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Toxoplasmosis Outbreak Associated With *Toxoplasma gondii*-Contaminated Venison—High Attack Rate, Unusual Clinical Presentation, and Atypical Genotype

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Abstract

Background.—During 2017, in response to a physician's report, the Wisconsin Department of Health Services, Division of Public Health, began investigating an outbreak of febrile illness among attendees of a retreat where never frozen, intentionally undercooked, locally harvested venison was served. Preliminary testing tentatively identified the illness as toxoplasmosis.

Methods.—Confirmatory human serology panels and testing of the venison to confirm and categorize the presence and type of *Toxoplasma gondii* were completed by French and American national reference laboratories. All 12 retreat attendees were interviewed; medical records were reviewed.

Results.—All attendees were male; median age was 51 years (range: 22–75). After a median incubation period of 7 days, 9 (82%) of 11 exposed persons experienced illness lasting a median of 12 days. All 9 sought outpatient healthcare for symptoms including fever, chills, sweats, and headache (100%) and ocular disturbances (33%). Testing confirmed the illness as toxoplasmosis and venison as the infection source. Multiple laboratory results were atypical for toxoplasmosis, including transaminitis (86%), lymphocytopenia (88%), thrombocytopenia (38%), and leukopenia (63%). One exposed but asymptomatic person was seronegative; the other had immunity from prior infection. The *T. gondii* strain was identified as closely related to an atypical genotype (haplogroup 12, polymerase chain reaction restriction fragment length polymorphism genotype 5) common in North American wildlife but with previously uncharacterized human clinical manifestations.

Conclusions.—The *T. gondii* strain contaminating the venison might explain the unusual clinical presentations. In North America, clinicians and venison consumers should be aware of risk for severe or unusual presentations of acute toxoplasmosis after consuming undercooked game meat.

Keywords

toxoplasmosis; genetic characterization; *Toxoplasma gondii* ; outbreak

Toxoplasmosis, a worldwide zoonosis caused by the protozoan parasite *Toxoplasma gondii*, is a leading foodborne cause of death in the United States [1]. Identified in 1939 as a causative agent of certain congenital abnormalities in humans [2, 3], *T. gondii* can infect any warm-blooded animal [4]. Its definitive hosts are the Felidae [4], and transmission to humans occurs by multiple routes, including accidental ingestion of oocysts from cat feces and consumption of undercooked meat containing viable tissue cysts. Most immunocompetent people experience subclinical infections, but some can experience chorioretinitis or unusual manifestations such as myocarditis, myositis, or hepatitis [5, 6].

Although *T. gondii* was initially categorized into three main clonal types (types I, II, and III) by polymerase chain reaction restriction fragment length polymorphism (PCR-RFLP) markers [7], research has revealed additional genotypic diversity with strains that do not cluster into these three clonal types referred to as atypical [8, 9]. *T. gondii* strains have been categorized into >200 different genotypes using PCR-RFLP or into 16 haplogroups using whole-genome sequencing, with haplogroups 1–3 corresponding to the 3 main clonal types and haplogroups 4–16 corresponding to atypical genotypes [8–10]. Illness severity varies by strain, with atypical strains associated with more severe human illness [11–13]. North American wildlife has been reported to be infected with atypical *T. gondii* genotypes [14]. One study identified atypical genotypes belonging to haplogroup 12 (referred to as type 12 [14] and corresponding to PCR-RFLP genotypes 4 and 5 [10]) in 47% of 97 samples from wildlife [15]. Because hunting and consumption of game meat is a common practice in North America [16], risk for exposure to *T. gondii*, especially strains with atypical genotypes, is substantial.

We describe an outbreak of toxoplasmosis among 9 of 11 men who consumed undercooked venison contaminated with an atypical *T. gondii* strain related to haplogroup 12 at a September 2017 weekend retreat in Wisconsin, USA. We present epidemiologic and laboratory (serologic and genotypic) components of the investigation and discuss implications for patient care and public health.

Outbreak Identification

On 8 October, a man aged 57 years (patient 1) sought treatment at a Wisconsin emergency department (ED) and reported multiple complaints, including sweats of 2 days' duration, difficulty sleeping, malaise, and intermittent hives. Laboratory test results showed mild leukopenia and transaminitis; a mononucleosis screen was negative. He reported recent exposure to wooded areas. He was discharged with a pending Lyme disease test result, an azithromycin prescription for suspected sinusitis, and instructions to follow up with his primary care physician (PCP).

On 9 October, patient 1 returned to the ED in lieu of following up with his PCP because of fevers up to 103°F, myalgia, body aches, headache, dark urine, fatigue, and self-reported rash. Influenza was ruled out, and additional test results showed worsening leukopenia, transaminitis, and elevated bilirubin. The consulting infectious disease physician (author D.L.) changed patient 1's treatment to a 10-day course of doxycycline (100 mg twice daily) for a suspected atypical tickborne illness, such as anaplasmosis. Patient 1 was discharged with a pending anaplasmosis test result and instructions to follow up with D.L.

On 11 October, patient 1 visited D.L. for follow-up and reported improvement of symptoms. However, he now reported similar illnesses among 8 other men who had spent the weekend of 29 September to 1 October with him at his cabin for a retreat. During the retreat, the men consumed never frozen, intentionally undercooked venison from a white-tailed deer that patient 1 had locally harvested and self-processed the week prior.

On 19 October, patient 2, a man aged 58 years who was also a retreat attendee, was treated by D.L. Patient 2 had been prescribed doxycycline at an urgent care clinic on 10 October based on his reports of the care patient 1 had received. He reported significant symptom improvement for himself and other attendees who had sought the same treatment at local urgent care clinics and emergency departments. Because patients 1 and 2 had negative *Anaplasma*/*Ehrlichia* panel test results, the hypothesized diagnosis was rejected.

On 19 October, D.L. called the Wisconsin Department of Health Services, Division of Public Health (DPH) to discuss potential diagnoses and report a suspected outbreak. DPH's differential diagnosis included leptospirosis, trichinellosis, and toxoplasmosis. By 30 October, 3 patients from the retreat were being treated at D.L.'s clinic. All 3 were tested for immunoglobulin (Ig) M anti-*T. gondii* antibodies at a commercial laboratory; results were positive. However, confirmation of *T. gondii* infection requires additional tests at a reference laboratory because IgM assays can produce false-positive results, and IgM levels can remain elevated for approximately 18 months after infection [17, 18]. Hypothesizing the outbreak cause was toxoplasmosis after consumption of undercooked venison, DPH conducted an investigation in collaboration with Jackson County Health and Human Services (JCHHS) to confirm the outbreak cause and source.

METHODS

Epidemiologic Investigation

From patient 1, JCHHS collected frozen venison that remained from the harvested and partially consumed deer along with a list of retreat attendees, meals, and activities. With input from the Centers for Disease Control and Prevention (CDC), DPH used this information to construct a standardized questionnaire covering exposures, illness symptoms, and treatment. All 12 retreat attendees were interviewed.

Confirmation of Human *T. gondii* Infections

For the 11 attendees with available serum samples, testing was performed at the Palo Alto Medical Foundation Toxoplasma Serology Laboratory, National Reference Center for the Study and Diagnosis of Toxoplasmosis (PAMF-TSL). The following assays were performed: Sabin-Feldman IgG dye test (DT), IgM double-sandwich enzyme-linked immunosorbent assay (ELISA), IgA double-sandwich ELISA, IgE double-sandwich ELISA, IgG differential agglutination (of methanol [AC]-fixed vs that of formalin [HS]-fixed tachyzoites) test, and IgG avidity (bioMérieux) [19]. Results were categorized as acute *T. gondii* infection (IgG+/IgM+) confirmed with additional testing diagnostic of a recently acquired infection (low IgG avidity, acute pattern differential agglutination [AC/HS], positive IgA, and/or positive IgE), chronic *T. gondii* infection (IgG+/IgM−), or never infected with *T. gondii* (IgG−/IgM−) [19]. Testing of 5 serum samples was also performed by the US Department of Agriculture (USDA) Animal Parasitic Diseases Laboratory by using the modified agglutination test (MAT) [20] and IgG ELISA [21].

Medical Record Abstraction

DPH requested medical records for all outpatient and ED visits corresponding with evaluation and treatment for illness during October 2017–February 2018 from all facilities that patients had reported visiting during their illness. Signs, symptoms, and treatments were abstracted by 2 abstractors, with differences resolved by consensus, and synthesized with interview results.

Confirmation and Characterization of Venison *T. gondii* Contamination

The frozen venison was shipped to the PAMF-TSL and USDA. PAMF-TSL performed *T. gondii*-PCR testing [22] of the venison samples. USDA performed MAT testing of venison meat juice and inoculated mice with meat digests [20]. *T. gondii* DNA extracted from the venison was sent to the French National Reference Center for Toxoplasmosis (CNR Toxoplasmose). CNR Toxoplasmose performed *T. gondii* genotyping based on 15 microsatellite markers. Although genotyping based on microsatellite markers cannot be translated into a PCR-RFLP genotype or haplogroup, neighbor-joining trees can identify the PCR-RFLP genotype or haplogroup to which a strain is most closely related. Neighbor-joining trees were constructed to examine the association between the isolated strain and (1) reference strains from 16 *T. gondii* haplogroups identified worldwide [23] and (2) 46 strains collected from animals and humans in North America (Supplementary materials, Table S1).

RESULTS

Epidemiologic Investigation

All retreat attendees were male; median age was 51 years (range: 22–75). Eleven (92%) of the 12 retreat attendees consumed the venison; the attendee who did not consume the venison did not become ill (attendee 12). Among the 11 men who consumed venison, 9 (82%) were ill (patients 1–9), and 2 (18%) were well (attendees 10–11; Table 1). Ten (91%) of 11 men who consumed the venison as grilled kabobs (Supplementary materials, Figure S1) on the evening of 29 September reported that it was possibly or definitely undercooked. Six (67%) of 9 men who ate the venison for breakfast the following morning reported that it was possibly or definitely undercooked. All ill men sought outpatient healthcare and were treated with doxycycline, and none had immunosuppressive conditions or were taking immunosuppressive medications.

Confirmation of Human *T. gondii* Infections

PAMF-TSL *T. gondii* testing results for patients 1, 2, and 4–9 were diagnostic of acute *T. gondii* infection (Table 2). In patients 1, 4, 6, and 7, in addition to avidity, AC/HS IgA, and/or IgM test results consistent with an acute infection, a significant rise in IgG was observed further supporting the diagnosis of acute infection in these patients. The commercial laboratory *Toxoplasma*-IgM result for patient 3 was positive, suggestive of recent *T. gondii* infection. Attendee 10, who consumed the venison but did not become ill, tested negative for IgG anti-*T. gondii* antibodies at both a commercial laboratory and PAMF-TSL at 76 days after exposure. Attendee 11, who also consumed the venison and did not become ill, had serologic results consistent with *T. gondii* infection acquired in the

distant past (>1 year). Attendee 12, who did not consume the venison and did not become ill, showed no serologic evidence of *T. gondii* infection when tested at 54 days after the retreat. USDA serologic results were consistent with PAMF-TSL results. For patients tested, all vectorborne disease panels and brucellosis, influenza, Epstein-Barr virus, and leptospirosis testing results were negative for acute infection.

Clinical Manifestations

Median incubation period was 7 days (range: 6–9 days; Table 1). Median illness duration was 12 days (range: 5–22 days). All 9 patients reported headache, muscle aches, fatigue, fever, sweats (including drenching night sweats), and chills. Other reported symptoms included decreased appetite, arthralgias, dark urine, lymphadenopathy, and sore throat (see Table 1).

Lymphadenopathy was noted during clinical examination for 3 patients (33%). One patient reported chest pain, shortness of breath, coordination issues, and confusion. Although no rash was visible during examination, one patient described to ED personnel intermittent hives or maculopapular rash.

Ocular symptoms reported by 3 patients (33%) included eye pain, blurred vision, and photophobia. Patient 1 described diplopia that had mostly resolved by 6 weeks postexposure. On examination of extraocular movements, there was evidence of exophoria supporting patient 1's complaint of diplopia.

Laboratory Findings

Complete blood counts with differential (CBC) were available for 8 of 9 patients, with repeat bloodwork available for 3 patients. Liver function tests (LFT) were available for 7 of 9 patients, with repeat bloodwork available for 2 patients. Table 1 contains the most extreme values for each patient's relevant results; Figure 1 plots CBC and LFT values over time. Five patients (63%) had leukopenia (<4000 white blood cells/mm^3), 7 (88%) had lymphocytopenia (<1500 lymphocytes/mm^3), and 3 (38%) had thrombocytopenia (platelet count <150,000/mm^3). Six (86%) patients with available LFTs had transaminitis, defined by elevation of either aspartate aminotransferase (AST) or alanine aminotransferase (ALT) above the upper limit of normal (>48 U/L for AST and >55 U/L for ALT). Only patient 1 (14%) had hyperbilirubinemia (>1.2 mg bilirubin/dL).

Confirmation and Characterization of Venison *T. gondii* Contamination

PCR results from PAMF-TSL and MAT results from USDA confirmed that the venison was contaminated with *T. gondii*. Mice inoculation tests performed by USDA were negative as the venison had been frozen. In the neighbor-joining tree with reference strains from 16 *T. gondii* haplogroups, the venison strain clustered with ARI, the haplogroup 12 reference strain. In the second neighbor-joining tree with the North American strains (Figure 2), the venison strain clustered with TgWolfMN26, a strain isolated from a wolf harvested in Minnesota. TgWolfMN26 was previously characterized as PCR-RFLP genotype 5 [26], which corresponds to haplogroup 12 [10].

DISCUSSION

During October 2017, an outbreak of toxoplasmosis was identified among a group of men who consumed undercooked *T. gondii*-contaminated venison while attending a weekend retreat. This outbreak, which was found to be associated with an atypical *T. gondii* strain, had a high attack rate of 82% and both clinical and laboratory findings atypical of acute toxoplasmosis. Unlike previous published reports of similar illnesses that only suspected venison as the vehicle [27–30], venison was confirmed as the infection source for this outbreak. This outbreak highlights the potential for exposure to an atypical strain of *T. gondii* common among wildlife in North America to result in an unusually severe presentation of acute toxoplasmosis among immunocompetent people.

The signs and symptoms exhibited by patients in this outbreak differed in multiple ways from the expected clinical manifestation of toxoplasmosis. Although infection in immunocompetent persons is usually asymptomatic or mildly symptomatic [31, 32], all infected patients experienced a febrile illness severe enough to seek outpatient healthcare. Fewer patients than expected presented with lymphadenopathy [32], whereas more patients than expected presented with transaminitis, ocular manifestations, and hematological abnormalities (eg, leukopenia, lymphocytopenia, and thrombocytopenia) [31, 32]. These unusual clinical manifestations and laboratory results likely contributed to diagnostic delay. Before public health consultation, none of the 16 healthcare providers involved in the care of ill attendees had included toxoplasmosis in the initial differential diagnosis.

Because of the delayed diagnosis and sharing of treatment information among the affected men, all patients received doxycycline instead of a standard treatment for toxoplasmosis (eg, pyrimethamine-sulfadiazine, TMP-SMX). Multiple patients reported improvement after starting treatment, which might have been a placebo effect or the nature of a self-limited illness. However, it is biologically plausible that clinical symptoms and severity of disease were attenuated by an anti-*T. gondii* effect derived from doxycycline therapy. Experimental models of murine toxoplasmosis have documented antimicrobial effects of doxycycline [33, 34], and in 1992 during the surge of human immunodeficiency virus and AIDS-related *Toxoplasma*-encephalitis, a recommendation was made to examine the role of doxycycline for toxoplasmosis based on several case-reports [35]. A case report of venison-related toxoplasmosis published in 2018 also reported similar improvement with doxycycline [28].

The genotype is a potential explanation for the high attack rate and unusual presentation. In neighbor-joining analyses of microsatellite marker genotype data, the venison strain was unrelated to the 3 clonal types, and closely related to an atypical strain from North America with a genotype corresponding to haplogroup 12 [26]. Haplogroup 12 seems to be common in wildlife in North America [15], and virulence testing in mice has demonstrated a substantial range of mortality (10%–71.4%) [14]. Transmission to humans on the continent has occurred, as evidenced by a recent study of toxoplasmosis among North American residents [36]. The relative frequency of haplogroup 12 strains identified in North American wildlife samples highlights the importance of considering game consumption as a risk factor for toxoplasmosis.

Recent seroprevalence studies of North American wildlife have reported *T. gondii* infection rates in white-tailed deer of 23%–74% in Minnesota [20, 37], Iowa [37], and Ohio [20, 38]. Approximately 300 000 deer were harvested during the 2017 Wisconsin deer season alone (https://dnr.wi.gov), and the number of people potentially exposed yearly to *T. gondii*, and atypical strains including haplogroup 12, is quite high. Although *T. gondii* tissue cysts can be killed by freezing meat for several days at subzero (0°F) temperatures or fully cooking meat (https://www.cdc.gov/parasites/toxoplasmosis/prevent.html), anecdotal evidence from this outbreak and others indicates that hunters sometimes eat raw or rare venison. In 4 other studies of suspected venison-transmitted toxoplasmosis, consumption of undercooked or raw venison was reported [27–30]. In a follow-up study of the coworkers of the patients in one report, *T. gondii* seropositivity was significantly associated with having eaten raw or rare venison [30]. Another of the reports describes a toxoplasmosis outbreak among a group of healthy male Canadian hunters who consumed undercooked venison in Illinois, USA [27]. The Canadian outbreak is almost identical to this outbreak in attack rate, incubation period, symptom severity, and the inclusion of one seronegative but presumed exposed individual. As Wisconsin and Illinois share a border, it is possible that similar strains of *T. gondii* caused the outbreaks. Increased awareness of toxoplasmosis and risk for consuming undercooked venison and other game is needed among hunters, consumers of game, and healthcare providers. In the absence of such awareness of game as a potential source of exposure, patients might not disclose relevant risk factors in an expedient manner.

This investigation had multiple strengths and limitations. The willingness of patients in this outbreak to be interviewed and to provide blood and venison samples and the access to necessary testing to determine acute human infection and venison contamination led to confirmation of both the infectious agent and source of exposure. Retreat attendees were interviewed 41–128 days after the retreat, making recall bias a possibility for both exposures and illness symptoms. In addition, recovery dates were challenging to ascertain because of lingering symptoms like fatigue and individual variation in tolerance for symptoms. The combination of interview and medical record data increased confidence that the fullest possible picture of symptoms was acquired. However, it is possible that more extensive medical examinations and follow-up might have revealed additional manifestations such as subclinical retinitis, myocarditis, myositis, or encephalitis, but extensive medical examinations and follow-up did not fall into the purview of this outbreak investigation.

This outbreak highlights potential for the underdiagnosis of toxoplasmosis acquired through consumption of undercooked *T. gondii*-contaminated venison or other game, and particularly the possibility of atypical clinical manifestations because of infection with atypical strains. Healthcare providers should ask patients about recent consumption of undercooked game when diagnosing febrile illness, and hunters and other consumers of game should be educated by both the medical and public health communities to fully cook game meat to prevent toxoplasmosis and other illnesses.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments.

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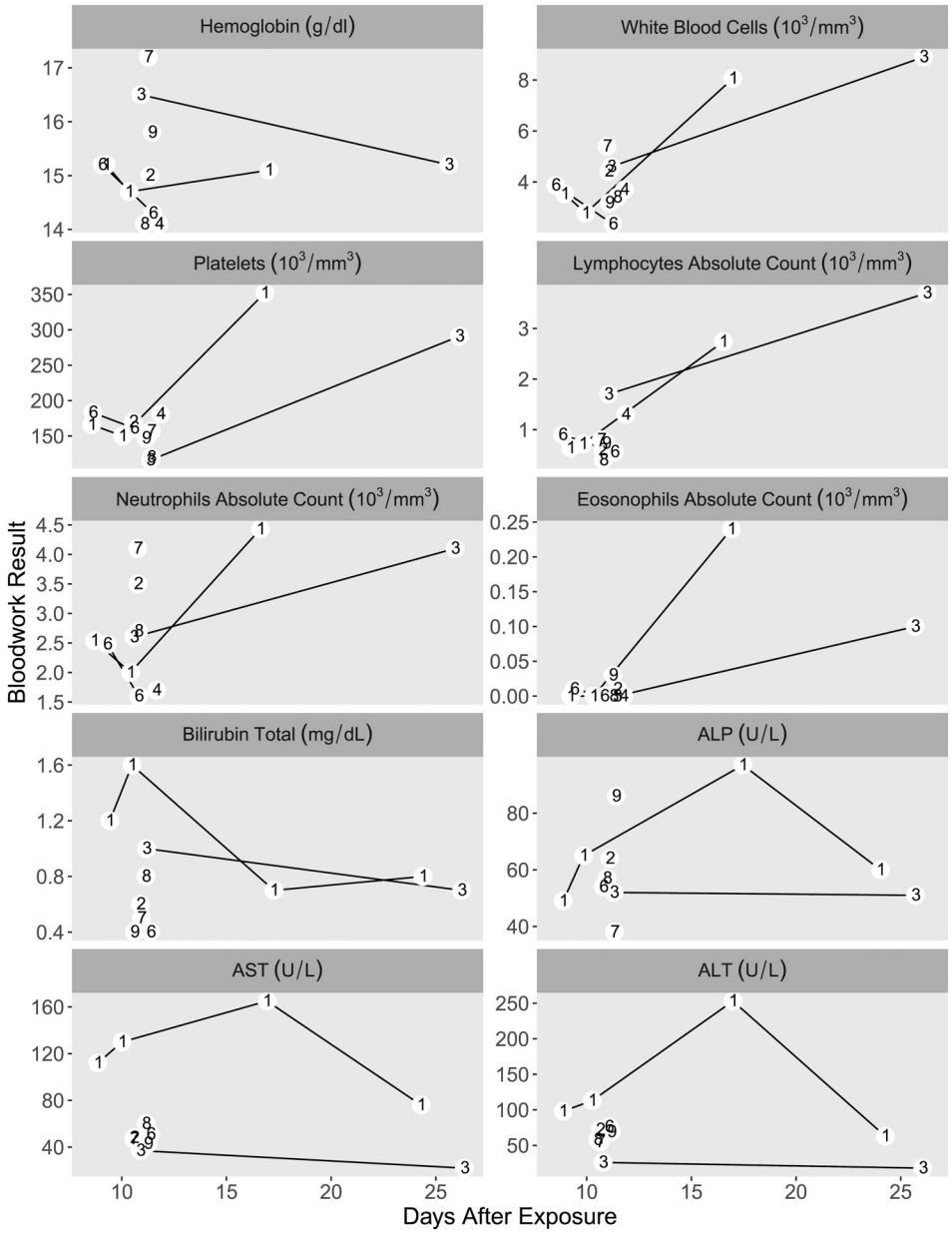

Figure 1.

CBC and liver function test abnormalities and resolution of Toxoplasma gondii infection in retreat attendees. Numbers in circles correspond to the patient IDs in Table 1. Blood test results were available for 8 of 9 cases, with repeat CBC with differential available for 3 patients (1, 3, and 6) and repeat LFTs available for 2 patients (1 and 3). Days after exposure was calculated as the number of days from 29 September to the date of blood sample. Points were jittered on the x-axis (days after exposure) to decrease overlapping of points. Graphs were produced using *R* (version 3.5.2). Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CBC, complete blood counts; ID, identification; LFT, liver function test.

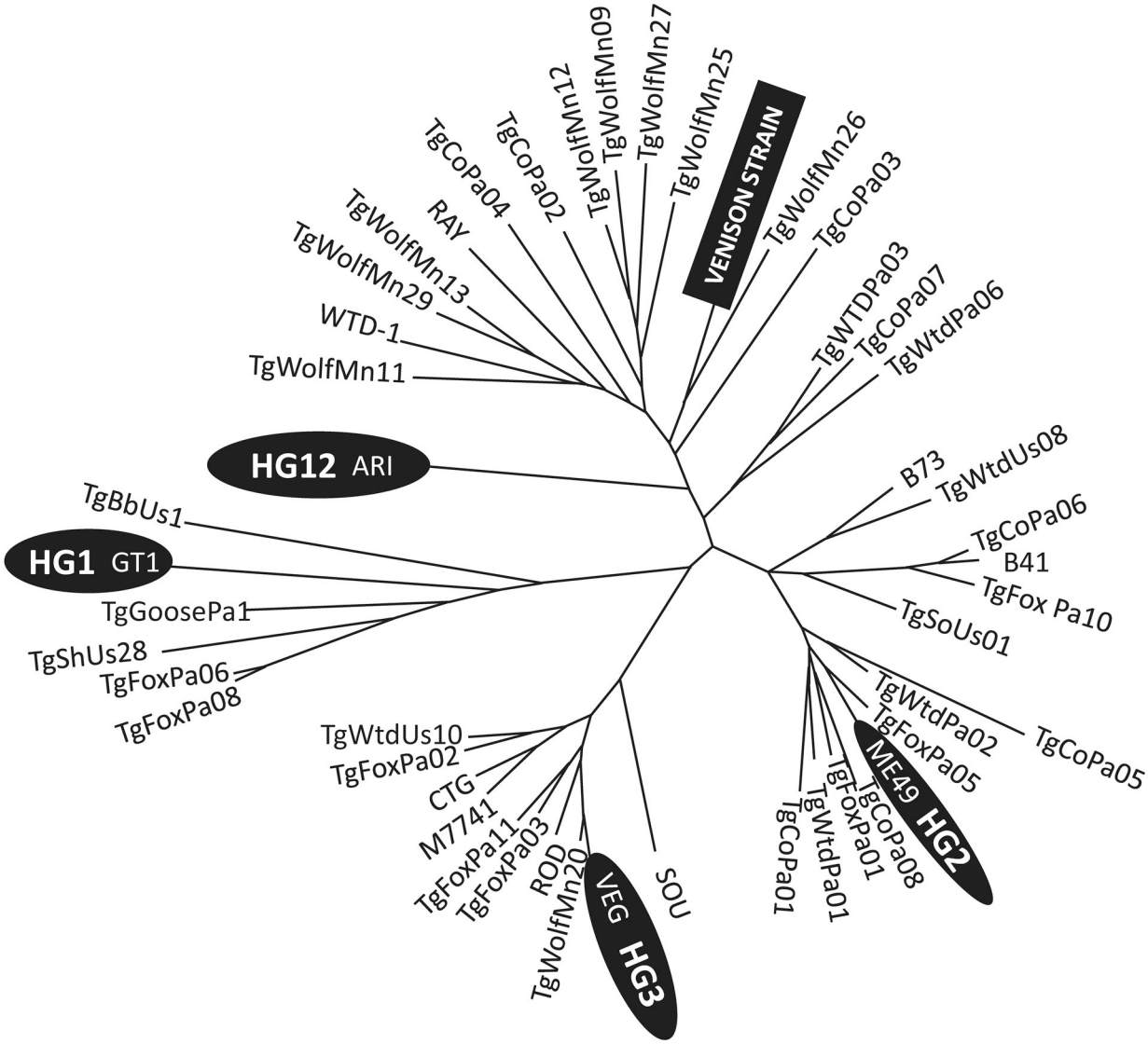

Figure 2.

Neighbor-joining clustering based on 15 microsatellite markers of the *Toxoplasma gondii* strain isolated from the Wisconsin venison sample and 46 strains collected from humans and animals in North America. The tree was constructed with populations 1.2.32 (http://bioinformatics.org/populations/) based on Cavalli-Sforza and Edwards chord-distance estimator [26] and generated with MEGA 6.05 (http://www.megasoftware.net/history.php) software. The *T. gondii* strain isolated from the venison sample is highlighted in black and the reference strains for haplogroups 1, 2, 3, and 12 are labeled.

Table 1.

Epidemiologic, Serologic, and Clinical Description of *Toxoplasma gondii* Infection in Retreat Attendees—Wisconsin, 2017

	Patient/Attendee											
	Ill									Not Ill		
Findings and Test Results[a]	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
Age (y)	57	58	69	53	22	34	65	49	33	43	37	75
Exposure and diagnosis												
Consumed venison	+	+	+	+	+	+	+	+	+	+	+	−
Venison possibly undercooked	+	+	+	+	+	+	+	+	+	+	−	NA
Serology[b]	A	A	A[c]	A	A	A	A	A	A	N	I	N
Illness course and treatment										Median	% Present	
Incubation period (d)	7	6	6	6	6	7	9	7	7	7	…	
Illness length (d)	5	11	11	19	22	6	20	12	14	12	…	
Days between exposure and antibiotic initiation	10	11	11	12	11	9	11	11	11	11	…	
Symptoms												
Highest reported temperature (°C)	39.6	40	40	39.6	40	39	37.8	40	40.3	40	…	
Headache	+	+	+	+	+	+	+	+	+	…	100%	
Fever, sweats, and chills	+	+	+	+	+	+	+	+	+	…	100%	
Fatigue	+	+	+	+	+	+	+	+	+	…	100%	
Muscle aches	+	+	+	+	+	+	+	+	+	…	100%	
Decreased appetite	+	+	+	+	+	+	u	+	+	…	89%	
Joint aches	−	+	−	+	+	−	+	+	+	…	67%	
Dark urine	+	+	−	+	−	+	−	−	−	…	44%	
Sore throat	+	−	−	−	−	−	+	+	−	…	33%	
Swollen lymph nodes	+	+	−	u	−	u	−	u	−	…	22%	
Blurred vision	+	+	u	−	−	−	−	−	−	…	22%	
Sensitivity to light	u	+	−	−	−	+	−	u	−	…	22%	
Cough	−	−	−	+	−	u	−	−	−	…	11%	
Chest pain	−	−	−	+	−	−	−	−	−	…	11%	
Shortness of breath	−	−	−	+	−	−	−	−	−	…	11%	
Coordination issues	−	−	−	+	−	−	−	u	−	…	11%	

	Patient/Attendee											
	Ill									Not Ill		
Findings and Test Results[a]	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
Confusion	−	−	−	+	−	−	−	−	−	…	11%	
Eye pain	−	−	−	−	−	+	−	−	−	…	11%	
Rash	u	−	−	−	−	−	−	u	−	…	0%	
Seizures	−	−	−	−	−	−	−	−	−	…	0%	
Clinical measures and findings[d]												
Measured temperature (°C)[e]	37.8	38.3	36.9	36.2	38.8	37.8	37.1	38.9	37.6	37.6	…	
Lymphadenopathy on exam	+	+	+	−	−	−	−	−	−	…	33%	
White blood cells (10^3/mm^3)	**2.78**	4.42	**4.6**	**3.7**	NA	**2.35**	5.4	**3.4**	**3.18**	3.55	…	
Platelets (10^3/mm^3)[f]	150	170	**116**	181	NA	161	157	**120**	147	153.5	…	
Hemoglobin (gm/dL)[e]	15.2	15	16.5	14.1	NA	15.2	**17.2**	14.1	15.8	15.2	…	
Absolute lymphocytes (10^3/mm^3)[f]	**0.64**	**0.6**	1.7	1.3	NA	**0.56**	**0.8**	**0.4**	**0.73**	0.69	…	
Absolute neutrophil (10^3/mm^3)[f]	**2**	3.5	2.6	**1.7**	NA	1.6	4.1	2.7	NA	2.6	…	
Absolute eosinophil (10^3/mm^3)[f]	**0**	**0.01**	0	**0**	NA	0	*NA*	**0**	**0.03**	0	…	
AST (U/L)[e]	**165**	**48**	**37**	NA	NA	**51**	**47**	**60**	**43**	48	…	
ALT (U/L)[e]	**253**	**72**	26	NA	NA	**76**	55	58	**69**	69	…	
ALP (U/L)[e]	49	64	51	NA	NA	54	**38**	57	86	54	…	
Bilirubin total (mg/dL)[e]	**1.6**	0.6	1	NA	NA	0.4	0.5	0.8	0.4	0.6	…	

Abbreviations: ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; lymph, lymphocytes.

[a] + = present, − = absent, u = patient unsure.

[b] A = acute, N = negative, I = immune.

[c] From commercial laboratory.

[d] Bolded = abnormal according to laboratory of origin.

[e] Highest measured.

[f] Lowest measured.

Table 2.

PAMF-TSL *Toxoplasma* Serology Panel Quantitative Results and Qualitative Interpretations

			Test Results[a,b,c,d]					
			IgG					
Patient/ Attendee	Panel Interpretation[d]	Days After Exposure	Sabin-Feldman Dye Test	Differential Agglutination (AC/HS)	Avidity	IgM ELISA	IgA ELISA	IgE ELISA
1	A	17	1:512 (+)	<50/<100 (Nonreactive)	Unable to perform[e]	9.9 (+)	2.4 (+)	3.6 (+)
		188	1:2048 (+)	200/>3200 (Nonacute)	23.2 (Equivocal)	2.2 (+)	0.0 (−)	0.0 (−)
2	A	18	1:512 (+)	<50/<100 (Nonreactive)	8.4 (Low)	10.0 (+)	4.9 (+)	5.2 (+)
		151	1:512 (+)	200/800 (Equivocal)	3.7 (Low)	11.3 (+)	1.9 (Equivocal)	3.7 (+)
4	A	59	1:2048 (+)	800/800 (Acute)	4.4 (Low)	10.2 (+)	7.0 (+)	3.0 (+)
		138	1:16 000 (+)	> 1600/3200 (Acute)	14.2 (Low)	7.2 (+)	5.5 (+)	3.8 (+)
5	A	188	1:1024 (+)	1600/>3200 (Equivocal)	26.9 (Equivocal)	1.5 (−)	0.0 (−)	0.0 (−)
6	A	60	1:2048 (+)	400/400 (Acute)	3.6 (Low)	8.8 (+)	4.3 (+)	3.6 (+)
		139	1:8000 (+)	1600/1600 (Acute)	9.9 (Low)	6.9 (+)	2.3 (+)	1.4 (−)
7	A	52	1:256 (+)	<50/<100 (Nonreactive)	4.3 (Low)	5.9 (+)	0.7 (−)	3.7 (+)
		143	1:8000 (+)	800/1600 (Acute)	10.5 (Low)	7.5 (+)	3.4 (+)	3.2 (+)
8	A	194	1:2048 (+)	100/400 (Equivocal)	10.5 (Low)	8.5 (+)	1.3 (−)	1.2 (−)
9	A	49	1:512 (+)	200/100 (Acute)	1.8 (Low)	9.9 (+)	3.1 (+)	2.0 (+)
10	N	76	<1:16 (−)	<50/<100 (Nonreactive)	Unable to perform	0.0 (−)	0.0 (−)	0.0 (−)
11	I	123	1:256 (+)	<50/800 (Nonacute)	42.6 (High)	0.0 (−)	0.0 (−)	0.0 (−)
12	N	54	<16 (−)	<50/<100 (Nonreactive)	Unable to perform	0.0 (−)	0.0 (−)	0.0 (−)

Abbreviations: AC/HS, the differential agglutination (of acetone [AC]-fixed versus that of formalin [HS]-fixed tachyzoites; ELISA, enzyme-linked immunosorbent assay; Ig, immunoglobulin; PAMF-TSL, Palo Alto Medical Foundation Toxoplasma Serology Laboratory.

[a] A = acute, N = negative, I = immune.

[b] + = positive, − = negative.

[c] Reference values from PAMF-TSL serological testing: IgG dye test (positive ≥1:16 dilutions; negative <1:16 dilutions); IgG AC/HS (see published interpretation [24]); IgG avidity (low avidity: <20%); IgM-ELISA (positive ≥2.0 units; indeterminate 1.7–1.9 units; and negative ≤1.6 units); IgA-ELISA (positive ≥2.1 in adults); IgE ELISA (positive ≥1.9 units, equivocal 1.5–1.8 units, negative 0.0–1.4 units).

[d] Patient 3 did not have testing performed at the PAMF-TSL.

[e] IgG avidity could not be performed because IgG antibody titer was below the minimum required for testing.