
Leslie M. East, DVM; Catherine J. Savage, BVSc, MS, PhD; Josie L. Traub-Dargatz, DVM, MS; Charles E. Dickinson, DVM, MS; Charles A. Kuntz, DVM, PhD; and Robert P. Ellis, PhD

Clostridium perfringens was a common gastrointestinal pathogen in foals younger than 10 days of age presenting with clinical signs of acute onset of colic, diarrhea, or obtunded mentation. Foals diagnosed with C. perfringens enterocolitis associated disease had an overall mortality rate of 53%. Because of the rapid and fatal progression of this disease, early recognition, treatment, and prevention are quintessential. Authors’ address: Depts. of Microbiology (Ellis) and Clinical Sciences (all other authors), Colorado State University, College of Veterinary Medicine and Biomedical Sciences, 300 West Drake, Fort Collins, CO 80523. © 1997 AAEP.

1. Introduction
Over the past several years, Clostridium perfringens has been recognized as a gastrointestinal pathogen in foals younger than 10 days of age.1–7 Common presenting clinical signs include acute colic, obtunded mentation, and hemorrhagic or nonhemorrhagic diarrhea. Many foals either die or require euthanasia early in the course of this disease because of the rapid progression and gastrointestinal deterioration.1–7 A 10-year retrospective study of foals presenting to Colorado State University Veterinary Teaching Hospital (CSU/VTH) was performed to identify common presenting clinical signs, physical and laboratory abnormalities, and medical treatments given, as well as the discharge status and in some the necropsy findings. The goal of this retrospective study was to improve early recognition, assess efficacy of treatment, and develop preventative methods to improve the outcome of this disease.

2. Materials and Methods
A retrospective study on 47 foals younger than 10 days of age presenting to CSU/VTH from 1988 to 1997 was performed. A search of the diagnostic laboratory bacterial culture results as well as a log of all neonatal foals entering the hospital was used to identify cases. Case selection for inclusion into the study included positive anaerobic fecal or gastrointestinal culture for C. perfringens as well as clinical signs or necropsy findings consistent with this gastrointestinal disease.
3. Results

Most of the foals' dams were multiparous and had a normal parturition. The majority of foals were >330 days in gestation, ≤5 days of age, and ill for less than 24 h at presentation. Multiple breeds were represented.

Reasons for hospitalization of the foals included an acute history of obtunded mentation, colic, nonhemorrhagic diarrhea, or hemorrhagic diarrhea. Physical examination findings at admission revealed that most foals were obtunded, normothermic, tachypneic, and had a normal heart rate. Many foals were clinically dehydrated >5%, not suckling, had signs of colic with or without abdominal distention, and had hemorrhagic diarrhea.

Hematologic abnormalities of leukopenia, neutropenia, moderately toxic cells—Doehle bodies, and fibrinogen >450 mg/dl were found in greater than one third of the foals; however, band neutrophils >200/µl were found in over half of the patients on admission. Approximately 50% of the foals had normal hematologic parameters when hospitalized; however, following hospitalization the majority of foals eventually became leukopenic, neutropenic, had band neutrophils >200/µl, and had hypoproteinemia. Most foals had minimal biochemical abnormalities and an IgG >800 mg/dl at admission.

Additional diagnostic tests such as abdominocentesis and abdominal radiographs were performed on one quarter of the foals. The majority of foals had peritonitis based on fluid analysis as well as gas- and fluid-distended small and large intestine on radiographs. All of the foals on which a necropsy was performed had a fibrinonecrotic enterocolitis, with less than one third having concurrent mural emphysema.

Most of the blood cultures and fecal rotavirus tests were negative. The most common bacterial species isolated with C. perfringens were Clostridium difficile (24%) and Escherichia coli (24%), with Salmonella spp. only occurring rarely. Polymerase chain reaction (PCR) typing of three C. perfringens isolates from 1997 revealed both type C (3/6) and type A (4/6).

The majority of foals were treated with antimicrobials, which included penicillin often in combination with another antimicrobial. Gastric ulcer medications, including H₂ receptor antagonists and sucralfate, were frequently given. Most foals received intravenous fluid support, with approximately half receiving intravenous plasma. Very few foals received C. perfringens type C and D anti-toxins (5/47) or antiolestridial—antirotaviral plasma (4/47) intravenously or by mouth. Nonsteroidal anti-inflammatory drugs and analgesics were required to control abdominal pain in approximately half of the foals. Despite the above outlined intensive medical care, less than half of the foals survived (47%). The remaining 53% were either euthanized or died while hospitalized.

4. Discussion

Our study confirmed previous reports¹–⁷ that C. perfringens associated enterocolitis frequently resulted in a rapidly progressive disease with a fatal outcome, despite intensive care measures. Therefore, it is important that early recognition of this disease occur so that appropriate treatment and farm preventative measures may be instituted without delay.

In order to detect this disease rapidly, fecal cytology may show large numbers of spores or large gram-positive rods,⁸ which occurred in 3/4 foals. The definitive diagnosis requires anaerobic along with an aerobic fecal culture. Most foals in our hospital have had a heavy growth of C. perfringens in the feces.

Most foals have adequate serum IgG levels, >800 mg/dl, indicating adequate passive transfer. This supports the theory that the presence of trypsin inhibitor in the dam's colostrum, which protects immunoglobins from gastrointestinal breakdown, may potentially allow C. perfringens' beta toxins to persist and these bacteria to overgrow.⁷,⁹

The medical management of these foals for the most part is similar to that for septic foals; however, because milk overload in the intestine is suspected as a predisposing factor to this disease, foals are often not allowed to suckle the mare for at least 12–24 h.⁷ New and promising treatments such as plasma from hyperimmunized horses against C. perfringens type C and D and rotavirus⁸ given both intravenously and orally and treatment with oral metronidazole have to be assessed further.

Currently, we are recommending vaccination of the broodmares at 6 weeks and 3 weeks prepamur with C. perfringens type C and D toxoid; who are housed on farms that have had multiple foals affected with this disease. Optimal hygiene of the foaling stall and the dam should be undertaken to decrease the degree of exposure of the foal to fecal pathogens.⁷ Oral administration of Lactobacillus acidophilus to minimize C. perfringens overgrowth has been studied successfully in chickens.¹⁰ In heavy milking mares, the foal should be prevented from overeating and these mares should receive low to moderate digestible energy rations the first week postpartum.

Future studies will include PCR typing of all C. perfringens isolates from foals in order to achieve a better understanding of the occurrence of this disease and its variability caused by different types. Preliminarily, those foals that were C. perfringens type C (n = 3) on PCR died, whereas those with type A (n = 4) survived. This type of trend will be essential in designing specific immune treatments and herd prevention. Given the high mortality rate of this disease, prevention may prove more essential than disease treatment in severely affected foals.

Note that foals for 1997 in our study only include those presenting from January through April 1st.
References and Footnotes


ªDone by Robert Ellis.
ªThese are off-label medications.
ªBio-Ceutics, St. Joseph, MO 64506. This bovine product is not approved in horses.
ªNote that four out of six foals were C. perfringens type A positive; however, the fourth was also type C positive and died.