ISSN 2316-9281

Scientific Electronic Archives
Issue ID: Vol.19, January/February 2026, p. 1-7
DOI: http://dx.doi.org/10.36560/19120262082
+ Corresponding author*:* giovana.cambuy@hotmail.com

Cytological evaluation of cerebrospinal fluid in a dog infected with toxoplasmosis and distemper – Case Report

Giovana Cambuy de Andrade [+], Fernando Nardi Drummond, Alessandra Kataoka

Federal University of Mato Grosso – Campus Sinop

Abstract.Toxoplasmosis is a zoonotic disease caused by the protozoan *Toxoplasma gondii*, which affects various species. Dogs are intermediate hosts and, due to their role as sentinel animals, help monitor the environmental condition. The infection is more common in cases of immunosuppression and coinfection, and infected dogs are usually asymptomatic. Although rare, the infection, when associated with distemper, significantly increases the severity of neurological symptoms. In the present case, a four-year-old male Golden Retriever dog was treated for symptoms including apathy, anorexia, cachexia, vomiting, and abdominal pain upon palpation. In addition to nonspecific symptoms, the dog exhibited severe neurological symptoms. The first complete blood count revealed intense pancytopenia, and the second showed worsening with intense normocytic normochromic non-regenerative anemia, as well as severe leukopenia and thrombocytopenia. Elevated ALT and urea levels were also observed. Due to the severely debilitated condition, the animal died. Immediately after death, cerebrospinal fluid (CSF) was collected for analysis. The sample was xanthochromic, turbid, and exhibited intense pleocytosis, with 156 mg/dL of proteins, a density of 1.016, and a pH of 6.5. Cytoscopy revealed a predominance of mononuclear cells, mainly reactive lymphocytes and heavily activated, foamy macrophages, with the presence of erythrophagocytosis and leukophagocytosis. The neutrophils present were degenerated. Additionally, *Toxoplasma sp.*bradyzoites were observed, both phagocytized and free in the background, as well as Lentz bodiesexclusively intracellularly. The report concludes that canine toxoplasmosis, associated with immunosuppressive diseases like distemper, causes severe neurological symptoms, often confused with other diseases, which can delay diagnosis and lead to death. Therefore, toxoplasmosis should be considered an important differential diagnosis. CSF examination is crucial in cases of severe neurological symptoms, especially when previous treatments have failed, as it was essential for the diagnosis guidance in this case.
Keywords:*Toxoplasma gondii*, CSF, Lentz bodies.

Introduction

Toxoplasmosis is a zoonotic disease with cosmopolitan distribution and opportunistic characteristics, caused by an obligate intracellular parasite known as *Toxoplasma gondii*. It affects various species, with homeothermic animals serving as intermediate hosts and felids as the definitive hosts. In Brazil, the seroprevalence in animals is high, and genotypes with varying patterns of virulence have been detected (RODRIGUES, 2022; FERREIRA, 2016; BRESCIANI, 2008).

In the case of dogs, infection by *T. gondii* is of great epidemiological importance, as they act as sentinel animals and indicators of environmental conditions, serving as a direct or indirect source of infection for humans. The main risk factors for the acquisition of the disease in dogs include the consumption of raw meat, free access to the streets, food or water contaminated by feces, and/or cohabitation with cats (RODRIGUES, 2022; FRADE, 2015).

As an opportunistic parasite, its proliferation occurs mainly in cases of immunosuppression. Thus, the acute phase of the disease and the clinical symptoms are generally associated with the presence of other illnesses such as canine distemper and ehrlichiosis (MORETTI, 2002).

Infected dogs are often asymptomatic, and toxoplasmosis is considered a rare condition in this species. However, a study conducted by the Animal Infectious Diseases Department at São Paulo State University showed that 41.1% of dogs with concurrent toxoplasmosis and distemper experienced a significant increase in symptom severity (SCHUSTER, 2020; AGUIAR, 2012).

In Brazil, studies on dogs show a varying prevalence of toxoplasmosis, ranging from 20.8% to 88%. The prevalence in dogs is correlated with highly contaminated environments that favor infection due to dietary habits and contact with soil (SORTE, 2015).

Serological studies show a prevalence of 88.5% of canine toxoplasmosis in the state of Mato

Grosso. Despite this, it remains a disease with rare symptomatology in dogs and is very similar to distemper. As a result, toxoplasmosis is often overlooked as a possible differential diagnosis (FRADE, 2015).

The aim of this report is to clarify the importance of early identification of canine toxoplasmosis, demonstrate the severity of clinical symptoms when associated with distemper, and emphasize the diagnostic value of cerebrospinal fluid analysis in cases of severe neurological symptoms.

Case Report

On April 24, 2023, a male, unneutered Golden Retriever, four years old and weighing 14.7 kg, was brought to the Veterinary Hospital of UFMT – Sinop. The dog presented with complaints of apathy, hyporexia, and later anorexia with progressive weight loss, emesis, abdominal pain upon palpation, and neurological signs such as head pressing against the wall, circling, photosensitivity, head tilt, ocular globe rotation, vocalization, and aggressiveness.

The vaccination protocol was carried out with a multivalent and rabies vaccine when the patient was a puppy, but there had been no vaccination history for over 12 months. The dog also had a history of a tick-borne disease treated during puppyhood, but the owner was unable to specify which disease it was or what treatment was administered.

According to the owners' report, the described symptoms had begun in a milder form approximately one month earlier, coinciding with the date of moving to a rural property. Two weeks after the onset of symptoms, the dog was taken to a private veterinary clinic, where a rapid test for distemper (brand not informed) returned a negative result, and a SNAP 4DX Plus test was positive for antibodies against *Ehrlichia* sp., leading to the initiation of symptomatic treatment along with doxycycline.

The complete blood count performed during the first visit to the private clinic, on April 8, 2023, is shown in Table 1, and the biochemical tests in Table 2. The results indicated normocytic hypochromic anemia, leukopenia due to marked lymphopenia, eosinopenia, and monocytopenia, as well as thrombocytopenia with a moderate presence of macroplatelets. The biochemical tests were within reference values. Despite treatment, the animal experienced a significant worsening of symptoms, at which point it was taken to the Veterinary Hospital.

Table1. Complete blood count results of the Golden Retriever patient from the first consultation at a private clinic on April 8, 2023.

	Results	Reference Interval *
Red Blood Cells(/µL)	3.400.000	5.500.000 – 8.500.000
Hemoglobin (g/dL)	6,2	12 – 18
Hematocrit (%)	21,43	37 – 55
MCV (fL)	63	60 – 77
MCHC (g/dL)	29	31 – 36
Total leukocytes(/µL)	4.150	6.000 – 17.000
Segmented neutrophils(/µL)	3.735	3.000 – 11.500
Band neutrophils(/µL)	83	0 – 300
Lymphocytes(/µL)	166	1.000 – 4.800
Eosinophils(/µL)	83	100 – 1.250
Monocytes(/µL)	83	150 – 1.350
Platelets(/µL)	137.000 (moderate presence of macroplatelets)	200.000 – 500.000

*SCHALM's Veterinary Hematology (2000).

Table 2. Biochemical test results (ALT and creatinine) of the Golden Retriever patient from the first consultation at a private clinic on April 8, 2023.

	Results	Reference Interval *
ALT (U/L)	23	21 – 102
Creatinine (g/dL)	1,4	0,5 – 1,5

Upon arrival for care at the UFMT Veterinary Hospital (HOVET) on April 24, 2023, the animal was cachectic, apathetic, with extremely pale mucous membranes, depressed, and exhibiting abnormal posture and movement (paresis of the hind limbs and a curved posture with head tilt). Upon hospitalization, treatment for ehrlichiosis was continued along with symptomatic care (support for muscle mass gain, dietary supplementation, and anti-nausea medications), while other possible differential diagnoses were investigated (other infectious agents, tumors, and organ involvement).

During the two weeks of hospitalization, the patient's neurological symptoms worsened, remaining only in lateral recumbency, becoming increasingly aggressive, and exhibiting localized myoclonus in the right eye. When attempting to stand, the dog showed paresis and abdominal pain detected by palpation. A new complete blood count and other biochemical tests were performed, shown

in Tables 3 and 4, respectively.

The results demonstrated pancytopenia with intense normocytic normochromic non-regenerative anemia, as well as severe leukopenia and thrombocytopenia. Additionally, increases in ALT and urea levels were observed.

On ultrasonographic evaluation, the animal showed changes indicative of prostatitis, lymphadenomegaly, biliary concretions, and pancreatic enlargement that may be related to an inflammatory process, as well as renal alterations compatible with possible chronic kidney disease.

After the exams, a blood transfusion was performed on April 30, 2023, resulting in a slight improvement in hematological parameters, but the patient remained severely pancytopenic. Due to the extremely debilitated state, along with possible bone marrow hypoplasia and intense immunosuppression, the animal died on May 9, 2023.

Immediately after death, cerebrospinal fluid was collected from the cisterna magna region for analysis. Because the animal was at high anesthetic risk, it was not possible to collect the sample earlier.

The sample appeared xanthochromic (Figure 1A), turbid, and with intense pleocytosis (13,000 nucleated cells per microliter), protein concentration of 156 mg/dL, specific gravity of 1.016, and pH of 6.5. Cytoscopy revealed a predominance of mononuclear cells, mainly reactive lymphocytes and intensely activated, foamy macrophages, with evidence of erythrophagocytosis and leucophagocytosis. The neutrophils present were degenerated. Additionally, bradyzoites of *Toxoplasma* sp. were observed, both phagocytosed and free on the slide background, as well as Lentz bodiesexclusively intracellularly (Figure 1B). The diagnosis of toxoplasmosis was later confirmed through histopathological examination.

Discussion

In the present study, a case of co-infection by toxoplasmosis and distemper in a dog was reported. This case is of particular interest because both diseases have clinical presentations that can be easily confused with other conditions, making differential diagnosis challenging (FERREIRA, F. 2016).

In this case, toxoplasmosis became a suspicion only after all other therapeutic protocols and differential diagnoses failed, when the condition was already severe and leading to the animal's death.

According to the owners, the symptoms began when the animal changed its place of residence, moving to a rural property. Although it is not possible to know exactly what caused the infection in this patient and there is no detailed information about its habits or environment in the medical record, the dog was exposed to risk factors after the move, since rural properties are places conducive to contagion, especially of toxoplasmosis.

Table3. Complete blood count and reticulocyte count results of the Golden Retriever patient on April 28, 2023, performed at the Clinical Pathology Laboratory of the UFMT Veterinary Hospital, Sinop – MT.

	Results	Reference Interval [1]
Red Blood Cells(/µL)	2.310.000	5.500.000 – 8.500.000
Hemoglobin (g/dL)	4,9	12 – 18
Hematocrit (%)	15,5	37 – 55
MCV (fL)	67,1	60 – 77
MCHC (g/dL)	31,6	31 – 36
Total leukocytes(/µL)	600	6.000 – 17.000
Segmented neutrophils(/µL)	498	3.000 – 11.500
Band neutrophils(/µL)	0	0 – 300
Lymphocytes(/µL)	66	1.000 – 4.800
Eosinophils(/µL)	12	100 – 1.250
Monocytes(/µL)	24	150 – 1.350
Platelets(/µL)	19.000	200.000 – 500.000
Reticulocytes(/µL)	6.930	> 60.000[2]

[1] SCHALM's Veterinary Hematology (2000).
[2] THRALL, M. A. Hematologia e Bioquímica Clínica Veterinária (2015).

Table 4. Biochemical test results (ALT, creatinine, alkaline phosphatase, and urea) of the Golden Retriever patient on April 28, 2023, performed at the Clinical Pathology Laboratory of the UFMT Veterinary Hospital, Sinop – MT.

	Results	Reference Interval
ALT (U/L)	205	21 – 102
Creatinine (g/dL)	1,4	0,5 – 1,5
Alkaline phospatase (U/L)	52	20 – 156
Urea (mg/dL)	66	21 – 59,9

* KANEKO, J. J. Clinical Biochemistry of Domestic Animals (2008).

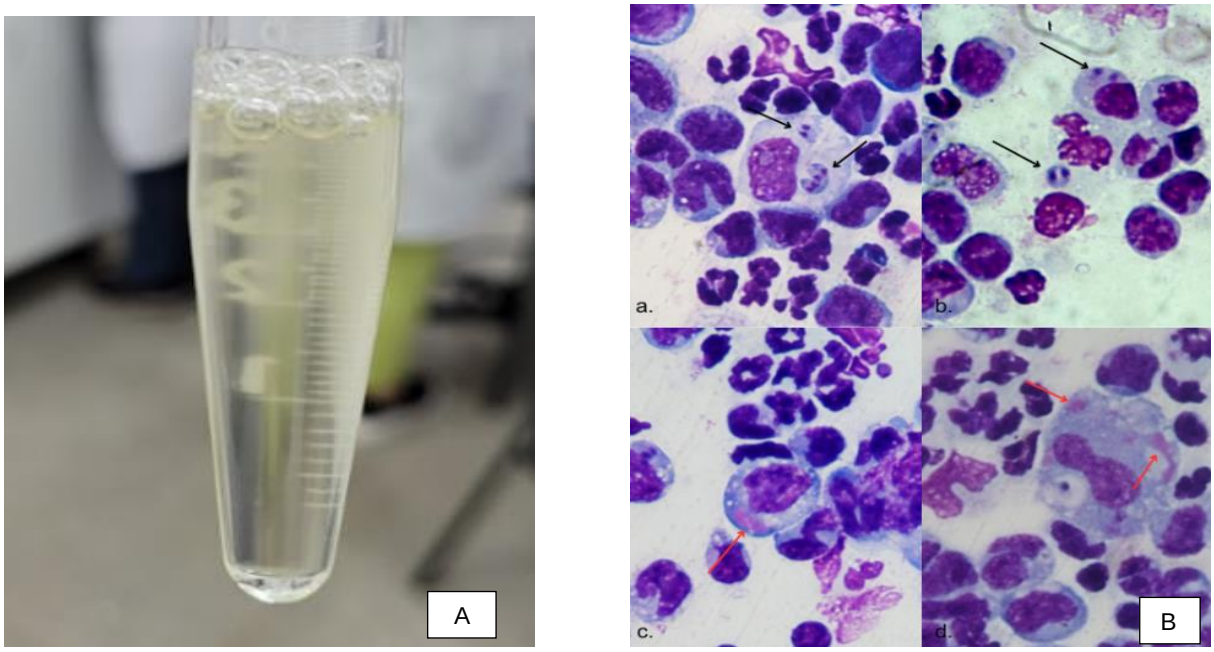

Figure 1. (A) Xanthochromic cerebrospinal fluid sample from the Golden Retriever patient on May 9, 2023, performed at the Clinical Pathology Laboratory of the UFMT Veterinary Hospital, Sinop – MT.; (B) Toxoplasma gondii bradyzoites (black arrows) and Lentz bodies (red arrows) in a cerebrospinal fluid sample

Brazilian rural areas concentrate many habits that can be sources of infection, such as contact with contaminated water and soil, contact with animals carrying the parasite, and ingestion of contaminated food. Similarly, Olbera (2020) obtained results that support these statements. In that study, a higher prevalence was observed in areas with lower socioeconomic status, poor infrastructure, presence of street or semi-domiciled dogs and cats, environmental pollution, and absence of impermeable soil — factors more frequently found in rural zones. These factors are also present in urban areas with habits similar to rural zones and are associated with the consumption of non-industrialized animal products and free access to the streets (ARAÚJO, 2011).

Studies by Sorte (2015) also demonstrated an infection distribution in dogs in Mato Grosso of 62.4% in rural areas and 40.4% in urban areas, with these urban areas presenting the same predisposing factors similar to the rural zones mentioned earlier.

Furthermore, the animal's vaccination protocol was not up to date, and consequently, it was vulnerable to the canine distemper virus. Distemper is the main immunosuppressive disease associated with toxoplasmosis, causing a significant worsening of neurological symptoms (SCHUSTER, 2020; FRADE, 2015; AGUIAR, 2012).

Although the patient was vaccinated as a puppy, it is not possible to know if there were any vaccination failures at the time of administration (such as improper refrigeration, incorrect application, vaccine inefficacy, inadequate antiviral immune response, immunocompromise of the dog, or stress). Even if the entire protocol was correctly followed, the absence of booster shots is a risk factor for infection since the vaccines currently used do not induce absolute immunity (MARTINS, 2009).

Toxoplasmosis is widely distributed in the environment, which poses a high risk of infection, although healthy dogs do not usually show clinical signs. In this species, the disease is difficult to diagnose due to its nonspecific symptoms and chronic nature. Canine distemper, on the other hand, can present in acute, subacute, or chronic phases and causes intense immunosuppression, which in this report may have triggered the opportunistic proliferation of the protozoan and the clinical signs of toxoplasmosis (MORETTI, 2002; MANGIA, 2008). According to Dubey and Beattie (1998), the association of both diseases occurs in 99% of reported clinical cases of toxoplasmosis.

When clinical signs are present in dogs with toxoplasmosis, the symptoms are nonspecific (such as vomiting, diarrhea, apathy, anorexia, neurological signs) and very similar to other diseases since they involve multiple systems including the nervous, reproductive, digestive, respiratory, ocular, muscular, hematopoietic, hepatic, and cardiovascular systems (FERREIRA, 2016; SORTE, 2015).

The patient in the present report showed alterations in the nervous, muscular, hematopoietic, and hepatic systems. The main symptoms related to toxoplasmosis observed in the animal were aggression, paresis of the hind limbs, lymphadenomegaly evidenced by ultrasound, and hyperesthesia upon palpation, similar to those reported by Moretti (2002) and Bresciani (2008).

In addition to the clinical signs, complementary exams also revealed alterations compatible with toxoplasmosis, such as anemia varying from normocytic hypochromic to normocytic normochromic associated with lymphopenia, increased serum ALT activity, lymphadenomegaly on imaging exams, as well as pancreatic and hepatobiliary involvement also evidenced by ultrasound (GALVÃO, 2014; MORETTI, 2002; ABREU, 2001).

Among the hematological changes mentioned, the severe, persistent, and non-regenerative normocytic normochromic anemia is likely a consequence of the infectious agents (virus and protozoan) that led to bone marrow hypoplasia or aplasia. The leukopenia due to lymphopenia, which was present from the first complete blood count, may have occurred due to the migration of cells to the inflamed tissue (which, in this case, was the cerebrospinal fluid), possibly caused by atrophy or necrosis of lymphoid tissues or due to the cytotoxic action of the parasites. Monocytopenia and eosinopenia, also observed in both exams, have little diagnostic significance, although eosinopenia may also result from diseases that cause bone marrow hypoplasia or aplasia (STOCKHAM AND SCOTT, 2011; THRALL, 2015).

In the second complete blood count, the patient also presented neutropenia, which was probably caused by the co-infection with virus and protozoan, possibly leading to granulocytic hypoplasia. The same mechanisms may apply to thrombocytopenia, which was likely caused by infection (immune-mediated destruction and consumption) or decreased production. The combination of all these changes justifies the pancytopenia and the inefficacy of the blood transfusion (STOCKHAM AND SCOTT, 2011; THRALL, 2015).

Regarding the biochemical exams, the increase in ALT can be explained by the fact that the protozoan causes lesions and focal necrosis in various organs as it disseminates (with the liver being one of the most affected), resulting in enzyme leakage (THRALL, 2015; GALVÃO, 2014; BRESCIANI, 2008; MORETTI, 2002).

The increase in urea not accompanied by creatinine is probably explained by the animal's cachexia, since creatinine originates from the conversion of creatine and creatine phosphate in the muscles. It is assumed that this increase does not stem from non-renal causes, as ultrasound showed that the patient had changes compatible with chronic kidney disease (THRALL, 2015).

Regarding the serological tests performed in a private clinic, the SNAP 4DX Plus test detects antibodies against *Ehrlichia canis/ewingii*, *Anaplasma phagocytophilum/platys*, and *Borrelia burgdorferi*, as well as the antigen of *Dirofilaria immitis*. In other words, if the animal has been exposed to the disease in the past two years, the test will be positive even if the disease is no longer active. The patient in this report had already been treated for tick-borne disease previously, which explains the positive result for *Ehrlichia* sp. (IDEXX Laboratories, 2022). Furthermore, the worsening of the clinical condition despite doxycycline treatment supports the suspicion that the animal did not currently have the disease.

As for the serological test for canine distemper, although it is not possible to determine the brand used, a study by Curti et al. (2012) showed that immunochromatographic tests for detecting distemper antigen (commonly used for screening in private clinics due to ease and speed) presented many false negatives in animals with neurological symptoms. Even the PCR technique, considered the diagnostic gold standard because of its high sensitivity and specificity, can yield false negatives depending on the type of clinical sample submitted for analysis (NEGRÃO, 2007).

Although, in general, cerebrospinal fluid (CSF) analysis for toxoplasmosis has low diagnostic value since the blood-brain barrier prevents the passage of antibodies and defense cells, when this barrier is breached, CSF analysis reveals mononuclear pleocytosis, which may include neutrophils and elevated protein levels, as in this case. In distemper, the alterations (when present) are similar to those in toxoplasmosis (SCHUSTER, 2020; GIRALDI, 2002; TUDURY, 1997).

Besides the previously mentioned changes, the presence of Lentz bodies and *Toxoplasma* sp. bradyzoites were fundamental in guiding the diagnosis, since no inclusions and/or bradyzoites were found in the circulating blood, and the serological test for distemper was negative.

Conclusion

With this report, it can be concluded that canine toxoplasmosis associated with immunosuppressive diseases (such as canine distemper) causes severe neurological symptoms. Because the symptoms are very similar to those of other more common diseases, clinicians are often directed toward other more likely diagnostic possibilities. This can delay an accurate diagnosis and lead the animal to death. For this reason, toxoplasmosis should be considered an important differential diagnosis. Furthermore, it is extremely important to request cerebrospinal fluid analysis in cases of severe neurological symptoms, especially when previous treatments have been unsuccessful. In this case, cerebrospinal fluid analysis was essential for the patient's diagnosis.

References

ABREU, C. B. et al. Aspectos clínicos, patológicos e sorológicos da toxoplasmose experimental em cães jovens. Semina: Ciências Agrárias, Londrina, v.22, n. 2, p. 123-130, 2001.

AGUIAR, D. M. et al. Canine distemper vírus and *Toxoplasma gondii* co-infection in dogs with neurological signs. Arquivo Brasileiro de Medicina Veterinária e Zootecnia, Belo Horizonte, v. 64, n. 1,

p. 221-224, 2012.

ARAÚJO, D. A. et al. Investigação dos fatores associados à infecção pelo *Toxoplasma gondii* em cães e seres humanos de Porto Filgueira, PR. Veterinária e Zootecnia, Botucatu, v. 18, n. 1, p. 98-111, 2011.

BRESCIANI, K. D. S. et al. Toxoplasmose canina: aspectos clínicos e patológicos. Semina: Ciências Agrárias, Londrina, v. 29, n. 1, p. 189-202, 2008.

CURTI, M. C.; ARIAS, M. V. B.; ZANUTTO, M. S. Avaliação de um kit de imunoensaio cromatográfico para detecção do antígeno do vírus da cinomose em cães com sinais sistêmicos ou neurológicos da doença. Semina: Ciências Agrárias, Londrina, v. 33, n. 6, p. 2383-2390, 2012.

DUBEY, J. P.; BEATTIE, C. P. Toxoplasmosis in dogs *(Canis familiaris*) - Toxoplamosis of animals and man. Boca Raton: CRC Press, 1998. cap.8, p.127-42.

FERREIRA, F. P. etal. Frequência de anticorpos anti-*Toxoplasma gondii* em cães com sinais clínicos compatíveis com toxoplasmose. Ciênc. Anim. Bras., Goiânia, v. 17, nº 4, p. 640-646, 2016.

FRADE, M. T. L. et al. Caracterização clínica, patológica e imuno-histoquímica da toxoplasmose em cães com cinomose no semiárido da Paraíba, Brasil. Semina: Ciências Agrárias, v. 36, nº 6, suplemento 2, p. 4251-4256, 2015.

GALVÃO, A. L. B. Aspectos da toxoplasmose na clínica de pequenos animais. Semina: Ciências Agrárias, v. 35, n. 1, p. 393-410, 2014.

GIRALDI, J. H. et al. Sorologia e histopatologia de *Toxoplasma gondii* e *Neospora caninum* em cães portadores de distúrbios neurológicos. Semina: Ciências Agrárias, Londrina, v. 23, nº 1, p. 9-14, 2002.

IDEXX Laboratories. SNAP 4Dx Plus Test Accuracy. 2022. Disponível em:<https://www.idexx.com.br/files/snap-4dx-plus-test-accuracy.pdf.> Acesso em: 10 fev. 2025.

MANGIA, S. H.; PAES, A. C. Neuropatologia da Cinomose. Vet. E Zootec., v. 15, n. 3, p. 416-427, 2008.

MARTINS, D. B.; LOPES, S. T. A.; FRANÇA, R. T. Cinomose canina – Revisão de literatura. Acta Veterinária Brasilica, v. 3, n. 2, p. 68-76, 2009.

MORETTI, L. A. et al. Toxoplasmose em cães co-infectados com o vírus da cinomose. Semina: Ciências Agrárias, Londrina, v. 23, n. 1, p. 85-91, 2002.

NEGRÃO, F. J.; ALFIERI, A. A.; ALFIERI, A. F. Avaliação da urina e de leucócitos como amostras biológicas para a detecção ante mortem do vírus da cinomose canina por RT-PCR em cães naturalmente infectados. Arq. Bras. Med. Vet. Zootec., v. 59, n. 1, p. 253-257, 2007.

OLBERA, A. V. G. et al. Cumulative incidence and spatial distribution of dogs exposed to *Toxoplasma gondii*. Brazilian Journal of Veterinary Parasitology, 2020.

RODRIGUES, N. J. L. et al. Atualizações e padrões da toxoplasmose humana e animal. Vet. e Zootec., v. 29, 2022.

SCHUSTER, D. M. Meningoencefalites infecciosas em cães. Trabalho de Conclusão de Curso (Graduação). Curso: Medicina Veterinária. Universidade Federal do Rio Grande do Sul, Porto Alegre, 2020.

SORTE, E. C. B. et al. Serological and molecular detection of *Toxoplasma gondii* in dogs of urban and rural areas of Cuiabá, Mato Grosso. Semina: Ciências Agrárias, Londrina, v. 36, n. 6, p. 3705-3712, 2015.

STOCKHAM, S. L.; SCOTT, M. A. Fundamentos de Patologia Clínica Veterinária. 2ª edição. Rio de Janeiro: Guanabara Koogan, 2011.

THRALL, M. A. Hematologia e Bioquímica Clínica Veterinária. 2ª edição. Rio de Janeiro: Guanabara Koogan, 2015.

TUDURY, E. A. et al. Observações clínicas e laboratoriais em cães com cinomose nervosa. Ciência Rural, Santa Maria, v. 27, n. 2, p. 229-235, 1997.