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An Outbreak of Toxoplasmosis Linked to Cats

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ABSTRACT. Clinical, serologic, and epidemiologic evidence documents an outbreak of toxoplasmosis involving ten of 30 members of an extended family. The index patient had unusual clinical manifestations including brain abscesses, progressive chorioretinitis, seizures, neurologic deficits, hepatosplenomegaly, pneumonitis, and eosinophilia. Toxoplasmosis was confirmed by demonstrating the organism in brain tissue and cerebrospinal fluids; clinical and serologic evidence also indicated infection with *Toxocara* (visceral larva migrans). This outbreak of toxoplasmosis was confined largely to preschool-aged children. Of the 11 such children, seven (68%) were seropositive, six of whom had high acute-phase titers (≥ 1024) to *Toxoplasma* and a disease consistent with acute toxoplasmosis. All six of the latter group required specific chemotherapy. Geophagia was associated statistically with acute toxoplasmosis among the children; it also increased the risk of infection with *Toxocara* and enteroparasites. Two school-aged children and two adults had serologic evidence of acute toxoplasmosis, but only one of the group was symptomatic. Epidemiologic evidence indicates that this outbreak was probably caused by ingesting oocysts from cat feces. We suggest that the severe and unusual clinical manifestations of the index patient resulted from simultaneous infection with *Toxoplasma* and *Toxocara*. *Pediatrics* 65:706-712, 1980; *toxoplasmosis, outbreak, cats, brain abscess, toxocariasis*.

Although *Toxoplasma* is one of the most common agents of human parasitic infection, toxoplasmosis is seldom recognized as a clinical entity.^{1,2} Clinical manifestations of acute toxoplasmosis in the immune-competent host frequently include lymphadenitis, asthenia, myalgia, and headache.³ Prominent pneumonitis and/or encephalomyelitis

are more characteristic of toxoplasmosis in the immunosuppressed host.⁴

Under natural conditions toxoplasmosis can be transmitted to people in poorly cooked or raw infected meat,^{5,6} through the placenta during a primary maternal infection,⁷ and through direct exposure to *Toxoplasma* oocysts excreted in the feces of infected cats.⁸⁻¹⁰ Outbreaks of toxoplasmosis are rare. Of the two that have been reported in the United States, one involved five medical students who ate rare meat, and the other involved 29 patrons of a riding stable whose infections were epidemiologically linked to exposure to oocysts.^{11,12} In this paper we discuss the clinical and epidemiologic characteristics of an outbreak of toxoplasmosis involving ten of 30 members of an extended family. The unusual clinical picture of the index patient and the high frequency of clinical disease for other family members suggest that toxoplasmosis is a less benign infection than the literature indicates.

CASE REPORT

The index patient was a previously healthy, normal sized, 3½-year-old white boy who developed fever, non-productive cough, wheezing, nasal congestion, and cervical lymphadenopathy in August 1976. Two weeks after onset of illness, he was admitted to a hospital in Dothan, AL. He had diffuse bilateral rales and radiographic evidence of extensive bilateral peribronchial infiltrates; his white blood cell count (WBC) was 7,000, with 52% eosinophils. After *Ascaris* worms were observed in his stools, he was given an eight-day course of piperazine for possible *Ascaris* pneumonitis. He was discharged on the fourth day of therapy. During the next three weeks, he remained febrile (101 F), became progressively irritable and weak, and developed hepatosplenomegaly. After two episodes of generalized self-limited seizures he was readmitted to the hospital with fever (105 F), irritability, inability to walk, and slurred speech. On physical examination the most salient features were a right hemiparesis with spas-

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ticity, cervical lymphadenopathies, and splenomegaly. Laboratory work-up revealed leukocytosis (23,900 WBC) with marked eosinophilia (38%), radiographic evidence of diffuse, bilateral pneumonitis; and elevated levels of SGOT (92 IU/liter), lactic dehydrogenase (960 IU/liter), and creatinine phosphokinase (390 IU/liter). Negative results were obtained with blood cultures for bacteria, lupus erythematosus preparation, and the tuberculin skin test, and for antinuclear antibodies and febrile agglutinins. Total bilirubin was 0.3 mg/100 ml, and total protein was 8.2 gm/100 ml, with 4.9 gm/100 ml of globulin. IgA was 210 mg/100 ml, IgG was 3,300 mg/100 ml, and IgM was 750 mg/100 ml. Cysts of *Giardia lamblia* were found in stool specimens. The tentative diagnosis was visceral larva migrans, and the patient was treated with thiabendazole (50 mg/kg/day). However, the patient's neurologic condition continued to worsen. On Sept 7, 1976, he was transferred to Children's Hospital in Birmingham, AL, where a neurologic examination revealed a stuporous, irritable, aphasic patient with motor-spastic right hemiparesis and clonus of the right knee and ankle; he could not stand or walk even with support. Fundoscopic examination of the right eye revealed papilledema and two marginal chorioretinal scars. The left eye had two non-pigmented marginal lesions and a large (three disc diameters) scar, nasal of the disc, with overlying vitreous haze (both maculae appeared intact). Pertinent laboratory tests revealed moderate leukocytosis (13,800 WBC) with eosinophilia (16%), sedimentation rate 38 mm, isohemagglutinin A 1/64 and B 1/64, IgA 74 mg/100 ml, IgG 2,000 mg/100 ml, IgM 420 mg/100 ml, IgE 620 units/ml,

C₃ 98 mg/100 ml, CH₅₀ 268 units, and titers measured with indirect immunofluorescence of 1,024 for *Toxoplasma* IgG antibodies and 1,024 for *Toxoplasma* IgM antibodies. The cerebrospinal fluid (CSF) was clear and contained 18 WBC/ml (100% mononuclear cells), 72 mg/100 ml of protein and 75 mg/100 ml of glucose. Seven days after CSF was placed in tissue culture tubes containing human fibroblast monolayers, *Toxoplasma gondii* was positively identified by hematoxylin and eosin staining. A computed axial tomogram performed on the second day of admission revealed three large, low-density lesions (right parietal, left frontal, and left posterior parietal). Material from the right parietal area obtained in a brain biopsy performed on the following day revealed extensive encephalitis and contained multiple free and encysted forms of *T gondii* (Figs 1 and 2). Therapy was begun with pyrimethamine (1 mg/kg/day), sulfadiazine (150 mg/kg/day), folinic acid (5 mg/day), and prednisone (2 mg/kg/day). After a month of therapy, the CSF contained 29 WBC (79% mononuclear cells), 57 mg/100 ml of protein, and 10 mg/100 ml of glucose. Fundoscopic examination revealed three new lesions in the left eye and two new lesions in the right eye; the older lesions became pigmented, and vitreous haziness disappeared. However, the patient's neurologic status did not improve.

On Jan 3, 1977, the patient was readmitted to Children's Hospital for evaluation. Neurologic examination

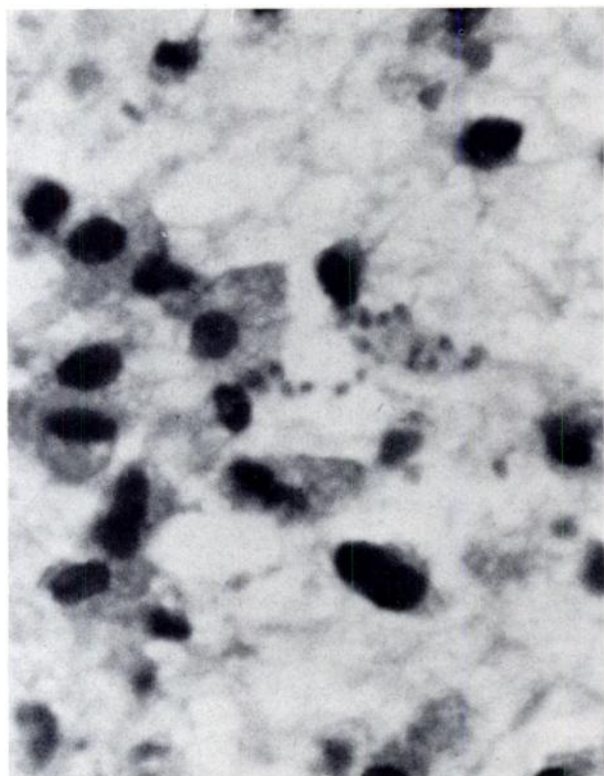


Fig 1. Brain biopsy: Trophozoites of *Toxoplasma gondii* in area of brain necrosis (hematoxylin-eosin, original magnification $\times 400$).

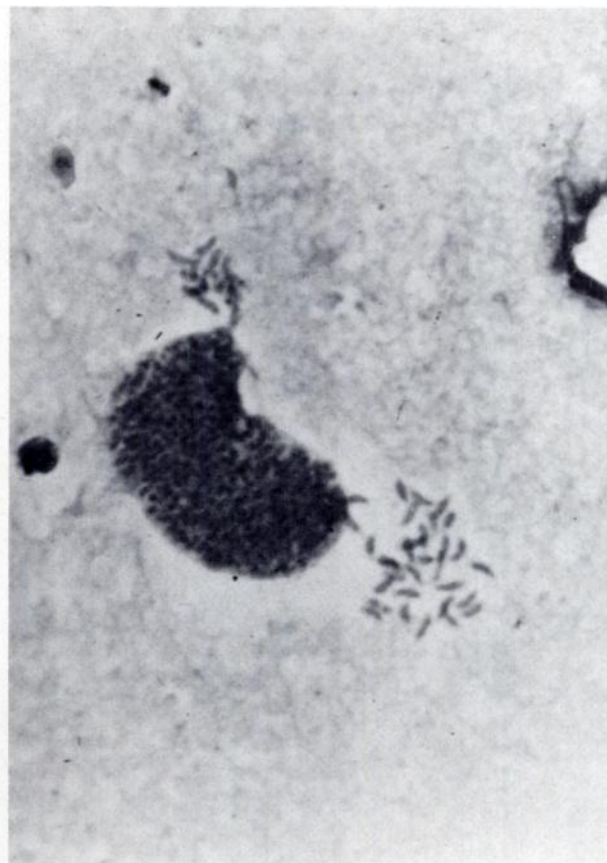


Fig 2. Brain biopsy: *Toxoplasma* cyst in cerebral cortex; note proximity of blood vessel and lack of inflammatory response (toluidine blue, original magnification $\times 1,000$).

to ten of the 11 preschool-aged children of the study family. Immunofluorescence tests for antibodies against *Toxoplasma* were performed on serum specimens at the Bureau of Laboratories, Alabama Department of Public Health, The University of Alabama Medical Center, and the Center for Disease Control. Sera were also tested for antibodies against *Toxocara* with the enzyme-linked immunosorbent assay at the Center for Disease Control.

Single stool specimens obtained from each of seven adults and 14 children within four weeks of the onset of the outbreak were examined for ova and parasites.

Samples of soil were checked for the presence of *Toxoplasma* oocysts by microscopic examination and by inoculating specimens into mice according to the method of Frenkel and Dubey.⁸

RESULTS

Serologic Studies

As illustrated in Table 1 and Fig 3, 57% of the 28 family members tested showed evidence of recent or past infection with *T gondii*. The highest rate of seropositivity (68%) was for preschool children; among them six of seven seropositive patients had antibody titers of at least 1,024 (indicative of recent infection), whereas only one of seven seropositive adults had as high a titer. On the initial screening all school-aged children were seronegative. Three months after these evaluations, three of 12 study subjects who had been seronegative had measurable titers against *T gondii* (two school-aged children and one adult). None of these patients had had clinical illness suggestive of toxoplasmosis, and their antibody titers ranged between 64 and 256.

When the second blood samples of 22 family members were tested with enzyme-linked immunoabsorbent assay, ten (45%) had antibody titers ranging from 32 to 256 against *Toxocara* (Table 1, Fig 3). The highest titer was measured in the index patient's serum. When serum samples of six of the seven preschool-aged children with acute toxoplasmosis were also tested for antibodies to *Toxocara*, only three were positive ≥ 32 . The serum of one other preschool-aged child was positive for *Toxocara* only.

None of the preschool-aged control children had

serologic evidence of *Toxoplasma* or *Toxocara* infections.

Symptomatology

At the time of the field investigation five children besides the index patient and one adult family member had histories and showed signs consistent with acute *Toxoplasma* infection (Fig 3). The adult, a 20-year-old woman, was the mother of the index patient; her symptoms included nasal congestion, bilateral cervical lymphadenopathy, nonproductive cough, and perihilar density on chest x-ray. Both her acute and convalescent-phase titers for *Toxoplasma* were indicative of active infection ($\geq 1,024$), and one titer measured for *Toxocara* was 32. She did not receive specific therapy. The six symptomatic children (four girls and two boys) ranged in age from 1½ to 4 years. Their symptoms included nonproductive cough (all six), nasal congestion (five), lymphadenopathies (five), malaise (four), pneumonitis (four), leukocytosis (three), eosinophilia (two), chorioretinitis (two), and central nervous system involvement (one). All six had acute-phase serum titers to *Toxoplasma* of $\geq 1,024$. Despite a one-month course of therapy with pyrimethamine, sulfadiazine, and foinic acid, when assessed two months later, the titers of all six had either remained stable or risen; however, five of the six children (all but the index patient) became asymptomatic.

Each of two asymptomatic seropositive adults (ages 21 and 50 years) had an inactive, noncentral, chorioretinal scar with the typical appearance of a healed *Toxoplasma* lesion. Their titers for *Toxoplasma* were 256 and 64 respectively.

Stool Examination

As illustrated in Table 1, one of seven adults and six of 14 children had laboratory evidence of enteroparasitic infections. *G lamblia*, the most common pathogen, was found in the stools of four persons. *Ascaris lumbricoides*, *Necator americanus*, and the commensal protozoans *Entamoeba coli* and *Endolimax nana* were found once each in the four stools from different family members. Stools of the index patient contained both *A lumbricoides* and *G lamblia*.

TABLE 1. Serologic Evidence of *Toxoplasma*, *Toxocara*, and Intestinal Parasitic Infections and Clinical Illness among Members of an Extended Family Who Were Tested

Age (yr)	Acute Clinical Illness	<i>Toxoplasma</i> Seropositivity	<i>Toxocara</i> Seropositivity	Intestinal Parasites
5	6/11 (55%)	7/11 (64%)	4/9 (44%)	5/9 (55%)
6-18	0/5	2/5 (40%)	3/4 (75%)	1/5 (20%)
≥ 19	1/14 (7%)	7/12 (58%)	3/9 (33%)	1/7 (14%)
Total	7/30 (23%)	16/28 (57%)	10/22 (45%)	7/21 (33%)

Epidemiologic Studies

In the hot, humid summer the children played in the yard of household A. At the time of the investigation, the yard was described as "a natural sand-box," a moist, loosely packed, sandy surface approximately 50 x 80 feet. The only domestic animal belonging to the family was a cat that had had a litter of six kittens in the first week of June 1976. A month later several of the kittens had been affected with a nonspecific illness, and one had died. The family, acting independently, had had all cats removed during the second week of October. However, when the female cat later returned home, her titer to *Toxoplasma* as measured with the indirect hemagglutination test was 1,024. Although before October the cats had been allowed to defecate at random in the yard, at the time of the investigation no cat feces could be identified. Microscopic examination of and mouse inoculation with nine samples of soil failed to demonstrate the presence of *Toxoplasma* oocysts.

Of 11 preschool-aged children, eight (73%) had a history of geophagia (Table 2). In fact, among the toys that the children played with in the yard were several tablespoons. Seven of these eight children were seropositive for *Toxoplasma*, whereas the three children with no history of geophagia had no evidence of infection ($P = .024$, Fisher's exact test). As illustrated in Table 2, geophagia was also associated with *Toxocara* and enteroparasitic infections. None of these parasitic infections occurred in the children who did not have a history of geophagia.

All but one family member liked meat "well done." Interestingly, this person had a positive, low (64) but stable antibody titer for *Toxoplasma*.

DISCUSSION

The clinical, serologic, and epidemiologic information contained in this report provide compelling evidence of a family outbreak of toxoplasmosis that probably resulted from ingesting oocysts from cat feces. The level of seropositivity to *Toxoplasma* is directly related to the age of the person, but rates vary by locale.¹³⁻¹⁷ In the area where this family

lives, prevalence studies indicate that only 25% of the residents who are between ten and 20 years of age are seropositive for *Toxoplasma*.¹⁴ The preschool-aged children in this family had a seropositivity rate of 68%, certainly representing a focus of infection. Congenital toxoplasmosis only occurs following primary infection acquired during pregnancy. Since only three of five mothers with affected children were seropositive, and two of these children were born to a mother who probably had acute toxoplasmosis during this outbreak, intrauterine transmission could not have caused all these infections. Infection via ingestion of rare or raw meat was equally unlikely because family members ate their meat cooked at least medium well, and most ate it cooked well done.

Therefore, ingestion of oocysts remains as the only likely route of infection. Although the roles of meat and oocysts as sources of *Toxoplasma* infection in people have not been specifically studied in Dothan, an epidemiologic study in nearby Birmingham, AL, in a population of similar cultural and socioeconomic background, indicated that exposure to cats was the only factor significantly associated with *Toxoplasma* infection.¹⁷

This outbreak of toxoplasmosis was largely confined to the children in the family. Therefore, it must be assumed that transmission of infection was closely associated with some particular activity of this age group. Two months before the index patient became ill, the seropositive cat had a litter of six kittens, several of which later developed a nonspecific illness of which at least one died. Kittens with acute toxoplasmosis may die from pneumonia, encephalitis, and diarrhea. More important, recently infected cats shed millions of oocysts per day in their feces for approximately two weeks; oocysts may remain infectious for years in soil with appropriate characteristics.^{8,9} The female cat and the kittens were allowed to defecate at random in the yard of household A, including in the preschool-aged children's regular play area. The distribution of toxoplasmosis was statistically associated with the children who had a history of geophagia. (The children who had no history of geophagia had no evidence of infection with either parasite at the

TABLE 2. Relationship of Geophagia and *Toxoplasma*, *Toxocara*, and Enteroparasitic Infections in Preschool-aged Children*

	<i>Toxoplasma</i> Infection		<i>Toxocara</i> Infection		Enteroparasitic Infection	
	Positive	Negative	Positive	Negative	Positive	Negative
History of geophagia	7	1	4	3	5	2
No history of geophagia	0	3	0	2	0	2

* Figures indicate number of patients

time of the epidemiologic investigation.) Toxocariasis was prevalent among the same children, and the only known route of infection with *Toxocara* is ingesting eggs deposited in the soil by dogs or cats.¹⁸ Nearly three months after all cats had been disposed of, no cat feces could be found in the yard. Our failure to demonstrate oocysts in samples of soil must be interpreted cautiously, because between the onset of the outbreak and the collection of soil samples bulldozers had graded the yard and the street in front of the house in order to solve a drainage problem.

Surprisingly, seven of ten members with serologic evidence of recently acquired toxoplasmosis (seroconversion or antibody titer of $\geq 1,024$) were symptomatic; the severity of their illness, particularly in preschool-aged children, was such that prompted specific chemotherapy. All symptomatic patients had high titers to *Toxoplasma*, whereas the two school-aged children and the adult who later became seropositive had low titers and remained asymptomatic.

Persons with toxoplasmosis or toxocariasis may have symptoms including fever, leukocytosis, cough, pneumonitis, rash and elevated levels of specific antibodies. However, eosinophilia, hypergammaglobulinemia, and hepatomegaly are associated only with *Toxocara* infections.¹⁸ On these grounds, the index patient had clinical and laboratory evidence of both visceral larva migrans and toxoplasmosis. However, double infection could not account for the illnesses of the six other symptomatic patients in this outbreak; only one of them had eosinophilia (minimal), and four had no serologic evidence of *Toxocara* infection.

Considering the immunologic competence and the absence of immunosuppressive medication, the severe clinical illness of the index patient is clearly unusual. In brain biopsy material, numerous free and encysted forms of *Toxoplasma* were found; the organism was also readily isolated from CSF in tissue cultures. Toxoplasmosis involving the CNS and leading to multiple extensive abscesses such as those found in this child had only been reported for patients with underlying systemic diseases requiring intensive immunosuppressive therapy.⁴ On the other hand, the clinical severity of toxocariasis in human beings is partially dependent on the number of larvae ingested.¹⁸ Certainly children with a history of pica, such as this patient had, are at a greater risk of acquiring severe infection. The few fatalities that appear to have been caused by *Toxocara* have resulted from extensive involvement of CNS and/or myocardium.¹⁸ In these cases, the presence of larvae and eosinophilic granuloma suggests that preceding neurologic disturbances were caused by actual larval invasion of nervous tissue. For the

patient reported on here, *Toxocara* infection of the CNS could not be documented with the small piece of brain tissue obtained at biopsy. Nevertheless, it seems reasonable that the unusual clinical syndrome suffered by the index patient resulted from a severe, simultaneous infection with both *Toxoplasma* and *Toxocara*.

The clinical morbidity rate for members of the family in this outbreak clearly justifies the measures outlined years ago for preventing toxoplasmosis during pregnancy,^{1,2,5} and the danger of toxocariasis in young children¹⁸ must be better appreciated by the public in general and by pet owners in particular. Because pica is an important risk factor for toxoplasmosis, toxocariasis, and other diseases, children with the problem should not be allowed in environments thought to be contaminated. Naturally, every effort should be made to eliminate this habit. Only through public awareness and education can these goals be reached.

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ON AIR INSUFFLATION FOR THE TREATMENT OF INTUSSUSCEPTION— AS REPORTED IN 1864

The first successful surgical operation for intussusception in an infant was reported in 1874.¹ Prior to this date, infants with intussusception were occasionally treated by introducing air into the rectum by means of a tube attached to a common pair of bellows.

In 1864 David Grieg² of Dundee, Scotland published the first detailed report of the successful treatment by insufflation of several infants with intussusception. This is how he described his first case.

Case I.—M.S.G., a stout, healthy, female child, 6 months old, always enjoyed good health, never having had a day's sickness; never had any food except breast milk; never troubled with diarrhoea or bowel complaint. Was in her usual good health on Monday, 13th October 1862, up to 6 o'clock in the evening, when, without any obvious cause, she suddenly became fretful, kicking with her feet, bending the body backwards, and screaming. In about ten minutes she became very sick and vomited severely. The skin became cold and clammy, the countenance pale, and the lips livid. . . . She seemed to have great pain in the abdomen, which came on in paroxysms, and to increase in intensity until she vomited, when she would seem relieved a little, or at least so faint and sick as not to scream. When given the breast, she would take it readily; but as the sickness and vomiting, with a paroxysm of pain, immediately came on, she latterly refused it. Immediately when she was seized a spoonful of castor oil was given, and hot fomentations were applied to the abdomen. The castor oil was soon ejected from the stomach, as was also a small purgative powder which was given. A warm-water enema was attempted to be administered, but the bowel seemed to be in such a state of spasm and none could be thrown up. About 8 PM, tenesmus came on, and she passed a little fluid blood, which continued to come with every paroxysm of pain during the night. . . . On the morning of the 14th, as the child was no better, and . . . as everything had been tried, and nothing had done any good, and as it was evident the child was fast sinking, it was proposed to use the air injection. . . .

The nozzle of a small pair of bellows was introduced into the anus, and air injected to a considerable extent. Contrary to our expectation the air passed readily into the bowel, and seemed to give the child great relief. After the injection it lay very quiet, as if asleep, and evidently quite free from pain. In about twenty minutes from the time the air injection was administered, a slight rumbling noise was heard in the child's abdomen, followed by a crack so loud and distinct as to alarm the attendants in the room, who thought something had burst in the child's bowels. The child, however, continued as if asleep and free from pain, and in about half an hour a large feculent fluid stool, slightly mixed with blood and mucus, was passed without pain, during the night the child rested pretty well, had no return of vomiting, took the breast as usual, and in two days was quite well.

Noted by T.E.C., Jr, MD

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