Advances in Management of Large Intestinal Colic

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Advances in treatment and management of colic caused by diseases localized to the large intestine include the availability of a novel antispasmodic medication, n-butylscopolammonium bromide; enteral fluid therapies; promising outcomes of surgical procedures; use of di-tri-octahedral smectite; dietary modifications; diagnostic use of ultrasonography; and new information about the use of non-steroidal anti-inflammatory drugs. Author’s address: Large Animal Surgery, Department of Large Animal Clinical Sciences, University of Florida, College of Veterinary Medicine, 2015 SW 16 Avenue, Box 100136, Gainesville, Florida 32610; e-mail: mortona@vetmed.ufl.edu. © 2009 AAEP.

1. Spasmodic (Gas) Colic

Spasmodic colic is the result of increased peristaltic contractions in the horse’s gastrointestinal tract and is thought to be one of the most common causes of colic.1 It can be the result of a mild gas buildup within the horse’s digestive tract. The signs of colic are generally mild and respond well to spasmolytic and analgesic medication, and diagnosis is often made based on response to treatment. A novel therapeutic, n-butylscopolammonium bromide, has recently become available in North America for the treatment of spasmodic colic. N-butylscopolammonium bromide is an antispasmodic and anticholinergic drug for IV use in horses. It is labeled for the control of abdominal pain associated with spasmodic colic, flatulent colic, and simple impactions in horses. This drug is an anticholinergic, causing competitive inhibition of parasympathetic activation (through muscarinic receptors) of smooth muscle cells.2 As a result, n-butylscopolammonium bromide impedes peristalsis and reduces the abdominal discomfort associated with abnormal gastrointestinal motility.2 In addition, n-butylscopolammonium bromide reduces rectal pressure and facilitates rectal examinations in horses. The major side effects of this drug are a transient tachycardia and decreased borborygmal sounds. Transient pupillary dilation has also been reported.2,3 The decreased borborygmal sounds are reported to last ~30 min after administration.2,3 Because of the effect of n-butylscopolammonium bromide on peristalsis, multiple dosing should be avoided, and repeated use in impaction colic is contraindicated.

2. Intraluminal Obstruction of the Large Colon

Intraluminal obstruction of the large colon is characterized by blocking of normal transit of ingesta by impaction, sand, enteroliths, course ingesta, or foreign bodies within the lumen. Obstruction usually occurs at sites of anatomic narrowing, including the pelvic flexure and transverse colon.4

Large Colon Impaction

Impaction with dehydrated ingesta is a common cause of colic in horses and may be associated with ingestion of rough fiber (straw, poor quality hay),
ingestion of sand, poor mastication, systemic dehydration, alterations in colonic motility, prolonged stall confinement, abnormal narrowing of the colonic lumen, and non-steroidal anti-inflammatory drug (NSAID) therapy. Diagnosis of large colon impaction is made based on clinicopathological signs, rectal palpation, and, when necessary, ultrasonography. The goals of treatment are management of pain, restoration of systemic hydration and electrolyte balance, hydration and softening of ingesta, and promotion of colonic motility. Enteral fluid therapy administered through a nasogastric or nasoesophageal tube is the most effective way to achieve ingesta hydration and promote colonic motility. This therapy can also restore fluid and electrolyte balance and is less expensive than IV fluid therapy. An effective electrolyte solution is made by combining 53.7 g of NaCl salt, 37.8 g of NaHCO₃, and 3.7 g of KCl salt in 10 l of water. Administration can be performed similarly to IV fluid therapy by connecting a coiled fluid administration line to the container of prepared fluids and to the nasogastric tube. Enteral therapy should be started slowly (5–10 ml/kg/h); if well tolerated, the rate can be increased (10–15 ml/kg/h) after 1–2 h. If signs of abdominal pain persist or worsen, the horse should be evaluated for accumulation of excessive fluid in the stomach. If >2 l of fluid is retrieved from the stomach, enteral therapy should be temporarily discontinued or administration rate reduced, and it might be necessary to start IV therapy in place of or in conjunction with enteral therapy. Response to enteral therapy should be seen within 24–48 h, if no improvement is seen or if, at anytime, signs worsen, the horse should be re-evaluated to ensure accurate diagnosis and for potential need of surgical intervention.

**Enterolithiasis**

Enterolith impaction can be a common cause of colic in certain regions of the country, such as California. Arabians and Arabian crosses, Morgans, American Saddlebreds, donkeys, and miniature horses are more susceptible to enterolith formation than other breeds. Smaller enteroliths may be passed in feces without clinical signs, and large enteroliths may even reside temporarily in the large colon without causing obstruction. Enteroliths are usually composed of struvite, (NH₄)MgPO₄ and are formed and found in the right dorsal colon. Obstruction usually occurs when an enterolith moves distally to the narrower transverse or small colon. Long-term ingestion of large quantities of an alkalinizing feed rich in protein, phosphorous, and magnesium, such as alfalfa, is an important contributing factor to formation of enteroliths. Drinking water with high magnesium content is also associated with development of enteroliths. Intermittent colic may be a common historical finding in many horses with enterolithiasis. Diagnosis of enterolith obstruction is made based on history, demographics, clinicopathological signs, rectal palpation, abdominal radiography, and ultrasonography. Surgery is the only option for removal of a large or obstructing enterolith. Prevention of enterolith formation may be attempted by instituting dietary modifications. In horses at risk, as outlined above, alfalfa should not constitute the majority of roughage intake, and >50% of the roughage should be provided as grass or grass hay. In areas with high magnesium content in the water, an alternate source of water should be provided. Wheat bran should be minimized because of its high phosphorous content. If these modifications are not possible, twice daily administration of an acidifying agent, such as 1 cup of apple cider vinegar, has been recommended, although the benefits of vinegar have not been confirmed.

**3. Extraluminal Obstruction of the Large Colon**

Extraluminal obstruction of the large colon is characterized by obliteration or collapse of the lumen secondary to displacement, volvulus, or herniation, or, less commonly, by deformation or thickening of the colonic wall from stenosis, neoplasia, or abscessation. Because of its lack of mesenteric attachment to the body wall, the equine large colon is freely mobile within the abdomen and prone to displacement. Large colon displacements are common causes of colic and have been classified into left dorsal displacement, right dorsal displacement, and non-strangulating volvulus of the large colon, although other intermediate locations may be seen as well.

**Right Dorsal Displacement of the Large Colon**

Right dorsal displacement of the large colon (RDDLC) occurs when the pelvic flexure and left colons migrate cranially and to the right abdomen until the right colons are located between the cecum and body wall. Depending on the location of the colon and degree of gas distension, horses with RDDLC can exhibit variable degrees of pain. Diagnosis of RDDLC is made based on clinical signs and rectal palpation. Some horses may present with nasogastric reflux and elevated γ-glutamyltransferase related to partial obstruction of the duodenum. When horses with RDDLC are presented early, medical therapy may be attempted using intravenous fluid therapy, analgesics, and withholding of feed. If pain is severe, or there is marked large colon distension or secondary impaction, surgical intervention is recommended.

**Left Dorsal Displacement of the Large Colon**

Left dorsal displacement of the large colon (LDDLC), also termed nephrosplenic or renosplenic entrapment, occurs when the left dorsal and ventral
colons migrate lateral to the spleen in a dorsal direction until they become entrapped in the nephroplenic space. Horses with LDDLC show variable degrees of pain, depending on the location of the colon, amount of colonic gas distension, and presence of secondary gastric distension.9 The diagnosis of LDDLC is based on the presence of abdominal pain, rectal palpation, and ultrasonographic examination of the abdomen.9 Ultrasonography was reported to be diagnostic in 88% of horses with LDDLC.12 A 2.5- or 3.5-MHz ultrasound probe is placed over the 15th to 17th intercostal space in a direction parallel to the ground, and the spleen is imaged. In the normal horse, the kidney is imaged deep to the spleen. In horses with LDDLC, the presence of gas-filled colon dorsal to the spleen excludes imaging of the spleen and is consistent with LDDLC.12 Once a diagnosis of LDDLC is made and the clinician is confident that there are no other abnormalities present, options for therapy are evaluated. These options include medical therapy, rolling under general anesthesia, or surgical correction.

Medical therapy includes administration of IV fluids with electrolyte supplementation, withholding feed, analgesia, and potentially the administration of phenylephrine with or without vigorous exercise. Phenylephrine is an α1-adrenergic receptor agonist that causes vasoconstriction and splenic contraction when administered at 3 μg/kg/min over 15 min.14 Phenylephrine should be administered slowly over 5–10 min at 10 to 20 mg diluted in 60 ml saline for an adult horse ~500 kg while monitoring vital signs.9 Vigorous exercise in the form of lunging at the walk, trot, and canter in both clockwise and counter-clockwise directions for 10- to 15-min intervals may be used in horses free of lameness as tolerated and is more successful in the early course of the disease before the colon becomes distended.15,16

Correction by rolling under general anesthesia is another non-surgical option for LDDLC.15,16 The phenylephrine may be administered to the horse before rolling to aid in correction. The horse is anesthetized and placed in right lateral recumbency, the horse’s left flank and abdomen is vigorously “shaken” as the hind legs are hoisted dorsally until the body reaches a 60° vertical position, and the shaking is continued for a few minutes. The horse may be placed either into right lateral recumbency or placed into left lateral recumbency, rolled to sternal position, and subsequently rolled into right lateral recumbency. This procedure is repeated two more times, and after the last manipulation, the horse is returned to left lateral recumbency for recovery.16 If non-surgical treatment is unsuccessful, surgical correction is indicated. Recurrence rates of LDDLC have been reported between 7.5% and 8.5%.15,19 Prevention of recurrence should include management and feeding practices to minimize the risk of gas distension of the large colon.9 Procedures that have been advocated for the prevention of recurrence of LDDLC include closure of the nephroplenic space, large colon resection, and colopexy.

Large Colon Volvulus

Strangulating large colon volvulus (LCV) is one of the most fatal causes of colic in horses. Large colon volvulus occurs when the colon rotates around its longitudinal axis, usually with the ventral colon moving dorsomedially. Moderate to intense and continuous pain, marked abdominal distension, severe hemodynamic changes, and signs of endotoxemia may be observed early in the course of the disease.11 The prevalence of LCV is increased in geographic areas with high concentration of broodmares, and risk factors include recent parturition, recent dietary changes, and recent access to a lush pasture.9 Diagnosis is based on presence of marked pain, hemodynamic changes, rectal palpation, and, in certain cases, transabdominal ultrasonography. Early in LCV, rectal palpation and hemodynamic parameters can be normal. In cases where diagnosis is unclear, transabdominal ultrasonography can provide an objective assessment of colon wall thickness. From the ventral abdomen, a colon wall thickness of ≥9 mm was 100% specific for LCV in one study.11 In advanced cases, horses have hemoconcentration, electrolyte imbalances, and metabolic acidosis.11 The treatment of strangulating LCV is prompt surgical intervention. Once the colon has been returned to its normal position, the surgeon must decide between large colon resection, colopexy for prevention of recurrence, recovery of the horse without further intervention, or euthanasia.11 The decision about the treatment at surgery is based on the surgeon’s assessment of colonic viability and prognosis, intended use of the horse, and financial constraints. Colopexy to prevent recurrence may be more suitable for non-athletic horses, such as broodmares, than for athletic horses that may be at increased risk of colonic rupture at the colopexy site during athletic activities such as jumping.

Earlier studies report survival rates after surgery of ~35%, although more recent studies have reported survival rates from 56% to 84%, because of prompt recognition, referral, and surgical intervention.21,22 In a recent study of 73 horses with strangulating LCV that underwent large colon resection, short-term survival rate was 74%, with 67.8% of the horses alive after 1 yr.23 In this study, colonic compromise was graded as severe in all horses, and resection was considered essential for survival, and none of the perioperative variables measured were associated with survival.23 In an earlier study, pre-operative variables indicative of poor survival included packed cell volume (PCV) of >50%, rectal temperature >102° F, and a heart rate >80 beats/min.24 Recent use of plasma lactate as a predictor of colonic viability and survival after strangulating LCV indicated plasma lactate concentrations were significantly lower in survivors compared with non-
survivors. Plasma concentration was related to survival with <6 mM having a sensitivity of 84%, a specificity of 83%, and a positive predictive value of 96%. None of the horses in the study on lactate were treated with large colon resection.

4. Cecal Impaction

Cecal impactions may occur primarily (type 1) from excessive accumulation of solid ingesta or secondarily (type 2) as a result of apparent cecal dysfunction. Horses with type 1 cecal impaction typically have a 5- to 7-day onset of abdominal pain similar to large colon impaction, and their impactions consist of solid ingesta. Type 2 cecal impactions are often more difficult to detect because they are most frequently diagnosed in post-operative patients being treated for an unrelated condition, and the cecum is frequently fluid-filled. It is crucial to differentiate cecal impaction from large colon impaction during rectal palpation because cecal impactions have a tendency to rupture before evidence of marked abdominal pain or signs of systemic shock. Medical therapy is often successful in treatment of horses with type 1 cecal impaction characterized by dehydrated cecal contents; however, immediate surgical management is the treatment of choice for cecal dysfunction (type 2) and should be pursued early because it may be difficult to evaluate the integrity of the cecal wall based on clinical examinations, including rectal palpation, alone.

Surgical treatment includes typhlotomy with or without a cecal bypass procedure. If cecal viability is compromised, ileocecal bypass is recommended. Prognosis for horses with cecal impaction is guarded to good depending on integrity of cecum and type of impaction, with a better prognosis for horses treated promptly and horses with type 1 cecal impaction.

5. Enterocolitis

Enterocolitis, also known as acute colitis, is a general term referring to inflammation of the cecum and/or colon with acute onset of diarrhea. Infectious causes of acute colitis in the horse most commonly include Salmonella spp., Clostridium spp., and Neorickettsia risticii. Other causes include parasite infestation, antimicrobial administration, cantharidin toxicosis, NSAIDs, excessive sand ingestion, and Colitis-X. It is a common cause of rapid, severe debilitation and death in horses in severe cases. Clinical characteristics include moderate to severe abdominal discomfort, profuse watery diarrhea, endotoxemia, leucopenia, and hypovolemia. The fatality rate for horses with enterocolitis may exceed 90% in untreated horses, although, depending on severity, many horses that are treated appropriately often respond and gradually recover over a 7- to 14-day period. Treatment of enterocolitis in the horse may be extremely costly because large volumes of IV fluids are often required. There is no specific treatment for any particular cause of enterocolitis in the horse, and treatment strategies should be aimed at rehydration, electrolyte replacement, maintenance of colloid oncotic pressure, amelioration of entero- and endotoxemia and their effects, nutritional support, anti-secretory or adsorptive agents, re-establishment of flora, and antimicrobials, when indicated. A novel commercially available product containing di-tri-octahedral (DTO) smectite is an intestinal adsorbent available to veterinarians for prevention and treatment of diarrhea in horses. In an early study, DTO smectite prevented the development of lincomycin-induced colitis, whereas all untreated (control) horses developed diarrhea and died or were euthanized because of severe colitis. More recent studies have shown that DTO smectite binds Clostridium difficile toxins A and B and Clostridium perfringens enterotoxin, and post-operative administration to horses treated for surgical disease of the large intestine significantly reduced the prevalence of diarrhea (10.8%) compared with placebo-treated controls (41.4%). DTO smectite should be administered early in the course of disease, at a dose of 0.5 kg/500 kg body weight in 4 1 of water through a nasogastric tube one to two times daily until resolution of enterocolitis.

Rapid institution of therapy improves the prognosis for horses with acute colitis; however, horses that continue to have frequent, profuse, watery diarrhea; systemic signs of endotoxemia; ongoing hemoconcentration; and hypoproteinemia have a poor prognosis for survival. Horses with antimicrobial-associated diarrhea may have a worse prognosis than those with other types of acute colitis. Frequent complications include laminitis, thrombophlebitis, debilitation, and marked weight loss.

6. Right Dorsal Colitis

Right dorsal colitis (RDC) is characterized acutely by mucosal and submucosal ulceration, inflammation, edema, and thickening of the right dorsal colon, although it may affect other segments of the colon as well. In chronic disease, fibrosis and stricture formation may be seen. RDC has been associated with administration of excessive doses of NSAIDs, most commonly phenylbutazone, but may also be seen in horses treated with appropriate doses and other types of NSAIDs. NSAIDs cause intestinal ulceration through the inhibition of cyclooxygenase (COX) and subsequent suppression of prostaglandin production. Prostaglandins are important for intestinal mucosal barrier function maintenance, and ulceration may occur in any portion of the gastrointestinal tract after NSAID administration. It is unknown why the right dorsal colon has a predilection for disease. Clinical signs seen commonly in horses with acute RDC include depression, inappetence, lethargy, diarrhea, moderate to severe abdominal pain, pyrexia, and endotoxemia. Weight loss, chronic intermittent colic, diarrhea, lethargy, inappetence, and ventral and distal limb edema are seen in horses with chronic RDC. Hypoalbuminemia is a hallmark of RDC.
and may be severe (<1.5 g/dl). Presumptive diagnosis is usually made based on clinical signs, laboratory findings, and history of NSAID administration. Transabdominal ultrasonography may aid in confirming diagnosis. With a 3.5- to 5.0-MHz transducer, the right 12th to 15th intercostal spaces below the margin of the lung and axial to the liver are imaged, and the right dorsal colon is examined. A mural thickness of >0.5 cm and evidence of colonic edema is supportive of RDC.

Discontinuation of NSAID administration and dietary management are the basis for treatment of RDC and are successful in resolving many cases. In acute cases, fluid and electrolyte therapy may be necessary. Additional pharmacological approaches are aimed at reducing inflammation and promoting mucosal healing. Misoprostol (5 μg/kg, PO, q 6 h) is a synthetic prostaglandin that has been shown to promote mucosal recovery in experimental models of colitis. Sucralfate (22 mg/kg, PO, q 6 h) may promote ulcer healing, although evidence is derived from human studies. Metronidazole (15 mg/kg, PO, q 6 h) may decrease colonic inflammation, as has been shown in people with Crohn’s disease. Hypoproteinemia and decreased colonic oncotic pressure may require treatment with plasma and other colloids, such as hetastarch. Pain control should not include NSAIDs and may be addressed using butorphanol (0.05–0.1 mg/kg, IM or IV, or 0.1 mg/kg, IV, followed by continued rate infusion [CRI] of 13.2 μg/kg/h in isotonic crystalloidal fluids) and/or a lidocaine CRI (1.3 mg/kg, IV, slowly over 5 min and then 3 mg/kg/h in isotonic crystalloidal fluids). Dietary management consists of eliminating fibrous roughage (hay) and feeding exclusively a complete pelleted diet containing at least 30% dietary fiber for at least 3 mo. Corn oil (1 cup, once to twice daily) may be added to increase caloric intake if needed. Psyllium mucilloid may be added to increase production of short-chain fatty acids (5 Tbl, q 24 h or q 12 h). Horses that have severe or recurrent abdominal pain or that are non-responsive to therapy are candidates for surgical resection or bypass of the right dorsal colon. Complications of RDC include laminitis, chronic colic, colonic stricture, colonic rupture, and colonic infarction. The prognosis for horses with either acute or chronic RDC is guarded.

7. NSAIDs and the Colon

Horses with colic are routinely treated with NSAIDs, most commonly (in North America) flunixin meglumine, to provide analgesia and to reduce the effects of endotoxemia. Traditional “non-selective” NSAIDs such as flunixin meglumine and piroxicam do not inhibit COX-1 and COX-2, whereas novel “selective” NSAIDs (coxibs) preferentially inhibit COX-2. Selective COX-2 inhibitors were developed to decrease the gastrointestinal (GI) toxicses experienced by many people after administration of non-selective NSAIDs. Although their use in human medicine has diminished after the discovery of serious cardiovascular side effects, their use is associated with a significant decrease in adverse GI events. These results would seem to support the central tenant that COX-1–derived prostanoids are produced constitutively and responsible for physiologic maintenance of homeostatic mechanisms such as mucosal barrier function, whereas COX-2–derived prostanoids are inducible in nature and responsible for inflammation and pain. Unfortunately, the story is not that simple. In multiple experimental and clinical studies in humans and other species, COX-2 has been shown to be produced constitutively, as well as to be critical for repair of damaged gastric and colonic mucosa. The use of COX-2 inhibitors in human patients with GI disease has been associated with increased risk for intestinal perforation. In horses, flunixin meglumine has been shown to retard recovery of ischemic-injured jejunum, whereas the preferential COX-2 inhibitor meloxicam did not. Although the mechanisms of ischemic-injury and repair are similar between the small and large intestine of the horse, the colon does not behave in the same manner as the jejunum when compared experimentally. In a similar model of ischemia to that reported in equine jejunum, equine colonic mucosal recovery was not retarded by flunixin meglumine both in vivo and in vitro, and results support its continued use in clinical disease characterized by ischemic injury. This is important, because strangulating disease of the colon is different from strangulating disease of the jejunum in horses. Not only does the mucosal recovery appear to be different, but in the majority of instances, strangulating disease of the equine jejunum can be treated surgically by complete resection of injured intestine. In large colon volvulus, resection is often not performed, or, when performed, resection is rarely complete, meaning that some amount of ischemic-injured intestine almost always remains in the horse. In these horses, ongoing absorption of endotoxin is inevitable until the injured mucosa recovers. Until we identify a better therapeutic with comparable good qualities, flunixin meglumine remains an important component of treatment of these horses post-operatively to reduce the morbidity and mortality associated with ongoing endotoxemia.

References and Footnotes
