Equine Medicine Field Tactics 102: Procedures and Therapies for Gastrointestinal Conditions

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Take Home Message

Liver disease, chronic diarrhea and foal diarrhea can be further diagnosed and managed in a field setting using available, basic, economical instrumentation.

Liver Disease

Introduction

Hepatic disease is uncommonly recognized in the horse. When suspected, serum biochemical analysis should be completed. Common causes of liver disease include: pyrrolizidine alkaloid toxicosis, cholelithiasis, bacterial hepatitis, Theiler’s disease (serum sickness) and neoplasia.

Clinical Signs

Liver disease may present with icterus of the mucous membranes, photosensitization, depression, head pressing, abnormal behavior, colic, coagulopathies, weight loss or anorexia.

Diagnostic Testing

As mentioned, biochemical analysis is the first step in establishing liver disease. Liver enzymes include:

- **λ-Glutamyl transpeptidase (GGT)** normal <50 IU/L- liver specific and elevations commonly reflect cholestatic disease. Elevated in foals due to colostrum intake.
- **Alkaline phosphatase (SAP, ALP)** normal <300 IU/L- elevations may be due to steroid use, cholestatic disease, bone turnover (young animals have higher values) and intestinal disease.
- **Aspartate aminotransferase (AST, SGOT)** normal <300 IU/L- elevations occur commonly after muscle damage, acute hepatocellular disease, steroid use and hemolysis. Liver derived levels >2000 IU/L carry a poor prognosis.
- **Bilirubin** normal 0-2 mg/dL- Elevations are due to cholestatic disease, hepatocellular disease, anorexia or hemolysis.

When liver disease is suspected based on physical exam, history and biochemical analysis, several other tests should be performed

- **Serum bile acids (normal <15 µmol/L)**- 90% of bile acids are normally removed by the liver from the enterohepatic circulation. Increased serum bile acids are
highly specific for liver disease but do not reveal whether the disease is hepatocellular or cholestatic in origin. Fasting should not affect the results unless it is >24 hours. Levels >50 µmol/L are typically associated with a poor prognosis.

- **Sorbitol dehydrogenase (normal <8 IU/L)** - Indicates active hepatic disease. Requires analysis within 12 hours or, if spun down and serum removed within 48 hours.

- **Bilirubin (Total Bilirubin normal 0.2-5.0 mg/dl, Direct Bilirubin <25% or 0.0-0.4 mg/dL)** - When elevations in total bilirubin are encountered, a determination of direct vs. indirect should be made. Bilirubin is derived from heme pigments and arises from the conversion of biliverdin to bilirubin by macrophages. The indirect (or unconjugated) form of bilirubin is then bound to albumin and sent to the liver for conjugation. Bilirubin fractions, direct and indirect, may be requested from laboratories. Indirect bilirubin may be elevated due to hemolysis, hepatocellular disease, fasting, intestinal displacement or obstruction, and drug administration—steroids, heparin, halothane. Direct (or conjugated) bilirubin levels should represent <25% of the total serum bilirubin. If the direct levels are >25% of total bilirubin value, hepatic disease should be considered likely, and non-hepatic differentials become less likely. Elevations >30% are indicative of cholestatic disease. Some horses with chronic liver disease may have normal bilirubin levels.

- **Liver ultrasound** - This procedure is useful in identifying choleliths, hepatoliths, diffuse hepatocellular disease or neoplastic masses. Multiple hyperechoic foci, known as a ‘starry sky’ appearance, may be an incidental finding due to parasite migration.

- **Liver biopsy** - This tool is of great benefit in determining the origin of liver disease and establishing an etiologic agent in cases of bacterial hepatitis when cultured. Ultrasound guidance on the right (or sometimes the left) side of the thorax provides easy access to transcutaneous biopsy instrument use (PGI E Z Core- EZ 1415-15-T: 14g x 15cm www.productsgroup.com). At least two biopsies should be obtained, one for histopathology and one for culture. Culture is extremely low yield.

**Treatment**

Treatment is dependent on the cause of the disease.

- Bacterial hepatitis should be treated with penicillin and either gentamicin or enrofloxacin until culture results are available
- Cholelithiasis may represent a surgical case if unrelenting colic signs are present. Otherwise, long-term antibiotic administration may provide a cure.
  - Enrofloxacin (5 mg/kg IV q 24 h, 7.5 mg/kg PO q 24 h), ceftiofur (2 mg/kg IM q 24 h, Crystalline Free Acid 6.6 mg/kg IM q 4-5 d) or trimethoprim-sulfamethoxazole (30 mg/kg PO q 12 h) are recommended.
  - Continue antibiotics until liver enzymes are normal.
  - DMSO (1 gm/kg IV or via NGT as 10% solution) has been shown to dissolve calcium bilirubinate stones in vitro.
• Chronic liver disease should be managed by dietary control and restriction of UV radiation to prevent photosensitization
  ▪ Eliminate high protein diets
  ▪ Feed grass hay free choice
  ▪ 1 part cracked corn and 2 parts beet pulp with molasses @ 2.5kg/100kg fed in 4-6 feedings has been recommended by Divers. Regardless of diet change, feeding frequency should be increased in order to offer smaller meals.
  ▪ Branched chain amino acids may be supplemented.
• Hepatic encephalopathy or severe acute hepatitis cases should also have their diets changed in addition to the following:
  ▪ Control of abnormal behavior by administering xylazine or detomidine
  ▪ Correct electrolyte abnormalities and fluid deficits
  ▪ Provide glucose in IV fluids to anorexic horses
    • This is easily accomplished by spiking 5 L bags with 1 L 50% Dextrose solution, 250 mL CMPK and a daily Vit B complex spike (10 mL B-12, 10 mL B-complex)

**Chronic Diarrhea**

*Introduction*

In the vast majority of horses presenting with chronic diarrhea we do not secure a definitive diagnosis. We are often left to try symptomatic therapy. These patients normally present with good attitudes, appetites, most maintain their weight reasonably well but they have increased fecal water and scalding of the perineum.

*Diagnostic Plan*

*Physical Examination*

Rarely is the exam rewarding as far as finding a cause but certainly you want to assess dental wear, hydration levels, auscultate the ventral abdomen for sand, look for edema that could be due to hypoproteinemia and perform a rectal exam to identify abdominal masses, thickened rectal mucosa, emerged small strongyles on your glove (seen as small red worms), sand, and to collect a fecal sample for analysis.

*Fecal Analysis*

Feces can be collected for fecal flotation, fecal occult blood (right dorsal colitis) and sent to a PCR lab for a “Diarrhea Panel” which includes most pathogens of interest. The turnaround is very quick and the results provide at least some due diligence towards a diagnosis. PCR labs include:

• Equine Diagnostic Solutions, LLC
  1501 Bull Lea Rd., Suite 104
Sand Flotation

Sand accumulation does occur in certain geographies and when in the field setting, where abdominal radiographs are not available, auscultation and sedimentation in a rectal sleeve are the gold standards.

Rectal Biopsy

Occasionally infiltrative bowel disease can occur in the horse. These patients typically have weight loss and low albumin levels. A small piece of rectal mucosa can be taken by infusing 60 mL lidocaine into the rectum and then using a uterine biopsy instrument to obtain a sample between 10 and 2 o’clock approximately 10 cm oral to the anus (caudal to the peritoneal reflection). The samples are then submitted for histopathology and/or salmonella culture or PCR analysis. Histopathology results often come back as mild proctitis, which doesn’t really help in determining an etiology. You may run into the odd case of intestinal lymphoma or granulamatous inflammatory bowel disease. Although these cases exist, they are extremely rare.

Blood Work

A complete blood count, fibrinogen, chemistry with electrolyte levels and bicarbonate values should be monitored. Many of the abnormalities you will find in the blood work are secondary to the diarrhea-azotemia, acidosis, electrolyte abnormalities. Hypoproteinemia indicates a loss of mucosal integrity and may be a sign of right dorsal colitis, segmental colitis, sand enteropathy, infiltrative bowel disease, cancer or parasitism (small strongyles).

Treatment

In most cases a definitive diagnosis will not be obtained, yet this should not stop us from looking. In the middle of our quest we still need to institute some basic therapies that should help resolve the increased fecal water content. Some of my preferred treatments are listed below.

1. Metronidazole (25 mg/kg PO q 12 h)
2. Fenbendazole (10 mg/kg PO q 24 h x 5 days)
3. Dental float
4. Diet- adjust to a bland diet of grass hay and a complete pelleted feed
5. Saccharomyces boulardii (50-100 billion cfu PO q 12 h)
6. Psyllium (tube with 1-2#, mix with oil for easy delivery)
7. Bismuth subsalicylate, smectite clay (tube with 1 gallon bismuth subsalicylate and 3# smectite clay)
8. Transfaunate (administer Gastrogard 4 hours prior to reduce stomach acid)
9. Clioquinol/Iodochlorohydroxyquin/Rheaform- (10 gm/adult PO q 24 h until feces normalize)
10. Loperamide- (0.1-0.2 mg/kg PO q 6-12 h)
11. Codeine- (200 mg-2 grams/adult PO daily, monitor for obstipation)

I personally begin with 1-5 on almost all patients. If sand is suspected, then I will start with tubing with psyllium, otherwise I will use the bismuth/smectite mix. Tubing is continued once daily for three days or diarrhea resolves, whichever occurs first. Transfaunation can be instituted at any time. Numbers 9-11 are used individually in that order, typically trying one course of seven days and switching if diarrhea fails to resolve.

A diagnosis of *Salmonellosis* may be obtained and is often subjected to lengthy courses of antibiotics. In truth, we do not know that antibiotics will stop fecal shedding of *Salmonella*. In fact, treatment with fluoroquinolones may increase fecal shedding. I prefer to treat these patients with probiotics, such as Saccharomyces boulardii, unless they are a foal or are systemically ill, in which cases I will use chloramphenicol orally (50 mg/kg PO q 6-8 h). I recommend isolating the horse and repeating either a series of 5 cultures or 2 PCR analyses in monthly intervals until the results are negative.

### Foal Diarrhea

**Introduction**

Neonatal foal diarrhea should always be treated aggressively as these patients are more prone to become systemically ill and/or septic. Older foals with diarrhea may be treated less aggressively depending on their severity of illness. Neonates may be infected with *Salmonella, Clostridium difficile or perfringens, Rotavirus*, or a variety of gram negative bacteria such as *E. coli* that have entered the gut and proceeded to cause sepsis. Older foals may be affected with *Salmonella, Clostridium difficile, Rotavirus, Lawsonia*, ascarids, sand or other non-specific causes.

**Diagnostic Tests**

**Physical Examination**

Neonates may develop collapse from the systemic effects of sepsis or dehydration. Carefully monitor for any secondary sites of infection - joints, physes, or bones. Typical signs of hypovolemia will include elevated heart rate, dry and injected mucus membranes and scleral injection. Older foals, especially those with *Salmonella* or *Lawsonia*, may develop ventral edema secondary to hypoalbuminemia.
Blood Work

A complete blood count, fibrinogen, chemistry, lactate and either a blood gas or at least a TCO₂ level should be performed. *Lawsonia intracellularis* IPMA antibody titers should be assessed on older foals/weanlings by submitting a sample to the University of Minnesota Diagnostic Laboratory. Some foals with this disease will test positive (>1:120) on only the blood test and not the fecal (see below), and also vice versa.

**Address:** Veterinary Diagnostic Laboratory  
College of Veterinary Medicine  
University of Minnesota  
1333 Gortner Avenue  
St. Paul, MN 55108-1098  
**Phone:** (612) 625-8787; (800) 605-8787  
**Fax:** (612) 624-8707  
**E-mail:** vdl@umn.edu  
**Website:** www.vdl.umn.edu

Fecal

The equine PCR “Diarrhea Panels” are also useful in foals. Be careful to recognize that many of these foals will have co-infections where they test positive for more than one organism. *Coronavirus, Cryptosporidium, Rhodococcus* may be positive and have unclear meaning. I concentrate on *Clostridial species, Salmonella, Rotavirus, Lawsonia* as principle culprits. *Lawsonia intracellularis* PCR is also available at the University of Minnesota Diagnostic Laboratory. A fecal egg count is warranted in foals over 45 days of age if parasites are a concern.

Blood Culture

Neonates that are showing systemic signs of sepsis should have a blood culture performed if at all possible. This test has always been viewed as a referral type of test but they are really very easy to perform in the field. Simply collect 5-10 ml of blood using sterile technique (I take mine just prior to placing an IV catheter) and place into a blood culture bottle (BBL Septi-Check BHI pediatric). The information gained with a blood culture provides you not only a diagnosis but also an excellent guide for an appropriate treatment plan for prolonged antibiotic therapy. Do not be discouraged by the idea of doing a blood culture, they are easy to perform and can be extremely helpful in the medical management of the foal.

Treatment

I always recommend broad-spectrum antibiotics for foals under 30 days of age with diarrhea, other than run of the mill foal heat diarrhea. Penicillin (20,000 IU/kg IV or IM q 6-12 h) or ampicillin (22 mg/kg IV q 6-8 h) with amikacin (25 mg/kg IV q 24 h) or gentamicin (12 mg/kg in neonates IV q 24 h) are good combinations to start with. Alternatively, ceftiofur (ceftiofur sodium 5 mg/kg IV, IM, SQ q 12 h, or ceftiofur crystalline free acid 13.2 mg/kg SQ q 36 h) with
or without an aminoglycoside to enhance gram spectrum may be used. Metronidazole (20 mg/kg PO q 12 h or 10 mg/kg IV q 12 h) should always be used if *Clostridium spp* infections are suspected. The intravenous route is preferred if ileus or bloat are detected. Chloramphenicol (50 mg/kg PO q 6-8 h) is preferred as a broad-spectrum oral antibiotic. As mentioned earlier, older foals may or may not require antibiotics depending on their physical exam findings, presence of a pathogen such as *Salmonella, Clostridium* or *Lawsonia* or leukopenia. Aminoglycoside doses are reduced for foals over 2 weeks of age (gentamicin 6.6 mg/kg, amikacin 10 mg/kg IV q 24 h).

**Anti-Inflammatories**

Flunixin meglumine (1.5 mg/kg IV q 24 h in neonates, 1.1 mg/kg IV q 12 h) or firocoxib (0.1 mg/kg IV q 24 h after 0.3 mg/kg IV loading dose) should be used to minimize systemic inflammatory responses and for analgesia. Butorphanol (0.05 mg/kg IV or IM PRN) can be used to control pain related to ileus and bloat.

Nutrition is a major concern with neonatal diarrhea. Many foals have an ileus along with bloat that prevents suckling or nasogastric tube feeding. In addition, milk may lead to an osmotic diarrhea if the small bowel is sufficiently injured to prevent proper absorption. I prefer to not feed foals under a week of age while they have diarrhea if severe depression, recumbency, bloat or ileus is detected. Instead, I will use a crude version of intravenous nutrition to maintain them for a short period of time, usually <36 hours, while the diarrhea reduces in volume and normal motility patterns return (parenteral nutrition recipes are available in the fluid therapy notes). While this level of care may seem to be too involved for a field setting, I truly believe this to be a worthwhile and manageable endeavor when the owner’s economical situation precludes referral. A small enclosure can be built in the mare’s stall out of shavings bags or bales of hay to keep the foal confined for 12-36 hours while the fluids are being administered. A gravity fluid control dial (Rely-A Flow 200 ml/hr by I-Flow 1-800-448-3569 or Dial-a-Flo by Hospira) can be used in lieu of a fluid pump or a drip rate can be calculated. Regular replacement fluids can be given intermittently if fluid deficits occur while on the nutrition drip.

**Fluids**

The fluid therapy section will cover most of the fluid requirements. In general, foals with diarrhea should be given a fluid bolus based on administering ½ of the estimated fluid deficit (Liters= % dehydration x BW kg). A replacement fluid such as Normosol-R, Plasmalyte-A, LRS or Hartmann’s solution is preferred. Do not use regular 5% dextrose in water as a resuscitative fluid. These patients need salt to preserve their circulating volume. Many times a follow-up bolus can be administered as needed to keep the foal hydrated. I will not spike rapid infusions with dextrose or electrolytes in most circumstances. If the foal is collapsed, you may need to begin a 10% dextrose drip (add 200 ml of 50% dextrose to a 1 L bag of LRS or other replacement fluid) while you are restoring the foal’s volume deficit. This can be done by starting the dextrose drip to empty over 30-60 minutes and then piggy-backing wide open replacement fluids to meet your calculated fluid requirements.

Foals with hypoalbuminemia require either plasma (20-40 ml/kg) or hetastarch (max 10 ml/kg per day) to preserve their oncotic pressure. These colloid fluids are administered on a daily basis until their attitude, fluid needs and edema return to normal.
Anti-Diarrheal Agents

a. Bismuth subsalicylate (BismuKote Paste)- 10 ml/100 kg PO q 6-12 h
b. Smectite clay (Hagyard Antidiarrheal Gel)- 30 ml/foal PO q 12 h
c. Saccharomyces boulardii - PO q 12 h
d. Lactobacillus rhamnosus - 2 capsules/50 kg PO q 12
e. Lactaid- 1 tablet PO q 2-6 h
f. Loperamide- 0.1-0.2 mg/kg PO q 6-12 h
g. Omeprazole (Gastrogard)- 4 mg/kg PO q 24 h

Agents a-d are always used initially as long as the foal is not refluxing. Lactaid tablets can be administered when milk is reintroduced to the diet or if there is a clinical suspicion of absolute or relative (villous tips produce lactase and are often damaged from enteritis) lactase deficiency. Loperamide is recommended for recalcitrant diarrhea. Omeprazole should be used in any foal that is not nursing normally.