THE OLDER FOAL: DIAGNOSIS AND MANAGEMENT OF COMMON DISEASES
Kelsey A. Hart, DVM, PhD, DACVIM
Department of Large Animal Medicine
University of Georgia College of Veterinary Medicine

I. GENERAL EVALUATION OF THE OLDER FOAL
Evaluation of the older foal is not strikingly different from evaluation of the adult horse, though some procedures (such as rectal examination) are obviously limited by the size and behavior of the patient. Important additional historical information to obtain in foals includes deworming and vaccination history for the foal, dam and other horses on the farm, signs of any infectious disease in older horses on the farm, and historical occurrence of common foal diseases such as Rhodococcus equi infection on the farm. As foals age, vital parameters approach those of adult horses, and are typically in adult ranges by approximately 6 months of age. In foals less than 6 months of age the systems-based physical examination is comparable to examination of neonatal foals. In weanlings and yearlings, examination is similar to that of adult horses, but certain procedures such as rectal examination are usually limited by the foal’s size and behavior. For that reason, trans-abdominal ultrasound is very useful in the older foal with colic or diarrhea; reasonable image quality of the majority of the abdomen can often be obtained with a rectal probe in many foals.

II. COMMON DISEASES IN OLDER FOALS

Rhodococcus Equi Infection

Presentation and Diagnosis:
The classic presentation for R. Equi infection in foals is a 1-2 month old foal with signs applicable to pneumonia (lethargy, fever, tachypnea, and cough). However, R. equi infection can present in foals as young as a few weeks or age and can impact a number of other body systems in addition to or instead of the respiratory tract, with quite variable presentation. Affected foals may present with uveitis, diarrhea, colic, weight loss, joint effusion, and/or lameness. Leukocytosis, characterized by a mature neutrophilia, and hyperfibrinogenemia are consistent laboratory abnormalities. Immune-mediated anemia and/or thrombocytopenia may also be present, and if severe enterocolitis and diarrhea are a feature, electrolyte and acid-base derangements and hypoalbuminemia may be noted. R. equi should be considered as a differential in any foal with fever, lethargy, respiratory tract disease, diarrhea, weight loss, intermittent colic, joint effusion, or lameness, and aggressive diagnostics undertaken to rule R. equi in or out.

Foals with pulmonary R. equi infection typically have a granulomatous pneumonia, with multifocal pulmonary nodules apparent on thoracic radiographs or ultrasound. Trans-tracheal aspirate is consistent with a septic suppurative or granulomatous inflammation,
with intracellular cocci frequently seen. Mesenteric lymphadenopathy and thickened, sometimes nodular, small intestine and colon are usually found on trans-abdominal ultrasound in foals with *R. equi* enterocolitis, and in some cases large, discrete intrabdominal abscessation (usually in a mesenteric lymph node) is present. *R. equi* infection can be confirmed by culture of the organism or PCR detection of *R. equi* DNA from transtracheal aspirate samples, peritoneal fluid, synovial fluid, or other affected tissues. Because foals can shed virulent *R. equi* organisms in the absence of clinical disease, fecal culture of virulent *R. equi* is not diagnostic for clinical disease due to *R. equi* in an individual foal.

**Management:**
Successful management of foals with *R. equi* depends on early and appropriate antimicrobial therapy. The antimicrobial therapy regime of choice includes a macrolide and rifampin. Recommended dosages are as follows:

1) macrolides:
   a) erythromycin 25-30 mg/kg Po q 6-12 hours
   b) or azithromycin 10 mg/kg PO q 24 hours for 5 days, then q 48 hours
   c) or clarithromycin 7.5mg/kg PO q 12 hours

2) rifampin 5-10 mg/kg PO q 12 hours

Recent studies based on minimum inhibitory concentrations effective against virulent *R. equi* suggest that erythromycin or clarithromycin may be more effective than azithromycin in some cases. In addition, some virulent *R. equi* isolates exhibit resistance to macrolides – isolates that are resistant to one macrolide appear to exhibit some degree of resistance to all three agents. Doxycycline (5-10 mg/kg PO q 12 hours) may be effective these cases.

On farms with recurrent *R. equi* problems, all foals should be monitored closely for fever or signs of respiratory disease. Some large farms find that serial complete blood counts or thoracic ultrasonography in all foals is the best means for early identification of affected foals. Treatment should be continued until the complete blood count and plasma fibrinogen concentration are normal and all clinical evidence of disease is resolved. If pulmonary and/or intraabdominal abscessation is identified with imaging, foals should be treated until radiographic and ultrasonographic abnormalities are resolved, which may take weeks to months. Unfortunately, some foals, especially those with *R. equi* granulomatous enterocolitis, fail to improve even with prolonged appropriate antimicrobial therapy; thus, the prognosis for *R. equi* in foals should be considered guarded in foals with severe disease or gastrointestinal signs.

**Proliferative Enteropathy (PE, *Lawsonia intracellularis* infection)**

**Presentation and Diagnosis:**
PE is occurs most often in weanlings. Clinical disease is most often reported in the autumn months, but this may be related to the presence of susceptible foals based on the northern breeding season rather than true seasonal variation. Affected foals present with fever, weight loss, lethargy, and ventral edema. Increased small intestinal
and large colonic mural thickness is a common feature on transabdominal ultrasound. Leukogram, electrolyte, and acid-base abnormalities are variable, but profound hypoproteinemia and hypoalbuminemia is a common feature due to a severe protein-losing enteropathy. Detection of the organism in the feces with PCR or positive serology are necessary to confirm a diagnosis of PE; both tests are available at the University of Minnesota Veterinary Diagnostic Lab, and are increasing available at other veterinary labs. Since the organism may be variably shed in the feces and some healthy foals may seroconvert in the absence of clinical disease, both tests are useful in suspect clinical cases.

**Management:**
The mainstays of therapy for PE include colloidal support, antimicrobial therapy, and supportive care. Plasma, synthetic colloids, or a combination of the two may be used for colloidal support. Plasma may initially be provided at 20-40 ml/kg, but in many cases multiple transfusions are needed and may be cost prohibitive in some cases. Synthetic colloids such as hetastarch (5-10 mg/kg IV bolus q 24 hours or 0.5 – 1 ml/kg/hr IV CRI) may also be used, but require the ability to monitor colloidal oncotic pressure to tailor therapy as plasma protein concentration will be no longer reflect oncotic pressure. Tetracycline antimicrobials are recommended for PE in horses; in most cases, doxycycline (5-10 mg/kg PO q 12 hours) is effective, but in severe disease intestinal absorption of doxycycline may be impaired and intravenous oxytetracycline (6.6 mg/kg IV diluted q 12 hours) may be warranted for initial therapy. Other antimicrobial agents such as chloramphenicol, erythromycin and rifampin may also be effective against *L. intracellularis*. Additional management in some cases may include fluid and electrolyte therapy, nutritional support, or analgesic therapy. Prognosis for PE in horses is guarded; some affected foals may respond well to early and aggressive therapy, but in some severe cases the intestinal lesions are irreversible and absorptive function can not be restored.

**GASTRODUODENAL ULCER DISEASE**

**Presentation and Diagnosis:**
Gastroduodenal ulcer disease (GDUD) is most common in foals aged 1-6 months, but is occasionally seen in younger foals. Risk factors for development of gastric ulceration include previous or concurrent gastrointestinal disease (especially diarrhea), NSAID therapy, and persistent inappetance or anorexia, but many foals with GDUD develop disease in the absence of these risk factors for unknown reasons. Affected foals are often in poor body condition with rough hair coats, and usually present with mild to moderate – and often episodic – colic and inappetance. Some owners report that the spends increased amounts of time recumbent. Many foals also exhibit fever, bruxism, hypersalivation, inappetance, or diarrhea. Hematologic abnormalities may include mild anemia or leukocytosis and hyperfibrinogenemia, but are not consistent findings. Biochemical parameters are usually within reference intervals, but severe and protracted cases resulting in pyloric or duodenal strictures and pyloric outflow obstruction, a hypochloremic metabolic alkalosis may be present. Evidence of
irregularity or thickening of the gastric wall may be seen on trans-abdominal ultrasound, but rugal folds in normal stomachs may also appear this way, and as the entirety of the stomach cannot be visualized sonographically, lesions are easily missed with this modality. Thus, while findings may be supportive, ultrasound should not be used for definitive diagnosis of GDUD. Fecal occult blood test is also neither sensitive nor specific for GDUD, and thus is not recommended.

Definitive diagnosis of GDUD requires gastroduodenoscopy. In most foals, a 2-3 meter flexible endoscope is necessary for comprehensive examination of the stomach and proximal duodenum. Feed/milk should be withheld for 2-6 hours prior to gastroduodenoscopy (1 hour in very young foals), and may need to be withheld for 24 hours or more in foals with delayed gastric emptying due to pyloric outflow obstruction. Ulceration may occur anywhere in the squamous or glandular mucosa, pylorus, or duodenum. Ulceration in the cardia and distal esophagus may also be present, and is often associated with gastroesophageal reflux due to pyloric outflow obstruction. Pyloric sphincter dysfunction, pyloric stricture, or duodenal stricture may be visible endoscopically, but barium administration and contrast radiography may be necessary to definitely diagnosis such conditions.

**Management:**
Appropriate management of gastroduodenal ulcer disease in foals requires a high index of suspicion for the condition in any febrile, colicky, or poor-doing foal. The mainstays of medical therapy include gastric acid suppression with a proton pump inhibitor (omeprazole 4 mg/kg PO q 24 hours or pantoprazole 1.5 mg/kg IV q 24 hours) and/or a H2-receptor antagonist (ranitidine 6.6 mg/kg PO q 8 hours, cimetidine 6.6 mg/kg PO q 6-8 hours; famotidine 3-4 mg/kg PO q 8-12 hours), gastroprotectant administration (sucralfate 20 mg/kg PO q 6-8 hours), and administration of broad spectrum antimicrobials (as for septic foals above). Additional supportive care with fluid therapy, oncotic support, analgesic therapy, or nutritional support may be indicated. Therapy should be continued until resolution of gastroduodenal ulceration is confirmed endoscopically. If gastric impactions occur or if gastric ulceration does not resolve with 30-60 days of the above therapy, delayed gastric emptying due to pyloric or duodenal stricture should be suspected and additional diagnostics (repeat duodenoscopy, contrast radiography) performed. Pyloroplasty, pyloric bypass (gastroduodenostomy) or duodenal bypass (gastrojejunostomy) may be indicated in foals with pyloric or duodenal stricture. However, these surgeries are technically challenging and extensive post-operative care is often needed until normal gastroenteric motility patterns are established at the anastamosis site.

**References are available from the author.**