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CHAPTER • 3

Gastrointestinal and Peritoneal Infections

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The common infectious diseases of the gastrointestinal (GI) tract and their pathogenesis are covered in detail individually in other chapters of this text. The primary goals of this chapter are to identify the normal microflora throughout the GI tract and briefly discuss an approach to the diagnosis and management of the primary clinical syndromes associated with these infectious processes.

ORAL CAVITY

Normal Flora

Most information regarding the normal flora of bacteria in the equine pharynx relates to upper respiratory tract infection and lower respiratory tract infection attributed to aspiration. Comparatively few studies have examined the normal flora of the equine oral cavity. Several aerobic and facultatively anaerobic organisms have been isolated from various locations throughout the pharynx, most notably *Streptococcus equi* subsp. *zooepidemicus*.[1,2] Anaerobic bacteria isolated from the normal equine pharyngeal tonsillar area include bacteria from the genera *Bacteroides*, *Eubacterium*, *Fusobacterium*, *Clostridium*, and *Veillonella*.[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 infection from *Actinobacillus lignieresii*, the organism associated with swollen or "wooden" tongue[5,6] (Fig. 3-1); various fungal organisms such as *Candida* spp., which can cause thrush in foals (see Chapter 53); viral diseases such as vesicular stomatitis[7] (see Chapter 24); and infectious causes of dysphagia such as *Clostridium botulinum*[8,9] (botulism, see Chapter 46), equine protozoal myeloencephalitis (see Chapter 59), and West Nile virus (see Chapter 21).

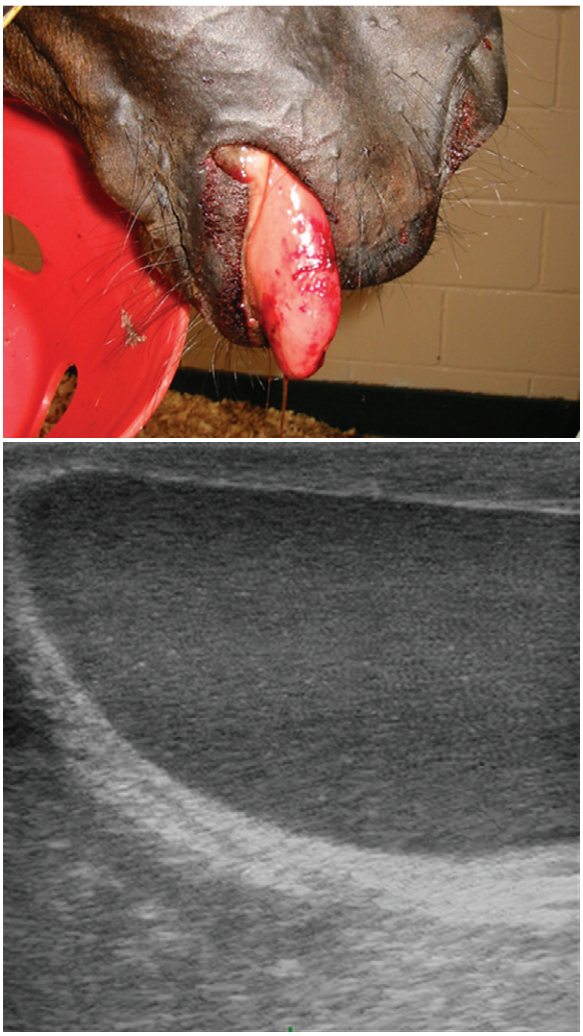

Fig. 3-1 **A,** Swollen tongue secondary to foreign body penetration. **B,** Ultrasound image demonstrating large abscess in the tongue. (Courtesy Dr. Steeve Giguere.)

ESOPHAGUS AND STOMACH

Normal Flora

The esophagus and stomach are not sterile environments. In one study, 2.8×10^9 total and 2×10^8 viable bacteria per gram of ingesta were recovered from the fundic region of normal ponies, with 1.9×10^9 total (but only 10×10^6 viable) bacteria/g recovered from the pyloric region.[10] In both regions, gram-positive organisms (rods and cocci) predominated, and very few cellulolytic bacteria (100-300/g) were isolated,[10] suggestive of the capacity for fermentation but minimal ability to utilize forage. Colonization of and attachment to the gastric squamous mucosa by several indigenous *Lactobacillus* spp. were recently described.[11]

Infectious Disorders

Infectious diseases of the esophagus mainly occur secondary to perforation and involve a mixed population of aerobic and anaerobic bacteria. 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.[12,13] One case of emphysematous gastritis from *Clostridium perfringens* has been reported.[14]

SMALL INTESTINE

Normal Flora

Few studies have evaluated normal microbial populations in the equine small intestine. Total bacterial counts and proportion of gram-positive bacteria recovered from the ileum were similar to those 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.[15] Proteolytic bacteria composed a high proportion of the total bacteria in all regions, but accounted for almost all bacteria in the duodenum. Numbers of bacteria identified from the GI lumen outnumbered those recovered from the mucosa in all segments.[15]

Infectious Diarrhea in Foals

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

Bacterial Disorders

Clostridial organisms can act as primary pathogens in foals, causing disease in individual animals or as outbreaks on affected farms (see Chapter 44). *Clostridium perfringens* typically affects foals under 10 days of age. Types A and C are most often implicated, with type C resulting in more severe disease, hemorrhagic diarrhea, and higher mortality than type A.[16] Type A *C. perfringens* is typically isolated from the feces of normal foals, but the organism in general is more often isolated from foals with diarrhea.[17] A diagnosis is usually confirmed with the combination of clinical signs and culture of the organism from feces, preferably with genotyping of the obtained isolate. Observation of large, gram-positive rods on a fecal Gram stain should prompt the clinician to consider clostridial enteritis[16] (Fig. 3-2).

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, and thus toxin detection in feces is useful for confirmation of a diagnosis.[18]

Commercial immunoassays are available for the detection of toxins A and B in feces as well as the enterotoxin of *C. perfringens* (CPE). 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.[19]

The other predominant bacterial cause of enterocolitis in foals is *salmonellosis* (see Chapter 38). In addition to diarrhea, affected foals typically display clinical signs of sepsis. Diagnosis is confirmed by aerobic culture of blood and feces. Treatment is supportive and should include directed systemic antimicrobial therapy. Foals with systemic sepsis may develop diarrhea in association with their primary disease, with a reported incidence between 16% and 38%.[20-23] Although *Escherichia coli* is the most common etiologic agent associated with sepsis (see Chapter 6), it is not typically recognized as a primary cause of enteritis or enterocolitis in foals. There is an increased probability of diarrhea in foals with *Actinobacillus* sepsis compared with foals from which other organisms are isolated.[20]

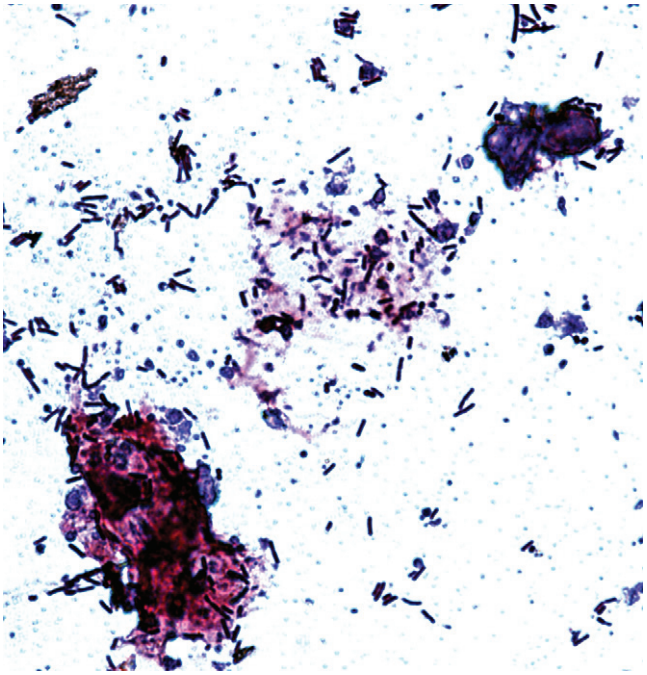

Fig. 3-2 Photomicrograph of Gram-stained feces of foal with *Clostridium difficile* enteritis. Note the numerous gram-positive rods. (Courtesy Dr. Michael Porter.)

Infection of older foals with *Lawsonia intracellularis*, an obligate intracellular pathogen, results in proliferative enteropathy[24-27] and should be considered in weanling-age foals with severe hypoproteinemia (see Chapter 36). Clinical signs include weight loss, ill thrift, depression, colic, peripheral edema, and variable fecal consistency, ranging from soft, normal stool 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 with erythromycin estolate and rifampin or chloramphenicol.[25,28]

Viral Disorders

The most common viral pathogen associated with diarrhea in foals is *rotavirus* (see Chapter 17). Typically, rotavirus affects foals between 5 and 35 days of age, with most foals at the younger end of this spectrum.[29] 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 similar findings. The virus causes blunting of the small intestinal microvilli, with 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, with extra emphasis placed on biosecurity protocols. Quaternary ammonium compounds are ineffective as disinfectant agents for equine rotavirus. The virus is extremely contagious, with morbidity approaching 100% in farm outbreaks. Prognosis is good with supportive care, and mortality is typically low in uncomplicated cases.

Other viral causes of diarrhea occur much less frequently and include coronavirus[30,31] and adenovirus[32,33] (see Chapters 18 and 16, respectively).

Fig. 3-3 Horse with duodenitis/proximal jejunitis (DPJ) with 20 to 30 liters of spontaneous reflux when nasogastric tube was placed.

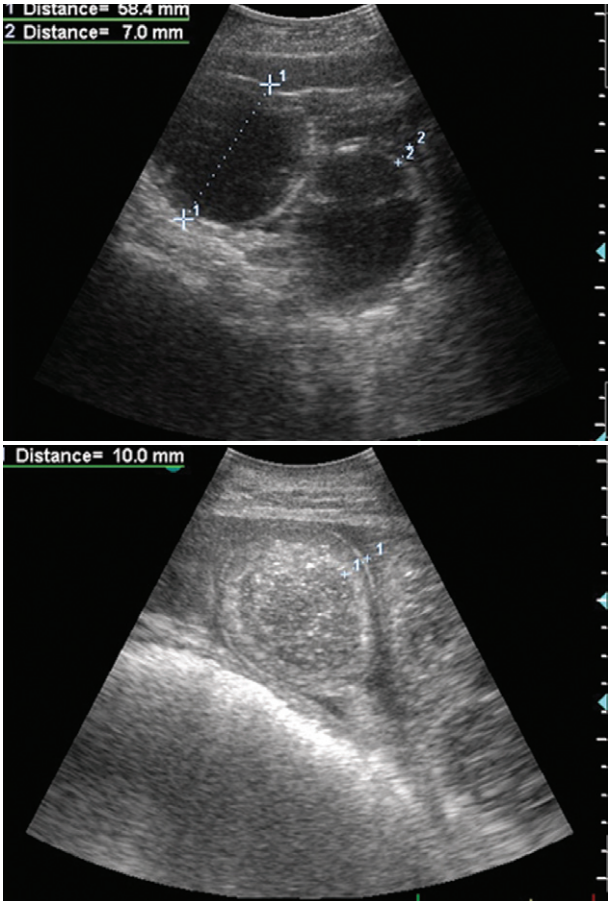

Fig. 3-4 Abdominal ultrasound images from horse with duodenitis/proximal jejunitis (DPJ). **A,** Dilated loops of small intestine. **B,** Thickened small intestine.

Protozoal Disorders

Cryptosporidium spp. are the major protozoal cause of diarrhea in foals (see Chapter 61).[17] These organisms are generally regarded as less significant relative to the major bacterial and viral diseases discussed previously.

Infectious Small Intestinal Disease in Adult Horses

Etiology and Pathogenesis

Proven infectious disorders of the small intestine of adult horses are rare. Horses do not appear predisposed to small intestinal bacterial overgrowth, which is common in dogs and humans. One equine disorder that has a suspected, but to this point unsubstantiated, infectious origin is *duodenitis/proximal jejunitis* (DPJ, also known as *anterior enteritis* or *proximal enteritis*), a syndrome of small intestinal inflammation characterized by copious quantities of gastric reflux (Fig. 3-3). In most affected horses, an underlying etiology cannot be determined and the syndrome of DPJ may include a wide variety of inflammatory small intestinal disorders resulting in a similar clinical presentation. In some horses, *Salmonella* spp. or *Clostridium* spp. are isolated from gastric reflux samples (see Chapters 38 and 44). Recently, toxigenic strains of *Clostridium difficile* were isolated from the reflux in five of five horses with DPJ and from none of six control horses with other causes of nasogastric reflux.[34] Mycotoxins of *Fusarium moniliforme* may also play a role in some cases of DPJ.[35]

Regardless of the initiating cause, intestinal inflammation results in changes in secretory activity and motility that contribute to a functional obstruction. Intestinal inflammation can change normal sensory-motor function, mucosal function, ion transport, and transepithelial permeability.

Clinical Findings

The most characteristic clinical signs in horses with DPJ include moderate to severe pain, which improves after gastric decompression; large volumes of gastric reflux; clinical signs of endotoxemia (see Chapter 37); and small intestinal distention evident on rectal palpation and ultrasonographic examination (Fig. 3-4).

Abnormal clinicopathologic findings in horses with DPJ can include hemoconcentration, neutropenia, acidemia, prerenal azotemia, hyponatremia, hypochloremia, hypokalemia, and increased hepatic enzyme activities.[36] Typically, peritoneal fluid has a mild to moderate increase in total nucleated cell count (TNCC, up to 20,000/μL), with a moderate to marked increase in total solids (up to 5 g/dL). However, the nucleated cell count may vary widely. These findings may help to differentiate horses with DPJ from horses with strangulating small intestinal disease, which tend to have higher numbers of red blood cells as well as a higher TNCC. However, the wide fluctuation in results obtained with these disorders may make differential diagnosis difficult in many horses, necessitating exploratory celiotomy.[37]

Therapy

Treatment of DPJ consists primarily of supportive care, with an emphasis on fluid therapy and gastric decompression. Particular care should be taken to administer maintenance fluid requirements and replace the fluid volume lost through gastric reflux. Therapy should also include nonsteroidal antiinflammatory therapy for analgesic and antiinflammatory effects,

as long as renal function remains normal, and directed therapy to combat endotoxemia. If the affected horse's condition either deteriorates or fails to improve with medical therapy, surgical exploration can be considered.[38] Surgical exploration can offer manual decompression of the small intestine and rule out any physical obstruction. In protracted cases or in horses with increased serum triglyceride concentrations, intravenous (IV) parenteral nutritional support should be considered. Prokinetic therapy with erythromycin lactobionate, metoclopramide, bethanechol, or lidocaine may also be considered.[39,40]

With prompt medical therapy, horses with DPJ generally have a good prognosis for survival. Factors associated with a decreased risk of survival include increased peritoneal fluid protein concentration, increased anion gap,[41] and failure to respond to prokinetic therapy within 24 hours.[39] Potential complications of DPJ include laminitis, thrombophlebitis, peritonitis, adhesions, pharyngitis or esophagitis, and cardiac arrhythmias.

LARGE INTESTINE

Normal Flora

Much more is known about the resident microflora in the equine large intestine than the small intestine or the more orad portions of the GI tract. The cecum and colon have a large capacity and the capability for extensive fermentation by bacteria and protozoa. Total protozoal concentrations in the large intestine appear to increase in horses fed a diet high in forage, relative to a diet high in concentrate.[42] The colon has concentrations of both total and cellulolytic fungi more than 10 times greater than those found in the cecum.[42] At least two species of anaerobic phycomycetes capable of digesting plant cellulose and hemicellulose have been isolated from the equine cecum.[43] *Ruminococcus flavefaciens* has recently been identified as the predominant cellulolytic cecal bacterial species.[44] At least two types of spirochetes have been identified in the equine cecum.[45] Bacteriophages infecting spirochetes within the equine cecum[45] and bacteriophage-like particles have been demonstrated in various regions of the large intestine by electron microscopy.[46]

Acute Diarrhea in Adult Horses

Etiology

The principal infectious agents associated with colitis in horses include *Salmonella* spp. (see Chapter 38), *Neorickettsia risticii* (see Chapter 43; equine monocytic erlichiosis, Potomac horse fever), *Clostridium difficile*, and *Clostridium perfringens* (see Chapter 44). *Aeromonas* spp. are often isolated from horses with diarrhea, but their significance has not been fully determined. Parasites are not typically associated with acute diarrhea in adult horses, with the exception of larval cyathostomiasis in Europe and the northern part of the United States and Canada (see Chapter 62). The most common cause of outbreaks of colitis in horses is salmonellosis. Outbreaks of Potomac horse fever (PHF) and clostridial colitis are rare, although the latter may occur as a clustering of cases of foals or hospitalized horses. Because each of these agents is covered in depth in other chapters, this chapter focuses on a diagnostic and therapeutic approach to an individual horse presenting with acute diarrhea.

Diagnostic Approach

In all horses with acute diarrhea, a minimum database includes complete blood count (CBC) with fibrinogen and a biochemical profile. If available, venous blood gas analysis is desirable. Additional diagnostic tests to identify a specific etiologic agent can be performed on blood and feces. The clinician should remember the potential for co-infections within the same patient.

Diagnostic Tests on Whole Blood or Serum. Although an enzyme-linked immunosorbent assay (ELISA) has been described for diagnosis of *N. risticii* infection in horses, an immunofluorescent assay (IFA) for detection of specific antibody or polymerase chain reaction (PCR) assay for detection of organism is the preferred test. IFA utilizes serum, and PCR is performed on buffy coat or feces. Most laboratories that perform PCR use buffy coats isolated from standard ethylenediaminetetraacetic acid (EDTA)–treated whole-blood tubes. Infected horses develop high IFA titers (>1:640) within days of infection, 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. It is generally believed that horses with PHF should have a titer of 1:80 or greater at the onset of signs; consequently, a negative titer indicates this disease is unlikely. Vaccination for PHF results in positive titers that usually disappear by 6 to 9 months. PCR offers the advantage of excellent sensitivity without the potential for interference from vaccination.[47,48]

Diagnostic Tests for Feces. Fresh fecal samples from horses with diarrhea should be submitted for aerobic culture, with a specific request for *Salmonella* spp. identification. These cultures require special media and antigens for serogroup identification and are readily available through most, if not all, commercial laboratories. Multiple cultures are preferable. Recovery of pathologic organisms can be difficult when feces are very watery, and thus the most productive cultures are performed on feces with at least some substance. Culture of a rectal mucosal biopsy sample may also improve the recovery rate.[49] PCR is reported to be a more sensitive method for detection of salmonella in feces.[50-53] The diagnostic significance of horses positive by PCR but negative by culture of multiple fecal samples remains to be determined. (Chapter 38 discusses *Salmonella* spp. diagnostic tests and their interpretation in detail.)

Anaerobic culture of feces should also be requested to facilitate detection of clostridial organisms. Strict anaerobic handling of the feces is critical to successful culture, especially for *Clostridium difficile*.[54] Recovery of *C. difficile* organisms is dramatically reduced after storage for 72 hours in aerobic conditions at 4°C.[54] Because clostridial organisms can be cultured from the feces of some normal horses, toxin detection is preferred for a diagnosis of clinically relevant disease. Commercial ELISA assays are available for detection of *C. difficile* toxins A and B, as well as the enterotoxin of *Clostridium perfringens* (CPE). Genotyping of *C. perfringens* isolates is also commercially available. Aerobic storage of fecal samples is unsuitable if samples are intended for culture of *C. difficile* because of the short length of time that organisms remain viable when stored under those conditions. However, toxins remain stable for at least 30 days when fecal samples are stored aerobically.[54] Many diagnostic laboratories will perform toxin testing, and some will provide packages including both culture and toxin analysis. (Chapter 44 discusses diagnosis of enteric clostridial infections in detail.)

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 (see Chapters 58 and 62).

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 therapy may be indicated, depending on the etiologic agent identified. For many horses with acute colitis, initial IV fluid replacement is required because of tremendous volume losses. Typically, mild to moderate acidemia is corrected by restoration of plasma volume with an alkalinizing solution such as lactated Ringer's or Normosol-R. Sodium chloride solutions (0.9%) should be avoided because they can be acidifying and may worsen edema. In horses with severe dehydration, initial therapy with hypertonic saline may be used to restore circulatory volume, but must be followed by administration of isotonic fluids. Alternatively, hydroxyethyl starch (Hetastarch) can also be used for quick expansion of plasma volume while also inducing a rapid increase in colloid oncotic pressure.

Other goals of therapy include reducing inflammation, pain control, and limiting the effects of endotoxemia (see Chapter 37). Drugs used for theses purposes include nonsteroidal antiinflammatory drugs (NSAIDs), such as flunixin meglumine, which have analgesic, antiinflammatory, and antiendotoxemic properties.[55] As with other NSAIDs, the clinician must take care to avoid use of flunixin in horses with renal compromise, moderate to severe dehydration, NSAID toxicity, or right dorsal colitis. Adjunctive therapy with polymyxin B sulfate[56,57] and pentoxifylline[55,58,59] is suggested to combat the effects of endotoxemia.

Chronic Diarrhea in Adult Horses

Etiology

Chronic diarrhea is usually defined as diarrhea lasting longer than 4 weeks.[60] Fecal consistency can vary widely. Although many specific diseases can result in chronic diarrhea, identification of the inciting cause in a patient frequently remains elusive. Occasionally, problems of a non-GI nature, such as hepatic disease or abdominal abscessation, result in diarrhea. Infectious causes of chronic diarrhea include chronic salmonellosis (see Chapter 38) and parasitism with large or small strongyles (see Chapter 61). Recently, the spirochete *Brachyspira pilosicoli* was implicated in a herd outbreak of chronic diarrhea in weanling-age horses.[61] Noninfectious inflammatory causes of chronic diarrhea include granulomatous enteritis or colitis, neoplasia (predominantly lymphosarcoma), sand irritation, and right dorsal colitis (Fig. 3-5). Noninflammatory causes include a range of problems, with the common theme of disruption in the flora of the large intestine. This may or may not be related to a dietary disruption, and many affected horses have few other clinical signs. Regardless of the inciting cause, horses with chronic diarrhea remain very difficult to treat and have a guarded prognosis.

Diagnostic Approach

A minimum database 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 these diagnostic procedures are frequently normal, and further recommended analyses include a comprehensive fecal examination and rectal biopsy.

Comprehensive fecal analysis should include assessment for parasites (grossly and by fecal flotation or McMasters quantification), aerobic culture for *Salmonella* (five samples at a minimum 12-hour interval, as for acute diarrhea), water suspension for sand, unstained wet mount for protozoa and parasites, new methylene blue stain for fecal leukocytes, and Gram stain to determine the ratio of gram-positive to gram-negative bacterial flora.

Rectal biopsy is a simple, relatively noninvasive procedure.[62] Two samples should be obtained and submitted for culture *(Salmonella)* and histopathology. Histopathologic examination is most helpful for diagnosis of inflammatory and neoplastic bowel diseases.

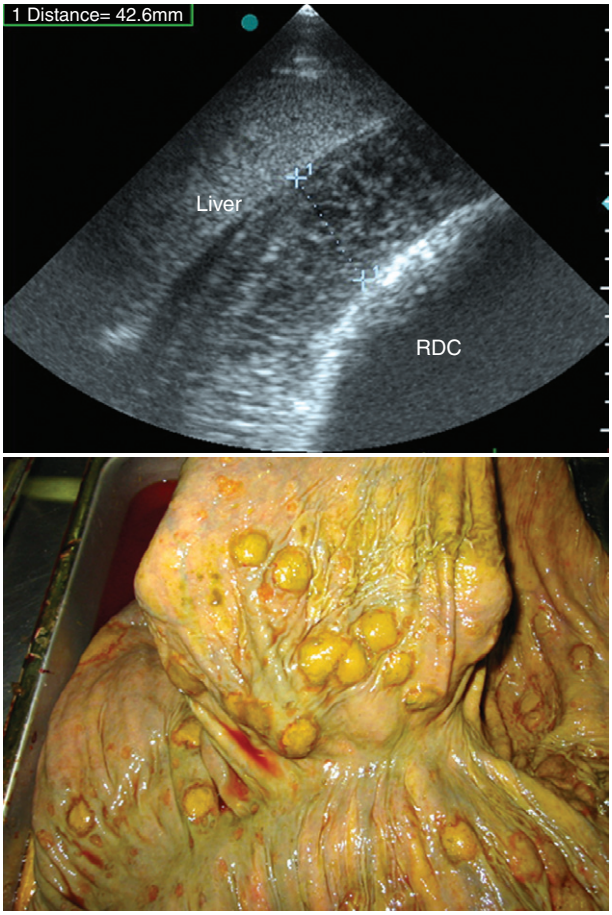

Fig. 3-5 **A,** Abdominal ultrasound image from aged pony with liver disease, low white blood cell count, and fever demonstrating greatly thickened right dorsal colon. **B,** Right dorsal colon of horse with thickened colon and actual granuloma formation in wall of intestine. (Courtesy Dr. Michael Porter.)

Therapy

If a specific diagnosis is achieved, directed therapy should be initiated. (See individual chapters for a more detailed description of directed therapy based on the causative organism.) In all cases, free-choice access to fresh water is critical to maintenance of hydration. Many horses will consume balanced, isotonic electrolyte water, and such a solution should be offered in addition to fresh water. 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 provide a cure.

Nonspecific therapy for horses with chronic diarrhea may include transfaunation or administration of iodochlorhydroxyquin. Detailed descriptions of *transfaunation* procedures 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. Because these sources are rarely available in proximity to the affected animal, the procedure itself may be a fairly daunting task. After appropriate transfaunate is

obtained, the clinician must 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 in the horse. However, the potential value of transfaunation was recently highlighted during a herd outbreak possibly related to the spirochete *Brachyspira pilosicoli*.[61]

Iodochlorhydroxyquin, an 8-hydroxyquinolone derivative (also called clioquinol) originally recommended for treatment of trichomoniasis, has long been recommended for the treatment of chronic diarrhea.[63] Although chronic diarrhea in horses is more likely to result from disruption of the normal intestinal flora than from infection, some horses responded favorably to therapy. The response to treatment with iodochlorhydroxyquin is highly variable; it may worsen diarrhea in some horses. Therapy may result in improvement in fecal consistency, with reversion to diarrhea within a few days of discontinuing drug administration.[61]

Prognosis

Regardless of the inciting cause, if a horse has diarrhea for at least a month, the prognosis for complete recovery is guarded. The prognosis worsens with the duration of diarrhea.

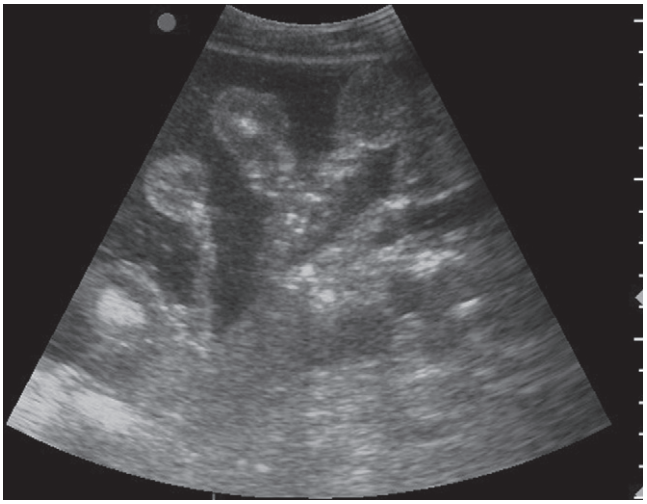

Fig. 3-6 Abdominal ultrasound image from a horse with septic peritonitis demonstrating increased quantities of hyperechoic fluid, with small intestine floating throughout the fluid (intestinal buds).

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 may include onset (acute or chronic), distribution (localized or diffuse), origin (primary or secondary), and infectious nature (septic or aseptic). Acute, diffuse, septic peritonitis secondary to GI disease is the most common manifestation.[64]

Etiology and Clinical Findings

Most cases of peritonitis occur secondary to a GI event (e.g., perforation of any portion of GI tract), intestinal ischemia, DPJ, colitis, neoplasia, verminous arteritis, intestinal mural abscess, or other causes.[65,66] 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 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, often with no clear predominance of one type. *Enterobacteriaceae*, *Streptococcus* spp., and *Staphylococcus* spp. are most often isolated from peritoneal fluid samples.[66,67] Common anaerobic isolates include *Bacteroides*, *Clostridium*, and *Bacillus* species. In foals, peritonitis is most frequently associated with *Streptococcus* and *R. equi* infections. Several case series describing peritonitis associated with *Actinobacillus equuli* have been reported.[68-70] Initial reports of *A. equuli* peritonitis originated solely in Australia, but one case was recently reported from the United Kingdom.[71]

Clinical signs of peritonitis in horses are variable and may include fever, depression, abdominal pain, diarrhea, and weight loss.[65] Depending on severity and localization, signs may also include those of endotoxemia and shock. Clinical signs in horses with *A. equuli* peritonitis include depression, inappetence, lethargy, and mild to moderate abdominal pain acutely or weight loss in a chronic form.[69,70] Postpartum mares with peritonitis secondary to a uterine perforation typically present with fever and depression, with or without abdominal pain.

Diagnosis

Definitive diagnosis of peritonitis is based upon an elevated TNCC in peritoneal fluid (>10,000 cells/μL). Culture of peritoneal fluid should be performed in all suspected cases, but this procedure has a low sensitivity, with only 9.5% to 32.5% of samples yielding positive growth.[65-67] Total cell count can be increased after enterocentesis, abdominal surgery, or open castration.[72-76] Thus, additional parameters must be considered in these populations. Abundant hypoechoic or variably echogenic peritoneal fluid (evident on abdominal ultrasound examination), fever, depression, and abdominal pain can all support the diagnosis (Fig. 3-6). A decrease in peritoneal fluid pH (<7.3) or glucose (<30 mg/dL) suggests the presence of septic peritonitis.[76] Peritoneal fluid cytology will typically reflect a septic process, with abnormalities ranging from the presence of bacteria or plant material to degenerate neutrophils (Fig. 3-7). If GI contents or plant material are evident, the clinician should take care to differentiate between GI rupture and enterocentesis. At sampling, alterations in TNCC and cytology should indicate peritonitis; enterocentesis can result in an elevated TNCC within 4 hours.[75] If a differentiation 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 can be increased for up to 7 days, but the total protein and TNCC should remain within normal limits.[77]

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.

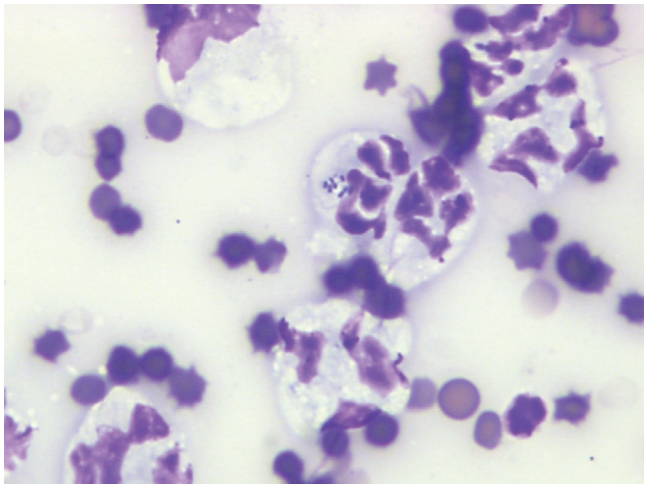

Fig. 3-7 Photomicrograph of peritoneal fluid from horse with septic peritonitis demonstrating toxic neutrophils with intracellular bacteria. (Courtesy Dr. Michael Porter.)

This should include correction of fluid deficits, acid-base and electrolyte imbalances, and colloid oncotic pressure. Anti-inflammatory and antiendotoxic therapies are also clearly of benefit (see Chapter 37). 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 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. Because many *Bacteroides* species are resistant to β-lactam antimicrobials, metronidazole should be included in the antimicrobial therapy plan if anaerobic involvement is suspected. Enrofloxacin may replace gentamicin in the treatment regimen if warranted. The lipophilic nature of enrofloxacin can provide increased penetration into the peritoneal cavity. Neonatal foals with peritonitis should receive an antimicrobial regimen similar to that suggested for adults, although amikacin is frequently substituted for gentamicin because of increased sensitivity of commonly isolated organisms.[78] A combination of azithromycin or clarithromycin plus rifampin provides reasonable coverage for older foals or weanlings, because *Streptococcus* and *R. equi* are often associated with disease in these populations if a primary GI lesion is not suspected.[78] 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 because of the resistance of some isolates.[70]

Abdominal drainage and lavage can help remove excess fluid, foreign materials, fibrin, and bacterial products from horses with peritonitis. Postoperative lavage decreases the incidence of experimentally induced abdominal adhesions in horses undergoing exploratory celiotomy[79] (Fig. 3-8). Open surgical exploration provides the most effective and thorough examination of all peritoneal surfaces and is recommended if GI perforation or ischemia is suspected, as well as in any other horses in which correction of a primary lesion is indicated. A ventral abdominal drain can either be placed at surgery or in the standing horse with sedation and local anesthesia. Techniques are described in detail elsewhere.[78,80]

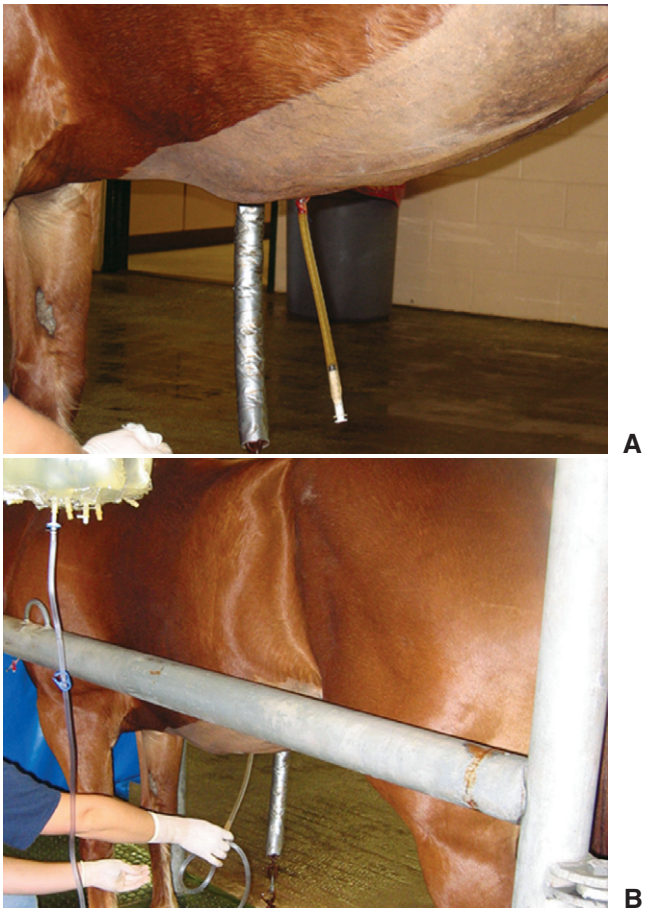

Fig. 3-8 **A,** Abdominal drain placement in most ventral point of abdomen in horse with septic peritonitis. **B,** Abdominal lavage system using the drain shown in *A*.

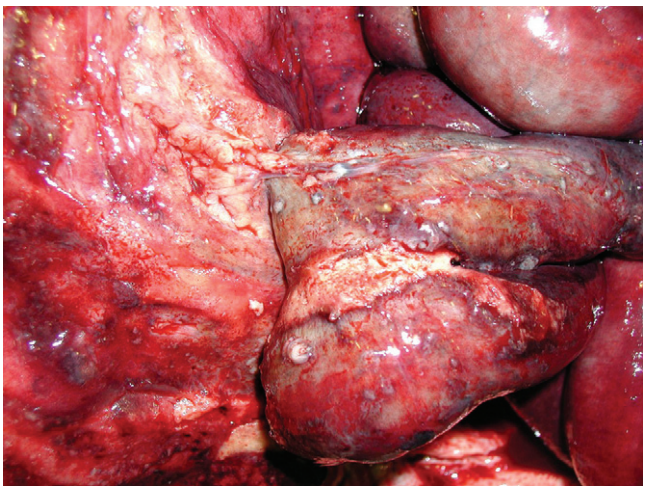

Fig. 3-9 Two-year-old horse with septic peritonitis and orchitis. The initiating cause was unknown; *Streptococcus equi* subsp. *zooepidemicus* was cultured from the abdomen of this horse.

Peritoneal lavage is typically performed by infusion of 10 to 20 liters of a balanced isotonic electrolyte solution (e.g., lactated Ringer's, Normosol-R) into the peritoneal cavity twice a day for 3 to 5 days, until the lavage solution becomes clear, or until the catheter becomes clogged with fibrin or omentum. Hypertonic solutions should be avoided because they may cause fluid shifts into the peritoneal cavity. The addition of povidone-iodine to a balanced solution should be avoided; concentrations as low as 3% may induce peritoneal inflammation.[81] Other agents, such as antibiotics and heparin, have also been suggested as components of peritoneal lavage solution, but data demonstrating their benefit are lacking. Active (or closed-suction) abdominal drains have also been advocated, with similar benefits and potential complications to other methods.[80] Lavage with a plain isotonic solution did not alter the pharmacokinetics of gentamicin administered systemically.[82]

Prognosis

The prognosis is grave for horses with peritonitis secondary to GI rupture. Reported survival rates for horses with peritonitis vary but can be as high as 59.7%[66] (Fig. 3-9). Some of the variability in reported survival percentages may be related to inclusion criteria, mainly whether or not horses with GI rupture were included. Septic peritonitis after abdominal surgery is reportedly associated with high mortality (56%).[66] Peritonitis associated with *A. equuli* carries a very favorable prognosis, and all horses in these reports responded to medical therapy, if attempted.[68-70]

REFERENCES

See the CD-ROM for a list of references linked to the abstract in PubMed.

CHAPTER • 4

Central Nervous System Infections

Kathy K. Seino

Infections of the central nervous system (CNS) of horses, although uncommon, are some of the most devastating and frequently fatal diseases in horses. Diseases such as *equine protozoal myeloencephalitis* (EPM) and *West Nile virus encephalomyelitis* (WNE) have had a significant economic impact on the equine industry in recent years and stimulated investigations into preventive, diagnostic, and therapeutic alternatives for CNS infections in horses.

Viral, bacterial, rickettsial, protozoal, parasitic, and fungal pathogens may cause CNS infections in horses (Table 4-1). In small animals and in humans the causes of meningoencephalitis, in order of decreasing frequency, are viral, bacterial, protozoal, rickettsial, parasitic, and fungal, whereas in the horse the most frequently diagnosed CNS infections are probably of viral and protozoal origin.[1,2] In an Australian study, 30 of 450 horses with neurologic disease had an infectious or inflammatory disease, and 11 of these 30 had meningitis.[3] This study did not reflect the emergence of *West Nile virus* (WNV) in the United States in 1999 or account for CNS diseases that are present in North America, such as *Eastern equine encephalomyelitis* (EEE) or EPM.

Regardless of the type of etiologic agent involved, CNS infections require an accurate and rapid diagnosis and implementation of an appropriate course of treatment by the attending clinician. CNS infection should be suspected in horses with abnormal mentation, seizures, blindness, multiple cranial nerve abnormalities, and general proprioceptive deficits. Infections involving primarily the spinal cord may manifest as limb weakness, incoordination, and stiffness, with or without associated cerebral dysfunction. The reader is referred to chapters on individual diseases for detailed description and discussion of EPM (see Chapter 59), WNV (see Chapter 21), alphavirus encephalitides (see Chapter 20), rabies (see Chapter 19), equine herpesvirus myelopathy (see Chapter 13), *Streptococcus equi* subsp. *equi* (see Chapter 28), and *Anaplasma phagocytophilum* (see Chapter 42). This chapter provides an overview of CNS infection, pathogenesis, diagnosis, and treatment, with discussion of miscellaneous CNS infections not covered elsewhere in this text.

The appropriate term for infection and resultant inflammation of the CNS is determined by the specific area of the nervous system affected. Inflammation of the brain, meninges, spinal cord, and peripheral nerves is termed *encephalitis*, *meningitis*, *myelitis*, and *neuritis*, respectively. *Rhomboencephalitis* and *cerebellitis* refer to localized inflammation of the brain stem and cerebellum, respectively.[4,5] Frequently, more than one tissue or anatomic site may be affected. *Meningoencephalitis* is inflammation of the meninges and brain, and *meningoencephalomyelitis* is inflammation of the meninges, brain, and spinal cord. Inflammation of the brain and spinal cord, without meningeal involvement, is termed *myeloencephalitis*.

Infection of the CNS can also result in focal suppuration of the brain parenchyma or spinal cord and formation of abscesses. Localized areas of infection between the outermost meningeal layer (dura mater) and the skull and vertebral column are termed *epidural abscesses*. Inflammation between the outer two layers of the meninges (dura mater and arachnoid) is termed *subdural empyema*.[1]

NEUROANATOMY AND DISEASE

Brain and Meninges

Inside the protective barrier of the skull, the brain is surrounded by three layers of meninges: the outermost dura mater, or *pachymeninges*, and the *leptomeninges*, consisting of the inner