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Adult Equine Diarrhea Workup

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Basic elements involved in the clinical evaluation of cases of acute and chronic diarrhea are covered. Major etiological considerations are listed, and important aspects of prognostication are discussed. Author's address: Island Whirl Equine Colic Research Laboratory, College of Veterinary Medicine, University of Florida, Gainesville, FL 32610. © 1999 AAEP.

Basic Historical Information

A. Other Animals Sick?

The major question is as follows: Does this look like an infectious/parasitic disease situation or is it something affecting only the patient under consideration? Just because the animal under consideration is the only one involved at present does *not* rule out the possibility of the cause of its diarrhea may be due to an infectious/parasitic agent, however. Often this question is easier to answer if it involves a regular client where the history of premises is well known.

B. Significant Management Actions or Weather Changes Associated with Onset of Problem?

A change in routine, particularly if it is very different and/or rapid in occurrence, can provoke the full-blown clinical manifestations of a GI problem that a horse had, up until that time, been able to keep under control. The critical factor is often what impacts on the balance of the intestinal bacterial flora, such as dietary changes, treatment with certain antibiotics (e.g., macrolides), shipping, changing deworming routine. It is also important to ask about previous treatment with medications other than antibiotics that are known to have potential pathogenic effects on the equine GI tract, such as the NSAIDs and cathartics.

C. Been Colicky?

It has been our experience that some horses with chronic active salmonellosis may experience recurrent periods of low-grade colic that may respond quite adequately to one treatment of flunixin. If one bothers to check the temperature or the WBC during one of these episodes, a mild fever and/or significant leukopenia may be found. These animals are just waiting for some situation, such as mentioned in "B" above to push them into a full-blown acute enterocolitis/diarrhea syndrome where, again, colic may be part of the clinical presentation. In addition, large and small strongyle infestations can cause episodes of recurrent colic and may also lead to diarrhea of varying severity and duration.

D. Lost Weight?

Any horse that develops a severe acute diarrheal disease is going to lose weight as a consequence of the attendant water loss and anorexia. This question is directed more at whether the patient had a tendency to lose weight before the development of any diarrhea or whether the weight loss occurs or persists even though the volume of the diarrhea is

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not great and the animal's electrolyte balance is not precarious. The point is that a chronic weight loss situation indicates the need to rule out conditions such as chronic inflammatory bowel disease, neoplasia, parasitism, and NSAID-induced enteropathy that could be at the root of the diarrheal disease. The finding of attendant ventral edema, for example, would support such an alternative.

Basic Clinical Evaluation as a Guide to Immediate Patient Management

Basic premise: An adult horse will *not* show diarrhea as a clinical sign unless there is colonic malfunction.

Physical Examination

The experienced practitioner knows the importance of general observation of attitude and vital signs in evaluating how acutely ill the patient really is. If we assume that severe enterocolitis induces excess production of numerous prostanoids and cytokines that are part of the arachadonic acid cascade, we can appreciate how bad some of these horses may feel, and what effect these agents have on attitude and vital signs. Also very important here is to note whether the horse shows any signs of foot pain indicative of developing laminitis, a well-recognized serious sequela of severe endotoxemia. It is this author's opinion that the finding of strong, bounding digital pulses is a much more useful indicator of developing laminitis than is evaluation of hoof temperature. Again, as indicated above, assessment of body condition and information on loss of weight and development of ventral edema in relation to onset of diarrhea will be useful in judging the severity of the problem, and perhaps in making a more definitive diagnosis. In chronic diarrhea cases, take a good look at the skin, since eosinophilic enteritis commonly manifests a nonpruritic dermatopathy. It is extremely important to make some judgment about fecal consistency and volume, not only for the purposes of diagnostic ruleout, but also to provide some guidance about therapeutic management. For instance, a high volume watery diarrhea, typical of acute onset infectious colitis, is going to require intensive IV fluid therapy, and perhaps plasma, whereas a patient with low volume fecal output, irrespective of consistency, may be successfully managed by providing a free-choice intake "electrolyte bucket" alone. Also, as the experienced practitioner knows, observation of the color of the mucous membranes and their capillary refill time (CRT) is an important adjunct to assessment of degree of endotoxemia and dehydration. For most cases of acute onset, severe diarrheal disease a rectal examination is not very helpful diagnostically; for the clinical evaluation of more chronic cases, however, it is essential. Finally, auscultation of the lungs should be part of the routine clinical evaluation of a horse with severe acute diarrhea. Most cases with acute diarrhea have clear lungs; the auscultation of wheezes indicates the need for a tracheal wash and/or thoracic radiographs to determine if the pulmonary problem is directly related or not to the disease condition causing the diarrhea (see "Prognostication" for acute, severe diarrheal disease below).

Clinical Pathology

Basic clinical pathology for any equine diarrheal disease includes the following: (1) CBC, (2) plasma total protein and fibrinogen, and (3) serum chemistry with special attention to Na, K, Cl, total CO_2, creatinine, and albumin concentrations. The utility of these needs little explanation, but a couple of things deserve mention, particularly in relation to equine diarrheal disease. In this context, perhaps the most useful aspect of a CBC is the total WBC count, especially the degree of leukopenia in an acutely ill patient, which can give some idea about the severity of the endotoxemia. This can be further evaluated by a look at the serum creatinine concentration which, unless there is attendant renal damage, is a reasonably accurate indicator of the degree of peripheral circulatory collapse (endotoxic shock). Total plasma protein measurement is extremely important for indicating the degree of intestinal mucosal damage; that is, is there a significant protein-losing enteropathy, which has major implications regarding prognosis, whether the case is acute or chronic. Knowing whether the loss is preferentially albumin or not is also useful, in that albumin will leak more readily (with less damage) than will globulin. Both hyponatremia and hypokalemia, the degree of which also reflects the severity and extent of the colonic lesion, commonly occur as a result of acute, severe diarrheal disease in the horse; thus, the need to measure serum Na and K concentrations. A look at the serum chloride and total CO_2 concentrations, particularly the latter, is useful in evaluating the degree of metabolic acidosis induced by the hypovolemia and electrolyte losses. Remember that the total CO_2 is a reasonably accurate indicator of the plasma HCO_3 concentration, being normally ~25 mEq/l in the horse. Severe cases of metabolic acidosis may yield total CO_2 values of 10–15 mEq/L.

Problem: Rapid Onset—Very Sick + Diarrhea of Few Hours/Days Duration

Major Ruleouts

A. Salmonellosis
B. *Clostridium difficile* overgrowth
C. *Ehrlichia risticii* infection
D. Cyathostomiasis
E. Antibiotic induced
F. Sand induced
G. Blister beetle intoxication
H. *Clostridium perfringens A* overgrowth
I. Peritonitis
J. Mycotic enterocolitis (very rare)
K. Equine viral arteritis (GI manifestations very rare)

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Important Readily Achievable Tests/Procedures to Consider Re: Determining Etiology

A. Fecal examination with special attention to:
   1. Salmonella culture; at least five separate samples (and PCR?)
   2. *Clostridium difficile* toxin; in human medicine, it has been found that testing for both A and B toxins is necessary to prevent missing the diagnosis (keep sample cool and transfer to lab ASAP)
   3. Presence of sand (swirl test); a negative finding does not rule out sand
   4. Presence of strongyle eggs; do not rely on fecal concentration alone to determine the severity of infestation; historical and physical exam points covered above (including rectal exam) are indicated here
B. Response to 6 mg/kg tetracyclene IV q 24 h; marked improvement within 24 hours is strong support for diagnosis of *Ehrlichia risticii* infection
C. Peritoneal fluid analysis; WBC counts > 10,000/µl are indicative of significant peritoneal inflammation
D. EVA titer

Prognostication

Initial prognosis depends upon how endotoxic (status of membranes; degree of neutropenia and azotemia), hypoalbuminemic, and hyponatremic the patient is and whether or not there are signs of laminitis. Severe hypoalbuminema (<1.5 g/dl) implies the need to give plasma in addition to the usual IV fluids to enhance the outcome. Severe hyponatremia (<120 mEq/L) implies a very widespread colitis. Conversely, as indicated above, an encouraging response to a single dose of 6 mg/kg tetracycline IV improves the prognosis and warrants continued q 24 h treatment for 3–4 more days. Short of that, the best that can be hoped for is metabolic stabilization of the patient for the first day or two, with no development of laminitis, and then gradual improvement in response to chosen therapeutic regimen with resolution of the diarrhea within 1 week. During this time, a close monitoring of the respiratory tract is also indicated, as the occasional case will develop a systemic mycosis (due to disruption of the colonic mucosal barrier?) that will manifest as pneumonia. This development makes the prognosis virtually hopeless. Furthermore, the longer the diarrheal condition persists, even with metabolic stabilization of the patient, the more concerned one must become about the development of a "chronic" diarrhea syndrome (see below).

Problem: Rapid or Insidious Onset—Variable Degree of Sickness + Diarrhea >2 Weeks Duration (Chronic Diarrhea)

Major Ruleouts

A. Cyathostomiasis
B. Salmonellosis
C. Sand induced
D. Chronic inflammatory bowel disease (CIBD)
   a. Cause unknown: noneosinophilic vs eosinphilic: the eosinophilic form commonly has attendant nonpruritic dermatopathy
   b. *Mycobacterium avium* infection
   c. NSAID-induced (right dorsal colitis): history is very important here
E. Giardiasis
F. *Lawsonia intracellularis* infection (yearlings and younger?)
G. Peritonitis
H. Persistent colonic floral disruption (indicating unhealthy intracolonic environment?)
I. Enteric lymphosarcoma involving the colon
J. *S. vulgaris* induced arteriopathy (now quite rare)

Important Readily Achievable Tests/Procedures to Consider Re: Determining Etiology

A. Rectal palpation (may need lidocaine enema pretreatment)
   1. Status of cranial mesenteric root: arteries and lymph nodes; any evidence of arteriopathy is compatible with *S. vulgaris* larval migrans; mesenteric lymph nodes are normally not palpable and their enlargement is compatible with chronic inflammatory bowel disease (CIBD) and lymphosarcoma
   2. Masses? size and location; this refers primarily to enlarged lymph node chains along the teniae of the large colon consistent with CIBD or lymphosarcoma, but discreet masses need to be noted and efforts made to determine whether they are primarily within the peritoneal cavity, gut wall, or gut lumen
   3. Thickened bowel? large vs. small?; appreciation of thickened small bowel indicates widespread intestinal involvement, which will have a negative impact on prognosis (see ultrasound)
   4. Degree of large colon fill; in some cases of chronic diarrhea the large colon cannot be readily palpated because it is so empty; in the author's opinion, this is a sign indicative of an extremely guarded prognosis
B. Transabdominal or transrectal ultrasound; thickened bowel?
C. Plasma total protein; low values are most consistent with CIBD, lymphosarcoma, or severe cyathostomiasis; high values are most likely due to an increased globulin fraction (see below) that would be consistent with chronic cyathostomiasis or peritonitis; knowing actual albumin and globulin values is very important
D. Serum chemistry with special attention to:
   1. Albumin/globulin; albumin is preferentially lost in protein-losing enteropathies, but both may be lost in severe cases of CIBD
   2. Liver enzymes: GGT, SDH: some cases of CIBD, especially if caused by *M. avium,* affect the liver as well as the gut

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E. Peritoneal fluid analysis; most useful here in ruling out peritonitis
F. Fecal examination with special attention to:
   1. Culture for salmonella; at least five separate samples (and PCR?)
   2. Presence of parasites; roundworm eggs; giardial cysts; crypto oocysts
   3. Presence of sand (swirl test); a negative finding does not rule out sand, but finding a large amount increases suspicion
   4. Protozoal presence/absence: ciliates vs. flagellates (e.g., trichomonads); needs to be done on fresh material[a] (see "Prognostication" below)
   5. PCR for *Lawsonia intracellularis,* especially young horses with hypoproteinemia
G. Rectal mucosal biopsy[b]
   1. Histopathology; a long shot on definitive diagnosis; be sure to include an acid-fast stain to look for *M. avium* (see "Prognostication" below)
   2. Culture for salmonella; reported to be more sensitive than fecal cultures
H. Sugar absorption test[c] indicates diffuse small intestinal involvement that would be most consistent with CIBD or lymphosarcoma. Some severe cases of *S. vulgaris* larval migrans will also show sugar malabsorption (Note: in cases of CIBD, lymphosarcoma, or parasitosis where the major lesions are primarily in the small bowel, diarrhea will not be seen.)

Additional Diagnostic Procedures to Consider, Short of Laparotomy, that Usually Require Hospital Involvement

A. Liver biopsy
B. Abdominal radiography for presence of sand

Prognostication

Initial prognosis depends primarily upon whether a specific etiology and/or disease process can be determined or not. Sand and parasitic causes have the best chance of desired response to specified therapy. Those cases where no protozoa can be found in the feces occasionally (<25%) respond to one or more "transfaunations," which are not that easy to accomplish effectively in their own right. Cases in which mesenteric lymph node enlargement and/or thickened bowel can be found, and are hypoproteinemic and malabsorb sugar, have a very poor prognosis. The finding of granulomas (eosinophilic or not) or acid fast organisms in rectal and/or liver biopsy specimens of such cases makes the prognosis even worse. Yearlings or younger that are hypoproteinemic may have *Lawsonia intracellularis* infection, which can be treated effectively with erythromycin. Some cases in which nongranulomatous proctitis is found will respond to a 2- to 3-week course of prednisolone (1 mg/kg PO, q 24 h). For a frustratingly large number of chronic diarrhea cases, however, a specific etiology/disease process cannot be determined. A few of these may be responsive to oral iodochlorhydroxyquin (20 mg/kg PO, q 24 h) so long as it is being administered, but many will not respond, and if they do not within a day or two, this treatment should be discontinued. Often, all attempts at symptomatic therapy are unsuccessful and the best advice the practitioner can give after covering all the things listed above and coming up empty-handed is to turn the animal out to pasture, making sure it has adequate nutrition and access to free-choice water and electrolytes, to see if the problem will resolve spontaneously within a few weeks' or months' time. Cases with the best prognosis for this happening are those in which the *volume* of soft feces is somewhat close to what would be expected normally and the patient has maintained electrolyte balance and good body condition without major nutritional supplementation. The hypothesis is that such cases have primarily a small colon malfunction, the abnormally soft feces reflecting an inability of the small colon to sufficiently dessicate contents entering from the right ventral colon into fecal balls.

Footnotes

[a]To look for fecal protozoa, place a drop of fresh fecal liquor on a slide and cover it with a cover slip. Observe through a microscope under low power with the condenser magnifier turned away and the condenser diaphragm almost closed. The majority of protozoa seen should be ciliates, which is normal. The absence of all protozoa, or the predominance of flagellates (*Trichomonas equi*), which are very difficult to see under low power, is not normal. [Note: The original rationale for the use of iodochlorhydroxyquin was to kill the trichomonads, which were considered to be pathogenic. *T. equi* is a normal inhabitant of the equine colon and it is now thought that its overgrowth at the expense of the ciliates is a reflection of a disturbed intracolonic environment that is manifesting as diarrhea, rather than the organisms being the cause of the diarrhea. There is no question that iodochlorhydroxyquin affects colonic bacterial fermentative activity, but how this all results in less watery fecal material still needs to be determined.]

[b]Rectal mucosal biopsy is easily accomplished using a uterine biopsy or analogous instrument. Clean the caudal rectum of feces and then insert the instrument protected within the hand. Once within the rectum, grab a small pinch of mucosa at either the 11 or 1 o'clock positions and snip a piece with the biopsy forceps, applying a firm closure pressure. Retract the closed forceps slowly being sure not to tear the remaining mucosa due to insufficient closure of the instrument. Drop the retrieved piece of mucosa onto a sterile gauze pad and bisect it with a scalpel blade: one piece into formalin and the other into a sterile cup or tube for culture. Sometimes a small amount of hemorrhage is seen following this, but perforation has never been reported.

[c]The sugar absorption test requires withdrawing sequential blood samples into oxalated (for glucose) or heparinized (for xylose) tubes. A baseline sample should be drawn before any sugar is given. Then, by nasogastric tube, deliver 1.0 g/kg glucose as a 20% solution or 0.5 g/kg xylose as a 10% solution into the stomach and withdraw a blood sample every 30 minutes for the next 3 hours. Keep the samples refrigerated until delivery to the lab. Normally, the plasma glucose concentration should peak at 2× baseline, and the plasma xylose at ~20 mg/dl after subtraction of the baseline value, between 60 to 90 minutes after intragastric instillation.

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