

# Diagnostic Exercise

## From The Davis-Thompson Foundation\*

Answer Sheet

Case: **174**; Month: **October**; Year: **2021**

**Title:** *Clostridial enterocolitis in an emu (Dromaius novaehollandiae)*

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**Clinical History:** A 25-year-old female emu (*Dromaius novaehollandiae*) presented with a 1-week history of lethargy, hyporexia, and recumbency. The bird had lived its entire life at the Winnie Carter Wildlife Center in Texas and had an unremarkable medical history, apart from bilateral congenital blindness.

**Necropsy Findings:** Within the coelomic cavity, the serosal surface of the intestines was coated with a moderate amount of tan to green, mucoid, opaque, foul-smelling fluid (fibrinoheterophilic coelomitis) (Fig. 1).



Figure 1:

The colon is markedly distended and covered with multifocal strands of fibrin. The large intestine was diffusely dilated and filled with dark red fluid and strands of yellow, friable material (fibrin). The mucosa of the small intestine, and most

severely the colon, was diffusely roughened, thickened, dull, and covered by a thick yellow to green, opaque, friable diphtheritic membrane (Fig. 2). The distal colon was diffusely, markedly distended and focally contained a 50x7x5 cm, semi-firm accumulation of feces coated by a 4-5 mm diameter layer of tan, friable material (fibrin) located 21 cm from the opening of the cloaca. Gross evaluation of the eyes revealed bilateral aphakia.



Figure 2: A thick diphtheritic membrane composed of fibrin and necrotic debris coats the small intestinal mucosa.

**Differential Diagnoses:** *Clostridioides difficile*, *Clostridium perfringens*, *Salmonella* spp., *Escherichia coli*, *Enterococcus* spp.

**Histologic findings:**

The majority of the small and large intestinal mucosa was effaced with replacement by a thick, diphtheritic membrane composed of abundant fibrin, necrotic cellular debris, and small numbers of heterophils (Fig. 3). Densely packed colonies of 1x2  $\mu$ m, gram-positive bacilli coated the surface of the necrotic membrane (Figs. 4 and 5).



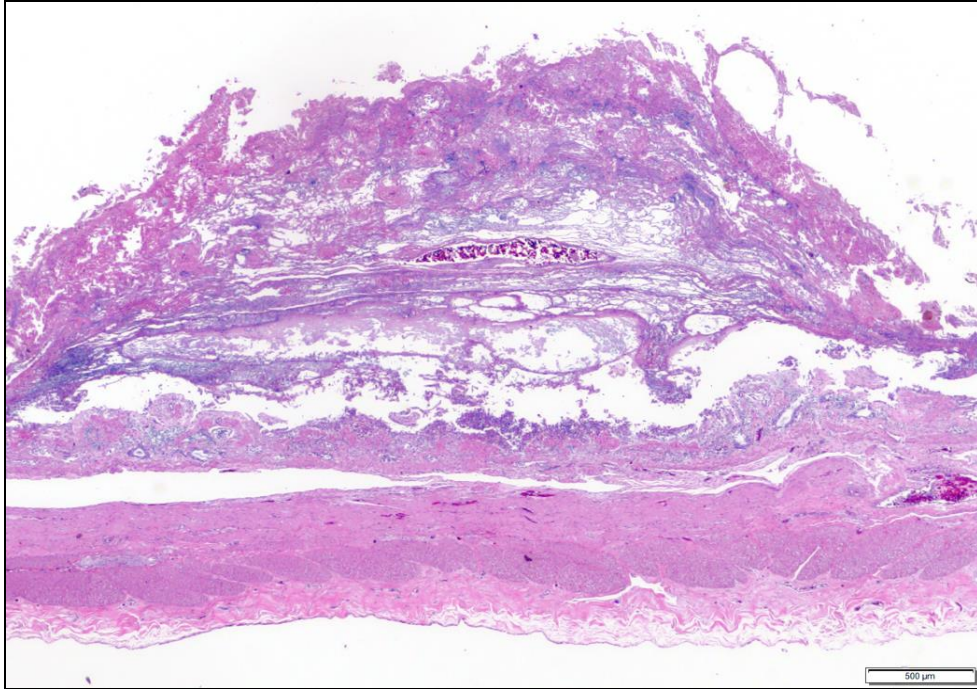


Figure 3: The small intestinal mucosa is effaced by a thick membrane composed of necrotic cellular debris and fibrin.

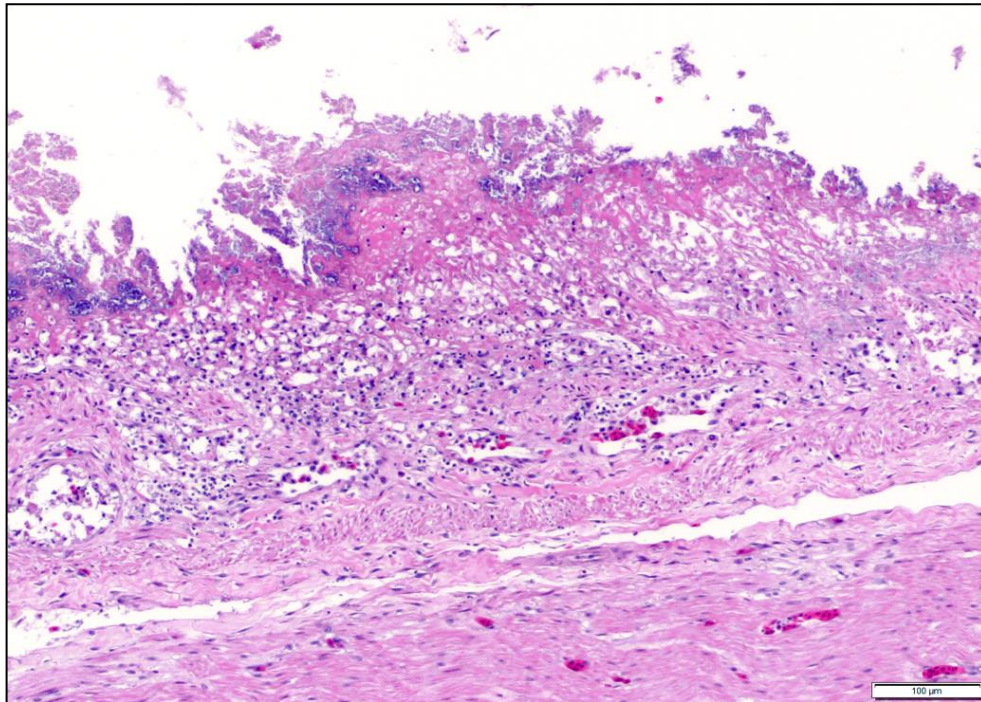


Figure 4: Large colonies of 1x2 μm bacilli coat the surface of the mucosa of the intestine.

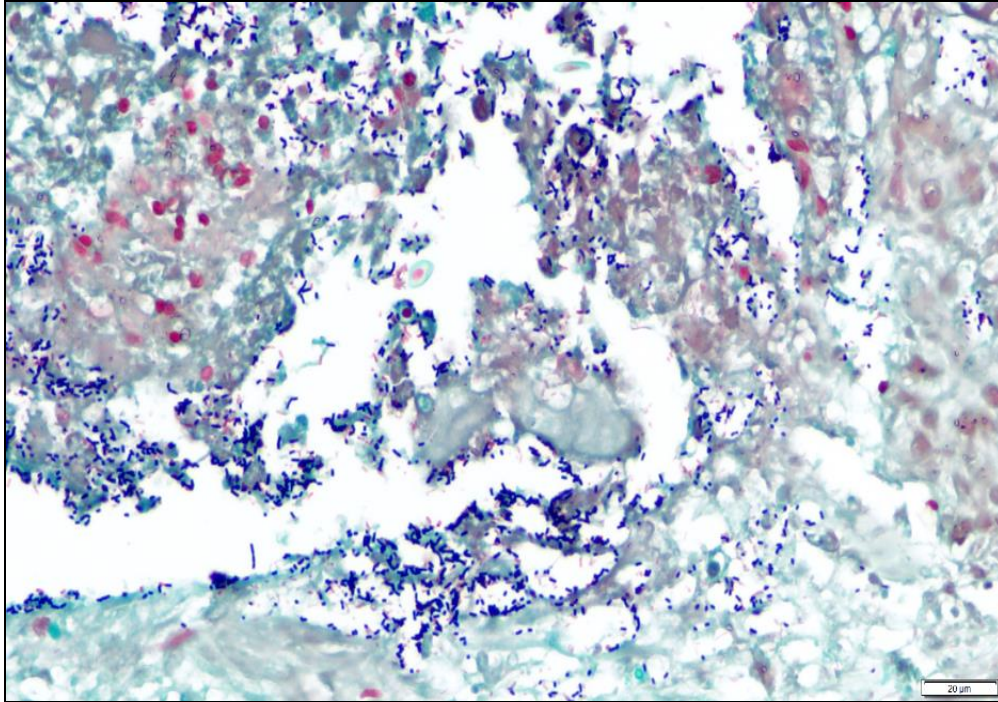


Figure 5: Gram-positive bacilli on the mucosal surface of the intestine.

**Morphologic diagnosis:** Marked, diffuse, acute, fibrinonecrotizing enterocolitis with myriad bacterial cocci.

**Ancillary test results:**

- PCR for toxins A and B of *Clostridioides difficile* on the small intestinal contents was positive
- PCR for *Salmonella* spp. on intestinal contents was negative
- Gram stain: numerous gram-positive bacilli coating the mucosa

**Etiology:** *Clostridioides difficile*

**Comments:**

*Clostridioides difficile* is a gram-positive, anaerobic bacillus that can cause colitis-associated diarrhea in humans and many other mammals including hamsters, guinea pigs, rabbits, horses, and neonatal pigs (Keel and Songer,, 2006, Uzal et al., 2016). Gastrointestinal lesions associated with a *C. difficile* infection can vary greatly in location and severity depending on the species, age, bacterial spore presence, and the individual animal response to disease. In general, necropsy findings include an aborally distributed colitis, typhlitis, and occasionally ileitis (Keel and Songer, 2006). Lesions in hamsters and guinea pigs most commonly include a hemorrhagic typhlitis that range from petechiation to diffuse hemorrhagic lesions in the cecum, colon, and ileum (Bartlet et al., 1978, Lusk et al., 1978, Lowe et al., 1980). Several reports describe chronic typhlitis associated with mucosal hyperplasia in addition to shivajonesRohwer, 1991, Godec, 2004, Ryden et al., 1991). Infections in the pig typically lead to a mild typhlocolitis that can sometimes

progress to transmural necrosis with microscopic, fibrin and neutrophil-secreting ulcers known as "volcano ulcers." The cecum and colon are often distended by edema. Pigs can clinically present with diarrhea, obstipation, or dyspnea associated with ascites or hydrothorax (Waters et al., 1998). Severe cases in adult horses can cause a hemorrhagic necrotizing typhlocolitis (Gustafsson et al., 1997, Perrin et al., 1999) while in foals and rabbits, lesions are typically seen in the small intestine (Jones et al., 1988, Magdesian et al., 2002, Perkins et al., 1995).

Various risk factors for development of *C. difficile* infection in animals have been identified, and include antimicrobial treatment, change in diet, and environmental stressors such as transportation (Kelly and LaMont, 1998). No known changes in this bird's routine occurred prior to development of enterocolitis and so the predisposing factors in this case are uncertain.

Although *C. difficile* infections are less common in avian species, there have been several reports of this disease in captive ostriches (Frazier et al., 1993) in addition to *C. difficile*-associated hepatitis in ostrich chicks (Shivaprasad 2003). It has also been isolated from the intestine of ostriches, rheas, and emus (Terio et al., 2018) and should be considered as a differential diagnosis when ratites present with enterocolitis.

While an abundance of clostridial growth on culture of the intestine allows for a presumptive diagnosis, ELISA or PCR on intestinal contents in order to detect clostridial toxins A and B (Sullivan et al., 1982, Post et al., 2002) is recommended for definitive diagnosis, as in this case.

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