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Clinical Characteristics of Horses with Gastrointestinal Ruptures Revealed During Initial Diagnostic Evaluation: 149 Cases (1990-2002) (21-Nov-2003)

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Abstract

Common clinical findings associated with acute intestinal rupture include depression, sweating, reluctance to walk, tachycardia, fever, hemoconcentration with normoproteinemia, leukopenia, abnormal mucous membranes, elevated peritoneal fluid protein concentration, and abnormal peritoneal fluid color. Abdominal radiography and ultrasound can be useful diagnostic aids for identification of pneumoperitoneum associated with intestinal rupture.

1. Introduction

In horses with colic associated with acute intestinal rupture, knowledge of the most common clinical parameters may be valuable in assessing colic and in making the decision to refer a patient to a surgical facility or to perform euthanasia. Definitive assessment of intestinal rupture may allow avoidance of prolonged suffering of the patient as well as additional expense to owners.

Familiarity with the common forms of colic leading to intestinal rupture may be useful in guiding initial diagnostics and in making therapeutic recommendations. Rupture of the gastrointestinal tract is not uncommon and has been reported for the stomach, [1,2] cecum, [3,4] and colon secondary to enterolithiasis [5]. Gastric ruptures may also be sequela to small intestinal strangulating obstructions [1,2].

Because of the large amount of luminal endotoxin [6] that is released into the peritoneum when the rupture occurs, the clinical features of most cases of gastrointestinal rupture are consistent with signs of endotoxemia. The signs of endotoxemia include the development of abnormal mucous membrane color with a "toxic" line, increased capillary refill time, tachycardia, tachypnea, decreased borborygmi, fever, and hemoconcentration [6]. As the disease progresses, some horses will have abdominal pain secondary to inflammatory mediators, and they may also seem depressed, be pyretic, and have a neutropenia with a left shift and hypotension [6].

Thousands of horses are presented to equine veterinarians every year for colic. Diagnosis, treatment options, and prognosis are of critical importance for the patient. To the authors' knowledge, the clinical features specific to horses with gastrointestinal rupture have not been previously described.

The purpose of this study is to describe the clinical findings of horses that are presented to equine veterinarians for colic and have gastrointestinal ruptures. The use of this information may help to guide the performance of diagnostic procedures in the field, and it may assist the veterinarian when weighing the accumulated clinical information and making recommendations to clients.

2. Materials and Methods

The medical records of 149 horses with gastrointestinal rupture that were admitted to the Veterinary Medical Teaching Hospital from January 1, 1990 through December 31, 2002 for treatment of colic were reviewed. All ruptures were confirmed during celiotomy or necropsy. Cases that were dead on arrival or ruptured intra-operatively or after medical treatment were excluded. Information extracted from medical records included age, breed, sex, clinical history, including duration of colic and level of pain, physical examination findings, transrectal examination findings, volume of gastric reflux obtained with

nasogastric intubation, and results of peripheral blood and peritoneal fluid analysis. Subjective assessment of the level of pain was recorded on a scale from 1 - 6, with 1 indicating no pain and 6 representing severe pain, or the horse was classified as "depressed" or "shocky". Physical examination findings recorded included temperature, heart rate, respiratory rate, mucous membrane color, and capillary refill time. Mucous membrane color was categorized based on color descriptions, and the presence or absence of a toxic line was noted. Pink and pale pink were considered normal. Other descriptions such as pale, injected, cyanotic, purple, and muddy or toxic were considered abnormal, as well as the presence of a toxic line with pink or pale coloration. Peripheral blood was analyzed for packed cell volume (PCV), total protein concentration, and total nucleated cell count. The color of peritoneal fluid was recorded as clear yellow, cloudy yellow, serosanguineous, bloody, or brown/green. Total nucleated cell count and total protein concentration of peritoneal fluid were analyzed, and the presence of bacteria was noted. Not all data was available for each case.

Statistical Analysis

A statistical software package [a] was employed for descriptive analysis of data using mean values and percentages for all clinical values evaluated. Median values were reported for continuous data containing extreme values. Data for several clinical parameters were separated into upper GI (stomach and small intestine) and lower GI (colon and cecum) groups to assess for potential differences in clinical presentation. Mean values and proportions were compared using the two sample t-test and χ^2 test. Significance was set at $P < 0.05$.

3. Results

There were 149 horses included in the study. Nineteen breeds were represented, but three breeds comprised 72% of the population. The most common breeds represented were Quarter Horses (43 of 149 horses, 29%), Thoroughbreds (36 of 149 horses, 24%), and Arabians (29 of 149 horses, 19%). There were 66 geldings, 60 mares, and 23 stallions. Ages ranged from 2 wk to 30 yr, with a mean age of 13 yr. All of the horses < 5 yr of age had gastric (eight horses) or small intestinal (four horses) ruptures.

Of the 149 cases, there were 100 (67.1%) ruptures of the colon, 31 (20.8%) gastric ruptures, 10 (6.7%) cecal ruptures, and 8 (5.4%) ruptures of the small intestine. Rupture of the descending colon occurred secondary to enterolithiasis in 87 of 90 cases. Of ruptures associated with enteroliths, 63 (72.4%) occurred in the small colon, 12 (13.8%) in the transverse colon, and 12 (13.8%) in the right dorsal colon.

Duration of colic signs before presentation to equine veterinarians varied from 3 h to 8 days. Time from onset of intestinal rupture to presentation was unknown. All of the horses were euthanized except for three that died during initial evaluation. Based on clinical findings associated with a grave prognosis, 106 (71.1%) horses were euthanized. The remaining 43 (28.9%) horses were euthanized intra-operatively when gastrointestinal rupture was definitively diagnosed.

Physical exam findings revealed an elevated temperature ($> 101.5^\circ\text{F}$, [$> 38.6^\circ\text{C}$]) in 47.8% (55 of 115 horses), tachycardia (> 50 bpm) in 92.0% (128 of 139 horses), tachypnea (> 24 bpm) in 67.2% (78 of 116 horses), increased capillary refill time (> 2 s) in 69.7% (76 of 109 horses), and abnormal mucous membrane color in 85.5% (112 of 131 horses) of cases (Table 1).

Mucous membrane color was described for 131 cases. Mucous membranes were described as muddy, toxic, cyanotic, or purple in 64.1% (84 horses) and as injected or pale in 22.1% (29 horses) of cases. A distinct toxic line was reported in only 14 (10.7%) cases. Horses were described as "shocky" or "depressed" in 65.6% (82 of 125 horses) of cases and severely painful in 17.6% (22 of 125 horses). Transrectal examination revealed a gritty feeling on intestinal serosa or free peritoneal gas in 48.4% (44 of 91 horses) of those horses who had results of a rectal examination recorded. A somewhat larger proportion of lower gastrointestinal ruptures (57.6%, 34 of 59 horses) had evidence or rupture on rectal examination than those with upper gastrointestinal rupture (31.3%, 10 of 32 horses); however, the difference was not statistically significant ($P = 0.10$). Other recorded rectal examination findings included small intestine or large colon distension, large colon impaction, hard mass palpated (enterolith), rectal mucosal tear, and pregnancy. No abnormalities were found in nine (9.9%) cases. Nasogastric intubation produced 3 l or more of gastric reflux in 56.3% (40 of 71 horses) of the cases. In cases of gastric rupture, reflux was obtained in 40.0% (10 of 25 horses) of the cases but was obtained for 65.2% (30 of 46 horses) of all other cases. This difference was not statistically significant ($P = 0.14$).

Analysis of peripheral blood revealed hemoconcentration ($> 46\%$ PCV) in 76.0% (92 of 121 horses), normal total protein concentration (5.8 - 7.7 g/dl) in 77.8% (88 of 113 horses), and leukopenia (< 5000 cells/ μl) in 78.6% (92 of 117 horses) of cases (Table 1). Cases with abnormal plasma protein levels were split between hypoproteinemia (13 horses) and hyperproteinemia (17 horses).

Abdominocentesis yielded peritoneal fluid with a markedly abnormal color in the majority (91 of 106 horses, 85.8%) of cases. Abnormal color was described as brown/green (51 of 106 horses, 48.1%), bloody (21 of 106 horses, 19.8%), or serosanguineous (17 of 106 horses, 16.0%). Cloudy yellow fluid was obtained in seven (6.6%) cases. No fluid was obtained in five cases, enterocentesis occurred in one case, and clear yellow fluid was obtained in one case. Peritoneal fluid nucleated

cell counts (Table 1) had a very wide range of 0 - 720,000 cells/ μ l, but these counts were elevated to >5000 cells/ μ l in only 28.4% (22 of 80 horses) or to >10000 cells/ μ l in 16.0% (12 of 80 horses). Overall, the total protein concentration of peritoneal fluid (Table 1) was elevated (> 2.5 g/dl) in 86.4% (76 of 88 horses) of cases, but it was elevated more commonly in lower gastrointestinal ruptures (93.8%; 60 of 64 horses) than in gastric and small intestinal ruptures (66.7%; 16 of 24 horses; P < 0.01). The presence of bacteria in peritoneal fluid was observed in 95.7% (66 of 69 horses) of cases that had cytologic evaluation.

Table 1. Values of Key Physical Examination and Clinicopathologic Data Evaluated for Cases of Gastrointestinal Rupture		
Physical Examination	Meant\pmSD	Range
Rectal temperature ($^{\circ}$ C)	38.6 \pm 16.9	36.1 - 41.4
Heart rate (beats/min)	84.3 \pm 25.4	36 - 200
Respiratory rate (breaths/min)	38.5 \pm 20.5	12 - 116
Capillary refill time (s)	3 \pm 1.3	1 - 10
Peripheral Blood		
PCV (%)	57.7 \pm 13.5	32 - 86
Protein concentration (g/dl)	6.8 \pm 1.0	4.6 - 10.4
Nucleated cell count (cells/ μ l)	3825 \pm 4215 2400*	495 - 34760
Peritoneal Fluid		
Protein concentration (g/dl)	4.4 \pm 1.4 4.7*	0.7 - 6.7
Nucleated cell count (cells/ μ l)	21610 \pm 85410 1430*	0 - 720000

*Median value

Abdominal radiography was performed in 29 cases, and suspected pneumoperitoneum was identified in the dorsal abdomen in 11 (37.9%) cases (Fig. 1). Abdominal ultrasound, when performed, commonly showed peritoneal fluid accumulation, sometimes containing echogenic debris. Presence of free peritoneal air identified using ultrasound was documented in one medical record.

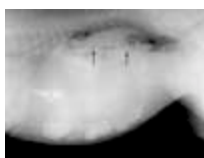


Figure 1. Lateral abdominal radiograph of an American Miniature foal with pneumoperitoneum secondary to gastric rupture. Note the increased fluid opacity in the ventral abdomen and a distinctive fluid line demarcating the peritoneal air/gas interface (arrows) separate from the gas contained within the intestinal loops. Serosal surfaces of intestine and viscera become more apparent, because the intraperitoneal gas provides contrast. - To view this image in full size go to the IVIS website at

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4. Discussion

Diagnosis of colic, accurately and rapidly, remains a challenge for equine practitioners. The equine clinician needs to be able to determine the most appropriate treatment and when appropriate, refer the patient to a hospital facility capable of intensive medical and surgical treatment. The majority of patients that are referred to our hospital for colic have had a physical and rectal examination and a nasogastric intubation, whereas very few have had abdominocentesis performed. Although not all cases of gastrointestinal rupture are easily determined, euthanasia was performed in the majority of cases with substantial evidence of rupture based on clinical findings and baseline laboratory data.

There were a number of important clinical findings that occurred in the majority of patients and contributed to a diagnosis of gastrointestinal rupture. A high proportion of cases (> 85%) presented with one or more of the following: fever, tachycardia, tachypnea, abnormal mucous membrane color, a "depressed" or "shocky" mentation, abnormal peritoneal fluid color, and increased peritoneal fluid protein concentration. Transrectal palpation confirmed a suspected intestinal rupture in nearly 50%

of cases when a feeling of gritty serosa or free air in the abdomen was identified. Additional supportive laboratory work included hemoconcentration, leukopenia, and identification of the presence of bacteria in peritoneal fluid. Little data has been published previously on the clinical and laboratory findings for cases of gastrointestinal rupture; however, our overall findings were similar to those described for cecal ruptures [3,4].

Four prognostic characteristics have been identified to aid in evaluating colic severity in horses [7]. They are heart rate, peritoneal protein concentration, blood lactate concentration, and mucous membrane appearance. These parameters were determined to be key prognostic indicators in predicting if a horse will die [7]. Heart rate and mucous membrane color are important indicators of cardiovascular status, are signs of endotoxemia, and have prognostic value regarding subsequent survival.

The clinical features of gastrointestinal rupture are primarily caused by endotoxemia from the large amount of luminal endotoxin that is released into the peritoneal cavity when rupture occurs. Endotoxemia may occur more rapidly after large colon and cecal ruptures compared with gastric or small intestinal ruptures, because there is a large amount of endotoxin normally in the intestinal lumen. The cecum and ventral colons of healthy horses contain more than 2 g of free endotoxin [6]. Although few significant differences were detected between horses with upper versus lower gastrointestinal tract rupture, this may be reflective of timing from rupture to presentation or the result of sequestration of contents within the omental bursa. The signs of endotoxemia include the development of abnormal mucous membrane color with a "toxic" line, increased capillary refill time, tachycardia, tachypnea, decreased borborygmi, fever, and hemoconcentration [6]. As the disease progresses, some horses will have abdominal pain secondary to inflammatory mediators and may also seem depressed, be pyretic, and have a neutropenia with a left shift and hypotension [6].

Abdominocentesis is an important diagnostic test in the assessment of colic cases and can be definitive in cases of rupture. Abdominocentesis can be accomplished in the field with low morbidity [8,9]. Color may be assessed immediately, and specific gravity and/or protein concentration can be determined with a refractometer. Total nucleated cell count has been shown to be useless in managing colic cases [10,11]. Abnormal color, especially brown-green and serosanguineous, were found to be reliably associated with surgical lesions and horses that died [7,10,11]. Close observation of brown-green fluid may reveal gross fecal contamination.

Enteroliths are primarily a regional problem in California, with a breed predilection for Arabian and Quarter Horses that have been fed a diet high in alfalfa [5]. In a recent retrospective study of 900 cases, 15% of enterolith-associated colics developed gastrointestinal rupture [5]. A significant number of horses with enteroliths have a history of colic during the year before admission or have passed enteroliths in their feces [5]. Clinical signs before rupture are often mild, and abdominal radiography is used to facilitate a diagnosis. Ruptures secondary to enterolithiasis dominated our study because of our location in California. The most common site of rupture was the descending colon.

Gastric ruptures have a reported prevalence of 1 - 8% of colic cases [1,2]. There are several etiologies behind gastric rupture including primary, idiopathic, obstructive, peritoneal, enteric, or traumatic and therefore, can present diverse physical and clinical parameters [1,2]. Peritoneal fluid may be relatively normal soon after rupture because of the sequestration of gastric contents in the greater omentum; this makes diagnosis of gastric ruptures difficult before referral [2]. In colic cases involving gastrointestinal ileus or distal obstruction, the possibility of gastric rupture should be considered, and persistent attempts to obtain gastric reflux may be appropriate [1]. Although the majority of horses with gastric rupture will not have reflux, the presence of gastric reflux does not rule out gastric rupture. The presence of an in-dwelling nasogastric tube does not prevent gastric rupture, and it is suggested that keeping the tube capped between refluxing may eliminate the trapping of gas in the stomach [1].

Cecal ruptures may occur in horses with concurrent but unrelated disease, with no concurrent disease, or associated with parturition subsequent to cecal impaction [3,4]. In one report, horses that had a ruptured cecum with concurrent, unrelated disease were all receiving phenylbutazone [3]. Of the mares reported with cecal rupture associated with parturition, 56% had dystocia and assisted foaling [3]. For all of the conditions except cecal impaction, the majority were found dead or with clinical and laboratory findings consistent with endotoxemia [3]. Cecal impaction may lead to rupture, and clinical signs are often subtle [3,4]. Transrectal palpation findings of increased tension of the ventral cecal band are suggestive of a developing cecal impaction, and findings of a taut ventral cecal band with firm ingesta filling the cecum are diagnostic for cecal impaction [3]. Medical or surgical therapy may be required depending on the underlying problem [3,4]. With early detection, a cecal impaction may respond to frequent administration of water through a nasogastric tube [4]. However, if the impaction is not responsive to conservative medical therapy, surgery may be required.

The use of ultrasonography has increased in equine practice. Use of a low frequency (2 - 3.5 MHz) probe is ideal for evaluation of the abdomen, although higher frequency probes (5 - 7.5 MHz) can be useful in evaluating superficial structures. In cases of gastrointestinal rupture, it is possible to determine if there is increased peritoneal fluid and assess the echogenic character. Fluid associated with a rupture is typically increased in volume, and digesta may appear as echogenic foci [12]. Additionally, free air may be appreciated in the dorsal abdomen when viewed transabdominally or transrectally. Although less available to most equine veterinarians, abdominal radiographs may reveal free abdominal air (Fig. 1). Some portable X-ray units may be of use in foals suspected of a having a gastrointestinal rupture.

In conclusion, common clinical signs associated with acute intestinal rupture include depression, abnormal mucous membranes, sweating, reluctance to walk, tachycardia, and fever. In approximately 50% of cases, transrectal abdominal palpation reveals the presence of free peritoneal gas, crepitus, or feed material on serosal surfaces. The most frequent clinicopathological findings are hemoconcentration with normoproteinemia, leukopenia, elevated peritoneal fluid protein concentration, identification of bacteria in peritoneal fluid on cytologic examination, and abnormal peritoneal fluid color. Abdominal radiography and ultrasound can be useful diagnostic aids for identification of pneumoperitoneum associated with intestinal rupture.


Footnote

[a] StatPlus V2.0, Data Analysis with Microsoft Excel by Berk K and Carey P, Duxbury, Pacific Grove, CA 93950.

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