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Case Report

Toxoplasma encephilitis in a Collared Peccary (*Dicotyles tajacu*): clinical and pathological findings

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Abstract
Toxoplasmosis is a widespread zoonosis affecting numerous mammalian species. Wild suoids have been confirmed as susceptible hosts of *Toxoplasma* spp., and their meat is frequently consumed by hunters, their families, and domestic dogs, posing a growing foodborne risk in South America. We conducted a pathological study on a young captive Collared Peccary that showed neurological signs. Severe brain lesions were observed, including parenchymal necrosis with demyelination, gliosis, meningitis, and perivascular cuffing. *Toxoplasma* cysts containing tachyzoites, as well as free tachyzoites, were identified within these lesions. Additionally, focal necrosis with inflammatory infiltration was noted in the liver and heart, along with marked pulmonary edema. Immunohistochemistry for canine distemper virus in the brain was negative. To the best of our knowledge, this is the first report detailing the pathological findings of *T. gondii* infection in a wild suoid (*Dicotyles tajacu*).

Keywords: Encephalitis, Pathology, Peccary, *Toxoplasma gondii*.

Introduction

Consumption of meat from *Sus scrofa* (a globally invasive wild suoid species) and native American wild suoids (peccaries) has become increasingly common in several countries, including those in South America (19). Their meat is typically consumed by hunters and their close social circles, and offal is often used to feed hunting dogs. Given the known association of foodborne diseases -such as toxoplasmosis- with wild suoid meat, assessing the incidence of these infections is crucial for evaluating the risks posed to humans, wildlife, and domestic animals (19).

Understanding *Toxoplasma* infections in both introduced and native wild suoid species is therefore essential. Toxoplasmosis in domestic pigs has been studied extensively since the 1950s (7, 8), and more recent research has focused on the molecular mechanisms underlying brain pathology caused by the parasite (11, 22).

Toxoplasma seroprevalence in European wild boars has been reported to range from 9% to 62%, depending on the region (1, 3, 6, 10, 17, 18). In South America, infection rates have been reported as high as 76.9% (19). Although numerous histological studies have described the effects of *Toxoplasma* in pigs (4, 5, 12, 14, 15, 16, 22, 23), reports detailing pathological findings in suoids, particularly peccaries, remain scarce.

The aim of this study was to characterize the pathological changes associated with *Toxoplama* infection of a captive juvenile Collared Peccary presenting with severe neurological signs.

Case description

The subject was a male captive Collared peccary (*Dicotyles tajacu*), less than one-year-old, referred from the Montevideo City Zoological System (Sistema Departamental

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Zoológico, Parque Lecocq, IMM, Montevideo, Uruguay). The animal exhibited lethargy, lateral recumbency, tremors and strabismus; due to the severity of which, euthanasia was performed using an intravenous overdose of xylazine and thiopental. The animal was submitted to the Diagnostic Pathology Laboratory, Faculty of Veterinary, Universidad de la República, Montevideo, for anatomopathological examination. Tissue samples from the brain, lungs, liver, and heart were fixed in 10% neutral buffered formalin, routinely processed, sectioned at 4 µm, stained with hematoxylin and eosin (H&E), and examined microscopically. Periodic acid-Schiff (PAS) staining was performed for special staining. Histopathological evaluations were conducted independently by six veterinary pathologists. Immunohistochemical (IHC) studies were performed on paraffin-embedded sections using previously described protocol (9). A rabbit polyclonal antibody specific for *T. gondii* (Polo Tecnológico de Pando, Udelar & DILAVE-MGAP, Uruguay) was applied overnight at 4°C, followed by the MACH 4 Universal HRP-polymer detection system (Biocare Medical, CA, USA). Additional IHC studies for canine distemper virus (CDV) were performed using a mouse anti-CDV monoclonal antibody (BIO-RAD, MCA 1893, 1:250 dilution), incubated overnight and detected with a mouse-on-canine HRP-polymer (Biocare Medical,CA, USA). GFAP (glial fibrillary acidic protein) immunostaining (PM 065 AA, RTU, Biocare Medical, CA, USA) was used to characterize astrocytic gliosis. Positive antigen-antibody reactions were observed by incubation with 3.3'-diaminobenzidine-tetrahidrochloride (DAB). All slides were counterstained with Mayer's hematoxylin.

Grossly, the lungs appeared dark red throughout all lobes. No significant macroscopic changes were noted in other organs.

Microscopically, severe lesions were observed in the brain, primarily in the cerebrum and cerebellum. These included parenchymal necrosis with demyelination, gliosis, meningitis, and perivascular cuffing. Necrosis and demyelination were distributed throughout both cortical and subcortical areas, with higher frequency in the cortex (Figs. 1 and 2). The cerebellum showed cortical degeneration, extending into the subcortical white matter, including Purkinje cell loss (Fig. 3). Necrotic changes with demyelination were progressed with infiltration of lymphocytes, plasma cells, Gitter cells and eosinophils accompanied with gliosis around the lesions. Gliosis, composed predominantly of proliferating astrocytes, was confirmed via GFAP-positive immunostaining. Reactive astrogliosis was indicated by positive GFAP immunostaining in the cytoplasm and cytoplasmic processes of reactive astrocytes (Fig. 4). Numerous *Toxoplasma* cysts containing tachyzoites were identified in and around necrotic foci. Cyst diameters ranged from 10.2 to 27.8 µm and were PAS-positive (Fig. 5). In some areas, ruptured cysts released tachyzoites into the parenchyma. The released tachyzoites were also found inside the cytoplasm of surrounding mononuclear cells of the necrotic areas. Abundant lymphocytes,

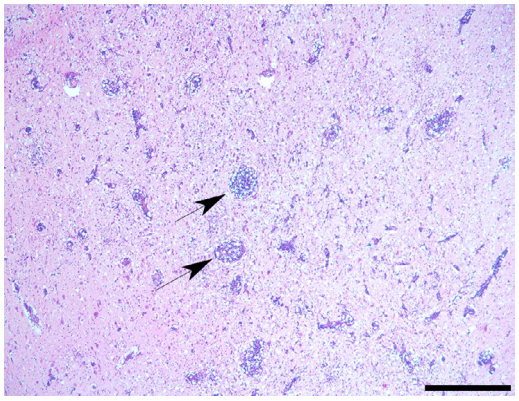

Figure 1. Parenchymal necrosis with demyelination is distributed throughout the white matter. Prominent perivascular lymphoplasmacytic cuffings are visible (arrows). Cerebrum. H&E stain. Original magnification 4×. Scale = 500 µm.

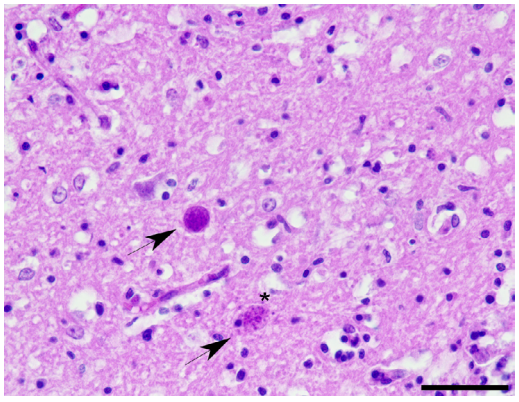

Figure 2. High magnification of Figure 1. *Toxoplasma* cysts are present (arrows); one cyst is ruptured with released tachyzoites (asterisk). Cerebrum. H&E stain. Original magnification 40×. Scale = 50 µm.

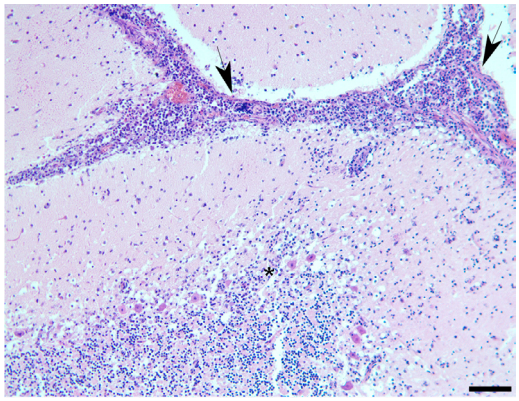

Figure 3. Severe meningitis (arrows) and degenerative changes (asterisk) extend throughout the cortex and into the subcortical white matter, with Purkinje cell degeneration and loss. Cerebellum. H&E stain. Original magnification 10×. Scale = 100 µm.

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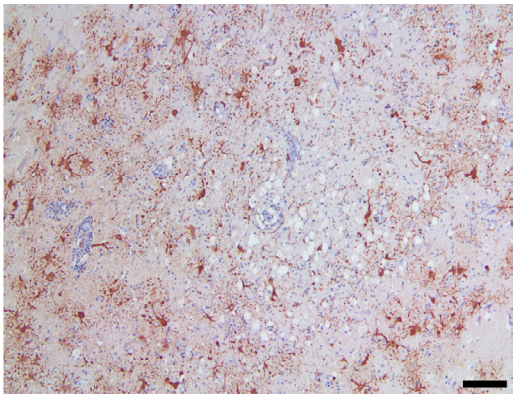

Figure 4. Reactive astrogliosis demonstrated by GFAP immunostaining in the cerebral cortex. Original magnification 10×. Scale = 100 µm.

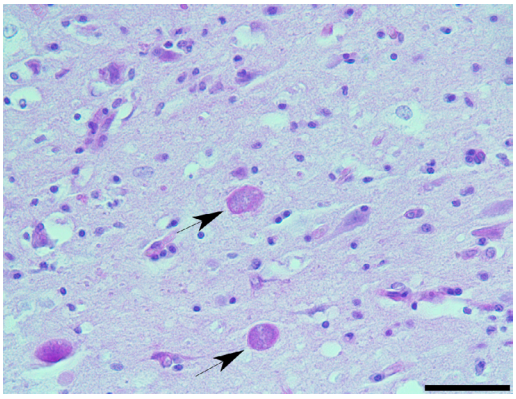

Figure 5. *Toxoplasma* cysts showing positive PAS staining (arrows). Cerebrum. PAS stain. Original magnification 40×. Scale = 50 µm.

plasma cells, and macrophages infiltrate the leptomeninges and surround their blood vessels in both the brain and cerebellum. Eosinophils were rare. Perivascular cuffings, mainly composed by lymphocytes and plasma cells, were one of the characteristic changes in this animal. Immunohistochemically, the cysts containing tachyzoites of *Toxoplasma gondii* and tachyzoites were immunopositive as fine brownish granules (Fig. 6). In addition, immunohistochemistry for CDV was negative in glial and nerve cells or in neuronal perikarya and their processes (axon and dendrites).

Although focal necrosis accompanied with mononuclear cell infiltration were observed in the liver, heart and lung, these were always mild inflammatory changes, characterized by lymphoplasmacytic infiltration, and did not spread to the surroundings, confirming that inflammatory changes in toxoplasmosis is not an important histological feature in these parenchyma. Severe hyperemia, congestion, and edema were also observed in the lungs. No significant changes were observed in the kidney. No *Toxoplasma* cysts and tachyzoites were detected by PAS staining and immunohistochemistry examination in the lung, heart, liver and kidney.

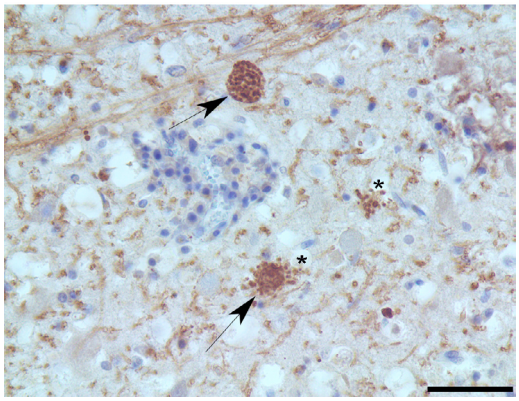

Figure 6. *Toxoplasma gondii* cysts (arrows) and released tachyzoites (asterisks) exhibit positive immunohistochemical staining in the cerebral cortex. Original magnification 40×. Scale = 50 µm.

Discussion

Although several studies reported *Toxoplasma* infection rate due to serum antibodies in wild suoids (1, 3, 6, 10, 17, 18, 19), no previous reports on pathological changes in adult peccaries infected with *Toxoplama* were found in scientific databases. In contrast, pathological changes in domestic pigs affected with this disorder have been reported since the 1950s, and the lesions were observed in multiple organs such as lung, liver, spleen, lymph nodes, heart, muscles and brain (5, 8, 14, 15, 16, 20). Furthermore, the inflammatory lesions in brain, heart, liver, lung and muscles were observed two weeks after inoculation of *Toxoplasma gondii* in an experimental study in piglets (4). It was also apparent in our study that *Toxoplasma* infection caused more severe and chronical changes in the brain than in other organs. Focal necrosis with mild mononuclear cellular infiltration was observed in the liver, heart and lung, but *Toxoplasma* was not detected in these organs. Progressive changes in the liver, heart and/or lung have been reported to be one of the characteristic lesions of *Toxoplasma* infection in pigs (4, 12, 22, 23), but no severe changes were observed in this study. In addition, it was a new finding that immunohistochemical examination revealed that the gliosis was primarily astrocytic in nature, as evidenced by GFAP staining. The association between demyelination and astrocytic gliosis in *Toxoplasma* infections remains an area for future research. The distribution of lesions, particularly their association with parenchymal and meningeal blood vessels, suggests hematogenous spread of the parasite into the central nervous system. The extension of cortical inflammation into subcortical white matter further supports this mechanism. Furthermore, the progressive lesions from the gray matter to the white matter in contact with severe meningitis was thought to be due to the infiltration of *Toxoplasma* from blood vessels in the meninges and formed these lesions.

The observed brain lesions -parenchymal necrosis, demyelination, gliosis, meningitis, and perivascular

cuffing- are reminiscent of changes associated with canine distemper virus infection. CDV has been reported in wild suoids, and its lesions overlap significantly with those of *T. gondii* (2, 13, 21). Histological examination in brain of dogs infected with CDV also revealed predominantly neurons with intracytoplasmic inclusions, parenchymal necrosis, perivascular cuffing, and gliosis (2). Although no inclusion bodies were seen in our case, many of the changes were resembled to the brain lesions of CDV infection. CDV infections are common in wild suoids and there is a possibility of co-infection of *Toxoplasama* and CDV, so the immunohistochemical examination for CDV may be necessary if brain changes are observed. However, in this case, CDV was excluded by immunohistochemistry, ruling out co-infection.

In conclusion, we present the first pathological description of *T. gondii* encephalitis in a Collared peccary. The findings highlight severe changes in the brain (parenchymal necrosis with demyelination, gliosis, meningitis and perivascular cuffing). *Toxoplasma gondii* were detected in the brain by histological and immunohistochemical examinations. Immunohistochemical staining did not detected CDV. Focal necrosis of the liver, heart and lung, and severe edema and congestion in the lung were also observed. These results underscore the importance of including *T. gondii* in differential diagnoses of neurological disease in wild suoids.

Conflict of Interest

The authors declared no conflicts of interest with related to the authorship and/or publication of this article.

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