HOW I TREAT...

Horses with Peritonitis

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Peritonitis can be induced by a number of infectious (bacterial, viral, fungal, parasitic) and noninfectious (traumatic, chemical, neoplastic) causes. Bacterial agents are the most well-recognized cause of peritonitis in horses, and bacterial peritonitis is often described as primary or secondary based on the mode of entry of the infectious agent. Primary peritonitis usually occurs only in association with impaired host defenses. Secondary peritonitis, by far the most common form in horses, occurs when intestinal contents leak through damaged or perforated intestinal wall or via inoculation from external trauma.[1]

Mortality from peritonitis in horses ranges from 30% to 67%.[1] Successful treatment and an accurate prognosis depend on correct identification of the cause as well as assessment of the severity of the inflammatory response (Figure 1).

CLINICAL SIGNS

Clinical signs and changes in the hematology profile and serum electrolyte concentrations may be minimal during the initial stages of peritonitis if inoculation of the peritoneal cavity did not involve concurrent visceral damage.[2] If contamination is limited and the host defenses can eliminate the initial contaminant, the episode may not be detected clinically. If bacteria trigger an extensive influx of neutrophils (and signs of bowel obstruction or rupture do not predominate), however, clinical signs will be consistent with the degree of peritoneal inflammation. Typical signs of peritonitis include mild abdominal pain (expressed most commonly as splinting of the abdomen), depression, anorexia, decreased borborygmi, and either decreased fecal production or diarrhea. Ileus may be sufficient to

cause gastric reflux.[3] Unless the peritonitis is accompanied by intestinal obstruction, signs of colic are usually not predominant.

A systemic response to peritonitis is characterized by pyrexia, tachycardia, metabolic acidosis, and, occasionally, decreases in serum sodium, chloride, and potassium concentrations. Although an inflammatory leukogram with neutrophilia is possible, involvement of gram-negative organisms within the peritoneal cavity may result in endotoxin release with resultant neutropenia. Initially, both the packed cell volume and serum protein concentration increase because of rapid losses of fluid from plasma. As this movement of fluid continues, however, serum protein concentration will decrease because of protein loss into the peritoneal cavity. Dehydration may become severe as protein-rich fluid enters the peritoneal cavity. Plasma fibrinogen concentration also may increase as a result of the severe systemic inflammatory response associated with peritonitis.[4]

Chronic peritonitis that has resulted in abdominal abscess formation may be difficult to diagnose because clinical signs tend to be mild and intermittent. A clinical history of intermittent, low-grade abdominal pain; pyrexia; tachycardia; anorexia; or weight loss is typical of horses with chronic intraabdominal abscessation. The presence and severity of these signs are affected by such factors as abscess wall integrity, location of the abscesses, and the effect of the localized infection on intestinal motility.[5] With chronic peritonitis, either mild anemia associated with a normal complete blood count or neutrophilia may be present. Plasma fibrinogen concentrations may be normal but are more often increased. A reduced serum albumin concentration and
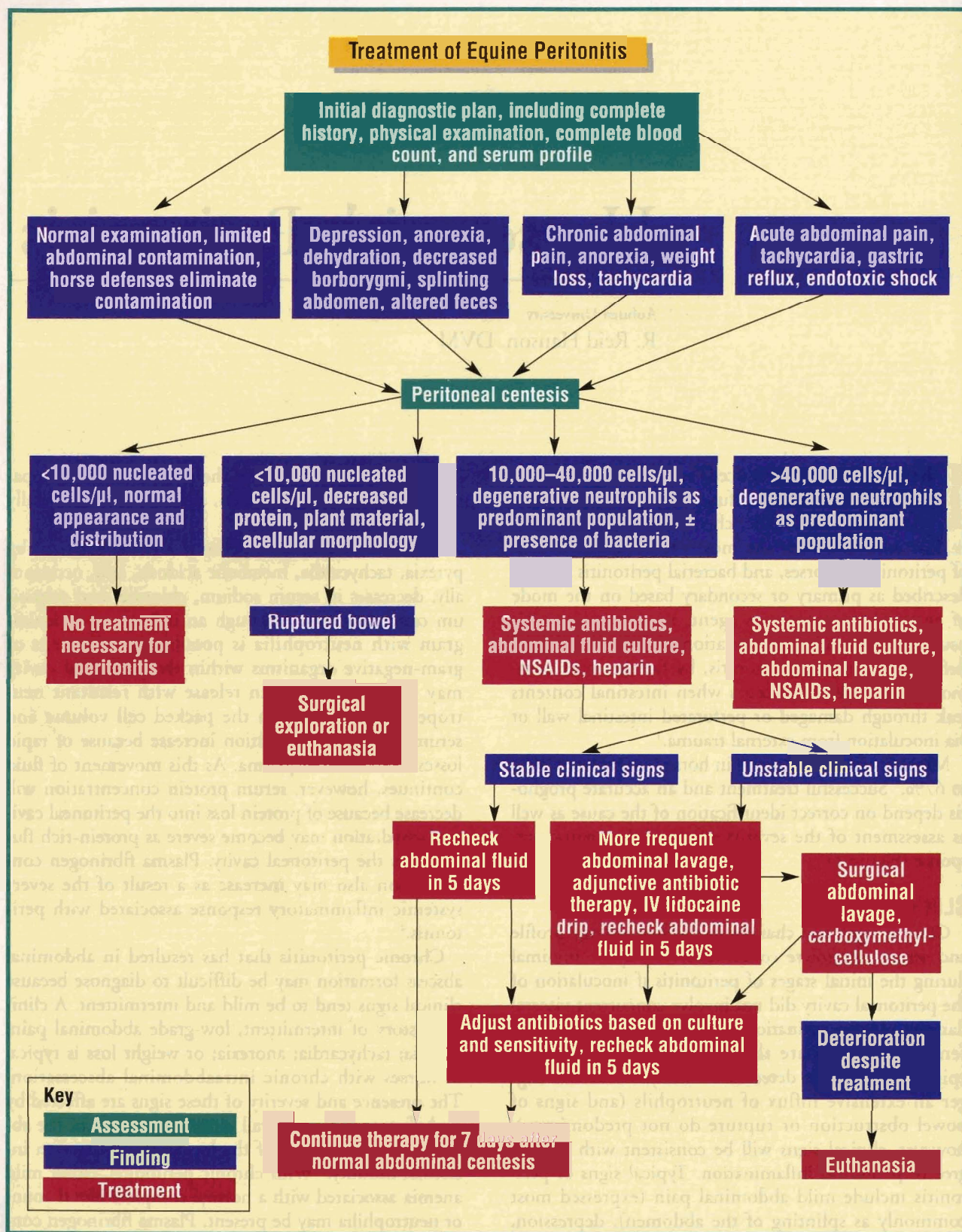
Treatment of Equine Peritonitis

Initial diagnostic plan, including complete history, physical examination, complete blood count, and serum profile

Normal examination, limited abdominal contamination, horse defenses eliminate contamination

Depression, anorexia, dehydration, decreased borborygmi, splinting abdomen, altered feces

Chronic abdominal pain, anorexia, weight loss, tachycardia

Acute abdominal pain, tachycardia, gastric reflux, endotoxic shock

Peritoneal centesis

<10,000 nucleated cells/μl, normal appearance and distribution

<10,000 nucleated cells/μl, decreased protein, plant material, acellular morphology

10,000–40,000 cells/μl, degenerative neutrophils as predominant population, ± presence of bacteria

>40,000 cells/μl, degenerative neutrophils as predominant population

No treatment necessary for peritonitis

Ruptured bowel

Surgical exploration or euthanasia

Systemic antibiotics, abdominal fluid culture, NSAIDs, heparin

Systemic antibiotics, abdominal fluid culture, abdominal lavage, NSAIDs, heparin

Stable clinical signs

Unstable clinical signs

Recheck abdominal fluid in 5 days

More frequent abdominal lavage, adjunctive antibiotic therapy, IV lidocaine drip, recheck abdominal fluid in 5 days

Surgical abdominal lavage, *carboxymethyl-cellulose*

Adjust antibiotics based on culture and sensitivity, recheck abdominal fluid in 5 days

Deterioration despite treatment

Continue therapy for 7 days after normal abdominal centesis

Euthanasia

Key

| Assessment |
| Finding |
| Treatment |

Figure 1—An algorithm for the treatment of peritonitis in horses. (*IV* = intravenous)

an increased serum globulin concentration are suggestive of chronic infection and loss of albumin into the peritoneal cavity.

DIAGNOSIS

Nucleated cell counts in the peritoneal fluid exceeding 10,000 cells/µl are indicative of inflammation. Identification of large numbers of degenerative neutrophils (karyorrhexis and karyolysis) associated with intra- or extracellular bacteria as the predominant population in the peritoneal fluid accompanied by clinical signs of depression and fever is strongly suggestive of clinically significant bacterial peritonitis. Peritoneal fluid nucleated cell counts in excess of 40,000 cells/µl with a predominant population of healthy neutrophils are also suggestive of peritonitis but should be considered with the previous history and clinical signs. Definitive diagnosis of bacterial peritonitis is generally based on identification of phagocytized bacteria in peritoneal fluid obtained via sterile centesis. Nucleated cell counts in excess of 100,000/µl with degenerate neutrophils may be detected in the peritoneal fluid as soon as 3 hours after accidental enterocentesis or surgical exploration in horses without evident infection.[1] Enterocentesis often causes septic peritonitis in which bacteria are seen.

Low peritoneal protein concentrations may occur in horses with peritonitis in which concurrent systemic hypoproteinemia or a large volume of fluid influx into the abdominal cavity has diluted the protein concentration in the peritoneal fluid. Therefore, peritoneal protein concentrations should be interpreted in conjunction with the serum protein concentrations and apparent peritoneal fluid volume.

Peritoneal fluid should be examined for cell types and foreign material. Plant material suggests bowel rupture or an enterocentesis. Serosanguineous peritoneal fluid with large numbers of erythrocytes in the absence of platelets suggests that vascular damage has occurred and supports a diagnosis of strangulating obstruction of bowel or bowel infarction. Because peritoneal fluid may be localized by loops of bowel or adhesions, cytology of peritoneal fluid should be interpreted with caution in horses with a history of either very acute (less than 4 to 6 hours) or more chronic (longer than 4 days) abdominal disease. Cytology of peritoneal fluid may be abnormal for up to 3 weeks after invasion of the peritoneal cavity for diagnostic or therapeutic reasons.[6]

When deciding on a course of treatment or developing an accurate prognosis, it is important to determine the potential causes of peritonitis and whether it is septic or nonseptic. Some procedures, such as castration, inadvertent enterocentesis, and routine celiotomy, typically cause subclinical peritonitis. These procedures usually result in peritonitis that is clinically insignificant, but evidence of sepsis is generally a reason to treat these horses for peritonitis. Samples of peritoneal fluid should be submitted for both anaerobic and aerobic cultures. In long-standing cases of peritonitis, cultures may be negative, reflecting either the dilution of organisms from the massive inflammatory fluid influx or clearance of bacteria before abdominocentesis.

THERAPY

The treatment of peritonitis involves elimination of known inciting causes, antimicrobial therapy, removal of inflammatory exudate, control of intestinal adhesions, and systemic support.[1] The relative importance of each and the specific methods used should be based on the stage of infection and the presence of concurrent disease.

Elimination of the Inciting Cause

Peritonitis of traumatic origin often results from abdominal surgery, castration, or blunt trauma; affected horses will frequently have signs that correlate with the severity and location of trauma. Debridement of penetrating wounds followed by either primary closure or diligent wound care is an essential part of therapy for secondary peritonitis. Blunt trauma to the abdomen can cause abscesses in the body wall and peritoneum.

Chronic peritonitis can lead to the development of intraabdominal abscesses. Because diagnostic confirmation of the presence of an abdominal abscess and surgical accessibility are limited in horses, treatment usually involves long-term administration of antibiotics. Drainage of intraabdominal abscesses by marsupialization is possible only in patients in which the abscess is focal, well encapsulated, and located in the vicinity of the ventral abdominal wall and in which no adhesions are present.

Uterine or vaginal tears without major vessel damage typically result in slow, progressive clinical signs of peritonitis because of the absence of intestinal distention and the presence of relatively low numbers of pathogenic organisms in the uterine lumen compared with the intestine. Complications related to surgery for ovariohysterectomy performed because of chronic pyometra associated with cervical abnormalities, uterine neoplasia, or removal of a macerated fetus can be reduced by removing as much of the uterus as possible, minimizing peritoneal contamination with uterine contents, and providing a secure closure of the caudal reproductive tract.

Peritonitis also occurs secondary to bladder rupture or intraabdominal urachal rupture, but clinical and

clinicopathologic signs of uroperitoneum are more likely to be noted than is peritonitis. Uroperitoneum is an important consideration in neonatal foals and to a lesser extent in postpartum mares. Correct diagnosis is based on recognition of the relevant facts in the history and detection of high peritoneal fluid creatinine concentrations compared with serum creatinine concentrations. Identification of calcium carbonate crystals in peritoneal fluid is consistent with rupture of the urinary tract. Treatment includes removal of peritoneal urine and surgical repair of the site of disease.

Antimicrobial Therapy

Antimicrobial agents are recommended in the treatment of equine peritonitis because host immune and phagocytic cell capacities may be depressed or overwhelmed. The organisms most commonly isolated from horses with peritonitis include *Escherichia coli* and *Staphylococcus epidermidis. Bacteroides fragilis* is estimated to be present in 10% to 20% of equine patients with positive culture results.[5,7] Synergistic relationships between *E. coli* and *B. fragilis* in which each organism plays an important role in the survival of the other have been reported.[8] Knowledge of specific organisms that persist in horses allows more effective direction of therapy.

As the peritoneum becomes inflamed, drug penetration from the circulation into the peritoneum increases. Intravenous administration of antimicrobial agents provides the most rapid tissue concentrations and should be considered over orally administered drugs, especially during the contamination stage when rapid, effective antibacterial therapy may stop the progression of the process. Broad-spectrum antimicrobial agents are recommended for treatment of the acute, diffuse stages of peritonitis when contamination with multiple organisms is suspected or demonstrated by Gram's stain. A combination of intravenous potassium penicillin (22,000 to 44,000 U/kg four times daily) and an intravenous aminoglycoside, such as gentamicin (6.6 mg/kg once daily) or amikacin sulfate (6 to 15 mg/kg three times daily), is the typical choice for broad-spectrum coverage in the early stages of peritonitis. To avoid nephrotoxicity in horses undergoing treatment with aminoglycosides, care must be taken to maintain adequate hydration and peripheral perfusion.[9]

Oral trimethoprim sulfadiazine (30 mg/kg twice daily) may also provide broad-spectrum coverage when used alone or in combination with penicillin. Oral chloramphenicol (25 to 50 mg/kg four times daily) and oral enrofloxacin (7.5 mg/kg once daily) are broad-spectrum antimicrobial agents that have good peritoneal penetration and can be useful if the culture and sensitivity results warrant their use. Enrofloxacin administered par-

enterally at 5 mg/kg twice daily is not detrimental to the metabolism of articular cartilage in vitro, and reported adverse effects of enrofloxacin on articular cartilage have not been scientifically documented.[10]

Penicillin is an effective drug against most anaerobic organisms with the exception of *B. fragilis.* Oral metronidazole (15 to 25 mg/kg three to four times daily) is effective against most of the penicillin-resistant anaerobes and should be included in the treatment regimen along with appropriate aerobic antimicrobial coverage. A more selective antimicrobial is appropriate for horses with peritonitis associated with lymphadenitis or if a Gram's stain of peritoneal fluid demonstrates only gram-positive organisms. Intramuscular procaine penicillin G at a dose of 22,000 U/kg results in peritoneal fluid concentrations that exceed minimum inhibitory concentrations for streptococcal organisms for 12 hours. Antimicrobial therapy should be continued for at least 7 days after systemic and peritoneal leukocyte counts have returned to normal and clinical signs of peritonitis have resolved.

The effectiveness of systemic antimicrobial therapy for chronic localized peritonitis is less evident than for the acute diffuse form. At this stage, antibiotics must not only penetrate the peritoneal membrane but also the fibrin network surrounding the bacteria. After the antimicrobial agent penetrates the fibrin layer, it must also be effective in the presence of cellular debris in an acidic abscess cavity.[9] Aminoglycosides can penetrate an abscess capsule but are minimally active in the acidic environment of an abscess. Penicillin's efficacy during rapid bacterial growth is related to destruction of bacterial cell walls; thus it has limited efficacy in the abscess environment where bacteria may exist without cell walls and grow at a slow rate.[11] Drugs that penetrate fibrin, such as erythromycin and fluoroquinolones, are effective in the local abscess environment. Foals with intraperitoneal rhodococcal or staphylococcal abscesses can be effectively treated with a combination of oral erythromycin (25 mg/kg three to four times daily) and oral rifampin (3 to 5 mg/kg twice daily). Resistance to rifampin can develop rapidly, and thus the drug should not be administered alone. Protracted antibiotic administration for the treatment of localized peritonitis is necessary; the standard recommendation is 6 to 8 weeks, pending development of antibiotic toxicity or confirmed resolution of the abscess.

Removal of Inflammatory Exudate
Nonsurgical Abdominal Lavage

Mechanical removal of peritoneal inflammatory exudate and the toxic products of the associated cellular response can be beneficial during some stages of peri-

tonitis. Benefits of draining the peritoneal cavity include the removal of bacteria, toxins, debris, and inflammatory mediators; decreased risk of adhesion formation; and improved contact between antimicrobial agents and the peritoneal surface. The efficacy of mechanical removal during early stages of peritonitis can be improved by combining lavage with drainage; this combination has the added theoretic advantage of mechanically dislodging debris that may not be removed by drainage alone. During the acute contaminating stage of peritonitis, lavage will have a dilutional effect, thereby slowing the influx of pathogenic organisms and toxic products into the general circulation. Excess fluid in the peritoneal cavity, however, has the deleterious effect of diluting opsonins, which bind to many contaminants and enhance the ability of phagocytic cells to ingest the opsonized particles. Effective lavage therefore relies not only on achieving broad distribution of the lavage fluid but also on relatively complete removal of the inflammatory exudate.[7]

Nonsurgical lavage has the advantage of being repeatable as long as the ingress and egress portals remain patent. Mushroom or Foley catheters are effective for abdominal lavage and drainage. A 1-cm stab incision is made through an anesthetized area of skin, subcutaneous tissue, and linea alba using a #10 or #15 scalpel blade. The catheters can be placed over a Chamber's mare catheter, which acts as a stylet, to facilitate their insertion. Retrograde infection of the catheter has not been a clinical problem even in catheters remaining in place more than 7 days.[6]

To lavage the abdomen in horses and contact all surfaces, 20 to 40 L of fluid are needed. The addition of antibiotics to the lavage fluid has failed to show a significant benefit in the treatment of bacterial peritonitis beyond that achieved with systemic use of appropriate antibiotics.[12] Similarly, the addition of antiseptic solutions (e.g., povidone–iodine, chlorhexidine) to lavage fluids has not yielded superior results over standard treatment with systemic antibiotics and lavage with balanced electrolyte solutions. Furthermore, the addition of certain antiseptics to the lavage fluid carries a potentially high risk of excessive peritoneal inflammation and death.[12]

Signs of discomfort may be encountered after 10 L of fluid has been infused into the peritoneal cavity, especially if infusion is rapid; sedating the horse or slowing the infusion rate may be necessary. After infusing the lavage solution into the abdomen, the drain is injected with heparinized saline and closed. Walking the horse for 20 minutes may promote distribution of the fluid. The drain is then opened, and the fluid is allowed to

empty into a calibrated container to record the volume retrieved. The majority of infused fluid should be retrieved; however, some fluid may be absorbed. This process is repeated twice daily for 3 to 4 days or until there is cytologic improvement in the peritoneal fluid. Between treatments, the abdominal drain should be filled with heparin and sealed.

Surgical Abdominal Lavage

Surgical intervention should be performed to treat horses with peritonitis resulting from leakage of intestinal contents into the abdominal cavity and to remove vascular-compromised intestine. Surgical lavage has the advantage of direct access to the peritoneal cavity, ensuring optimal distribution of fluid and allowing visual confirmation of the effectiveness of removing gross debris. The principal disadvantage of surgical lavage is the required celiotomy, which limits its repeated use. Lavage is much less effective in the latter stages of peritonitis when contaminants and bacteria are surrounded by fibrin.[12]

Open peritoneal lavage, in which the abdomen is loosely apposed at the linea alba after celiotomy to allow drainage of peritoneal fluid, is used successfully to treat humans and small animals with generalized septic peritonitis.[13] The abdomen is usually closed in humans and small animals in 3 to 5 days, when cytology of the peritoneal fluid indicates resolution of the peritonitis. The primary advantage of open drainage is that all inflammatory debris can be effectively removed from the peritoneal cavity. Horses treated for peritonitis by open drainage of the peritoneal cavity using a knit polypropylene or woven plastic mesh, however, are prone to develop incisional infections after the mesh is removed and the abdomen closed; herniation of the abdominal wall usually does not occur.[14]

Control of Intestinal Adhesions

Intravenous heparin (40 IU/kg twice daily) is believed to inhibit intestinal adhesions, thereby minimizing the localization and entrapment of bacteria, which in turn decreases the effectiveness of antimicrobial agents. Use of systemic heparin in a bowel occlusion study in foals prevented adhesions in only one of four foals versus four of four foals treated with either dimethylsulfoxide or a combination of an NSAID and broad-spectrum antibiotics.[15] Although heparin was shown to decrease the formation of adhesions in a small group of ponies with experimentally induced intestinal ischemia, there are no controlled studies of its use in a large number of horses and no reports of horses with peritonitis treated with heparin.[16]

Sodium carboxymethylcellulose provides surface pro-

tection through a siliconizing effect and decreases contact between serosal surfaces. In an experimental study, instillation of sodium carboxymethylcellulose (7 ml/kg of a 1% solution) into the abdominal cavity after closure of the incision significantly decreased the incidence of adhesions in ponies.[17] In a clinical study of 136 horses undergoing exploratory celiotomy, however, intraperitoneal use of sodium carboxymethylcellulose did not affect the clinical outcome or long-term survival.[18] Although no side effects have been reported in the studies performed in horses, little is known about how carboxymethylcellulose is eliminated. Consequently, this treatment should be used with caution until more information is available.

Systemic Support

Initial patient management should be directed at stabilizing life-threatening conditions. Intravenous fluid therapy is indicated if fluid loss is severe. Care should be taken to avoid administration of excessive fluid volumes when hypoproteinemia exists. If replacement of fluid volume is critical in a hypoproteinemic patient, use of colloids or plasma should be considered. With the exception of hypoproteinemic patients or foals with uroperitonitis caused by bladder rupture, fluid therapy may be directed at replacing circulating volume using a balanced polyionic electrolyte (e.g., lactated Ringer's) solution. In foals with bladder rupture, severe hyperkalemia may be present and fluids containing potassium should be avoided.

The use of NSAIDs is an important adjunct in the treatment of horses with mixed or gram-negative peritonitis to combat the inflammatory consequences of gram-negative septicemia and endotoxemia. These drugs may also improve the overall response to therapy by enhancing the early host phagocytic cell response and by decreasing adhesions and abscess formation via enhancement of macrophage fibrinolytic and general proteolytic capacity.[19]

Lidocaine is useful in providing analgesic support of abdominal pain associated with peritonitis.[a] Administration of intravenous lidocaine (a loading dose of 1.3 mg/kg followed by constant-rate infusion at 0.05 mg/kg/minute) provides sufficient pain relief to inhibit paralytic ileus and anorexia associated with the disease. Lidocaine potentiates other analgesics such as xylazine and butorphanol and blunts the inflammatory response by reducing the chemotactic factors for lysosomes, macrophages, and neutrophils.[20,21]

CONCLUSION

Treatment of peritonitis relies on recognition of the

[a]Personal communication: Meyer G, Auburn University, Alabama, 1999.

stage of the disease and selection of the appropriate treatment option. Careful consideration of clinical history, peritoneal centesis, and fluid culture assists in determining the stage of infection. Successful treatment is based on recognition of the inciting cause, administration of the appropriate antimicrobial agent, removal of inflammatory exudates, control of adhesion formation, and provision of systemic support.

REFERENCES

1. Trent AM: The peritoneum and peritoneal cavity, in Kobluk CN (ed): *The Horse: Diseases and Clinical Management.* Philadelphia, WB Saunders Co, 1995, pp 373–404.
2. Mair TS, Hillyer FG, Taylor FGR: Peritonitis in adult horses: A review of 21 cases. *Vet Rec* 126:567–570, 1990.
3. Markel MD: Prevention and management of peritonitis in horses. *Vet Clin North Am Equine Pract* 4:145–156, 1988.
4. Dyson S: Review of 30 cases of peritonitis in the horse. *Equine Vet J* 15:25–30, 1983.
5. Hawkins JF, Bowman KF, Roberts MC, et al: Peritonitis in horses: 67 cases (1985–1990). *JAVMA* 203:284–288, 1993.
6. Hanson RR, Nixon AJ, Calderwood-Mays M, et al: Evaluation of peritoneal fluid following intestinal resection and anastomosis in horses. *Am J Vet Res* 53:216–221, 1992.
7. Valdez H, Scrutchfield WL, Taylor TS: Peritoneal lavage in the horse. *JAVMA* 175:388–391, 1979.
8. Sawyer RG, Spengler MD, Adams RB, et al: The peritoneal environment during infection. *Ann Surg* 213:253–260, 1991.
9. Semrad DS: Peritonitis, in Robinson NE (ed): *Current Therapy in Equine Medicine 3.* Philadelphia, WB Saunders Co, 1992, pp 236–237.
10. Beluche LA, Bertone AL, Anderson DE: In vitro dose dependent effect of enrofloxacin on equine articular cartilage. *Am J Vet Res* 60:577–582, 1999.
11. Ahrenholz DH, Simmons RL: Peritonitis and other intra-abdominal infections, in Simmons RL (ed): *Surgical Infectious Diseases.* East Norwalk, CT, Appleton-Century-Crofts, 1982, pp 795–801.
12. Schneider RK, Meyer DJ, Embertson RM, et al: Response of pony peritoneum to four peritoneal lavage solutions. *Am J Vet Res* 49:889–894, 1988.
13. Greenfield CL, Walshaw R: Open peritoneal drainage for treatment of contaminated peritoneal cavity and septic peritonitis in dogs and cats: 24 cases (1980–1986). *JAVMA* 191:100–105, 1987.
14. Chase JP, Beard WL, Bertone AL, Goltz K: Open peritoneal drainage in horses with experimentally induced peritonitis. *Vet Surg* 25:189–194, 1996.
15. Sullins KE, White NA, Lundin CS, et al: Treatment of ischemia induced peritoneal adhesions in foals. *Proc Int Symp Gynecol Surg Adhesion Prevent* 2:77, 1992.
16. Parker JE, Fubini SL, Carr B, et al: The use of heparin in preventing intra-abdominal adhesions secondary to experimentally induced peritonitis in the horse. *Vet Surg* 16:459–462, 1987.
17. Moll HD, Schumacher J, Wright JC, et al: Evaluation of sodium carboxymethylcellulose for prevention of experimentally induced abdominal adhesions in ponies. *Am J Vet Res* 52:88–91, 1991.

18. Mueller POE, Hunt RJ, Allen D: Intraperitoneal use of sodium carboxymethylcellulose in horses undergoing exploratory celiotomy. *Vet Surg* 24:112–117, 1995.

19. Rodgers KE: Nonsteroidal anti-inflammatory drugs (NSAIDs) in the treatment of postsurgical adhesion, in Dizerega GS (ed): *Treatment of Postsurgical Adhesions.* New York, Wiley-Liss, 1990, p 119.

20. Rimback G, Cassuto J, Tollesson PO: Treatment of paralytic ileus by intravenous lidocaine infusion. *Anesth Analg* 70:414–419, 1990.

21. Kahoru N, Mikawa K, Takao Y, et al: Intravenous lidocaine attenuates acute lung injury induced by hydrochloric acid aspiration in rabbits. *Anesthesiology* 88:1300–1309, 1998.

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