Cryptosporidium andersoni Associated Proliferative Abomasitis in a Roan Antelope (*Hippotragus equinus*)

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INTRODUCTION

Cryptosporidium spp. are zoonotic, intracellular and extracytoplasmic, apicomplexan parasites; infections are an important cause of potentially life-threatening diarrhoea in a wide variety of vertebrate species worldwide (1). Though many cryptosporidia have a tropism for small intestinal enterocytes, *C. andersoni*, in addition to *C. muris* and *C. serpentis*, preferentially infect abomasal and gastric epithelia. Abomasal and gastric infections with *C. andersoni* have, to date, mainly been described in cattle and camels, with occasional reports in humans (2).

In species other than cattle, clinical findings, as well as the gross and histologic lesions associated with *C. andersoni* have not been described.

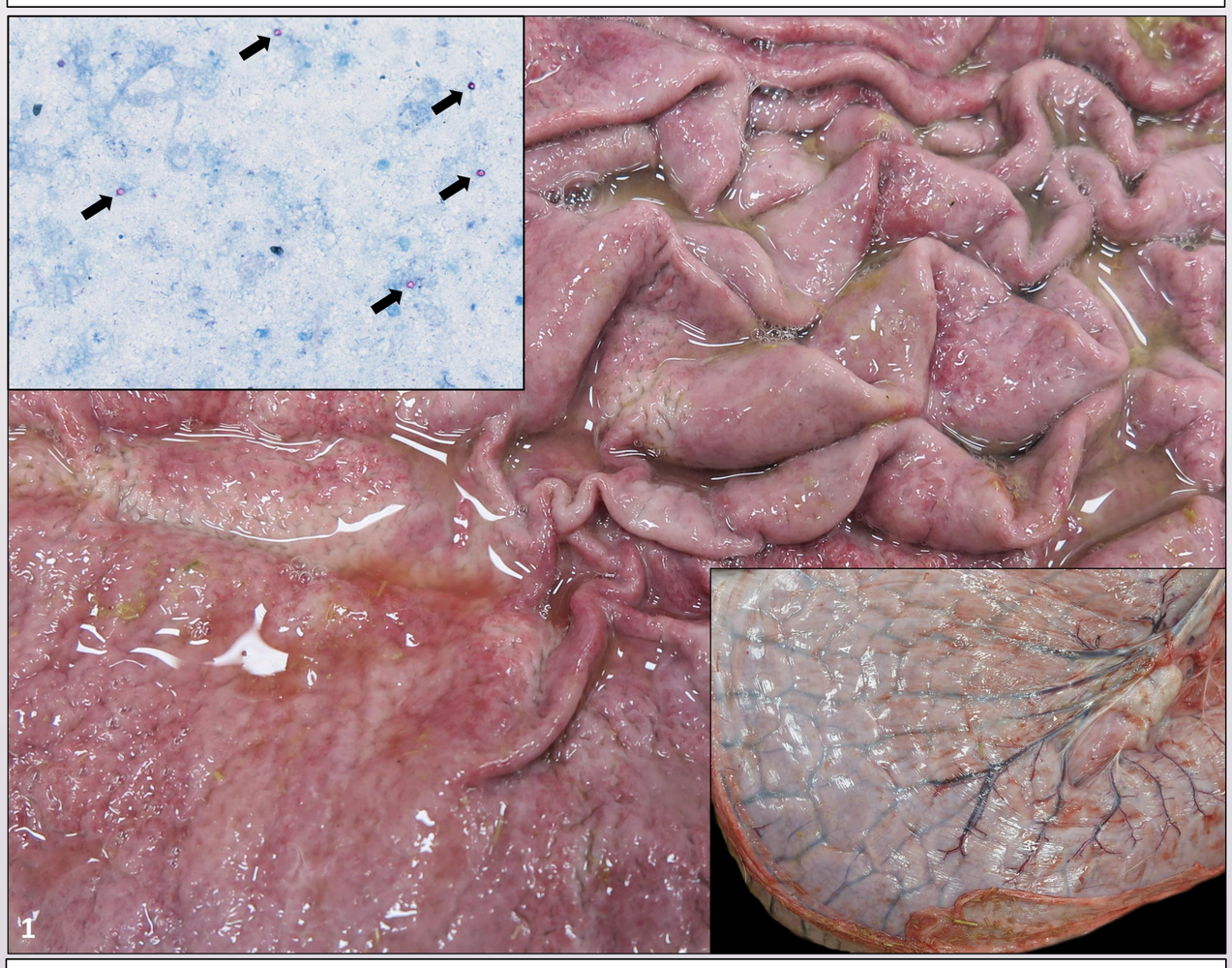

Fig. 1. Abomasal cryptosporidiosis, abomasum, roan antelope.

The abomasal mucosal folds are diffusely thickened. The mucosa itself has a cobblestoned texture. **Upper Inset:** Abomasal contents smear containing 6 to 8 µm diameter, acid-fast positive structures that are consistent with *Cryptosporidium* spp. oocysts. Modified Ziehl-Neelsen. **Lower Inset:** The serosa of the abomasum is swollen and glistening with prominent, engorged blood vessels bridging across the surface.

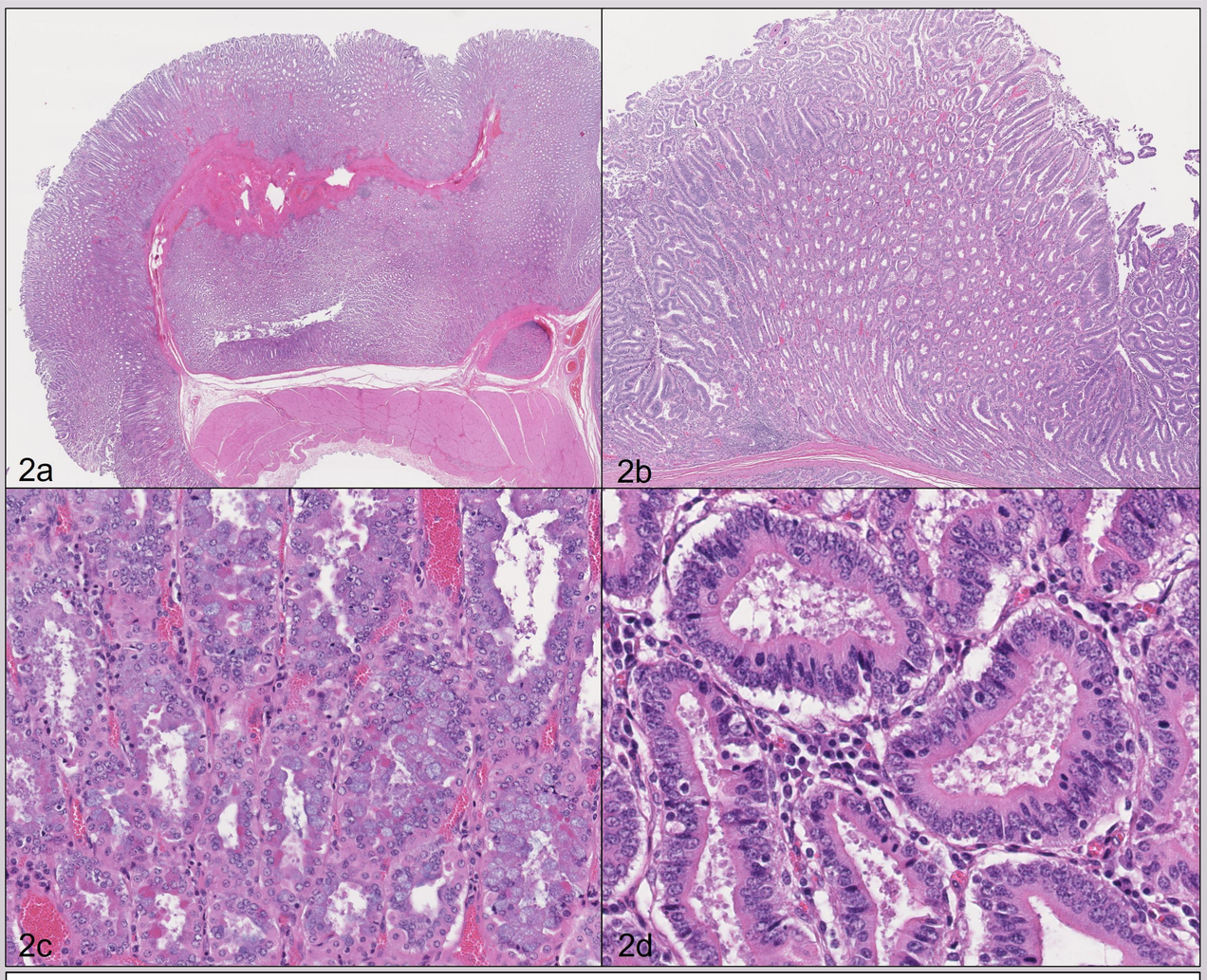

Fig. 2. Abomasal cryptosporidiosis, abomasum, roan antelope.

(a) Abomasal fold demonstrating diffuse mucosal thickening. HE. **(b)** Abomasal glands are densely packed with elongation of the fovelae, neck, and body. Occasional ciliates are present near the abomasal pits. The superficial laminal propria is mildly oedematous. HE. **(c)** Hyperplastic glands include a mixture of mucous cells and chief cells, the latter of which have prominent, apical, eosinophilic granules. HE. **(d)** Abundant 6 to 8 µm diameter structures line the apical epithelia and fill the glands. Lymphocytes and plasma cells multifocally expand the interstitium. HE.

ACKNOWLEDGEMENTS

Picture credit to Asha Stone (University of Surrey undergraduate) for the gross images in Fig. 1. Special gratitude to all of the members of the University of Surrey Veterinary Pathology Centre, in particular the histology lab and Ella May for performing the ZN staining of the abomasal contents.

CASE SUMMARY

History and Signalment: A 2-year-old, male-intact, zoo-housed, roan antelope was submitted for *post-mortem* examination after 5-months of diarrhoea and weight loss that was unresponsive to anthelminthics (ivermectin) and antibiotics (TMPS).

Gross Findings: Poor body condition with reduced adipose stores and skeletal muscle mass was confirmed. The serosa of the abomasum was traversed with congested, prominent blood vessels, imparting a cerebriform pattern to the surface (**Fig. 1 lower inset**). The abomasal mucosa was diffusely thickened (up to 3 mm) with a notably corrugated surface, consistent with a proliferative abomasitis (**Fig. 1**). Watery faecal contents (diarrhoea) were present in the spiral and descending colons and the mesenteric lymph nodes were firm and mildly enlarged.

Microscopic Findings: Ziehl-Neelsen staining of abomasal content smears highlighted abundant 6 to 8 µm diameter, acid-fast positive structures (**Fig. 1 upper inset**). Histologically, abomasal glands were diffusely and markedly elongated, affecting both the foveolae (pits) and the body of the glands (**Fig. 2a,b**). The epithelial cells lining the elongated abomasal glands were densely packed, occasionally jumbled and included a mixture of mucous cells and chief cells, the latter of which were characterized by their prominent apical, eosinophilic granules (**Fig. 2c**). Lining the apical surface of the abomasal epithelium, extending from the base of the glands to the pits were numerous 6 to 8 µm diameter, lightly basophilic structures (**Fig. 2d**). The gastric lamina propria was multifocally mildly to moderately expanded with lymphocytes and plasma cells and occasional intraepithelial lymphocytes were scattered throughout the mucosa. The foveolar lamina propria was mildly oedematous **(Fig. 2b).**

Ancillary Diagnostics: Genomic DNA was extracted from abomasal and intestinal contents and subject to PCR using primers specific for the 18S rRNA gene of Cryptosporidium spp., followed by Sanger sequencing. All samples tested were positive by 18S PCR and sequences were most closely homologous to *Cryptosporidium andersoni*. Faecal worm egg and coccidial oocyst counts were below the limit of detection (<50 oocysts or eggs per gram).

DISCUSSION

This is the first report of the gross and histologic lesions associated with *C. andersoni* infection in a roan antelope. Gross and histolomorphologic features of *C. andersoni* infections have been well documented in cattle with notable histologic similarities to those documented in this case, including the association of the organisms with abomasal pit elongation, epithelial hyperplasia, and lamina propria infiltration with lymphocytes and plasma cells (1). Bovine cases of *C. andersoni* typically present as chronic infections of juvenile to adult animals but have not been definitively linked to overt clinical disease nor consistent gross lesions, though association with reduction in milk production in dairy cattle has been reported (3,4). This is in contrast to the current case, where loss of body condition and intractable diarrhoea was significant enough to be the impetus for euthanasia and thickening of the abomasal mucosa was grossly apparent.

The diarrhoea in this case was attributed to the abomasal changes and speculatively, the pathophysiology may include a combination of maldigestion (osmotic diarrhoea) and increased permeability of the abomasal barrier. In cattle, *C. andersoni* clinicopathologically has been associated with elevated abomasal pH (4.5 to 5), and elevated plasma pepsinogen, similar to ostertagiasis (4). If similar pH changes occur in hippotraginids, decreased active pepsin and gastrin could have led to impaired digestive function of the abomasum and the increased pH may have allowed for overgrowth of other organisms (e.g. ciliates, **Fig. 2b**). Both alterations in the abomasal microbiota with resultant dysbiosis of the more distal intestinal tract and the passage of undigested feed into the lower intestinal tract could have contributed to diarrhoea. Additionally, compromised intercellular junctions in the hyperplastic and inflamed mucosa may have led to the movement of serum proteins into the abomasal lumen (5), though the exact role this feature may play in the pathophysiology of diarrhoea remains elusive.

Rare reports of unspeciated cryptosporidosis in captive hippotraginids have been documented, however the susceptibility of these animals to clinical disease remains unclear (6). Further research into the prevalence of infection with *C. andersoni* in zoo-housed roan antelope and the association of this organism with clinical disease is warranted. Based on this case, zookeepers and veterinarians should consider this pathogen in cases of diarrhoea in this species. This agent is a zoonotic pathogen and early diagnosis of animals in captivity may help to prevent human cases, especially in immunocompromised patients.

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