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INTRODUCTION
Right dorsal colitis (RDC) has been observed to occur in horses in association with non-steroidal anti-inflammatory drug (NSAID) administration; most notably, disease has been observed following high dose phenylbutazone administration. Clinical signs in horses suffering from RDC typically include poor appetite that may progress to complete anorexia, weight loss, and moderate to severe abdominal pain. Many horses develop diarrhea associated with RDC however, some horses maintain normal fecal character, and therefore the finding of diarrhea may not be apparent in all cases of RDC. Early recognition combined with implementation of appropriate therapy and dietary modification will provide the best chance for a favorable outcome.

CLINICAL MANIFESTATION
Common historical complaints include colic that is intermittent in nature, poor appetite, poor weight gain and in some cases ventral edema. In combination with the clinical course of “poor doing”, concurrent NSAID administration is generally in the history. Common case history includes a young, athletic performance horse that has some level of musculoskeletal pain requiring intermittent systemic anti-inflammatory therapy. In some settings, such as if the horse is under the care of a trainer, the owner might be unaware of drug administration, therefore this information must be determined by communication with the caretaker or trainer directly.

- Colic- intermittent mild to moderate in intensity
- Weight loss- particularly with chronic disease
- +/- Diarrhea
- Icterus- associated with inappetence and potential anorexia
- Ventral edema- depending on severity of protein losing enteropathy

ETIOLOGY OF RDC
Factors associated with the development of RDC continue to be investigated. Clearly an association with nonsteroidal medication is clear. In some cases ulcerative lesions involving the right dorsal colon have been induced following appropriate therapeutic protocols such as phenylbutazone at a dose of 4.4 mg/kg PO twice daily for one week. In many instances additional factors such as dehydration or physiologic stress such as competition or severe disease also play a contributing role in development of disease. Although less commonly implicated, flunixin meglumine is capable of inducing colonic ulcers as well. Concurrent administration of phenylbutazone and flunixin prolongs the pharmacologic effect of these drugs and increases the likelihood for the development of toxicity. Young performance horses, horses with chronic lameness, ponies and miniature horses appear most likely to develop RDC. Incompletely understood is the predilection for lesions to involve specifically the right dorsal colon.

Figure 1 demonstrates ulcerative lesions of the right dorsal colon secondary to NSAID toxicosis in two horses. a) Illustrates the transition from normal epithelium to the severely ulcerated epithelium. b) Diffuse ulceration of a severely affected colon.

Although somewhat confusing, at times, infectious disease might need to be investigated in select cases of suspect RDC. Some horses suffering from RDC have been identified to culture positive for Salmonella spp.; the significance of this finding is unknown. It is recognized that some horses will intermittently shed salmonella and yet there is no scientific data to demonstrate that the presence of salmonella will increase the likelihood for developing NSAID toxicosis. In some cases with protracted diarrhea, it might be worthwhile to culture the feces of affected horses since salmonella can cause a protein losing enteropathy and the etiology of protein loss might not be due to NSAID toxicosis.

SPECIFIC DIAGNOSTIC TESTING
Common hematologic abnormalities associated with RDC include anemia, hypoproteinemia, and hypo-albuminemia. Anemia is most likely due to chronic inflammation, although some low level blood loss from the ulcerated right dorsal colon might contribute to this abnormality. Occult blood can be identified in the feces of affected horses, unfortunately highly sensitive tests are unavailable at this time. Rectal palpation within 24 hours of assaying the feces for blood can cause an erroneous false-positive result for fecal occult blood.
The most common hematologic abnormality identified for horses with RDC is hypoproteinemia, which can be marked in select cases. When considering the history, physical examination, urinalysis, peritoneal fluid analysis, and serum biochemistry gastrointestinal loss of protein can be determined. Because of the small molecular size of albumin this is the protein that is preferentially lost in horses suffering from severe gastrointestinal inflammation. Secondary to a reduction in circulating oncotic pressure, due to albumin loss, moderate to severe edema may develop in affected horses. In most cases the leukogram remains within normal limits, however in some cases a leukocytosis might be observed secondary to significant inflammation. Severe ulceration can result in significant endotoxin absorption, therefore leukopenia, neutropenia and a left shift is observed in some cases.

Hypocalcemia is a common change observed on serum chemistry analysis in horses with RDC. Reduced feed intake with continued excretion, loss of protein bound calcium into the gastrointestinal tract and decreased protein-bound calcium associated with hypoalbuminemia and hypoproteinemia are all contributing factors for the reduced serum calcium levels in RDC cases. Since the ionized fraction of calcium typically remains within normal limits, clinical hypocalcemic tetany is rarely observed in horses with RDC. Some horses may be identified to have prerenal azotemia and hyperbilirubinemia secondary to reduced water and feed intake, respectively. In horses that are hemoconcentrated, anemia and hypoproteinemia may not be immediately identified; therefore careful serial monitoring is indicated in suspect cases. Cytologic analysis of peritoneal fluid rarely identifies abnormalities, unless colonic disease is severe enough to have resulted in full thickness damage and subsequent peritonitis develops. In most cases, gross examination of the colonic wall does not reveal abnormalities, yet when the mucosal surface is inspected significant ulceration is found to exist.

ULTRASOUND EVALUATION

Clinical suspicion for RDC can be quite strong with a consistent history of NSAID administration in a colicky, hypoproteinemic, horse, yet diagnostic confirmation can be difficult. Noninvasive techniques have been employed for examining suspect cases and include nuclear scintigraphy and ultrasonographic examination of the right caudal abdomen. The region of interest is between the 11th and 15th rib space in the right caudal abdominal quadrant. Once clipped and prepared with alcohol or contact gel, using a 5 MHz (or higher) linear or sector ultrasound probe, an image of the colon can be clearly defined against the body wall, just caudal and axial to the margin of the liver. As previously published, the approximate thickness for the colonic wall is < 5mm, many normal horses have been observed by this author to have measurements between 2-3 mm in thickness. Multiple measurements can be taken in a relatively rapid fashion so that a mean measurement can be calculated. Clinically affected RDC horses frequently have measurements of colon thickness of > 5mm. Serial monitoring following initiation of therapy can be easily implemented in addition to monitoring serum albumin measurements.

Figure 2 a) Measurement of the right dorsal colon adjacent to the liver in a horse with RDC 0.47 cm. b) More severe thickening of the right dorsal colon 0.64 mm

THERAPEUTIC MANAGEMENT OF RDC

Treatment of right dorsal colitis is initially managed by the discontinuation of all NSAIDs. Dietary modification should include a low residue diet consisting of pelleted complete (e.g. Senior pellet) feed and pelleted hay. Addition of prostaglandin precursors such as linoleic acid, contained within Safflower oil (1 cup daily), and psyllium (2 ounces 1-2 times daily) which contains short chain fatty acids are also advocated for the medical management of RDC in horses. Serial measurements of serum albumin concentrations will give an indication to the level of colonic healing that has occurred. During episodes of severe ulceration serum albumin concentrations might be dangerously low (< 1.5 g/dl), necessitating the need for plasma transfusion. Although protein levels may initially be very low, dietary modification will aid in epithelial healing. When healing is complete, serum albumin levels will remain within normal limits. In general, 4-6 months are required for healing to be complete. Once the protein loss and colonic healing is complete dietary reintroduction might include small amounts of chopped hay with gradual reintroduction of long stemmed hay. In rare cases, colonic stricture may yield an individual unable to properly digest hay and process hay through the damaged
right dorsal colon leading to protracted, chronic colic. Such cases may require permanent dietary modification or potentially surgical resection of the stenotic region of colon.

- Eliminate NSAID administration
- Plasma transfusion in cases with profound hypoalbuminemia (<1.5 g/dl)
- Minimize stress to horse
- Implement dietary modification of low residue diet (complete pelleted diet)
- Safflower oil- 1 cup added to feed daily
- Psyllium 2 ounces 1-2 times daily

PHARMACOLOGIC MANAGEMENT

Although ulceration may be confined to the right dorsal colon, many horses have concurrent gastric ulcers, so the listed pharmacologic agents are recommended for management of horses that have concurrent diseases. Gastroscopy is recommended for documentation and establishing a definitive diagnosis of gastric ulceration.

Prostaglandin E2 analogue

Misoprostol (2.5 µg/kg PO b.i.d) this drug is a synthetic analogue of prostaglandin E2 that has been shown to protect against gastric ulcers induced by NSAIDs in humans and dogs. There is some preliminary data to suggest similar protection in horses that are concurrently administered NSAIDs with misoprostol. Although side effects have not been reported, it has been this authors’ experience that abdominal cramping (colic) can be induced in equine patients, similar to reports for other species.

Sucralfate

Sucralfate (20 mg/kg PO q.i.d.) is a hydroxy aluminum salt of sucrose octasulfate that forms a sticky viscous gel at a pH of less than 4. This gel adheres firmly to the base of ulcers and forms an acid resistant layer that protects against acid and pepsin. Sucralfate is typically used in combination with another agent such as a proton pump inhibitor or histamine receptor type-2 antagonist. Rapid relief from pain associated with gastric ulcers can be observed following treatment with sucralfate. Although no data is available to confirm the effectiveness of sucralfate for the medical management of RDC, this is a common therapy for colonic ulcerative lesions in people. Several authors support the use of sucralfate, at least in the short term, for the clinical management of RDC, particularly in horses that demonstrate significant abdominal pain.

Proton Pump Inhibitor

**Omeprazole** (4 mg/kg PO daily) is a substituted benzimidazole that irreversibly binds to the parietal cell H’/K’ ATPase proton pump that blocks hydrogen ion secretion for the life of the cell. Therefore, omeprazole suppresses acid secretion for a period that outlasts its serum concentrations, therefore it is appropriately dosed at once daily administration. Omeprazole is currently available as an oral paste for horses that has been approved by the FDA as a prescription product to be sold through licensed veterinarians. Omeprazole given intravenously, intramuscularly, or orally can increase gastric pH in horses and ponies and hastens the healing of gastric ulcers induced by flunixin meglumine, and phenylbutazone. When given at the prescribed dose for a period of 28 days, significant ulcer healing has been observed in horses.

Histamine type-2 receptor antagonists (H2 Blockers)

**Cimetidine** (20 mg/kg PO q.i.d) acts as a histamine type-2 receptor antagonist, increasing gastric pH enabling gastric ulcer healing to occur.

**Ranitidine** (6.6 mg/kg PO t.i.d.; 10 mg/kg PO b.i.d.) this drug is also a histamine receptor type-2 antagonist that has been shown to increase gastric pH in adults and foals. Ranitidine has been suggested to be useful for preventing gastric ulcers associated with NSAID administration.

REFERENCES