…
No. of mice infected	4	4	1	1	…
No. of mice that died	4	4	1	0	…
Virulence in mice at isolation	Death at D19–D30 pi	Death at D16–D18 pi	Death at D18 pi	Chronic infection in the infected mouse	ND
Isolate	GUY-2004-ABE	GUY-2004-TER	GUY-2004-ANG	GUY-2004-TER1	GUY-2004- ANG1

NOTE. D, day; ND, not determined; PB, peripheral blood; pi, postinoculation.

vere infectious syndrome with visceral involvement (especially pulmonary and hepatic), leading to death in 1 case. However, there was diversity in clinical manifestations among the different cases, as 4 patients were found to be infected during investigation in the village, without any life-threatening symptoms or need for hospitalization. Because the identical strain was isolated from each patient, hypotheses other than that of virulence of the strain should be considered, including an inoculum effect. In addition, even though the inhabitants belonged to the same family, the 3 fatalities occurred in members

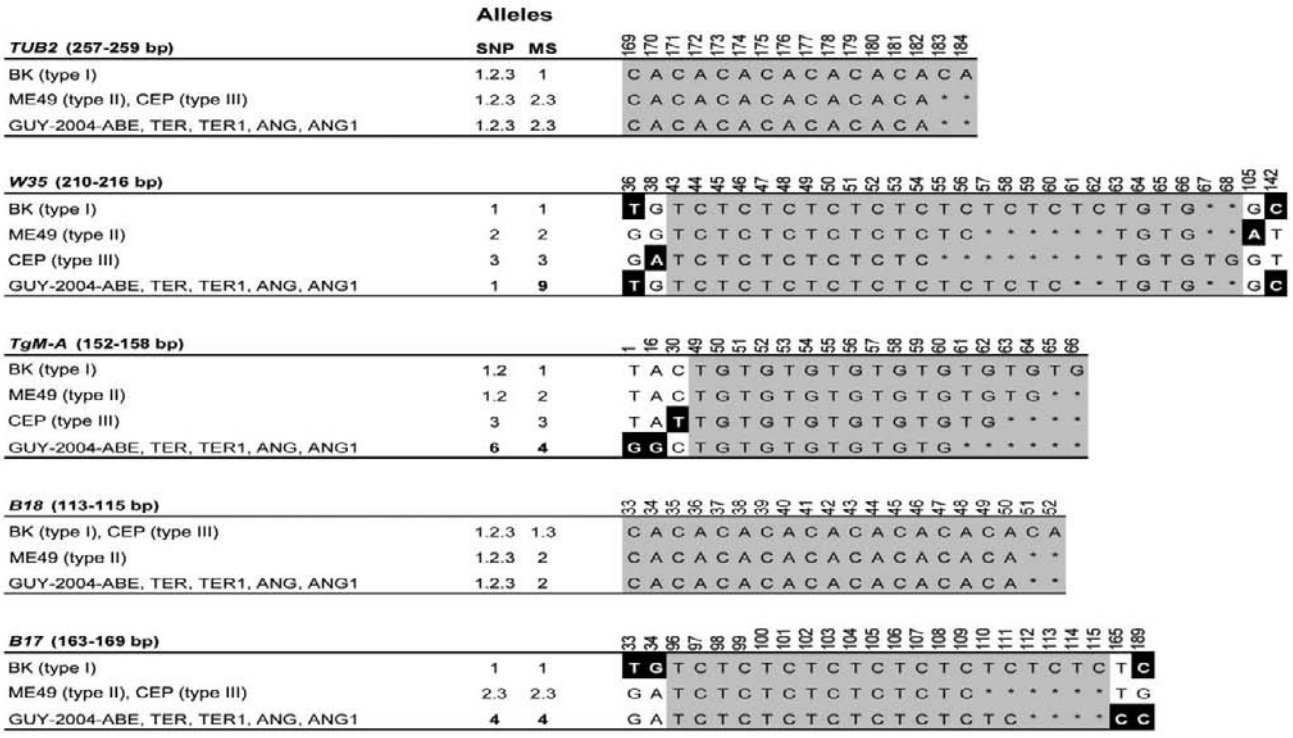

Figure 2. Nucleotide polymorphisms at 5 microsatellite markers *(TUB2, W35, TgM-A, B18,* and *B17).* Sequences from archetypal type I (BK), II (ME49), and III (CEP) strains were compared with sequences obtained from the GUY-2004-ABE, GUY-2004-TER, GUY-2004-TER1, GUY-2004-ANG, and GUY-2004-ANG1 isolates. SNP and MS alleles were defined by polymorphic sites in the flanking region and microsatellite sequences, respectively. BK, Me49, and CEP are reference type I, II, and III strains, respectively. Alleles 1, 2, and 3 are reserved for clonal lineages I, II, and III. For allele 1.2, types I and II share the allele; for allele 2.3, types II and III share the allele; and for allele 1.2.3, types I, II, and III share the allele. Alleles ⩾4 are used for atypical alleles. Boxes with gray background indicate microsatellite sequences. Boxes with black background indicate single-nucleotide polymorphisms not shared by at least 2 archetypal sequences. In each marker, sites demarcated by an asterisk *(*)* indicate deletion sites in comparison with the longest microsatellite sequence. Nucleotide sequence data are available on GenBank (accession numbers: EF523521–EF523545).

Table 4. Genescan analysis of length polymorphism of 3 additional polymorphic microsatellite markers *(N60608, N82375,* and *AA519150)* in GUY-2004-ABE, GUY-2004-TER, GUY-2004-TER1, GUY-2004-ANG, and GUY-2004-ANG1 isolates.

	Polymorphic microsatellite marker		
Isolate	*N60608*	*N82375*	*AA519150*
GUY-2004-ABE	133	109	160
GUY-2004-ANG	133	109	160
GUY-2004-ANG1	133	109	160
GUY-2004-TER	133	109	160
GUY-2004-TER1	133	109	160

of the family who were the furthest apart in age, indicating a host's susceptibility.

Most of the population of Patam were sensitive to *T. gondii,* because the prevalence of toxoplasmosis in this village was very low before the outbreak (8.3%). However, numerous risk factors for toxoplasmosis exposure were present in the village, such as regular close contact with soil, vegetation, and water in the Amazonian forest surrounding the village; the presence of wild cats or other felids around the village; the report of hunting activities and occasional consumption of game; and the use of run-off water or wastewater. Unfortunately, none of these risk factors was formally identified as being the source of this outbreak. The number and severity of cases and the religious interdiction on undercooked meat consumption suggest a likely contamination by oocysts rather than by tissue cyst ingestion. The occurrence of 1 subsequent case (in patient 11) might suggest a persistent source of contamination in the environment. However, no new cases were reported in the months following this outbreak. Although water analysis was not performed, the drinking water could have been contaminated with oocysts of wild cats or felids, because it was collected from rain or river water and was untreated.

In neighboring French Guiana, the incidence of these severe forms of primary *T. gondii* infection is high in this region of 200,000 inhabitants, with >40 well-documented cases in the past decade (M.D., unpublished data) [28, 29]. Such forms of infection seem to be linked to a forest-based cycle involving wild felids and their prey. Recent studies in French Guiana highlight this hypothesis, indicating that there is high toxoplasmosis seroprevalence in noncarnivorous wild mammals, such as peccaries *(Tayassu* species), pacas *(Agouti paca),* armadillos *(Dasipus noviemcinctus),* and deer *(Mazama* species), living in an uninhabited forest zone [30] and considered to be major human game species; terrestrial mammals were significantly more frequently exposed to *T. gondii* than were other mammals [31], which is concordant with oral exposure related to ground dwelling behavior. One *T. gondii* strain was isolated from a wild jaguar in French Guiana, but no isolations were attempted on game meat [32]. Ajzenberg et al. [27] revealed that most of the reported cases of disseminated toxoplasmosis in immunocompetent patients in French Guiana involved atypical genetic isolates of *T. gondii.* These atypical isolates are not related to the 3 main lineages (i.e., lineages I, II, and III) and have many unique polymorphisms at many markers. Allele 4 at Myosin-A marker was found only in isolates originating from South America (French Guiana, Brazil, and, now, Suriname) among >500 strains typed with these 5 microsatellite markers in the Biological Resource Center *Toxoplasma* (D.A., unpublished data); this genetic diversity and these atypical characteristics were also exhibited in the *T. gondii* strain isolated from the wild jaguar in French Guiana with the allele 4 at *TgM-A* [32]. Additional studies are needed to better describe this sylvatic cycle of *T. gondii* infection.

This outbreak of toxoplasmosis in an Amazonian rain forest region was characterized by its severity. Besides the epidemiological and clinical aspects, we report direct molecular evidence for the outbreak by describing the identical genetic profile of isolates collected from 5 patients. Although the likely hypothesis for the common source of contamination could be the ingestion of oocysts, the environmental investigation remained noncontributive. Physicians should be aware of such clinical presentations of acute toxoplasmosis that seem to be linked to a forest-based cycle involving wild felids and their prey in the Amazon rainforest.

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