



## Clinical Toxoplasmosis in Two Cats and its Treatment with Clindamycin

Mustafa Güven<sup>1</sup>  Ebubekir Ceylan<sup>1</sup> 

<sup>1</sup> Department of Internal Medicine, Faculty of Veterinary Medicine, University of Ankara, Turkey

Correspondence: Mustafa Güven (mustafaguvendvm@gmail.com)

Received: 27.02.2020

Accepted: 16.03.2020

### ABSTRACT

Toxoplasmosis is a zoonotic disease, affecting birds, human beings and most warm-blooded animals throughout the world. On the following case report *Toxoplasma gondii* infection was detected in two cats. Primary clinical findings were defined as involuntary and continuous contraction of the hind limb muscles, incoordination and pain. *Toxoplasma gondii* generally progresses asymptotically however when clinical signs do appear, *T. gondii* shows itself with neurological symptoms. In these cases, the diagnosis of the disease was made by enzyme-linked immunosorbent assays (ELISA) method. In both of the cases Clindamycin was given at a dose of 25mg/kg/24h for the first week and the dosage was rearranged to 12,5mg/kg/q12h. Clinical improvement was observed after one week and treatment was discontinued at the third week.

**Keywords:** *Toxoplasma gondii*, ELISA, Diagnosis, Treatment, Cats

### INTRODUCTION

Toxoplasmosis is caused by an intracellular protozoal parasite *Toxoplasma gondii* and it is one of the most common parasitic infections of man and other warm-blooded animals (Dubey, 1998). It has been found globally, infecting nearly one-third of the human population (Dubey, 1998; Tenter et al., 2000).

While *T. gondii* can multiply asexually in all of its hosts, the only known definitive ones are feline species, including the domestic cat, Iriomote cat, leopard, tiger and lions (Lukesová & Literák, 1998). Humans, birds, and all other animals are mostly infected by contamination of the environment with feces which passes through immune-competent cats (Smith et al., 1992). Also in neonatal or young dogs and cats (e.g., transplacentally infected kittens), clinical toxoplasmosis is often persistent, disseminated, and has a high mortality rate (Davidson, 2000).

In humans *T. gondii* infection can result with clinical toxoplasmosis in the fetus; stillbirths, intensive neurologic and ocular diseases are common if the mother is infected during pregnancy. Most humans acquire the disease by ingesting oocyst-contaminated soil and water, or from undercooked meat containing tissue cysts, by transplantation, blood transfusion, laboratory accidents, or congenitally (Elmore et al., 2010).

Clinical signs of feline toxoplasmosis include; anorexia, cough, dyspnea, fever followed by hypothermia, lethargy, vomiting, diarrhea, peritoneal effusion, icterus, myocardial dysfunction encephalitis and sudden death (Dubey et al., 1990, 1996; Patton et al., 1991). Ophthalmic manifestations consists of retinochoroiditis and both anterior and posterior uveitis (Piper et al., 1970; Dubey et al., 1990, 1996; Davidson, 2000; Michael R. Lappin, 2010).

Clinicopathological findings and test results are not definitive for toxoplasmosis diagnosis. However, Toxoplasmosis should be on the differential list for

cats with nonregenerative anemia, neutrophilic leukocytosis, lymphocytosis, monocytosis, neutropenia, eosinophilia, proteinuria, bilirubinuria, as well as increases in serum protein and bilirubin concentrations, and creatinine kinase, alanine aminotransferase, alkaline phosphatase, and lipase activities (Dubey et al., 2009; Michael R. Lappin, 2010).

Definitive diagnosis of feline toxoplasmosis is quite hard to make. Radiographic, hematologic, and biochemical changes sometimes occur but these results are not pathognomonic. High levels of Immunoglobulin G (IgG) antibody titers are not always an indication of the disease. ELISA can diagnose subclinical infections by using immunoglobulin M (IgM) antibody titers (Michael R. Lappin et al., 1989a) and circulating antigen (Ag) (Michael R. Lappin et al., 1989b) detection, but their use with clinically ill animals has not been reported (Michael R. Lappin et al., 1989).

## CASES

### First Case

The first case was a 1-year-old, female, mix breed cat showing convulsions, pain, head flexed towards the chest and instability on hind limbs without problems with defecation or urination. She was brought to a clinic in Hatay, Turkey with suspicion of trauma and the treatment was applied accordingly with methylprednisolone sodium succinate, vitamin B complex, ranitidine and fluid therapy. The condition of the cat did not improve and it was referred to Small Animal Hospital, Faculty of Veterinary Medicine in Ankara. First the cat's neurological examination was performed and the results was normal and according to that the cat's treatment started at the Department of Internal Medicine.

During general examination ptosis on the left eye, pale mucous membranes with hyperemic areas noted on the soft palate was observed. Lower back and pelvic pain was noted with palpation. Heart rate was 140 bpm, respiratory rate was 80/min and capillary refilling time was < 2 seconds. Femoral arterial pulse was normal and there was no color change on footpads of the hind legs. Crackles and dyspnea were detected after auscultation. In radiographic images of the thorax bronchial structures had an increased opacity.

A Complete Blood Count (CBC) showed Leukocytopenia, lymphocytopenia, anemia and

thrombocytopenia. Alanine aminotransferase (ALT) value was measured as 331 IU / L. FCOV / FIP IgG antibody titer was lower than 1:10 and Toxoplasma / Chlamydia IgG antibody titer was 1:64 resented by ELISA (Table 1, Table 2). Toxoplasma and Chlamydia antibody titer were checked. Toxoplasma antibody titer 1:64, Chlamydia antibody titer was less than 1:16.

Contractions of the hind limbs continued after the initial fluid and O<sub>2</sub> therapy initiated to improve the general condition and breathing problems. Following the test results Clindamycin 25 mg/kg/q24h IM was used for treatment. Within the first few days' response to treatment was positive but the treatment was continued for another 2 weeks 12.5mg / kg / 12h IM due to complaint of lameness in right hind leg. Lameness slowly decreased to normal at the end of third week of treatment. At the end of the third week Toxoplasma and Chlamydia antibody titer were checked again for control and the results were; Toxoplasma antibody titer 1:16, Chlamydia antibody titer was less than 1:16. Three months after the treatment, recurrence of disease has not occurred.

**Table 1.** Biochemical parameters of cases

Parameters	First Case		Second Case		Normal Range
	Lab. Value	Result	Lab. Value	Result	
Urea (mg/dl)	41.1	L	27.70	L	42.80- 64.20
Creatine (mg/dl)	0.71	L	12.24	N	0.80- 1.80
Total Protein (g/dl)	7.15	N	8.73	H	5.40- 7.80
Albumin (g/dl)	3.02	N	2.87	N	2.40- 3.80
ALT (IU/L)	331.0	H	53.00	H	0.00- 50.00
ALP (IU/L)	50.0	H	14.00	N	0.00- 70.00
CK (IU/L)	95.2	N	101.00	N	0.00 – 130.00

ALT alanine aminotransferase, ALP alkaline phosphatase, CK Creatine Kinase

### Second Case:

The second case was an 11 years old, male, mix breed cat showing convulsions and pain on the hind legs. Owners consulted to a veterinary clinic after the cat has lost its appetite. Clinic referred the patient to Small Animal Hospital, Faculty of Veterinary Medicine in Ankara suspecting from an underlying neurological condition after the general examination.

**Table 2.** Hematologic parameters of cases

Parameters	First Case		Second Case		Normal Range
	Lab. Value	Result	Lab. Value	Result	
WBC	1.60	L	18.10	N	5.50- 19.50
LYM	0.90	L	0.60	L	1.10- 7.00
MONO	0.30	N	0.40	N	0.20- 1.50
NEUT	0.40	L	15.80	H	2.80- 13.00
EOS	0.00	L	1.30	N	0.10- 99.90
LYM%	53.50	N	3.30	L	15.00- 60.00
MONO%	19.00	H	2.20	N	0.50- 11.00
NEUT%	27.10	N	87.20	H	25.00- 85.00
EOS%	0.40	N	7.30	N	0.10- 99.90
RBC	1.82	L	7.96	N	5.00- 45.00
HGB	2.60	L	12.70	N	8.00- 15.00
HCT	6.80	L	34.90	N	25.00- 45.00
MCV	37.40	L	43.80	N	39.00- 50.00
MCH	14.60	N	16.00	N	12.50- 17.50
MCHC	39.10	H	36.60	N	31.00-38.50
RDWa	17.00	L	20.30	L	20.00- 35.00
RDW%	14.60	N	13.80	L	14.00- 18.50
PLT	16.00	L	10.10	N	200.00- 500.00
WBC	1.60	L	18.10	N	5.50- 19.50

WBC White blood cells, LYM Lymphocytes, MONO Monocytes, NEUT Neutrophil, EOS Eosinophils, LYM% Lymphocytes percent, MONO% Monocytes percent, NEUT% Neutrophil percent, EOS% Eosinophils percent, RBC red blood cells, HGB Haemoglobin, HCT Haematocrit, MCV Mean corpuscular volume, MCHC Mean corpuscular haemoglobin concentration, RDWa Red blood cell distribution, RDW% Red blood cell distribution percent, PLT Platelet

General condition of the cat was good but it was struggling to urinate and defecate and it was not relating to a trauma or operation history. Right hind leg was hyperextended and show more pain compared to the other one. Lung auscultation revealed no abnormalities. The heart rate was measured as 120 bpm and the number of breaths were 64 per min. The color of the mucous membrane was normal and capillary refilling time was determined as normal. Femoral arterial pulse was normal and no color change was observed in the footpads of the hind legs.

Lymphocytopenia and thrombocytopenia was noted on CBC. Alanine aminotransferase was measured as 53 IU/L. (Table 1, Table 2). In

Toxoplasma/ Chlamydia test performed by ELISA method, IgG antibody titer was 1:64. Toxoplasma and Chlamydia antibody titer were checked. Toxoplasma antibody titer 1:64, Chlamydia antibody titer was less than 1:16.

Treatment started with Clindamycin 25mg/ kg/ 24h IM. Since oral intake did not reach to a sufficient level on the 5<sup>th</sup> day of treatment jaundice was detected on the mucous membranes. Clindamycin dosage was rearranged to 12.5 mg/kg/q12h IM. Ornipur Solution<sup>®</sup> was used 2 ml/sc /48h and forced feeding was performed. The treatment process was completed at the end of the 3rd week. General condition improved to normal and problems with hind limbs completely resolved. After treatment clinical recovery maintained. The patient didn't show up for controls therefore patient doesn't have antibody titer after treatment.

## DISCUSSION

Extraintestinal toxoplasmosis develops from intracellular replication of tachyzoites in hepatic, pulmonary, Central Nervous System (CNS) and pancreatic tissues (Michael R. Lappin, 2010). In both of our cases permanent contracture of the muscles of the hind limb and generalized fatigue were the first clinical signs of the disease. Symptoms relating to CNS problems might be seen in feline toxoplasmosis cases, *T. gondii* should be in the differential list when such symptoms are encountered.

Toxoplasmosis shows a variety of clinical signs, most commonly anorexia, lethargy, interstitial pneumonia, fever, hepatitis, gastrointestinal signs, hyperesthesia from myositis, and a variety of neurologic signs (Davidson, 2000). Decrease or loss of appetite and respiratory problems were also recognized in our cases and they are prevalent findings in other toxoplasmosis cases as well.

Variety of clinicopathologic abnormalities are recognized in feline toxoplasmosis including nonregenerative anemia, neutrophilic leukocytosis, lymphocytosis, monocytosis, neutropenia, eosinophilia, proteinuria, bilirubinuria, as well as in serum protein and bilirubin concentrations, creatinine kinase, alanine aminotransferase, alkaline phosphatase, and lipase activities might increase (Michael R. Lappin, 2010). Out of these nonspecific hematological and serum biochemical abnormalities we have recognized include leukopenia, lymphopenia and increase of alanine aminotransferase activity in our two cases. Our

second patient also developed jaundice due to loss of appetite which leads to hepatic lipidosis and damage to the paranchyma as a direct effect of the parasite.

Various publications for the treatment of feline toxoplasma suggest the administration of clindamycin at a dose of 10-12 mg / kg p12h for 4 weeks (Michael R. Lappin, 2010). During our treatment, clindamycin was administered intramuscularly at a dose of 25mg / kg p24h during the first week. For the next 2 weeks, 12.5mg / kg p12h was administered and the treatment was completed.

A study done in Ankara region with samples taken from 129 cats 43.4% had positive results in titer of 1/16, 5.4% had in 1/64 and 17.8% had in 1/256 (Yücesan et al., 2019). Another study on seroprevalence of *T. gondii* in humans obtained 30.80% IgG, 3.78% IgM and 7.02% IgG and IgM positive results (Yereli et al., 2006). *Toxoplasma gondii* seroprevalence was found to be high in these studies. This report is written to draw attention to *T. gondii* since it has high prevalence and asymptomatic symptoms which could be easily overlooked by the veterinarians.

---

## ACKNOWLEDGMENTS

**Conflict of Interests:** The authors declared that there is no conflict of interests.

**Financial Disclosure:** The authors declared that this study has received no financial support.

---

## REFERENCES

- Davidson MG.** Toxoplasmosis. In *Vet Clin North America - Small Anim Pract* 2000; 30(5), 1051–1062.
- Dubey JP.** *Toxoplasma gondii* oocyst survival under defined temperatures. *J. Parasitol* 1998; 84(4), 862.
- Dubey JP, Lindsay DS, Lappin MR.** Toxoplasmosis and other intestinal coccidial infections in cats and dogs. *Vet Clin North America - Small Anim Pract* 2009; 39(6), 1009–1034.
- Dubey JP, Mattix ME, Lipscomb TP.** Lesions of neonatally induced toxoplasmosis in cats. *Vet Pathol* 1996; 33(3), 290–295.

- Dubey JP, Schlafer DH, Urban JF, Lindsay DS.** Lesions in Fetal Pigs with Transplacentally-induced Toxoplasmosis. *Vet Pathol* 1990; 27(6), 411–418.
- Elmore SA, Jones JL, Conrad PA, Patton S, Lindsay DS, Dubey JP.** *Toxoplasma gondii*: epidemiology, feline clinical aspects, and prevention. *Trends Parasitol* 2010; 26(4), 190–6.
- Lappin MR, Greene CE, Prestwood AK, Dawe DL, Tarleton RL.** Diagnosis of recent *Toxoplasma gondii* infection in cats by use of an enzyme-linked immunosorbent assay for immunoglobulin M. *American J Vet Res* 1989a; 50(9), 1580–1585.
- Lappin MR, Greene CE, Prestwood AK, Dawe DL, Tarleton RL.** Enzyme-linked immunosorbent assay for the detection of circulating antigens of *Toxoplasma gondii* in the serum of cats. *American J Vet Res* 1989b; 50(9), 1586–1590.
- Lappin MR.** Update on the diagnosis and management of toxoplasma gondii infection in cats. *Topics in Comp Anim Med* 2010; 25(3), 136–141.
- Lappin MR, Greene CE, Winston S, Toll SL, Epstein ME.** Clinical Feline Toxoplasmosis. *J of Vet Internal Med* 1989; 3(3), 139–143.
- Lukesová D, Literák I.** Shedding of *Toxoplasma gondii* oocysts by Felidae in zoos in the Czech Republic. *Vet Parasitol* 1998; 74(1), 1–7.
- Patton S, Legendre AM, McGavin MD, Pelletier D.** Concurrent Infection with *Toxoplasma gondii* and Feline Leukemia Virus: Antibody Response and Oocyst Production. *J Vet Internal Med* 1991; 5(3), 199–201.
- Piper RC, Cole CR, Shadduck JA.** Natural and experimental ocular toxoplasmosis in animals. *American J Ophthalmol* 1970; 69(4), 662–668.
- Smith KE, Zimmerman JJ, Patton S, Beran GW, Hill HT.** The epidemiology of toxoplasmosis on Iowa swine farms with an emphasis on the roles of free-living mammals. *Vet Parasitol* 1992; 42(3–4), 199–211.
- Tenter AM, Heckeroth AR, Weiss LM.** *Toxoplasma gondii*: From animals to humans. *Int J Parasitol* 2000; 30(12–13), 1217–1258.
- Yereli K, Balcioğlu IC, Özbilgin A.** Is *Toxoplasma gondii* a potential risk for traffic accidents in Turkey? *Forensic Sci Intern* 2006; 163(1–2), 34–37.
- Yücesan B, Babür C, Koç N, Sezen F, Kılıç S, Gürüz Y.** Investigation of Anti-*Toxoplasma gondii* Antibodies in Cats Using Sabin-Feldman Dye Test in Ankara in 2016. *Turk Parazitoloj Derg* 2019; 43(1), 5–9.