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Severe interstitial pneumonia and necrohemorrhagic hepatitis due to Toxoplasma gondii infection in a blackbuck (Antilope cervicapra)

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Abstract

Herein, we report a 4-month-old male blackbuck (*Antilope cervicapra*) from a Zoo, with toxoplasmosis. Histopathological examination of the lung and liver revealed severe diffuse acute lymphohistiocytic interstitial fibrinous pneumonia with a hyaline membrane, associated with circular basophilic structures, 50µm in diameter (*T. gondii* bradyzoite cysts), and multifocal subacute fibrinous necrohemorrhagic hepatitis. Both the amplified 18S rRNA gene and the SAG2 sequence in the BLASTn analyses showed 100% identity to the *T. gondii* isolate, confirming the agent's identification. Toxoplasmic pneumonia and hepatitis were considered to be the primary causes of death.

Background

Toxoplasma gondii is a zoonotic intracellular protozoan found in various domestic, wild, or captive zoo animal species (Roman et al., 2024). *T. gondii* has a heteroxenous life cycle and requires a felid as a definitive host, and an intermediate host is necessary for reproduction (Silva et al., 2025). It can cause significant clinical and pathological effects in multiple animal species, including humans (Dubey and Jones, 2008). Herein, we report a blackbuck (*Antilope cervicapra*) with toxoplasmosis.

Case presentation

Animal Ethics. The *BioParque do Rio* veterinarians responsible signed a necropsy consent for research purposes for this *Antilope cervicapra* ID: 963003100034204 (IBAMA 15360).

Clinical History. The 4-month-old male blackbuck (*Antilope cervicapra)* had shown apathy and prostration for about 2 days. The ruminant was isolated for veterinary management and dietary monitoring. According to records, the blackbuck had reached sexual maturity and had experienced conflicts with the group's dominant male and died. The blackbuck carcass was provided by veterinarians from BioParque do Rio (Zoo), Rio de Janeiro, for necropsy.

Pathologic examination. The *BioParque do Rio* team conducted the necropsy. Tissue samples collected from the lungs, liver, brain, skeletal muscle, heart, tongue, esophagus, trachea, spleen, lymph nodes, kidney, rumen, reticulum, omasum, abomasum and intestines were sent to the Pathology Section (SAP) of UFRuralRJ for histological examination. The tissues were fixed in 10% buffered formalin, routinely processed for histology, embedded in paraffin, sectioned at 5µm, and stained with hematoxylin and eosin (HE).

Grossly, the lungs were diffusely enlarged and failed to collapse within the thoracic cavity. The liver exhibited randomly distributed millimetric white foci on both the capsular and parenchymal surfaces. Histopathological examination of the lung and liver revealed well-preserved tissues, allowing detailed evaluation of the lesions. Microscopic evaluation of the pulmonary tissue revealed severe, diffuse, subacute lymphohistiocytic interstitial pneumonia, characterized by marked expansion of alveolar septa due to infiltration by histiocytes, lymphocytes, and occasional neutrophils. Moderate amounts of hyaline

fibrillar material (fibrin) and amorphous eosinophilic material (edema) were also present in the alveolar interstitium.

Frequent structural alterations in the alveolar septa were noted, including septal discontinuity with pyknotic debris and loss of type I pneumocytes, indicating alveolar septal necrosis. In response to this loss, a clear, amorphous, eosinophilic mass delineating the alveolar sac (hyaline membrane) and moderate to marked proliferation of type II pneumocytes were observed. The airspaces contained a large number of extravascular red blood cells (hemorrhage) and moderate proliferation of lymphocytes and alveolar macrophages. Occasionally, cells with eosinophilic cytoplasm and two to five round to oval nuclei were identified.

Within the lymphohistiocytic infiltrate in the alveolar septum, multifocal areas containing circular basophilic structures, 50 µm in diameter, with a thin unstained capsule and many slightly elongated structures measuring 2–4 µm, consistent with *T. gondii* bradyzoite cysts, were observed (Fig. 1A). In the liver, lesions consistent with mild multifocal subacute fibrinous necrohemorrhagic hepatitis were observed. Small foci of hepatocytes with pyknotic nuclei (necrosis), interspersed with mild numbers of lymphocytes, histiocytes, extravascular red blood cells (hemorrhage), and eosinophilic fibrillar material (fibrin), were seen (Fig. 1B). No significant microscopic alterations were observed in the skeletal muscle, tongue, esophagus, trachea, spleen, kidney, rumen, reticulum, omasum, and intestines. Based on the histopathological findings, severe diffuse subacute lymphohistiocytic interstitial pneumonia with a hyaline membrane, associated with a *T. gondii* bradyzoite cyst (Fig. 1A, arrow), and mild multifocal subacute fibrinous necrohemorrhagic hepatitis (Fig. 1B) were diagnosed.

DNA extraction and endogenous gene verification. DNA was extracted from lung fragments using the phenol-chloroform method (Sambrook and Russell, 2001). After extraction, the DNA samples were packed into polypropylene microtubes, identified, and stored at -20°C until molecular analysis. The detection of amplifiable DNA was confirmed using a conventional PCR reaction targeting the endogenous GAPDH gene (Glyceraldehyde-3-phosphate dehydrogenase enzyme), which amplifies a fragment of 400 base pairs (bp), as described by Birkenheuer et al. (2003) (Table 1). The following reagents were used for amplification (final volume: 25µl): 0.8µM of each primer; 1.5mM MgCl2; 1x PCR buffer (5X Colorless Promega®); 0.2mM DNTPs; 1U Taq DNA polymerase (Promega®) and 2µL DNA. The reaction was carried out in a thermocycler with an initial denaturation step (95°C, 5min), followed by 35 cycles of denaturation (95°C, 30sec), annealing (55°C, 30sec), and extension (72°C, 25sec), with a final extension step (72°C, 5min). DNase-free ultrapure water was used as a negative control.

Table 1
Primers targeting endogenous genes, Sarcocystidae family, and Toxoplasma gondii used in this study

Primers	Gen	Nucleotide sequences (5'-3')	Approximate amplicon size	Reference
GAPDH-F	*GAPDH*	CCTTCATTGACCTCAACTACAT	400bp	Birkenheuer, Levy, & Breitschwerdt. (2003)
GAPDH-R	*GAPDH*	CCAAAGTTGTCATGGATGACC	400bp	Birkenheuer, Levy, & Breitschwerdt. (2003)
SarcoF	*18s rRNA*	CGCAAATTACCCAATCCTGA	700bp	Moré et al. (2011)
SarcoR	*18s rRNA*	ATTTCTCATAAGGTGCAGGAG	700bp	Moré et al. (2011)
TOXO 1	*B1*	GGAACTGCATCCGTTCATGAG	194bp	Burg et al. (1989)
TOXO 2	*B1*	TCTTTAAAGCGTTCGTGGTC	194bp	Burg et al. (1989)
SAG2ALTF	*SAG2*	GGAACGCGAACAATGAGTTT	729bp	Howe et al. (1997)
SAG2ALTR	*SAG2*	GCACTGTTGTCCAGGGTTTT	729bp	Howe et al. (1997)

Detection of the Sarcocystidae family. Amplification of an approximately 700 bp fragment of the *18S rRNA* gene was performed using primers previously described by Moré et al. (2011) (Table 1). PCR reactions were carried out in a final volume of 25 µL, containing 0.8 µM of each primer, 3 mM $MgCl_2$, 1× PCR buffer (5× Colorless, Promega®), 0.2 mM dNTPs, 1 U of Taq DNA polymerase (Promega®), and 3 µL of template DNA. DNase-free ultrapure water was used as a negative control. The reaction was carried out in a thermocycler with initial denaturation (94°C, 5min), followed by 40 cycles of denaturation (94°C, 1min), annealing (60°C, 45sec), and extension (72°C, 1min), with a final extension cycle (72°C, 5min). A *Sarcocystis* sp. sample from sheep esophageal cysts was used as a positive control for Sarcocystidae, and a microtube containing DNase-free ultrapure water was used as a negative control.

Toxoplasma gondii detection. PCR was performed using universal oligonucleotides, as previously described by Burg et al. (1989), to amplify a 194 bp fragment of the *Toxoplasma gondii*-specific *B1* gene (Table 1). The reaction was carried out in a final volume of 25 µL, containing 0.8 µM of each primer, 3 mM $MgCl_2$, 1× PCR buffer (5× Colorless, Promega®), 0.2 mM dNTPs, 1 U of Taq DNA polymerase (Promega®), and 3 µL of template DNA. DNase-free ultrapure water was used as a negative control. The reaction was carried out in a thermal cycler with initial denaturation (94°C, 5min), followed by 35 cycles of denaturation (94°C, 1min), annealing (60°C, 1min), and extension (72°C, 1min), with a final extension cycle (72°C, 10min).

PCR was also performed using oligonucleotides described by Howe et al. (1997) to amplify a 729 bp fragment of the *Toxoplasma gondii*-specific *SAG2* gene (Table 1). Reactions were carried out in a final

volume of 25 µL, containing 0.4 µM of each primer, 3 mM $MgCl_2$, 1× PCR buffer (5× Colorless, Promega®), 0.2 mM dNTPs, 1 U Taq DNA polymerase (Promega®), and 3 µL of template DNA. The reaction was carried out in a thermal cycler with an initial denaturation step (94°C, 5min), followed by 40 cycles of denaturation (95°C, 30sec), annealing (55°C, 30sec), and extension (72°C, 30sec), with a final extension cycle (72°C, 7min). A cell culture-isolated *T. gondii* strain from the Parasitic Diseases Laboratory at the Federal University of Santa Maria (UFSM) was used as a positive control, and a microtube containing DNase-free ultrapure water was used as a negative control.

Electrophoresis and Analysis of Results. The amplification products were separated on a 1.5% agarose gel (UltraPureTM LMP Agarose, Invitrogen®) under a current of 75 V (5 V/cm) for 45 minutes in TAE (Tris, acetate, and EDTA). They were then stained with ethidium bromide (0.5 µg/mL) and visualized using an ultraviolet light transilluminator (L-PIX Sti, Loccus). To determine the size of the amplified fragments, they were compared to a 100bp molecular weight standard (100 bp Ladder Plus, Ready-To-Use, M1071, Sinapse Inc®).

Genetic sequencing. The sequencing material was purified from 5 µL of the PCR product from the *18S rRNA* and *SAG2* gene-positive samples and treated with Exo-Sap-IT (GE Healthcare®) according to the manufacturer's protocol. The fragments were sequenced in both directions on an automated genetic analyzer (ABI 3500 Genetic Analyzer, Applied Biosystems®) at the Multiuser Molecular Biology Laboratory (PPGCV/ UFRuralRJ).

The amplified DNA from the *18S rRNA* gene showed 100% identity (Query coverage: 100%) in the BLASTn analysis with *T. gondii* sequences (accessions: KM875436, U03070, and XR_001974180). However, given the high conservation of primers across Sarcocystidae species, additional tests were performed using other molecular markers. Amplification targeting the *B1* and *SAG2* genes was successful. Due to the small size of the fragment generated for the *B1* gene, it was decided to sequence only the *SAG2* gene. BLASTn analysis of the SAG2 sequence obtained showed 100% identity (Query coverage: 100%) with the *T. gondii* isolate (accession: LN714498), confirming the agent's identification.

Discussion

The detection of *T. gondii* DNA by PCR in the blackbuck highlights the importance of this diagnostic method, especially for wild or captive animals. The molecular method has been widely used due to its high sensitivity and specificity. Ang et al. (2010) demonstrated that PCR targeting the SAG2 gene can achieve 100% specificity and 87.5% sensitivity for detecting *T. gondii*. In wildlife, PCR is essential, as shown by Morgado et al. (2017), who first detected *T. gondii* in a free-ranging giant anteater. These studies emphasize that healthy wild animals can harbor the parasite without being noticed unless sensitive molecular techniques are used.

Each diagnostic method has its strengths and limitations. Serological tests (e.g., IFAT, ELISA, MAT) are useful for epidemiological screening but only indicate previous exposure rather than active infection (Dubey and Jones, 2008; Sedlák and Bártová, 2006; Silva et al., 2025). Histopathology and

immunohistochemistry enable direct visualization of tachyzoites or tissue cysts (bradyzoites) and their correlation with lesions, aiding understanding of pathogenesis (Casartelli-Alves et al., 2014). However, these methods have limited sensitivity and may miss infections with low parasite loads or subtle lesions. In contrast, PCR can detect minimal amounts of *T. gondii* DNA, even in early stages or low-parasite-load samples, including those collected before seroconversion (Ang et al., 2010). In this case, few bradyzoite cysts were identified histologically, while PCR detected DNA in multiple samples, reinforcing the need for combined techniques.

Histopathological diagnosis confirmed a protozoal infection in the blackbuck, with severe interstitial pneumonia and necrohemorrhagic hepatitis. These histological features are typical of acute toxoplasmosis. The presence of bradyzoites in the lung and hepatic necrosis supports findings reported in wild animals (Dubey and Jones, 2008), and afterward, we confirmed this by molecular analysis. Similarly with the present report, a saiga antelope (*Saiga tatarica*) from Zoological Garden in Prague, Czech Republic, died suddenly from acute toxoplasmosis, with toxoplasmic pneumonia and necrotizing hepatitis considered the main causes of death (Sedlák et al., 2004). Due to the proliferation of type II pneumocytes, alveolar necrosis, and lung inflammation, we postulate that antelopes have a pattern of toxoplasmic interstitial pneumonia. Similarly, ring-tailed lemurs kept in a zoo in Italy died of acute fatal toxoplasmosis, presenting interstitial pneumonia and disseminated necrotic foci in the liver, spleen, and kidneys (Rocchigiani et al., 2022). Although toxoplasmosis in wildlife often goes unnoticed, the necropsies and cited studies reveal that it can cause pneumonia and necrotizing hepatitis, leading to acute death in vulnerable species, as was the case with the blackbuck. The mortality of rare antelopes due to toxoplasmosis can represent a significant loss for captive breeding programs.

On the one hand, neotropical monkeys with toxoplasmosis can present acute lesions of interstitial pneumonia, as in this blackbuck (Schiffler et al., 2023), or, if the diagnosis is made early, treated monkeys can develop a subacute clinical course (Moreira et al., 2022). On the other hand, the blackbuck (*Antilope cervicapra*) is an antelope, and it means that it is a ruminant species; the clinical picture differed from that of the small ruminant clinical presentation, compatible with abortions and lesions of brain malacia in the fetus (Caldeira et al., 2011; Pereira et al., 2021). From this perspective, female nilgais (*Boselaphus tragocamelus*), an Indian antelope, showed abortion or stillbirth and *T. gondii* DNA was demonstrated in the brain and liver of the fetus and neonates (Sedlák et al., 2004).

Zoo animals are highly susceptible to toxoplasmosis. Some may die acutely before brain lesions develop. In such settings, infection usually occurs via food or water contaminated with *T. gondii* oocysts. Clinical signs include pneumonia, myocarditis, hepatitis, and blindness; some animals die despite treatment (Dubey and Jones, 2008). Zoo animals are more exposed to *T. gondii* than to *N. caninum*, with seroprevalence reaching 42%, indicating environmental contamination (Sedlák & Bártová, 2006; Cano-Terriza et al., 2020). These findings underline the importance of prevention in zoos (e.g., controlling stray cats and ensuring hygienic feed) and surveillance for toxoplasmosis in vulnerable species.

Reports of diseases in captive wildlife are relatively scarce. Necropsy is an opportunity to understand threats to wildlife health. Epidemiological data are vital for accurate diagnosis and preventive strategies (Sharma et al., 2014). This case reinforces the importance of integrating histopathology with molecular diagnostics for a definitive diagnosis. Freitas et al. (2023) detected *T. gondii* DNA in tissues of slaughtered wild boars, underscoring the need for definitive methods to identify parasites in wild animals. Accurate diagnosis in captive or wild animals is essential for health management and conservation. Antemortem diagnosis is often more difficult in wild animals, underscoring the need for necropsy and postmortem testing.

Several studies have reported acute toxoplasmosis in zoo animals, including Cuvier's gazelles (Junge et al., 1992), nilgai antelopes and saiga (Sedlák et al., 2004), lemurs (Rocchigiani et al., 2022), and wallabies (Valenzuela-Moreno et al., 2022), often resulting in severe pneumonia, necrohemorrhagic hepatitis, abortions, and acute death. These cases underline the importance of post-mortem examination and molecular tools for definitive diagnosis, especially in species highly susceptible to *T. gondii*. Serological surveys have shown widespread exposure in zoo collections, with up to 42% of animals testing positive (Cano-Terriza et al., 2020). Preventive measures, including feral cat control, food hygiene, and environmental sanitation, are essential to reduce the risk of toxoplasmosis in captivity.

Based on a review of several scientific articles on the subject, we can conclude that *T. gondii* infection in antelopes and other zoo animals has a significant impact on individual health and species conservation. In captive wildlife populations, toxoplasmosis can lead to acute losses of valuable animals. In rare breeding antelopes, abortions and neonatal deaths due to toxoplasmosis represent losses beyond clinical concerns. However, a challenge remains: controlling *T. gondii* exposure in zoos, since oocysts shed by cats (domestic or visiting wild cats) can remain infectious in soil and water for months. Therefore, management strategies and preventive measures in zoos are recommended to minimize the effects of *T. gondii* infection. Accurate diagnosis of *T. gondii* in zoo animals helps mitigate risks, preserve animal health, and contribute to integrated conservation and One Health efforts.

Diagnosing toxoplasmosis in zoo hosts leads to discussion of the epidemiological scenario in Brazil, since regions reporting significant numbers of toxoplasmosis diagnoses in both wild and domestic carnivores are often those with greater diagnostic capacity and research activity. Veterinary diagnostic laboratories have contributed fundamentally to the diagnosis of animal diseases. Nevertheless, particularly regarding free-ranging and zoo animals, there are extensive areas of the country with limited investigative demand and scarce animal health data (Riet-Correa et al. 2025; Silva et al. 2025). One of the main characteristics of wildlife disease surveillance is the early detection of dead or sick animals and the identification of pathogens or diseases through necropsy and laboratory investigations. *Postmortem* examination is crucial for the investigation and specific diagnosis of *T. gondii* in zoo and wild animals, as it enables lesion detection and a complete tissue sampling to obtain a definitive diagnosis (Sharma et al., 2014; Silva et al. 2025).

This case report reinforces the importance of toxoplasmosis in the order Artiodactyla, family Bovidae, which includes 143 species. Toxoplasmosis has been reported in captive Asian and African antelopes: a fatal case in a Cuvier's gazelle, an outbreak in nilgai (an Indian antelope), and acute toxoplasmosis in saigas (a rare Eurasian antelope). Serological surveys have also indicated exposure to *Toxoplasma gondii* in blackbuck (*Antilope cervicapra*) from European zoos. (Junge et al., 1992; Sedlák et al., 2004; Sedlák et al., 2006).

Conclusion

The identification of *Toxoplasma gondii* bradyzoite cysts in the lungs, associated with histological lesions, PCR detection, and sequence analysis, confirmed fatal toxoplasmosis in a blackbuck (*Antilope cervicapra*). These findings support the inclusion of the blackbuck as a susceptible host to *T. gondii* infection and emphasize the importance of integrating clinical, pathological, molecular, and epidemiological data to improve the understanding of toxoplasmosis in zoo and wildlife species.

Declarations

Author Contribution

Andressa Silva, Asheley Pereira, Daniel Ubiali: Conception. Andressa Silva: Wrote the main manuscript text. Arthur Alves, Bruna Barbosa: clinical assessment. Asheley Pereira, Fernanda Rocha, Paula Retamero, Daniel Ubiali: pathological and histopathological assessment. Jônathan Chagas, Matheus Cordeiro, Bruna Baêta: molecular assessment. Daniel Ubiali: Project administration.

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Figures

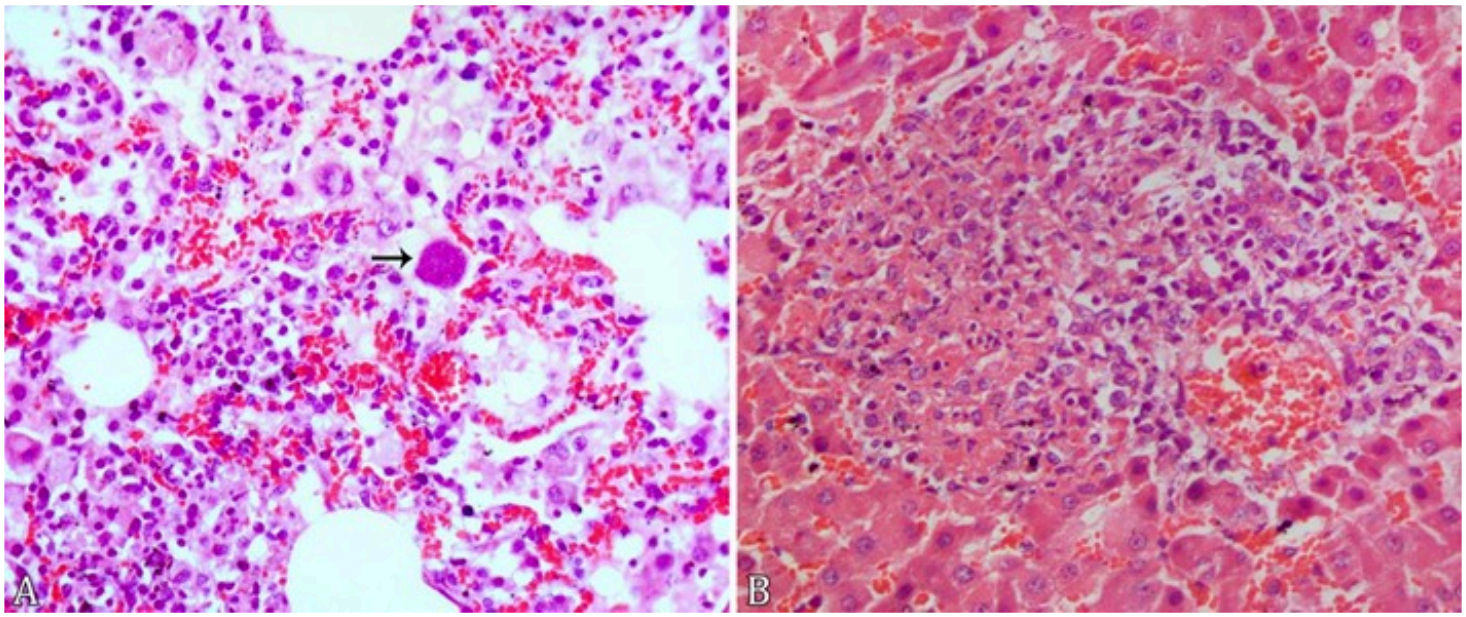


Figure 1

Histopathological findings in *Antilope cervicapra*. (A) Necrohemorrhagic and fibrinous interstitial pneumonia. A *Toxoplasma gondii* cyst containing bradyzoites (arrow) is present within the alveolar septum. (HE, obj. 20x). (B) Focus of hepatocytes with pyknotic nuclei interspersed with lymphocytes, histiocytes, fibrin, and extravascular red blood cells (HE, obj. 40x).