Duodenitis-Proximal Jejunitis in Horses  (16-Dec-2003)

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A. Causes
The cause(s) of duodenitis-proximal jejunitis (DPJ, also referred to as anterior enteritis, proximal enteritis, gastroduodenitis-jejunitis, hemorrhagic fibrinonecrotic duodenitis-proximal jejunitis, and proximal duodenitis-jejunitis) remains unknown. Bacterial (including clostridial agents and salmonellae), parasitic (nematodes), and toxic (mycotoxins such as fumonisin B1) have been proposed or implicated, often solely on the basis of their isolation or identification from affected cases. Controlled epidemiologic and experimental studies have yet to confirm a cause. The disease is diagnosed by finding characteristic gross and microscopic lesions at the time of celiotomy or necropsy. Alternatively, the diagnosis is made on the basis of the clinical signs and clinicopathologic findings of a syndrome of reflux. Because the diagnosis is either that of a clinical syndrome or an anatomical diagnosis at surgery/necropsy, it is possible if not probable that their may be multiple causes of the syndrome and it is likely that each of the causes is itself multifactorial.

B. Signalment & Anamnesis
Epidemiologic studies documenting predilection for DPJ by age, breed, or sex are lacking. Most horses with DPJ are < 10 but horses of any age may be affected. Although anecdotal reports of the syndrome in foals exist, to the author’s knowledge no such cases have been reported. Although feeding concentrates in large amounts has been implicated, we have not found horses with DPJ to be fed concentrate in amounts larger than horses with other types of colic. In the United States, the disease is considered to be more prevalent in the southeastern region; disease in this region of the country may differ from that of other parts of the United States and Europe, possibly reflecting different causes among regions. A seasonal distribution has not been identified and the incidence of admissions for DPJ has remained relatively constant at our clinic. The medical history of affected horses generally includes relatively rapid onset of signs of colic; severity of colic ranges from mild to severe and is often severe until after gastric decompression.

C. Clinical Signs
Horses with DPJ generally have signs of colic associated with the hallmark finding of profuse nasogastric reflux. Initial volumes of reflux may be unusually large (e.g., 20 liters). Horses with signs of severe colic often become lethargic after gastric decompression. Similarly, the heart rate of affected horses is commonly elevated but often diminishes following gastric decompression (although heart rates may remain elevated). Fever is an inconsistent finding. Rectal palpation generally reveals mild to moderate distension of small intestine. Arrhythmias that occur in some horses with DPJ (< 20%) tend to resolve as an affected horse’s condition improves. Non-specific signs of hypovolemia and endotoxemia (such as altered appearance of mucous membranes) occur in many cases. Laminitis develops in about 25% to 30% of horses and can occur early or late in the course of the disease.

D. Clinical Laboratory Findings
Clinicopathologic testing of horses suspected to have DPJ should include a complete blood count, serum biochemistry panel, urinalysis, and peritoneal fluid analysis (PFA). Results of CBC are variable and may be indicative of either localized or disseminated inflammation. Although characteristic electrolyte abnormalities have been reported, these are variable; horses may be academic or (uncommonly) alkalemic. Some affected horses are hypocalcemic. Most horses are azotemic, and this may be of renal or prerenal origin. Urinalysis can be helpful to identify casts or excess protein concentration in urine. Some DPJ horses have elevated liver enzymes. Although the ratio of nucleated cell count to protein in peritoneal fluid tends to be lower (i.e., there is a disproportionate elevation of protein concentration) for horses with DPJ than for
strangulating obstructions, results of peritoneal fluid analysis of DPJ vary among horses and with stage of disease. Sequential analysis of peritoneal fluid may be of prognostic value.

**E. Diagnosis**
Diagnosis of DPJ is clinically challenging and often presumptive. Definitive anatomic diagnosis often requires celiotomy; however, many clinicians and owners prefer to manage the condition medically, thereby relying upon a presumptive diagnosis. Combination of clinical signs, clinicopathologic findings, and ancillary diagnostic testing may be useful for presumptive diagnosis. Although often described as an adynamic ileus, it has been our experience that motility of the duodenum and jejunum in horses with DPJ is variable with respect to motion. Ultrasonographically, amotile loops of small intestine are as likely to be identified in horses with strangulating lesions of the small intestine. There is nothing characteristic about the reflux fluid of horses with DPJ.

**F. Treatment**
Treatment can be either medical or surgical. Medical treatment is aimed at decompressing gastric fluid accumulation; correcting fluid, electrolyte, and acid-base imbalances; and reducing inflammation and effects of endotoxemia. The frequency and duration of gastric decompression needed vary among cases and with stage of disease, but often must be hourly or every 2 hours early in the course of the disease. Food and water are withheld until reflux is absent or markedly diminished. Fluid therapy of affected horses can be challenging because the volume of fluid lost into the gastrointestinal tract can be large and because administration of large volumes of fluid may increase intracapillary hydrostatic pressure, thereby exacerbating intraintestinal fluid volume by passive secretion. The fluid composition must be based on the electrolyte and acid-base status of affected horses, which can be variable among horses and varying over time.

Use of anti-inflammatory drugs may be helpful. Flunixin meglumine (Banamine® or Finadyne®) may be useful to alter hemodynamic and ileus-inducing effects of endotoxin and counter effects of mediators (such as metabolites of cyclooxygenase) produced in response to endotoxemia. Lower doses may be useful to avoid toxic nephropathy and enteropathies associated with NSAID use. Use of other anti-endotoxin strategies (such as administration of fresh frozen plasma, polymyxin B, pentoxifylline) also may be useful. Because DMSO may have anti-inflammatory properties, it is administered to some horses with DPJ (at doses from 20 to 200 mg/kg).

Use of antimicrobial agents in horses with DPJ is controversial. At our clinic, most horses with DPJ are treated with broad-spectrum antimicrobial drugs intravenously. Use of metronidazole IV in conjunction with surgical decompression of small intestine has been advocated on the basis of the assumption that cases are associated with clostridial infection. This may reflect regional variation in the cause(s) of DPJ.

Because horses may be without oral intake of nutrients for prolonged periods, partial parenteral nutrition may be a useful adjunct for horses with protracted DPJ (e.g., a horse that refluxes for > 4 days). Partial parenteral nutrition can be accomplished in many ways (e.g., with or without lipids).

There has been great interest and enthusiasm for use of various drugs that alter gastrointestinal motility (so-called gastrointestinal prokinetic drugs), including bethanechol, cisapride, erythromycin, lidocaine, and metoclopramide. Evidence supporting the value of prokinetic drugs to treat DPJ remains limited. Evidence exists that prokinetic drugs may not be effective when the intestinal tract is inflamed: they may only affect non-inflamed, unaffected portions of the intestinal tract of affected horses). Thus, there apparent effectiveness may simply reflect a resolution of intestinal inflammation rather than a direct prokinetic effect. Lidocaine is touted because of anti-inflammatory effects, but these effects of this drug are considered modest. Prokinetic drugs have side-effects that can be detrimental to horses with DPJ.

Surgical management of horses with DPJ is occasionally used. Celiotomy of some horses with DPJ occurs because a mechanical obstruction of the intestine could not be excluded. Some advocate surgical management for horses with DPJ to confirm the diagnosis and to decompress the small intestine and remove (via enterotomy) the accumulated intra-intestinal fluid. Horses with DPJ may undergo celiotomy if medical management has not been successful. Such horses may be candidates for various techniques of bypassing the affected portion of small intestine. Although evidence that surgery is detrimental to horses with DPJ is lacking, development of intra-abdominal adhesions can occur as a sequela to celiotomy of horses with DPJ.
G. Complications
A number of complications have been documented among horses with DPJ, including: septic peritonitis, aspiration pneumonia, pleuropneumonia, ulcerative pharyngitis, esophageal ulceration and perforation, cardiac arrhythmias, and laminitis. Many of these can be life-threatening and vigilance for their development is advocated in the hope that early recognition may lead to improved outcome.

References


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