



Clinical and Some Laboratory Findings in Cats with Toxoplasmosis

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ABSTRACT

Objectives: The aim of this study was to draw attention to the clinical course of the disease and some laboratory findings in cats diagnosed with Toxoplasmosis.

Materials and Methods: *Toxoplasma gondii* seropositive 14 cats were used in this study. A serological evaluation was carried out to determine the presence of *Toxoplasma gondii* specific IgG using commercial diagnostic kits, by the enzyme-linked immunosorbent assay method. Hematological and clinical changes of those cats were recorded.

Results: Of 14 cats, neural symptoms such as behavioral changes, seizures, ataxia and nystagmus were detected in 11 cats, uveitis in 5 cats and diarrhea in 4 cats. Serum urea, creatinine and bilirubin levels were normal in all cats. However, Anemia (decreased Hb, RBC, PCV) in 8 cats (57.1%), monocytosis in 6 cats (42.8%), neutrophilia in 5 cats (35.7%), hypoalbuminemia in 5 cats (35.7%) and increased AST and ALT levels in 3 cats (21.4%) were detected.

Conclusion: It was concluded that clinical Toxoplasmosis in cats is characterized by neurological, ocular and gastrointestinal signs and hematological signs such as anemia, monocytosis, neutrophilia and hypoalbuminemia that clinically patient cats should also be evaluated in terms of Toxoplasmosis in cats.

Keywords: Toxoplasma Gondii, cats, neurological signs, gastrointestinal signs, ocular signs, anemia.

INTRODUCTION

Toxoplasma gondii (*T. gondii*) is a protozoan that is common worldwide and infects all species of birds and mammals (Dubey 2006; Lappin, 2010). The sexual phase of parasite is complete only in the gastrointestinal tract of the cats, and oocysts resistant to environmental conditions are excreted in the stool. Oocysts diffused around are an important source of infection for humans and other mammals (Dubey, 1998). It is estimated that the seroprevalence of *T. gondii* in cats is 30-40% worldwide (Lappin, 1999).

Both enteroepithelial and extraintestinal phases of *T. gondii* occur in cats. After extra intestinal phase, cysts form in many tissues (Dubey, 1986; Greene 1984). Cats carry cysts throughout their lives that cause constant antigen release and re-infection (Lappin, 1988).

The disease is generally subclinical in cats, and clinically healthy pets may also carry the agent. In some cats, the clinical form of the disease may be associated with feline immunodeficiency virus (FIV)

(Davidson et al., 1993) and feline leukemia virus (FeLV) (Witt et al., 1989). The disease progressing in subclinical form can convert to clinical form after immunosuppressive drugs (Beatty and Barrs 2003; Bernstein et. al., 1999). Clinical symptoms of the disease emerge as a result of necrosis and inflammation in the tissues during intracellular development of tachyzooids, and the clinical symptoms usually become evident when the cats become immunosuppressive (Greene, 1984). In general, the central nervous system, lung, muscles and eye is most affected by the disease while the liver and pancreas are less affected (Dubey and Carpenter, 1993).

While clinical symptoms vary according to the affected organ, immune system and presence of other diseases, the most common clinical symptoms are increase in body temperature, diarrhea, hyperesthesia in muscles, seizure, ataxia, respiratory distress, uveitis, icterus, lethargy, anorexia and weight loss (Hartmann, 2013).

Hematologic changes also occur with the disease. The most common hematologic changes are non-regenerative anemia, lymphocytosis lymphopenia, neutropenia, neutrocytosis, monocytosis and eosinophilia. Biochemical changes are hypoalbuminemia, hypoproteinemia and increase at alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels (Lappin, 1999).

The aim of this study was to investigate clinical, some biochemical and hematological parameters in 14 cats infected with *Toxoplasma gondii*.

MATERIALS AND METHODS

In this study, 14 cats ranging in age from 2-7 brought to Ankara University Veterinary Faculty Department of Internal Diseases Small Animal Clinic with at least one of the complaints of vomiting, loss of appetite, respiratory distress, central nervous system disease were used.

After taking the information of medical history, clinical examination of the patients was performed and history and clinical findings were recorded. After the anamnesis and clinical findings were evaluated, the blood collected from the cats for serum biochemistry and routine hematology were sent to Ankara University Veterinary Faculty Central Diagnostic Laboratory. Abdominal ultrasonography (USG) was performed to evaluate the liver, kidney and urinary bladder. Presence of FeLV, FIV and Corona Virus in blood samples collected was evaluated by PCR method in Ankara Sequence Laboratory. The blood smears were stained with DiffQuick or the investigation of the presence of *Haemobartonella*

Cat	Age	Sex	Breed	Clinical Sign
1	2	Male	Mixed	Diarrhea, Laterolateral nystagmus weight loss
2	3	Male	Mixed	Diarrhea, weight loss
3	4	Male	Mixed	Diarrhea, weight loss
4	2	Female	Domestic short hair	Behavioral changes, weight loss
5	5	Female	Mixed	Behavioral changes, Latero-lateral nystagmus, weight loss
6	2	Male	Domestic short hair	Bilateral uveitis, behavioral changes
7	2.5	Male	Persian	Bilateral uveitis
8	5	Female	Mixed	Laterolateral nystagmus, mydriasis.
9	4.5	Male	Mixed	Paralysis, Bilateral uveitis
10	3	Male	Mixed	Behavioral changes, seizures, ataxia, Bilateral uveitis
11	2.5	Female	Mixed	Diarrhea, weight loss, nystagmus
12	5	Female	Mixed	Vestibular syndrome
13	6	Female	Mixed	Unilateral uveitis, seizures
14	7	Male	Siamese	Behavioral changes, seizures, nystagmus, weight loss, Anorexia

Table 1: Signalment, History, and Physical Examination Abnormalities Associated With Clinical Toxoplasmosis in 14 Cats

felis.

In order to establish a final diagnosis, it was investigated whether anti-*T. gondii* IgG and *Chlamydomphila felis* IgG antibodies in blood samples were positive by commercial ELISA method (ImmunoComb® Feline *Toxoplasma* and *Chlamydomphila* Antibody Test Kit).

RESULTS

Ages of effected cats ranged from 2 to 7 years. The mean age of the cats was found to be 3.8 ± 1.6 . Eight of the 14 cats with *T. gondii* were male (57.1%) and 6 were female cats (42.8%). Many breeds were represented (Table1).

As a result of abdominal USG performed, there was no pathology in liver, kidney and urinary bladder. Serum urea, creatinine and bilirubin levels were found in the normal range. *Haemobartonella felis* was not detected in blood smears stained with DiffQuick. FeLV, FIV and Corona Virus were not detected in cats. On examination of the stool, *T. gondii* oocysts were detected in 3 cats but no *Isospora felis* was detected. *Chlamydomytila felis* IgG were negative in cats.

Of 14 cases, 11 cases presented neurological signs (78.5%) such as behavioral changes, seizures, ataxia nystagmus, vestibular signs and 5 cases presented uveitis (35%). Diarrhea was observed in 4 cases (28.5%). Of 11 cats with neurological signs, uveitis was found in 4 cats (36.3%) and diarrhea was observed in 2 cats (18.1%) (Table1).

As a result of the laboratory examination, anemia (decreased Hb, RBC, PCV) was detected in 8 cats (57.1%), monocytosis in 8 cats (57.1%), neutrophilia in 5 cats (35.7%), hypoalbuminemia in 5 cats (35.7%), and increase in AST and ALT levels in 3 cats (21.4%).

DISCUSSION

In this study, the clinical signs and some hematological serum biochemical changes in cats with *T. gondii* were examined. The neurological signs include a wide variety of clinical manifestations. Behavioral changes, seizures, nystagmus, ataxia, vestibular signs were the most common clinical symptoms in cats in the study. In one study on the cats with *T. gondii*, reported behavioral changes, seizures, nystagmus, ataxia and vestibular signs as the neurological symptoms of the disease (Cucos et al, 2015). Neurologic symptoms of the disease were same in both studies.

The neurological signs may be observed alone or along with the digestive or ophthalmological signs (Gunn-Moore et al, 2011; Lorenz et al., 2012). In this study, digestive or ophthalmological findings were also observed in the cats with neurological symptoms. This result shows that more than one system in cats with *T. gondii* have been affected.

In cats, *T. gondii* tends to form cysts in the liver, lungs, CNS, muscles and pancreas (Lappin et al., 1989; Lappin, 2010). Clinical signs of toxoplasmosis correlate with cell rupture secondary to organism

replication, cell necrosis associated with hypersensitivity reaction, and immune complex vasculitis (Greene 1989). CNS and ophthalmic disease were present in the cats were thought to have been caused by the damage the *T. gondii* cysts caused in the affected tissues.

In this study, the monocyte count was found to be higher in majority of the cats with *T. gondii*. Increase in the number of monocytes in humans and cats especially with toxoplasmosis is very common (Rajantie, 1992). In *T. gondii* infection, an increase or decrease in the number of the neutrophils may be seen. This condition varies depending on the presence of active infection or other bacterial and viral agents (Bliss et al, 2001). In this study, the increase in the number of neutrophils was determined in many of the cats. This condition was thought to be an indication of the presence of an active infection in many of the animals.

While biochemistry screening is not specific, hypoalbuminemia was determined as one of the most common findings in blood chemistry analysis. Dubey et al. (2009) reported that hypoalbuminemia can occur in acute phase of the disease. It was considered that hypoalbuminemia may be caused by the acute phase response in acute phase of the disease, and the increase in AST and ALT levels may be as a result of the damage caused by *T. gondii* in liver.

As a result, it has been concluded that cats with neurological and ophthalmological signs and anemia, neutrocytosis, monocytosis and hypoalbuminemia in the blood examination should be evaluated in terms of *T. gondii*.

Conflict of Interest: No conflict of interest was declared by the authors.

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REFERENCES

- Beatty J, Barrs V. Acute toxoplasmosis in two cats on cyclosporin therapy. *Aust Vet J* 2003; 81:339.
- Bernsteen L, Gregory CR, Aronson LR, et al. Acute toxoplasmosis following renal transplantation in three cats and a dog. *J Am Vet Med Assoc* 1999; 215:1123-1126.
- Bliss SK, Gavrilescu LC, Alcaraz A, et al. Neutrophil depletion during *Toxoplasma gondii* infection leads to impaired immunity and lethal systemic pathology. *Infect Immunol* 2001; 69:4898-905.

- Cucoş CA, Ionaşcu I, Mocanu J, Militaru M. Neurological and Ocular Form of Toxoplasmosis in Cats. *Scientific Works. Series C. Veterinary Medicine* 2015; 1:95-98.
- Davidson MG, Rottman JB, English RV, et al. Feline immune deficiency virus predisposes cats to acute generalized toxoplasmosis. *Am J Pathol* 1993; 143:1486-1497.
- Dubey JP. Toxoplasmosis in cats. *Feline Pract* 1986; 16:12-45.
- Dubey JP, Carpenter JL. Histologically confirmed clinical toxoplasmosis in cats: 100 cases (1952-1990). *J Am Vet Med Assoc* 1993; 203:1556-1566.
- Dubey JP, Lindsay DS, Speer CA. Structures of *Toxoplasma gondii* tachyzoites, bradyzoites, and sporozoites and biology and development of tissue cysts. *Clin Microbiol Rev* 1998; 11: 267-299.
- Dubey JP, Lappin MR. Toxoplasmosis and neosporosis. In: Greene CE (ed): *Infectious Diseases of the Dog and Cat*. 3th ed. Philadelphia: Saunders/Elsevier, 2006;754.
- Dubey JP, Lindsay DS, Lappin MR. Toxoplasmosis and Other Intestinal Coccidial Infections in Cats and Dogs *Veterinary Clinics of North America: Small Animal Practice* 2009; 39:1009-1034.
- Greene CE, Prestwood AK. Coccidial infections. In: Greene CE, ed. *Clinical Microbiology and Infectious Diseases of the Dog and Cat*. Philadelphia: WB Saunders, 1984; 826-840.
- Gunn-Moore D, Reed N. Central Nervous System Disease in the Cat. Current knowledge of infectious causes. *Journal of Feline Medicine and Surgery* 2011; 13:824-836.
- Lappin MR, Greene CE, Prestwood AK, et al. Enzyme-linked immunosorbent assay for the detection of circulating antigens of *Toxoplasma gondii* in the serum of cats. *Am J Vet Res* 1989; 50:1586-1590.
- Lorenz MD, Coates JR, Kent M. Handbook of veterinary neurology. 5th ed Missouri: Saunders Elsevier; 2012.
- Hartmann K, Addie D, Belák S, et al. *Toxoplasma gondii* infection in cats: ABCD guidelines on prevention and management. *J Feline Med Surg* 2013; 15:631-637.
- Lappin MR, Greene CE, Winston S, Toll SL, Epstein ME. Clinical feline toxoplasmosis: serologic diagnosis and therapeutic management of 15 cases. *J Vet Intern Med* 1989; 3: 139-143.
- Lappin MR. Update on the diagnosis and management of *Toxoplasma Gondii* infection in cats. *Top Companion Anim Med* 2010; 25:136-141.
- Lappin MR. Toxoplasmosis. In: Couto G, Nelson R (eds). *Small Animal Internal Medicine*, 4 th ed. St Louis: Mosby Elsevier, 2010; 1366- 1373.
- Rajantie J, Siimes MA, Taskinen W, et al. White blood cells in infants with congenital toxoplasmosis: transient appearance of cALL antigen on reactive marrow lymphocytes. *Scan J Infect Dis* 1992; 24:227-232.
- Witt CJ, Moench TR, Gittelsohn AM, Bishop BD, Childs JE. Epidemiologic observations on feline immunodeficiency virus and *Toxoplasma gondii* coinfection in cats in Baltimore. *Journal of American Veterinary Medical Association* 1989; 194: 229-233.