Mem. Inst. Oswaldo Cruz, Rio de Janeiro, Vol. 77(1) :29-36, jan./mar. 1982

OUTBREAK OF HUMAN TOXOPLASMOSIS IN A RURAL AREA. A THREE YEAR SEROLOGIC FOLLOW-UP STUDY

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Thirty-six persons living on a farm located in the state of Minas Gerais (Brazil) were studied. Nine of them had the glandular form of toxoplasmosis, between May and August, 1976. These nine cases of toxoplasmosis were confirmed serologically by immunefluorescence-IF-, presenting IgG antibody titres between 1:4096 and 1:32000 and IgM antibody titres between 1:16 and 1:8000. Twelve out of thirty-six persons studied were considered to be "dubious cases". They were defined either by presenting a clinical picture compatible with acquired toxoplasmosis, yet having low serologic titres, or inversely they did not have a clear clinical picture but had serologic evidence of recent Toxoplasma *infection. Fifteen out of thirty-six persons studied showed neither serologic nor clinical evidence of recent* Toxoplasma *infection. The epidemiologic information suggests two possible modes of transmission: a) poorly cooked pork at a barbecue party; b) farm vegetables and soil contaminated with* Toxoplasma gondii *oocysts (rat-cat cycle). Serologic follow-up nine months later in the human farm population demonstrated still high IgG titres, yet they tended to decline and IgM titres became negative. Three years later most of the IgG titres continued to decline and were almost compatible with the titres obtained in Brazilian population surveys.*

Toxoplasmosis is caused by the protozoan parasite *Toxoplasma gondii*. Human infection (after birth) is more frequent, being transmitted mainly by the ingestion of poorly cooked or raw meat containing parasite cysts or the ingestion of oocysts eliminated in faeces of infected cats (Frenkel, Dubey & Miller, 1970; Hutchinson et al, 1971; Work, 1971; Jacobs, 1974; Frenkel & Ruiz, 1981).

The clinical picture of acquired toxoplasmosis may vary from subclinical to severe disease. The mild glandular form of the disease has been identified as the most frequent symptomatic form of acquired toxoplasmosis (Kean, 1972; Remington, 1974).

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Received for publication August 28th and accepted November 30th, 1981.

Cases of acquired toxoplasmosis in the same home have already been described by Amato Neto, Rivetti & Malheiros (1967) and Masur et al (1978). Magaldi et al (1967, 1969) have described two outbreaks of toxoplasmosis among students in two schools in the state of São Paulo (Brazil). Kean, Kimbal & Christenson (1969) have described a cluster of five cases among medical students who ate poorly cooked hamburger.

Teutsch et al (1979) and Stagno, Sykes & Amos (1980) have described outbreaks of toxoplasmosis associated with infected cats.

During the present study special efforts were made to identify the possible mechanisms of transmission involved in this outbreak. Also serial serum samples were collected for a follow-up study.

MATERIAL AND METHODS

The dairy farm studied is located in the township of Além Paraíba, state of Minas Gerais. During the outbreak 6 families: *A* (landlord family), *B*, *C*, *D*, *E*, *F*, lived on the farm forming a total of 36 persons, whose ages varied between 1 and 70 years. Families *B*, *C*, *E* and *F* lived on the farm during the entire period of this study. Family *A* lived in Rio de Janeiro and commuted to the farm during weekends and holidays. Family *D* started living on the farm in July 1976. All persons mentioned above were submitted to a clinical examination and blood samples collected in Sept. 1976, June 1977, and July 1979. These sera were tested for IgG and IgM antibodies for toxoplasmosis by indirect immunefluorescence test (IF).

Another group was also studied, group Z, consisted of 7 persons whose age varied between 0.5 and 63 years living in the city of Rio de Janeiro. They were relatives, friends and domestic employees of family *A*. None of them visited the farm but had very frequent contact with group *A* in their house in Rio de Janeiro, including the period when two persons of group *A* had the glandular form of toxoplasmosis.

The farm residents organized a party on April 30th, 1976. A barbecue was prepared and only pork was served to all family groups including the landlord and family (group *A*). Group *D* was not present because they arrived on the farm during July, 1976. The pig used for the barbecue was butchered on the farm. There also were 40 milk cows and their calves, 3 horses, 2 donkeys, 6 dogs and 3 pigs, during 1976. Blood samples from a few of these animals were collected in September, 1976 for the Sabin Feldman dye test for toxoplasmosis.

The number of rats in the farm was extremely large, especially near the corn storage barn annexed to house C and another small corn storage near house E. This number increased especially in March and April, 1976 when an exceptionally large quantity of corn was harvested. Eight cats lived on the farm at that time. These cats were kept there in order to combat the increasing rat population. Only one cat could be examined for Coccidian parasites due to a misunderstanding.

RESULTS

Nine successive human cases of glandular toxoplasmosis were detected from May to August, 1976 according to the clinical histories and examinations performed on the human farm population.

The index case (C2), belonging to group *C*, appeared in the second half of May. The successive cases appeared one month later and lasted until August belonging to

groups *A*, *B*, *C*, and *E*. The diseases was characterized mainly by high fever lasting from 2 to 7 days, severe and persistent headache. These nine cases presented glandular hypertrophy, mainly of the cervical, axillar and inguinal lymph nodes, which appeared when the fever declined. Tables I, II, III and IV, show the serologic results, as well as the clinical data.

TABLE I

Outbreak of Toxoplasmosis. Serological (indirect immunofluorescence – IF) reactivity of the human population in 1976, 1977 and 1979 to Toxoplasma antigen

Reciprocal of titres	*September 1976*				*June 1977*				*July 1979*			
	IF-IgG		*IF-IgM*		*IF-IgG*		*IF-IgM*		*IF-IgG*		*IF-IgM*	
	Nº	%	*Nº*	%	*Nº*	%	*Nº*	%	*Nº*	%	*Nº*	%
SN	2	5.6	19	52.8	2	6.7	23	76.7	5	20.0	24	96.0
16	2	5.6	6	16.7	4	13.3	2	6.7	4	16.0	0	0
64	7	19.4	2	5.6	8	26.7	4	13.3	8	32.0	1	4.0
256	10	27.8	2	5.6	6	20.0	1	3.3	5	20.0	0	0
1024	2	5.6	5	13.9	4	13.3	0	0	3	12.0	0	0
4096	5	13.9	1	2.7	5	16.7	0	0	0	0	0	0
8000	3	8.3	1	2.7	1	3.3	0	0	0	0	0	0
16000	1	2.7	0	0	0	0	0	0	0	0	0	0
32000	4	11.1	0	0	0	0	0	0	0	0	0	0
Reactive sera Sub total	34	94.4	17	47.2	28	93.3	7	23.3	20	80.0	1	4.0
TOTAL TESTED	36	100	36	100	30	100	30	100	25	100	25	100

SN – serum negative at 1:16 dilution.

TABLE II

Outbreak of Toxoplasmosis. Clinical and serological (indirect immunofluorescence – IF – Toxoplasmosis) data of 9 subjects who were considered as "cases of toxoplasmosis" in 1976. Clinical and serological follow-up in 1977 and 1979

Nº	*Age*	*September 1976*					*June 1977*					*July 1979*				
		Clinical data			*Reciprocal of titre*		*Clinical data*			*Reciprocal of titre*		*Clinical data*			*Reciprocal of titre*	
		Fv	*L*	*H*	*IgG*	*IgM*	*Fv*	*L*	*H*	*IgG*	*IgM*	*Fv*	*L*	*H*	*IgG*	*IgM*
A3	16	+	+	0	8000	1024	0	0	0	4096	SN	0	0	0	64	SN
A4	08	+	0	+	4096	1024	0	0	0	64	SN	0	0	0	64	SN
B2	62	0	+	0	8000	64	0	+	0	1024	64	–	–	–	–	–
C2	23	+	+	+	16000	4096	0	0	0	4096	16	0	0	0	1024	SN
C3	02	+	+	0	32000	1024	0	+	0	4096	SN	0	0	0	1024	SN
E4	24	+	+	+	4096	16	0	0	0	1024	16	–	–	–	–	–
E5	20	+	+	+	4096	1024	0	0	0	4096	SN	0	0	0	1024	SN
E6	16	+	+	+	32000	8000	0	+	0	8000	64	0	0	0	256	SN
E13	01	+	+	–	8000	16	0	0	0	64	SN	0	0	0	SN	SN

Fv = fever; L = lymphadenopathy; H = headache; SN = serum negative at 1:16 dilution.

TABLE III

Outbreak of Toxoplasmosis. Clinical and serological (indirect immunofluorescence—IF—Toxoplasmosis) data of 12 persons who were considered as "dubious cases" of toxoplasmosis in 1976. Clinical and serological follow-up in 1977 and 1979

N°	*Age*	*September 1976*					*June 1977*					*July 1979*				
		Clinical data			*Reciprocal of titre*		*Clinical data*			*Reciprocal of titre*		*Clinical data*			*Reciprocal of titre*	
		Fv	*L*	*H*	*IgG*	*IgM*	*Fv*	*L*	*H*	*IgG*	*IgM*	*Fv*	*L*	*H*	*IgG*	*IgM*
A2	40	0	0	+	1024	16	0	0	0	256	SN	0	0	0	64	SN
B1	62	0	+	0	256	16	0	0	0	64	SN	–	–	–	–	–
D3	10	0	0	0	4096	SN	0	+	0	64	SN	0	0	0	256	SN
D4	09	0	0	0	64	16	0	0	0	1024	256	0	0	0	16	SN
D5	05	0	0	0	32000	SN	0	0	0	64	SN	0	0	0	64	SN
D6	07	0	0	0	32000	1024	0	0	0	256	SN	0	0	0	256	SN
E2	42	+	+	+	16	SN	0	0	0	16	SN	0	0	0	SN	SN
E3	17	+	0	+	16	SN	0	0	0	SN	SN	–	–	–	–	–
E9	10	0	0	+	64	64	0	0	0	256	64	0	0	0	16	SN
E10	08	0	0	0	1024	16	0	0	0	1024	64	0	0	0	SN	SN
E12	04	0	0	0	64	256	0	0	0	16	SN	0	0	0	SN	SN
E14	12	0	0	0	4096	256	–	–	–	–	–	0	0	0	256	SN

Fv = fever; L = lymphadenopathy; H = headache; SN = serum negative at 1:16 dilution.

TABLE IV

Serologic follow-up of 15 persons who were considered as "healthy subjects" in 1976

N°	*Age*	*Reciprocal of titres IF—Toxoplasmosis*					
		1976		*1977*		*1979*	
		IgG	*IgM*	*IgG*	*IgM*	*IgG*	*IgM*
A1	50	256	SN	256	SN	64	SN
A5	25	256	SN	64	SN	–	–
B3	40	64	SN	–	–	–	–
B4	48	256	SN	–	–	–	–
C1	28	64	SN	64	SN	16	SN
D1	34	256	SN	256	SN	16	SN
D2	28	256	SN	16	SN	64	SN
E1	47	256	SN	16	SN	64	SN
E7	12	SN	SN	4096	SN	64	SN
E8	14	256	SN	256	SN	256	64
E11	05	64	SN	64	SN	SN	SN
F1	65	256	SN	–	–	-	–
F2	70	64	SN	–	–	–	–
F3	29	SN	SN	SN	SN	–	–
F4	15	256	SN	–	–	–	–

SN = serum negative at 1:16 dilution.

The human farm population was divided into three groups according to the clinical data and the IgG and IgM antibody titres, as observed in September, 1976 by IF for toxoplasmosis: 1) "cases of acute toxoplasmosis" – 9 persons presenting clear clinical symptomatology with lymphadenopathy and serologic evidence of recent *Toxoplasma* infection (IF–IgG $\geqslant$ 1:4096 and IF–IgM $\geqslant$ 1:16) (Table II). 2) "dubious cases" – 12 persons presenting one of the following conditions: a) clear clinical symptomatology without serologic evidence of recent infection (persons: E2 and E3); b) only serologic evidence of recent infection (IF–IgG $\geqslant$ 1:4096 or IF–IgG and IF–IgM $\geqslant$ 1:16; (persons: D3, D4, D5, D6, E10, E12, E14); c) discrete clinical symptomatology and slightly elevated antibody titres for toxoplamosis (persons: A2, B1, E9) (Table III). 3) "healthy subjects" – 15 persons presenting neither clinical nor serologic evidence of recently acquired toxoplasmosis (11 persons distributed among groups *A*, *B*, *C*, *D* and *E* and the whole group *F* (persons F1, F2, F3, F4). Two of them were seronegative (Table IV).

Group *Z* did not present clinical symptomatology of toxoplasmosis in 1976. Four of the seven persons of group *Z* presented IF–IgG antiboy titres between 1:64 and 1:256 and the three other persons were seronegative. All persons of group *Z* were IF–IgM seronegative.

Ten faeces samples obtained from one cat examined in September, 1976 were negative for *Toxoplasma gondii* oocysts or for other *Coccidia* oocysts. The cat was sacrified after 20 days of observation. Histopathological examination of lungs, muscles, brain and intestine were negative for tissue forms of the parasite. Sabin Feldman dye test performed on this cat's serum presented 1:256 antibody titre. The dye test performed in 1976 on sera from 10 cows, 2 pigs, 1 horse, 2 donkeys, 1 dog, and 2 sheep presented 4 negative sera (21%). The highest titre among the positive sera was 1:1024 (5 cases: 3 cows, 1 donkey, and 1 sheep). The animals did not presented clinical symptomatology.

DISCUSSION

The initial clinical suspicion of acquired glandular toxoplasmosis in these nine patients was confirmed by the IF–IgG antibodies titres $\geqslant$ 1:4096 and IF–IgM $\geqslant$ 1:16 (Table II). No similar disease was observed in previous years and did not occur again after this outbreak.

According to Remington, Miller & Browlee (1968), Amato Neto et al (1972), Camargo & Leser (1976), and other authors, the presence of specific IgM Toxoplasma antibodies, even at low titres, might be considered as indication of recent infection. The present study demonstrates IgM antibodies still present after nine months in four cases but the titres did not exceed 1:64. In July 1979, the IF test performed on the seven sera available demonstrated negative IF–IgM and low titres of IgG antibodies not exceeding 1:1024. Case E13, one year old, became IF–IgG and IgM negative (Table II).

Some of the twelve "dubious cases" (Table III) of toxoplasmosis investigated in September 1976, had indications of recent infection, as demonstrated by elevated IgG and IgM antibody titres and decreasing titres in 1977 and 1979.

Dubious case D4, was also considered in this group because although no symptoms and inconclusive serology (IF–IgM = 1:16) were found in 1976, IgG and IgM titres increased in 1977.

"Healthy subject" – E7 (Table IV) presented seroconversion indicating that infection had occurred after Septembre 1976, even without any detectable clinical symptomatology.

Only two of the 36 individuals studied were IgG and IgM seronegative for *T. gondii* in September 1976 (Table I). There were 34 IF positive sera (94.4%). This percentage is rather high if compared to other Brazilian serologic surveys for toxoplasmosis, in which this percentage varied between 40% and 80% (Coutinho et al, 1981; Amendoeira & Coutinho, 1981). The existence of a toxoplasmosis outbreak among the human farm population might explain this high percentage.

The percentage of IF–IgG reactive sera was almost the same nine months later but this percentage became nearly compatible with the general Brazilian population two years and 10 months later (Table I).

The percentage of IF–IgM positive sera decreased more rapidly, being only 4% two years and ten months later (Table I)..

Members of group *Z* had intense contact with "acute cases" A3 and A4 during their febrile stage of the disease occurred in Rio de Janeiro. None of them had any clinical or serological evidence of acute recent toxoplasmosis. These data are consistent with the hypothesis that direct interhuman contact was not an important source of infection.

Two occurrences on the farm were considered as possible explanations for the mechanism of the disease transmission.

The first was the party barbecue. Individuals A4, B2, C2, E6, A2 and B1 were the principal meathandlers and the first four of them became "cases of acute toxoplasmosis" (Table II), whereas A2 and B1 were considered as "dubious cases".

On the other hand, the following facts show that the barbecue should not be considered as the only source of infection during this outbreak; a) other successive "cases of acute toxoplasmosis" occurrred until August of the same year, giving a too large variation in the possible incubation time, suggesting the existence of another source of *Toxoplasma* infection; b) group *D* was not present at the farm's party and only arrived on the farm in July, 1976. Nevertheless four out the six members from this group were considered as "dubious cases"; c) all individuals of group *F* were present at the party, eating the barbecue, but all were considered as "healthy subjects".

The second occurrence which might be considered as a mechanism of *T. gondii* transmission was the increased population of rats and cats in the farm. Wallace, Marchall & Marchall (1972), working in Pacific islands, Frenkel & Ruiz (1981) in Costa Rica and other authors have stressed the importance of the cat-rat cycle in the epidemiology of toxoplasmosis, causing soil contamination with oocysts (Ruiz, Frenkel & Cerdas, 1975).

If the farm's cats were eliminating oocysts they could contaminate vegetables which could be a source of human *Toxoplasma* infection.

The clinical "cases of toxoplasmosis" appeared gradually, from May to August 1976, pointing to several different episodes of contamination, which could be expected if the soil and vegetables were contaminated with oocysts. *Toxoplasma* was isolated from a soil sample, collected in the garden behind houses B and C, proving that the farm's soil was contaminated (Coutinho, Lobo & Dutra, 1982). Group *D* did not eat pork meat at the barbecue party. Nevertheless, at least two persons from that family had IF–IgM titres which strongly suggest recent *T. gondii* infection (D4, D6).

Probably both mechanisms of transmission occurred during this outbreak. First of all, the barbecue's poorly cooked *T. gondii* contaminated pork could infect both humans and cats. These cats eating part of the leftover pork initiated the sporogonic cycle

which in turn contaminated the soil and vegetable gardens with oocysts. Then the cat-rat cycle increased the soil contamination originating further infections.

The farm animals during this period were healthy and there was no increased abortion rate. The antibody titres for toxoplasmosis from most of the animals were not elevated. There is no clear explanation why in the present outbreak the disease was apparently only restricted to the human farm population.

RESUMO

Foram estudados 36 indivíduos habitantes de uma fazenda no interior do Estado de Minas Gerais nove dos quais adquiriram a forma linfonodular de toxoplasmose entre maio e agosto de 1976, apresentando títulos de anticorpos pela imunofluorescência indireta – IF – entre 1:4096 e 1:32000 na classe IgG e entre 1:16 e 1:8000 na classe IgM. Doze das 36 pessoas da fazenda foram consideradas como casos "duvidosos", assim definidos ou por apresentarem quadro clínico compatível com toxoplasmose adquirida apesar de baixos títulos de anticorpos, ou por não apresentarem nítido quadro clínico, mas com evidências sorológicas de infecção recente pelo *T. gondii.* As outras 15 pessoas estudadas não mostraram evidências clínicas ou sorológicas de infecção recente. As informações coletadas sugerem duas possíveis principais formas de transmissão: a) carne de porco mal cozida durante um churrasco na fazenda; b) solo e vegetais contaminados com oocistos de *T. gondii* (ciclo gato-rato). O acompanhamento sorológico da população humana, nove meses após, demonstrou títulos ainda elevados na classe IgG embora com tendência ao declínio e soros não reagentes na classe IgM. Após três anos os títulos na classe IgG eram quase compatíveis com os títulos observados em inquéritos sorológicos em outras populações brasileiras.

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