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CHAPTER 3

Gastrointestinal and Peritoneal Infections

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The pathophysiologic mechanisms of the primary etiologic agents responsible for infectious diseases of the gastrointestinal (GI) tract are covered in depth individually elsewhere in this text. Thus the primary goals of this chapter are to present the normal microflora throughout the GI tract and to briefly discuss an approach to the diagnosis and management of the primary clinical syndromes associated with these infectious processes.

Oral Cavity

Normal Flora

Most work describing the normal flora of bacteria in the equine pharynx relates to upper respiratory tract infection and lower respiratory tract infection attributed to aspiration. But, relatively few studies have examined the normal flora of the oral cavity in horses. Several aerobic and facultative anaerobic organisms have been isolated from various locations throughout the pharynx, most notably *Streptococcus equi* subsp. *zooepidemicus*.[1-3] Commonly isolated anaerobes included those from the genera *Bacteroides*, *Eubacterium*, *Fusobacterium*, *Clostridium*, *Veillonella*, *Peptostreptococcus*, and *Megasphera*.[3]

Infectious Disorders

Unlike in small animals, infectious diseases of the oral cavity are relatively rare in horses. Primary problems with a possible infectious etiology include periodontitis and tooth root abscesses, pharyngitis, and dysphagia. Anaerobic organisms are frequently associated with tooth root abscesses.[4] Other infectious problems with potential impact on the oral cavity include *Actinobacillus lignieresii*, the organism associated with wooden tongue[5,6]; various fungal organisms such as *Candida* spp., which can cause thrush in foals; viral diseases such as vesicular stomatitis[7]; infectious causes of dysphagia such as *Clostridium botulinum* (botulism)[8,9]; and equine protozoal myeloencephalitis.

Esophagus and Stomach

Normal Flora

The esophagus and stomach are not sterile environments, and the stomach harbors a relatively diverse microbial population. In one study, 2.78×10^9 total (2.00×10^8 viable) bacteria per gram of ingesta were recovered from the fundic region of normal ponies, with 1.92×10^9 total (1.0×10^7 viable) bacteria per gram of ingesta recovered from the pyloric region.[10] In both regions, gram-positive organisms (rods and cocci) predominated, and very few cellulolytic (100 to 300/g) bacteria were isolated.[10] Both the total anaerobic bacterial population and the lactobacilli concentration appeared to increase in a linear fashion in live horses after a meal.[11] Colonization of and attachment to the gastric squamous mucosa by several indigenous *Lactobacillus* spp. have also been documented.[12] Similar bacteria have been identified within different regions of the gastric mucosa using fluorescent in-situ hybridization,[13,14] although the microbial population can vary by individual.[13]

Infectious Disorders

Infectious diseases of the esophagus mainly occur secondary to perforation, thus involving a mixed population of aerobic and anaerobic bacteria. In the equine stomach, although polymerase chain reaction (PCR) fragments unique to gastric-dwelling *Helicobacter* spp. have been identified in horses, an association between *H. pylori* and ulceration has not been established in adult horses or foals.[15,16] More recent studies have failed to identify *Helicobacter* spp. in equine gastric mucosa using fluorescent in-situ hybridization,[13] including those with antral pathology.[14] One case of emphysematous gastritis due to *Clostridium perfringens* has been reported.[17]

Small Intestine

Normal Flora

Few studies have evaluated normal equine small intestinal microbial populations. Total bacterial counts and proportion of gram-positive bacteria recovered from the ileum were similar to that seen in the stomach,[10] but viable bacteria numbered 3.6×10^7. In a study analyzing only anaerobic bacteria, increasing numbers of both culturable and proteolytic bacteria were identified in the duodenum, jejunum, and ileum.[18] Proteolytic bacteria composed a high proportion of the total bacteria in all regions but accounted for almost all in the duodenum. Numbers of bacteria identified from the GI lumen outnumbered those recovered from the mucosa in all segments.[18]

Infectious Disorders Causing Diarrhea in Foals

Most infectious causes of diarrhea in foals, unlike those causes in adult horses, affect the small intestine either alone or in combination with the large colon.

Bacterial Disorders

Foals diagnosed with systemic sepsis (see Chapter 6) commonly develop diarrhea in association with their primary disease, with reported incidence between 16% and 42%.[19-23] As such, although *Escherichia coli* is the most common causative organism

associated with sepsis, it is not typically recognized as a primary cause of enteritis and/or enterocolitis in foals. One report demonstrated an increased probability of diarrhea in foals with *Actinobacillus* sepsis when compared to those foals from which other organisms were isolated.[19] Further, up to 50% of foals presenting with diarrhea have a positive blood culture at admission, supporting the usefulness of this diagnostic test for foals less than 30 days of age.[24,25]

Another common bacterial cause of enterocolitis in foals is *Salmonella enteritidis* (see Chapter 35). In addition to diarrhea, affected foals typically display clinical signs of sepsis. Diagnosis is confirmed via aerobic culture of blood and/or feces. Treatment is supportive and should always include directed systemic antimicrobial therapy.

Clostridial organisms can act as primary pathogens in foals (see Chapter 41), and these agents can cause disease in individual animals or present as outbreaks in affected farms. *Clostridium perfringens* typically affects foals younger than 10 days of age. *Clostridium perfringens* serotypes A and C are most commonly implicated, with serotype C resulting in more severe disease, hemorrhagic diarrhea, and higher mortality than that seen with serotype A.[26,27] Serotype A is commonly isolated from the feces of normal foals, but the organism in general is more commonly isolated from foals with diarrhea.[28,29] A diagnosis is typically based on the presence of compatible clinical signs and culture of the organism from feces, preferably with genotyping of the obtained isolate. Observation of large, gram-positive rods or spores on a fecal Gram stain should raise the index of suspicion.[26] *Clostridium difficile* has also been implicated as a cause of diarrhea in foals. Disease severity can vary from mild to hemorrhagic diarrhea. As with *C. perfringens*, *C. difficile* can be isolated from asymptomatic foals, thus toxin detection in feces is useful for confirmation of a diagnosis.[30,31] Commercial immunoassays are available for the detection of toxins A and B in feces, as well as the enterotoxin of *C. perfringens*.[32] Treatment is supportive with the addition of directed antimicrobial therapy, typically with metronidazole. In some geographic locations, documented metronidazole resistance in *C. difficile* isolates has prompted therapy with vancomycin in select cases.[33] Infection with *Lawsonia intracellularis* has been described in older foals.[34-37] Infection with this obligate intracellular pathogen results in a proliferative enteropathy and should be suspected in any weanling age foal with severe hypoproteinemia. Clinical signs include weight loss, ill thrift, depression, colic, peripheral edema, and variable fecal consistency, ranging from soft normal to watery diarrhea. Protein loss can be severe. Diagnosis is based on clinical signs in combination with results of fecal PCR and serum antibody testing. Treatment includes supportive care, predominantly with colloid replacement, and directed antimicrobial therapy. Common specific recommendations include chloramphenicol and oxytetracycline followed by doxycycline, erythromycin, or clarithromycin.[38-40] Duration of treatment is dictated by clinical status and improvement of clinicopathologic findings; a minimum of 3 weeks of treatment is typically indicated.

Viral Disorders

The most commonly encountered viral pathogen causing diarrhea in foals is rotavirus. Typically, rotavirus is thought to affect foals between 5 and 35 days of age,[41] although recent data suggest the mean age of affected foals can be as high as 81 days.[24] Older foals appear to be less severely affected.[42] The most common and obvious clinical sign is diarrhea, and fecal consistency can vary greatly. Other signs relate to disease severity, including depression, anorexia, dehydration, and other similar findings. The virus causes blunting of the small intestinal microvilli, resulting in malabsorption and maldigestion. Diagnosis can be confirmed with fecal electron microscopy, which has a significant lag time, or commercial immunoassays, also performed on feces. Treatment is principally supportive. Emphasis must be placed on biosecurity protocols to prevent spread of new outbreaks. Because this is a nonenveloped virus, typical antiviral disinfectant compounds, such as quaternary ammonium mixtures, are ineffective against rotavirus. The virus is extremely contagious, with morbidity often approaching 100% in farm outbreaks. Prognosis is very good to excellent with supportive care, and mortality is typically very low in uncomplicated cases. Other viral disorders occur much less frequently and include coronavirus[43-45] and adenovirus.[46,47]

Protozoal Disorders

Cryptosporidium spp. are the most common protozoal cause of diarrhea in foals.[28,45,48] These organisms are generally regarded as less significant than the major bacterial and viral diseases discussed previously.

Small Intestinal Disease in Adult Horses

Etiology and Pathophysiology

Proven infectious disorders of the small intestine in horses are rare. Horses do not appear predisposed to small intestinal bacterial overgrowth, which is common in dogs and human beings. One disorder that has a suspected, but to this point unsubstantiated, infectious origin is duodenitis/proximal jejunitis (DPJ), a syndrome of small intestinal inflammation primarily characterized by copious quantities of gastric reflux. This syndrome has also been termed *anterior enteritis* or *proximal enteritis*. In most cases, an underlying etiology cannot be determined. In some cases, *Salmonella* spp. or *Clostridium* spp. can be isolated from culture of gastric reflux. Salmonella has not been consistently identified in a majority of cases, and many horses with documented infections by these organisms do not develop DPJ. Recently, toxigenic strains of *C. difficile* were isolated from the reflux of 10 of 10 horses with DPJ and 1 of 16 control horses with other causes of nasogastric reflux.[49] Further investigation of this organism is clearly warranted. Another suspected infectious cause is *Fusarium* spp.[50] In one epidemiologic report, affected horses were fed significantly more concentrate and were more likely to have grazed pasture than unaffected controls.[51] Regardless of the initiating cause, inflammatory-mediated alterations in secretion and motility contribute to a functional obstruction and a vicious cycle of events. Intestinal inflammation can result in alterations in normal sensory-motor function, mucosal function, ion transport, and transepithelial permeability. The blanket term *duodenitis/proximal jejunitis* may actually encompass a wide spectrum of inflammatory small intestinal disorders resulting in a similar clinical syndrome.

Clinical Signs and Laboratory Findings

The most characteristic clinical findings in horses with DPJ include moderate to severe pain that often improves after gastric decompression, large volumes of gastric reflux, clinical signs of endotoxemia, and small intestinal distention evident on rectal palpation and ultrasonographic examination.

Abnormal clinicopathologic findings can include hemoconcentration, neutropenia, acidemia, prerenal azotemia, hyponatremia, hypochloremia, hypokalemia, and increased hepatic enzymes.[52] Typically, peritoneal tap findings include a mild to moderate increase in total nucleated cell count (TNCC), which can be elevated to 20,000/μL with a moderate to marked increase in total solids (up to 5 g/dL). However, the nucleated cell count has been reported to vary widely in some cases. These findings are often a useful method of differentiation from horses with strangulating small intestinal disease, which tend to have higher numbers of red blood cells, as well as increased TNCC;

however, peritoneal tap findings should be used in conjunction with other parameters.[53]

Therapy

Treatment consists primarily of supportive care, with an emphasis on fluid therapy and gastric decompression. Particular care should be taken to provide maintenance fluid requirements while also replacing the volume lost through gastric reflux. Therapy should also include nonsteroidal antiinflammatory therapy for analgesic as well as antiinflammatory purposes, as long as renal function remains normal, and directed therapy to combat endotoxemia. When cases either deteriorate or do not improve with medical therapy, surgical exploration can be considered.[54] Surgical exploration can offer manual decompression of the small intestine and rule out any physical obstruction, although surgical treatment did not improve patient outcome in one report.[55] In protracted cases or in horses with hypertriglyceridemia, supplemental parenteral nutritional support should be considered. Prokinetic therapy with either erythromycin lactobionate, metoclopramide, bethanechol, or lidocaine can also be considered[56-58]; one should note that these therapies may be less effective in the inflamed intestine.[59]

With prompt medical therapy, horses with DPJ generally carry a good prognosis. Factors associated with a decreased risk of survival include increased peritoneal fluid protein concentration and increased anion gap,[60] as well as failure to respond to prokinetic therapy within 24 hours.[56] Potential complications include laminitis, thrombophlebitis, peritonitis, adhesions, pharyngitis and/or esophagitis, and cardiac dysrhythmias.[61]

Large Intestine

Normal Flora

Much more is known about the resident microflora in the equine large intestine relative to the more orad portions of the GI tract. The cecum and large colon have a large capacity and the capability for extensive fermentation by bacteria and protozoa. Total protozoal concentrations in the large colon appear to increase in horses fed a diet high in forage, relative to a diet high in concentrate.[62] In the same study, the colon had concentrations of both total and cellulolytic fungi more than 10 times greater than those found in the cecum.[62] Increasing the grain component of a diet appears to have a greater impact in the colon, relative to the cecum; total bacterial concentration increased and cellulolytic bacterial concentration decreased in horses fed a primarily concentrate diet.[63] Cecal microflora remained predominantly cellulolytic regardless of diet.[64] At least two species of anaerobic phycomycetes capable of digesting plant cellulose and hemicellulose have been isolated from the equine cecum,[65] and *Ruminococcus flavefaciens* has recently been identified as the predominant cellulolytic cecal bacterial species.[66] At least two types of spirochetes have been documented in the equine cecum.[67] Bacteriophages have been documented infecting spirochetes from the equine cecum,[67] and bacteriophage-like particles have been demonstrated in various regions of the large intestine by electron microscopy.[68]

Acute Diarrhea in Adult Horses

Etiology

The most common infectious agents associated with colitis in adult horses include *Salmonella* spp. (see Chapter 35), *Neorickettsia* (formerly *Ehrlichia*) *risticii* (equine monocytic ehrlichiosis, Potomac horse fever [PHF]; see Chapter 40), C. *difficile* (see Chapter 42), and C. *perfringens* (see Chapter 42). *Aeromonas* spp. are often isolated from horses with diarrhea, but true causality as an etiologic agent in acute diarrhea has not been determined. Parasites are not typically associated with acute diarrhea in adult horses, with the exception of larval cyathostomiasis in Europe, the northern part of the United States, and Canada. The most common cause of outbreaks of colitis in horses is salmonellosis. Outbreaks of PHF and clostridial colitis are rare, although the latter may occur as a clustering of cases of foals or hospitalized horses.

Diagnostic Approach

In evaluation of horses with acute diarrhea, laboratory evaluation should include complete blood count (CBC) with fibrinogen and a biochemical profile. If available, venous blood gas analysis is desirable. Additional tests aimed at identification of an etiologic agent can be performed both on blood and feces. The potential for co-infections should be considered when determining an appropriate diagnostic plan.

Diagnostic Tests on Whole Blood or Serum

Neorickettsia risticii

Although an enzyme-linked immunosorbent assay (ELISA) has been described for the diagnosis of *N. risticii*, most laboratories use an immunofluorescent assay (IFA) or PCR.[69] Infected horses develop high titers (>640) within days, often before clinical signs are apparent. Paired serum samples (acute and convalescent) should be collected within 5 to 7 days rather than the conventional interval of 2 to 4 weeks because infected horses rapidly develop high titers. Several laboratories interpret a single serum titer of ≥80 at the onset of signs as consistent with PHF once horses develop diarrhea. However, given the variation in the immune response of individual horses and the variation in clinical signs wherein some horses do not develop diarrhea, paired serum must be utilized even if the first sample was negative. Vaccination for PHF results in positive titers that usually disappear by 6 to 9 months, and previous subclinical exposure has prolonged waxing and waning titers for over a year. Polymerase chain reaction detects the presence of antigen and has excellent sensitivity without the potential for interference from vaccination,[70,71] and both feces and blood should be tested since the organism may not be present in both fluids concurrently.[69,70] This testing can be performed on postmortem tissues even if formalin-fixed.[69]

Diagnostic Tests on Feces

Aerobic Culture

The main organism of interest is *Salmonella enterica*. Enrichment culture is required, and antiserum assays are required for identification to serogroup. Most accredited laboratories identify to serogroup, and the National Veterinary Services Laboratory (Ames, IA) will identify all isolates to serotype. Multiple cultures are preferable, and recovery of pathogens can be difficult when feces are very watery. Culture of a rectal mucosal biopsy sample may improve the recovery rate.[72] Polymerase chain reaction has been reported as a more sensitive method of detecting salmonella in feces relative to culture.[73-76] In a horse with clinical signs, detection using a validated real-time assay PCR recently had a specificity as high as 98%.[77]

Anaerobic Culture

Anaerobic culture of fecal samples is primarily used to identify the presence of *Clostridium* spp. Strict anaerobic handling of the feces is critical to successful culture, especially for C. *difficile*.[78] Recovery of C. *difficile* organisms is dramatically reduced after storage for 72 hours in aerobic conditions at 4° C.[78]

Because clostridia can be cultured from the feces of some normal horses, direct detection of specific clostridial toxins from

feces enhances diagnostic specificity. Toxins are detected by bioassay for the C. *perfringens* pathogens and by cytoxic bioassay or ELISA for C. *difficile*. *Clostridium difficile* toxins are more stable in aerobic conditions than the organisms themselves.[78] Commercial assays (ELISA) are available for C. *difficile* toxins A and B, as well as the enterotoxin of C. *perfringens*.[32] For C. *perfringens*, PCR analysis of isolates cultured from feces is the preferred method for detection of toxin and confirmation of a diagnosis of clinically relevant disease. Many diagnostic laboratories will perform toxin testing, and some will provide packages including both culture and toxin analysis.

Feces should be examined by sedimentation for sand and microscopically for increased fecal leukocytes. A Gram stain may be useful as an initial screen for *Clostridial* organisms (long gram-positive rods). Cyathostome larvae are best detected by direct examination of feces.

Therapy

The primary goal of therapy for adult horses with diarrhea is restoration and maintenance of fluid, electrolyte, and acid-base balance. Specific pathogen-directed antimicrobial therapy may be indicated, depending on the etiologic agent identified, but generalized, prophylactic antimicrobial administration is not recommended for most adult horses with diarrhea. For many horses with acute diarrhea, initial intravenous (IV) fluid replacement is required as the result of tremendous volume losses. Typically, mild to moderate acidemia is corrected by restoration of plasma volume with a balanced polyionic replacement fluid. Acidifying solutions (such as 0.9% sodium chloride) are not recommended but may be used if another fluid option is unavailable. In cases of severe dehydration, initial therapy with hypertonic saline or synthetic colloid (such as hydroxyethyl starch) can be used to restore circulatory volume, but these must be followed by administration of isotonic fluids.

Other goals of therapy include reducing inflammation, pain control, and limiting the effects of endotoxemia. Drugs commonly used for these purposes include nonsteroidal antiinflammatory drugs (NSAIDs), such as flunixin meglumine which has analgesic and antiinflammatory properties.[79] As with other NSAIDs, one must take care to avoid use of this agent in horses with renal compromise, moderate to severe dehydration, NSAID toxicity, or right dorsal colitis. Adjunctive therapy with polymyxin B sulfate[80-82] and/or pentoxifylline[79,83-85] is suggested to combat the effects of endotoxemia.

Chronic Diarrhea in Adult Horses

Etiology

Chronic diarrhea is usually defined as diarrhea persisting for more than 4 weeks.[86] Fecal consistency can vary widely. Although many specific diseases can result in chronic diarrhea, the actual inciting cause at the time of presentation is usually elusive. Occasionally, problems of a non-GI nature, such as hepatic disease or abdominal abscessation, result in diarrhea. More common infectious causes of chronic diarrhea include chronic salmonellosis and parasitism with large and/or small strongyles. Recently, the spirochete *Brachyspira pilosicoli* was implicated in a herd outbreak of chronic diarrhea in weanling age horses.[87] Noninfectious inflammatory causes include inflammatory bowel diseases (granulomatous enteritis or colitis, lymphocytic-plasmacytic enterocolitis, and eosinophilic enterocolitis), neoplasia (most commonly lymphosarcoma), sand enteropathy, and right dorsal colitis. Noninflammatory causes encompass a range of problems with the common theme of disruption of the large colonic intestinal flora or mucosal function. This may or may not be related to a dietary disruption, and many of these horses have few other clinical signs. Regardless of the inciting cause, horses with chronic diarrhea remain very difficult to treat and afford a guarded prognosis for long-lasting return to normal fecal consistency.

Diagnostic Approach

Laboratory evaluation for the individual horse with chronic diarrhea typically includes CBC with fibrinogen, serum biochemical profile, venous blood gas analysis, rectal examination, abdominal ultrasound, and analysis of peritoneal fluid. Results of all the aforementioned diagnostic procedures are commonly normal, and further recommended analyses include a comprehensive fecal examination, gastro-duodenoscopy with biopsy, and rectal biopsy.

Comprehensive fecal analysis should include assessment for parasites (grossly and by fecal flotation/McMasters quantification), aerobic culture for *Salmonella* (5 samples at a minimum 12-hour interval, as for acute diarrhea), sand sedimentation, unstained wet mount for protozoa and parasites, new methylene blue stain for fecal leukocytes, and Gram stain to evaluate the gram-positive:gram-negative bacteria ratio.

Gastro-duodenoscopy allows both visual inspection of the stomach and proximal duodenum and the collection of biopsy specimens for histopathology. Rectal biopsy is a simple, relatively noninvasive procedure.[88] Samples should be submitted for both culture *(Salmonella)* and histopathology. The main diagnoses obtained with use of histopathology are the inflammatory bowel diseases.

Therapy

If a specific diagnosis is achieved, directed therapy should be initiated according to the etiologic agent. In all cases, free-choice access to fresh water is critical to maintenance of hydration. Many horses will consume balanced, isotonic electrolyte-spiked water, if such a solution is offered. Alternatively, access to a salt or mineral block can serve as a substitute source of electrolyte replacement. Typical feeding recommendations include good quality grass hay with limited legume hay and concentrate intake. Dietary changes alone are unlikely to effect a cure but may provide improvement.

Nonspecific therapy for horses with chronic diarrhea can include transfaunation and iodochlorhydroxyquin. Exact recommendations for the transfaunation procedure are sparse in the veterinary literature, as are reported benefits. Typically, cecal liquor is obtained either from an animal recently euthanized for non-GI reasons or from an animal implanted with a cecal cannula. Once appropriate transfaunate is obtained, one must also decide whether to pretreat the recipient. Frequently, recipients are pretreated with acid-suppressing agents to enhance viability of transplanted bacteria and protozoa as they pass through the gastric environment. The efficacy of such treatment has not been validated to date in the horse, although the potential value of transfaunation was recently highlighted during a herd outbreak potentially related to the spirochete *Brachyspira pilosicoli*.[89]

Iodochlorhydroxyquin, an 8-hydroxyquinolone derivative, has long been recommended for the treatment of chronic diarrhea, although this therapy was initially proposed for the treatment of trichomoniasis.[90] Although the described disease entity likely involved disruption of the normal intestinal flora rather than an infectious cause, some horses responded favorably to therapy. The response to treatment with iodochlorhydroxyquin is highly variable, from no change to worsening of the diarrhea. Often, therapy will result in more normal fecal consistency, but unfortunately many will revert to diarrhea within a few days of drug withdrawal.[89]

Prognosis

Regardless of the inciting cause, the prognosis for complete recovery is guarded when the duration of diarrhea exceeds

1 month; the prognosis worsens as the course of disease progresses.

Peritoneal Infections

Peritonitis refers to inflammation of the mesothelial lining of the peritoneal cavity and is typically caused by mechanical, chemical, or infectious insult to the parietal peritoneum. In addition to classification based on the causative insult, further classification can include onset (acute or chronic), distribution (localized or diffuse), origin (primary or secondary), and infectious component (septic or aseptic). Acute, diffuse, septic peritonitis secondary to GI disease is the most common manifestation.[91]

Etiology and Clinical Findings

Most cases of peritonitis occur secondary to a GI event such as perforation of the GI tract, intestinal ischemia, DPJ, colitis, neoplasia, verminous arteritis, intestinal mural abscess, or other causes.[92-94] Iatrogenic causes include rectal tear, enterocentesis, castration, and abdominal surgery. Other causes include traumatic events (including uterine or vaginal perforation during foaling or breeding), mesenteric abscess (including those associated with *Streptococcus equi* subsp. *equi*), cholelithiasis, and others. Causes specific to the young foal include rupture of the urinary bladder or urachus, omphalitis and/or omphalophlebitis, sepsis, and *Rhodococcus equi* abscessation.

Organisms associated with GI rupture include a mixed population of gram-positive and gram-negative aerobic and anaerobic organisms, typically with no clear predominance. Enterobacteriaceae, *Streptococcus* spp., and *Staphylococcus* spp. have been most commonly isolated from septic peritoneal fluid samples.[93,95,96] Common anaerobes include *Bacteroides, Clostridium*, and *Bacillus* spp. In foals, peritonitis is more commonly associated with *Streptococcus* and *R. equi* infections. Several case series involving peritonitis associated with *Actinobacillus equuli* have been reported.[97-99] Initial reports of *A. equuli* peritonitis originated solely in Australia, but individual cases have been reported from the United Kingdom and United States.[100,101]

Clinical signs of peritonitis in horses are variable but include fever, depression and abdominal pain. Individuals may present with diarrhea or weight loss.[92,94] Based on severity and localization, signs can also include those of endotoxemia and shock. In horses with *A. equuli* peritonitis, clinical signs commonly include depression, inappetence, lethargy, and mild to moderate abdominal pain acutely or weight loss in a chronic form.[98,99] Postpartum mares with peritonitis secondary to a uterine perforation typically present with fever and depression, with or without abdominal pain.[102]

Horses with acute peritonitis are more likely to present with subclinical disseminated intravascular coagulation (DIC), as indicated by increased plasma and peritoneal D-dimer concentrations and prolonged clotting times, relative to those with other causes of acute colic.[111,112]

Diagnosis

Definitive diagnosis is based on an increased TNCC in peritoneal fluid (generally >10,000 cells/μL) with an increased total solids or protein that is often >5.0 mg/dL. Cytologic examination of the fluid is imperative to identify toxic changes and degenerating neutrophils and detect any intracellular or extracellular bacteria or plant materials. If GI contents or plant material are evident, one should take care to differentiate between GI rupture and enterocentesis. Aerobic and anaerobic bacterial culture of peritoneal fluid should be performed in all suspected cases, but this procedure has a low sensitivity, with only 9.5% to 41% of samples yielding positive growth.[92,93,95,96] Total cell count in peritoneal fluid may be increased following enterocentesis, abdominal surgery, or open castration.[103-107] It is important to differentiate between peritonitis secondary to gastrointestinal rupture or leakage and iatrogenic enterocentesis. If fluid samples show abnormalities in TNCC and cytology, peritonitis is most likely present; enterocentesis can result in an elevated TNCC in abdominal fluid within 4 hours.[106] If a differentiation between peritonitis and enterocentesis cannot be clearly made, a sample should be taken from an alternate location, preferably with ultrasound guidance. In postfoaling mares, the percentage of neutrophils in the peritoneal fluid may be increased for up to 7 days, but the total protein and TNCC should remain within normal limits.[108,109] Other parameters, if available, can aid in the diagnosis. Ultrasonographic findings of abundant hypoechoic or variably echogenic peritoneal fluid is supportive of a septic process. A decrease in peritoneal fluid pH (<7.3) or glucose (<30 mg/dL) or an increase in the peritoneal lactate concentration can indicate aseptic peritonitis.[107,110]

Therapy

Treatment of horses with peritonitis should begin with identification and correction of the underlying problem, if possible. If a GI source is suspected, an exploratory celiotomy is likely indicated. Supportive care is also critical to the treatment protocol. This should include correction of fluid deficits, acid-base and electrolyte imbalances, and colloid oncotic pressure. Anti-inflammatory and antiendotoxic therapy are also clearly of benefit. Additional analgesic and prokinetic drugs should be provided if necessary.

Antimicrobial therapy is critical to the management of septic peritonitis. Broad-spectrum coverage should be instituted pending results of peritoneal fluid culture and sensitivity. If positive results with sensitivity testing are obtained, therapy can be adjusted accordingly. A typical initial regimen includes penicillin, gentamicin, and metronidazole to cover gram-positive, gram-negative, and anaerobic spectrums, respectively (see Appendix D for dosages). Metronidazole should be included if anaerobic involvement is suspected because of the known resistance of multiple *Bacteroides* spp. to penicillins. Enrofloxacin can replace gentamicin in the treatment regimen because the lipophilic nature of this compound can provide increased penetration to the peritoneum. Neonatal foals with peritonitis should receive a similar regimen to that suggested for adults, although amikacin is frequently substituted for gentamicin due to increased sensitivity of commonly isolated organisms.[111] A combination of azithromycin or clarithromycin plus rifampin provides reasonable coverage for older foals or weanlings, as *Streptococcus* or *R. equi* are commonly associated with disease in that population if a primary GI lesion is not suspected.[111] Although *A. equuli* is typically sensitive to either penicillin or trimethoprim-sulfonamide combinations, initial broad-spectrum coverage with penicillin and gentamicin is suggested pending culture results as the result of microbial resistance of some isolates.[99]

Abdominal drainage and lavage can help remove excess fluid, foreign materials, fibrin, and bacterial products from horses with peritonitis, and postoperative lavage has been shown to decrease the incidence of experimentally-induced abdominal adhesions.[112] Open surgical exploration provides the most effective and thorough examination of all peritoneal surfaces and is recommended if GI perforation or ischemia is suspected or in any other case in which correction of a primary lesion is indicated. A ventral abdominal drain can either be placed at the time of surgery or in the standing horse with

sedation and local anesthesia. Techniques have been described in detail elsewhere.[111,113] Medical and surgical treatment of postpartum peritonitis had similar outcomes in 49 recently described cases; but the severity of injury that can safely be treated medically is unknown.[102]

Peritoneal lavage is typically performed with 10 to 20 L of a balanced isotonic electrolyte solution (such as lactated Ringer's solution or Normosol-R) twice a day for 3 to 5 days, until the lavage solution becomes clear or the catheter loses its patency because of fibrin deposition or omentum. Hypertonic solutions should be avoided because they can result in fluid shifts into the peritoneum. The addition of povidone-iodine to a balanced solution is contraindicated since concentrations as low as 3% can induce peritoneal inflammation.[114] Other agents, such as antibiotics or heparin, have also been suggested as components of the lavage solution, but data demonstrating their benefit are not currently available. Active (or closed suction) abdominal drains have also been advocated, with similar benefits and potential complications to other methods.[113] Lavage with a plain isotonic solution did not alter the pharmacokinetics of gentamicin administered systemically.[115] Thus alteration of antimicrobial dosing does not appear necessary if lavage with plain solutions is part of the therapeutic regimen.

Prognosis

The prognosis is grave for peritonitis associated with GI rupture. Reported survival rates for horses with peritonitis vary but can be as high as 84%.[93,94,96] Some of the variability in reported survival percentages can be related to inclusion criteria, mainly whether horses with GI rupture were included. Septic peritonitis following abdominal surgery is reportedly associated with high mortality (56%).[93] Peritonitis associated with *A. equuli* carries a very favorable prognosis, and all horses in these reports responded to medical therapy if attempted.[97-99]

The complete reference list is available online at www.equineinfectiousdiseases.com.

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