Scientific Report

Anterior uveitis in a kitten infected with *Toxoplasma gondii* (Tehran strain)

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Summary

Ocular lesions are a common manifestation of toxoplasmosis in cats. In this survey, 10 healthy Syrian mice were infected through intraperitoneal inoculation of bradyzoites of *Toxoplasma gondii* (Tehran strain). Mice were killed 30 days after inoculation and the suspension of their brains was prepared, after confirmation of the infection. Seven kittens were fed with the infected mice brain suspension. The kittens were 2.5–3 months old and had body weights of 650–900 g. All kittens shed oocyst 7–8 days after infection, and had antibody titers as high as 1/320–1/1280 30 days after the challenge. Serum chemistry and complete blood count were determined in all of the kittens. Examination of eyes was done with ophthalmoscope twice during one week. Only one kitten showed unilateral anterior uveitis on day 16 after the infection was diagnosed based on serology, oocyst shedding and other clinical signs.

Key words: Anterior uveitis, Kitten, *Toxoplasma gondii*

Introduction

Ocular lesions are a common manifestation of generalized toxoplasmosis in cats. Toxoplasmosis may lead to chorioretinitis, anterior uveitis or both (Dubey *et al*., 1996; Green, 1998). The prevalence of toxoplasmosis, as a cause of idiopathic anterior uveitis in cats, is not clear, although there is a significant association between exposure to *Toxoplasma gondii* and feline anterior uveitis. Anterior uveitis may represent a type of immune-mediated inflammation (Davidson *et al.*, 1993; Lappin, 2000). Despite improved diagnostic techniques, including determination of IgM class antibodies and polymerase chain reaction (PCR) testing, definite diagnosis of ocular toxoplasmosis remains a challenge. The objective of this study was to investigate the clinical, serologic and biochemical profile features of an experimental model of toxoplasmosis in cats.

Materials and Methods

In this survey, 10 mice were infected through intraperitoneal inoculation of bradyzoites of *T. gondii* (Tehran strain; this strain obtained from Faculty of Hygiene of Tehran University. It infects mice chronically). The infected mice were sacrificed 30 days after inoculation (Radkle *et al*., 2001). Cerebral infection of mice was verified by observation of bradyzoites in tissue smears under light microscope (Dubey, 2001; Dubey, 2002).

Then, seven mixed-breed kittens aged 2.5–3 months with a body weight of 650–900 g were selected. Before the experiment,

all kittens were examined clinically and paraclinically including a complete blood count (CBC), faecal examination for detection of oocyst or parasite eggs and antibody titers by indirect fluorescent assay (IFA). All the cats were healthy. They fed infected mice brain tissues. Antibody titers, serum chemistry including blood urea nitrogen (BUN), serum creatinine, alkaline phosphatase (ALP), aspartate amino-transferase (AST), alanine aminotransferase (ALT) and certain haematologic values were determined on days 1 and 30 of infection (Tables 1, 2 and 3).

Results

Before the trial, the results of CBC and all chemical profiles were normal in all cats. Also faecal examination was negative for oocyst and other egg parasites. None of the kittens had any antibody titers measured by IFA.

On day 30 of infection, the results were as follows:

Table 1: Results of CBC in kittens

Kitten number	1	2	3	4	5	6	7
PCV	31	32	31	31	28	26	34
WBC	27000	24500	7500	13750	9000	24000	23500
Neut.	68	58	48	73	54	84	60
Band	-	-	1	-	-	-	-
Lym.	25	33	41	22	39	14	33
Mon.	2	3	3	1	-	-	1
Eos.	5	6	7	4	7	2	4

CBC: leukocytosis (>19,500) in 4, neutrophilia (>11,500) in 3, lymphocytosis (>4,500) in 2 and eosinophilia (>1,250) in 3 of kittens.

Results of chemical profiles were as follows:

Table 2: Results of chemical profiles in kittens

Chemical profiles	Mean ± SD
BUN (mg/dl)	27.9 ± 5.08
Creatinine (mg/dl)	1.61 ± 0.6
ALP (IU/L)	54 ± 14.7
AST (IU/L)	31 ± 16.2
ALT (IU/L)	58.4 ± 2.3

Chemical profiles were normal.

Also all the kittens became sero-positive for *T. gondii* with titers ranging from 1/320–1/1280.

Table 3: Results of IFA test in kittens

The first titer (before inoculation)	The last titer (after inoculation)
-	1/640
-	1/320
-	1/1280
-	1/640
-	1/1280
-	1/320
-	1/640

All the kittens shed oocyst one week after the infection which lasted for 7–8 days.

The mean oocyst shedding was 3+ (2,000–3,500 oocysts/g faeces). *Cystoisos-pora felis* and other coccidians were not seen in kittens. Ocular ophthalmoscopic examinations were performed on kittens twice per week for 30 days after infection.

Among the kittens studied, only one (14.3%) developed ocular involvement (Fig. 1). The antibody coefficient (C-value) for antibody in this kitten was 1/1,280 on day 30 after the challenge. The animal developed unilateral anterior uveitis on day 16 after infection that continued untill day 30. CBC was abnormal (neutrophilic leukocytosis, lymphocytosis and eosinophilia) and oocyst shedding lasted for 8 days in this kitten.

Fig. 1: Anterior uveitis in *T. gondii* infected kitten inthe left eye.

Discussion

Generally, diagnosis of toxoplasmosis is made by observing compatible clinical findings and obtaining supportive findings on serologic tests (Green, 1998).

Clinical or laboratory evidences of immunodeficiency in these kittens were not detected. In our study, detection of a high

serum antibody titer against *Toxoplasma* (which had no titer before the trial), exclusion of other causes of uveitis, and experimental infection with *T. gondii*, oocyst shedding and other clinical signs (e.g., weight loss) suggest that the kitten have had toxoplasmosis resulting in uveal tract inflammation. It is worth to mention that the oocyst size of *T. gondii* (10 µ) is smaller than *Cystoisospora felis*. Because the infection with *T. gondii* was experimentally induced in the kitten and that the animal did not shed any oocysts before the challenge, the oocysts after the induction of infection shed by the animal were most likely those of *Toxoplasma*. All kittens had high antibody titers against *T. gondii* after infection.

It is mentioned that haematologic parameters may be abnormal in cats with toxoplasmosis. The changes may include non-regenerative anemia, neutrophilic leukocytosis, lymphocytosis, monocytosis, and eosinophilia (Green, 1998). In this study, among the haematologic parameters studied, we observed only a significant leukocytosis ($P = 0.04$). Neutrophilia, lymphocytosis and eosinophilia did not significantly change. Moreover, all the measured biochemical values including BUN, serum creatinine, ALP, ALT and AST were normal on day one and 30 of infection.

The factors that trigger development of uveitis in cats infected with *T. gondii* have not been elucidated, but infection by more than one organism may be contributory (Powel and Lappin , 2001; Hegab and Al-Mutawa, 2003). In the past, most cases of ocular toxoplasmosis were considered to result from reactivation of a congenital infection, but it is now believed that post-natal acquired infection accounts for many cases of this condition.

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