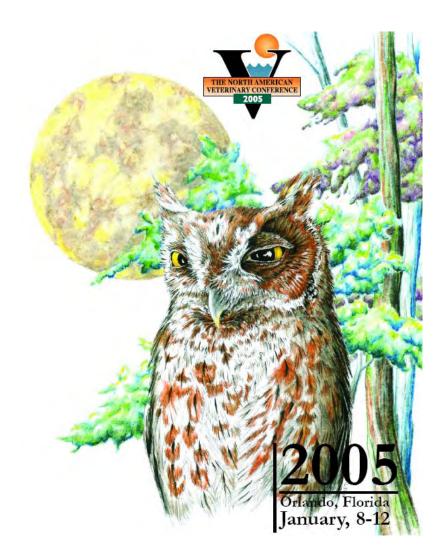
Proceeding of the NAVC North American Veterinary Conference Jan. 8-12, 2005, Orlando, Florida



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BACTERIAL ENTERITIS

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INTRODUCTION

The main etiological agents involved in bacterial enteritis are reported to be *Clostridium perfringens*, *Clostridium difficile, Campylobacter* spp., *Salmonella* spp and enteropathoegnic *Escherichia coli* (EPEC). However, given that these organisms can be part of the indigenous intestinal microflora, their role in causing clinical disease is controversial.

CLOSTRIDIUM PERFRINGENS

Clostridium perfringens is an anaerobic, spore-forming, gram-positive bacillus, which has been typed into five toxigenic phenotypes, A-E. Each elaborates a major toxin and other toxins, including *C. perfringens* enterotoxin (CPE), a well-characterized virulence factor. Several recent studies have shown an association between the presence of CPE in feces and diarrhea. Signs of large-bowel diarrhea predominate characterized by increased frequency of defecation, tenesmus, fecal mucus and hematochezia.

C. perfringens has also been implicated in the acute hemorrhagic gastroenteritis syndrome (AHG) in dogs; clinical signs include severe vomiting and diarrhea, often associated with blood. Onset of signs is peracute and animals can be markedly volume-depleted.

A number of methods to diagnose *C. perfringens*associated diarrhea have been reported including routine bacterial culture, identification of increased spore counts on fecal smears, and detection of CPE in feces. Although the presence of endospores could help support a diagnosis, detection of CPE is most reliable. An ELISA kit is available for detection of CPE in fecal specimens (Techlab Inc., Blacksburg, VA), but the assay has not yet been fully validated in dogs or cats.

For most cases of *C. perfringens*-associated diarrhea, animals respond to antimicrobial therapy. Recommended antimicrobials include macrolides (particularly tylosin), ampicillin, and metronidazole. Supportive measures such as dietary modification, is also recommended. Aggressive treatment is required for AHG, including intravenous fluid therapy (usually colloids), and a combination of intravenous bacteriocidal antibacterials (e.g. amoxicillin-clavulanate and enrofloxacin). Cases are initially maintained nil per os, but subsequently dietary management can be instigated.

CLOSTRIDIUM DIFFICILE

Clostridium difficile is a gram-positive, anaerobic sporeforming bacillus, which has been associated with diarrhea in dogs. Two toxins, toxin A and toxin B, are thought to be primarily responsible for manifestation of clinical signs, although other toxins may also play a role. C. *difficile*associated diarrhea can be diagnosed by routine bacterial culture, identification spores on fecal smears and detection of toxin A or toxin B in fecal specimens with ELISA. Again the latter is reported to be most reliable. Similar to *C. perfringens*, a strong association has been found between the detection of *C. difficile* toxin A and the presence of AHDS. *Clostridium difficile* seems to be less prevalent in cats compared to dogs. The antimicrobial of choice for therapy of *C. difficile*-associated diarrhea is metronidazole.

CAMPYLOBACTER spp.

Campylobacter is a small, curved, motile, microaerophilic, gram-negative rod, which is often isolated from healthy dogs and cats. Transmission occurs by the feco-oral route. Campylobacter species can be isolated in feces from animals with clinical signs and from up to 50% of normal dogs. Clinically significant disease is more common in young, immunocompromized individuals, or in colony dogs. Dogs with concurrent enteric infections e.g. endoparasitism and viral diarrhea are more severely affected. Although the organism is a potential zoonosis, most cases of human campylobacteriosis develop secondary to food poisoning with poultry, poultry products and unpasteurized milk. The organism most reported to cause clinical signs in companion animals is C. jejuni. However, other Campylobacter spp. are frequently isolated (e.g. C upsaliensis), and their pathogenic significance is less clear. Clinical signs range from mild transient diarrhea to mucous laden bloody stools with associated signs of colitis.

The organism can be detected on a direct fecal smear, as a 'seagull-shaped' bacterium. However, infection should be confirmed by culture of the bacterium from feces and, given the potential for intermittent excretion, multiple samples may be required. Given that the organism is fragile, falsenegative results can occur if culture techniques are not optimal.

In light of the fact that, in many cases, isolation of *Campylobacter* is incidental and that some cases spontaneously resolve, If antibacterials are thought necessary, the drugs of choice are the macrolides (erythromycin, azithromycin) and fluroquinolones (enroflo-xacin, marbofloxacin).

SALMONELLA spp.

Salmonella species are Gram-negative motile rods, which can cause significant clinical infections in dogs and cats. However, Salmonella spp. are not always associated with clinical disease, since organisms can be isolated from healthy dogs and cats. Clinical problems are most common in young, kenneled or immunocompromised animals, and concurrent viral infection (e.g. parvovirus) may increase disease severity. Transmission is via the feco-oral route, and some species have a zoonotic potential.

Clinical signs include acute diarrhea of variable severity, often but not invariably containing blood. If bacterial translocation from the intestine occurs, life-threatening septicemia may result. Diagnosis is made by isolation of the organism from feces, or blood if septicemic. Given problems with intermittent excretion, three fecal cultures are required to confirm a negative result.

If a *salmonella* species is isolated from healthy animals, or animals with acute diarrhea that are not systemically ill, no treatment is needed, since antibacterial use could promote resistance and a carrier state. Treatment is only necessary when there is evidence of sepsis, severe hemorrhagic diarrhea, PLE or neutropenia. The choice of antibacterial should be governed by culture and sensitivity testing, but fluoroquinolones (e.g. enrofloxacin, marbofloxacin) are usually suitable, and the feces should be re-cultured to verify eradication. Given the zoonotic risk, owners should be advised to take appropriate precautions to avoid selfinfection.

ENTEROPATHOGENIC E. COLI

It is controversial whether enteropathogenic (EPEC) and enterotoxigenic *E. coli* (ETEC) are pathogenic in dogs and cats. Attachment of ETECs and subsequent release of heatlabile, heat-stable and Shiga-like toxins may cause acute diarrhea. Further, EPECs may attach to the mucosa causing effacement of microvilli, and leading to profound malabsorption without morphological abnormalities on histopathological examination. Identification of pathogenic strains requires specialized assays, such as bioassays for toxins and genome probes for identification of pathogenicity markers. However, because these organisms can be seen in healthy animals, a positive result does not necessarily prove a role in clinical disease.

References available on request.