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Primary gastric rupture in 47 horses (1995–2011)

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Abstract — The purpose of this retrospective case-control study was to identify factors associated with primary gastric rupture and to investigate if there were differences between etiologies of primary gastric rupture. Compared to the general colic population, Quarter horses were under-represented and Friesians and draft breeds were over-represented in 47 cases of primary gastric ruptures. Horses with primary gastric rupture typically presented with severe clinical and clinicopathological derangements. There were 24 idiopathic gastric ruptures, 20 gastric impaction associated ruptures, and 3 perforating gastric ulcers. Thoroughbred horses were over-represented in the idiopathic gastric rupture group compared to other breeds and etiologies. This study suggests the presence of important breed predispositions for development of gastric rupture. Further study is necessary to identify if these predispositions are associated with management factors or breed-specific disorders.

Résumé — Rupture gastrique primaire chez 497 chevaux (1995–2011). Le but de cette étude rétrospective de cas témoins était d'identifier les facteurs associés à la rupture gastrique primaire et de faire une enquête afin de déterminer s'il y avait des différences entre les étiologies de la rupture gastrique primaire. Comparativement à la population générale de coliques, les chevaux Quarter horse étaient sous-représentés et les Frisons et les races de trait étaient surreprésentées dans 47 cas de ruptures gastriques primaires. Les chevaux atteints de rupture gastrique primaire étaient présentés avec de graves dérangements cliniques et clinicopathologiques. Il y avait 24 ruptures gastriques idiopathiques, 20 ruptures associées à l'impaction gastrique et 3 perforations ulcéreuses gastriques. Les chevaux pur-sang étaient surreprésentés dans le groupe des ruptures gastriques idiopathiques comparativement à d'autres races et étiologies. Cette étude suggère la présence de prédispositions importantes des races pour le développement de la rupture gastrique. De nouvelles études sont nécessaires pour identifier si ces prédispositions sont associées aux facteurs de gestion ou à des troubles spécifiques aux races.

(Traduit par Isabelle Vallières)

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Introduction

Full thickness gastric rupture is a fatal cause and complication of equine colic, with a reported prevalence of 1% to 8% (1–3). Gastric rupture is caused by loss of tissue integrity due to severe gastric ulceration and perforation, localized infarction, or marked distension of the stomach wall (4,5). Gastric dilation and subsequent rupture may be primary or secondary. Primary gastric rupture is caused by excessive intake or fermentation of ingesta or reduced outflow from physical or functional obstruction within the stomach. Secondary gastric rupture is associated with intestinal obstruction aboral to the stomach and therefore is a complication of a primary lesion elsewhere in the intestinal tract. Primary gastric rupture has not been previously evaluated in a case series separately from secondary gastric rupture.

Proposed causes of primary gastric dilation and rupture include overconsumption of grain, ingestion of indigestible or expansible feeds, and ingestion of large quantities of cold water (6,7). Idiopathic gastric ruptures include those cases in which no underlying cause can be identified (3,6,8). Two previous studies examined risk factors for gastric rupture and found no associations with age, breed, or season (6,8); although geldings may have been over-represented in one study (6). Kiper et al (8) suggested that a diet of grass hay or grass alfalfa hay, and drinking water from a bucket, stream, or pond are predisposing factors for gastric rupture. Both of these studies included secondary causes of gastric rupture in their analysis, which may confound identification of risk factors for primary gastric rupture, and both studies had geographically specific breed populations (6,8).

The purpose of this study was to identify factors in the history, signalment, physical examination, and results of routine

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diagnostics performed on colic patients (ultrasound, radiographs, abdominocentesis) associated with a diagnosis of primary gastric rupture. Primary gastric rupture includes cases of gastric rupture caused by gastric pathology that disrupted the gastric wall, including gastric impaction, and perforating gastric ulcers (known causes) and gastric rupture in which no underlying pathology to cause the rupture could be identified (idiopathic causes). Our hypotheses were that draft breeds and Friesian horses would be over-represented in the primary gastric rupture population, there would be a seasonal distribution of primary gastric rupture cases compared to the general colic population, and that horses with known causes of primary gastric rupture would have a longer duration of colic prior to presentation than would horses with idiopathic causes of gastric rupture. The suspected breed predisposition was based on clinical impressions of the authors. A secondary hypothesis was that horses with gastric rupture would have more abnormalities in the results of physical examination, clinical pathology, and abdominocentesis suggestive of systemic inflammatory response syndrome (SIRS) and gastrointestinal compromise than would horses evaluated for colic from other etiologies.

Materials and methods

A search of electronic medical records of horses presenting to the William R. Prichard Veterinary Medical Teaching Hospital (VMTH), University of California, Davis between January 1995 and December 2011 was performed. Search criteria included "gastric rupture," "stomach rupture," and "gastric impaction" searched in parallel. Each phrase in quotations was used and output recorded and checked for inclusion criteria. Inclusion criteria included surgical or necropsy confirmation of primary gastric rupture. Horses that were less than 1-year old and cases with secondary gastric rupture (rupture secondary to obstruction/pathology elsewhere in the gastrointestinal tract) were excluded. This study included a subset of cases that were previously published in a study on gastrointestinal ruptures, although primary gastric ruptures were not separated from other gastrointestinal rupture in that study (9). Information collected from the medical record included month and year of presentation, duration of clinical signs prior to presentation (< 12 h or ≥ 12 h), signalment, admission clinical examination findings, transrectal palpation examination findings, presence and character of gastric reflux, results of initial hematologic values [packed cell volume (PCV), total protein (TP), blood lactate, peripheral white blood cell (WBC)], abdominocentesis (color, peritoneal TP), peritoneal total nucleated cell count (TNCC), and abdominal radiographic findings. Physical examination findings included rectal temperature, heart rate, respiratory rate, mucous membrane color (toxic/injected, pale pink/normal), and presence of borborygmi. Horses were also examined for criteria of systemic inflammatory response syndrome (SIRS), characterized by 2 or more of the following abnormalities: temperature > 38.6°C, heart rate > 60 beats/min, respiratory rate > 30 breaths/min, peripheral WBC $> 12.5 \times 10^9$ or $< 4.5 \times 10^9$ cells/L or $> 10\%$ band neutrophils (9,10). Results of necropsy examination, including the location of the rupture in the stomach, were also recorded. Etiology of the gastric rupture was recorded as known cause (causative gastric pathology present such as perforating ulcer or secondary to gastric impaction) or idiopathic (no causative gastric pathology identified).

Two sets of comparison populations were used. The first comparison group was all horses presenting for diagnosis and treatment of colic at the VMTH for the same time period (colic population) to compare signalment and season of presentation between the gastric rupture population and the colic population. Season was defined as Winter (December through February), Spring (March through May), Summer (June through August), and Fall (September through November). The second comparison group (case-controls) consisted of the colic cases examined immediately prior to and after each gastric rupture case (2 case-controls per gastric rupture) which allowed comparison with history and colic examination findings. Horses were only included once in the study (readmissions were excluded). Data recorded for each case-control were the same variables recorded for each gastric rupture case, including assessment of SIRS criteria.

Statistical analysis

Summary statistics were calculated for all variables. Normality of data was assessed using Q-Q plots. Signalment and season of presentation were compared between gastric rupture cases and the colic population presenting during the same time period using Chi-square test or Student's *t*-test for categorical and continuous data, respectively. Duration and colic examination findings for gastric rupture cases and case-controls were compared using Student's *t*-test (parametric data) or Wilcoxon rank-sum (nonparametric data) for continuous variables and Chi-square test or Fisher's exact test for categorical variables. Bonferroni corrections were made for individual pairwise comparisons if the overall comparison was significant. Gastric rupture etiologies (impaction *versus* idiopathic) were compared for signalment, season, and duration, using Chi-square test and Student's *t*-test for categorical and continuous data, respectively. All statistical analyses were performed using a commercial statistical software program (Statistical Analysis Systems 9.3; SAS Institute, Cary, North Carolina, USA). Level of significance was set at $P \leq 0.05$.

Results

Forty-seven cases met the inclusion criteria of confirmed primary gastric rupture that was not associated with disease elsewhere in the gastrointestinal tract. Three horses were seen exclusively in the field by the Equine Ambulatory Service, and the remaining horses were examined in the hospital by Equine Medicine or Equine Surgery Services. There were 9305 horses presented for colic during the same time period; therefore, primary gastric rupture represented 0.5% of all colic cases during this time. There were 21 geldings, 19 mares, and 7 stallions. Breed distribution was as follows: 12 Thoroughbreds, 8 Warmbloods, 8 Arabians or Arabian crosses, 6 Quarter horses or Paints, 5 Draft horses, 3 Friesians, and 5 horses of other breeds. Mean age was 12.3 ± 6.7 y and median age was 12 y (range 1–27 y). Thirty-two percent presented in Winter, 17% in Spring, 17% in Summer, and 34% presented in Fall.

Breed distribution was significantly different between the gastric rupture cases and the colic population. Quarter horses

Table 1. Comparison of continuous variables in colic examination and clinicopathological findings for horses with confirmed gastric rupture compared to contemporaneous colic cases

Variable	Reference interval	Gastric rupture (mean ± standard deviation)		Colic control (mean ± standard deviation)		*P*-value
Temperature (°C)	37.2 to 38.3	$n = 36$	37.6 ± 1.1	$n = 74$	37.8 ± 0.8	$P = 0.54$
Heart rate (beats/min)	28 to 44	$n = 42$	77.3 ± 21.4	$n = 86$	53.4 ± 17.7	$P < 0.0001$
Respiratory rate (breaths/min)	8 to 16	$n = 36$	32.4 ± 13.9	$n = 77$	24.7 ± 12.6	$P = 0.005$
PCV (L/L)	0.30 to 0.46	$n = 36$	0.59 ± 0.15	$n = 79$	0.38 ± 0.09	$P < 0.0001$
TP (g/L)	58 to 75	$n = 35$	75 ± 16	$n = 79$	69 ± 9	$P = 0.038$
Blood lactate (mmol/L)	< 2	$n = 15$	9.5 ± 7.1	$n = 19$	4.0 ± 3.5	$P = 0.013$
Peripheral WBC ($\times 10^9$ cells/L)	5.0 to 11.6	$n = 29$	3.9 ± 4.0	$n = 78$	9.3 ± 4.0	$P < 0.0001$
Peritoneal TNCC ($\times 10^9$ cells/L)	< 2.5	$n = 19$	23.0 ± 49.0	$n = 59$	67.0 ± 17.0	$P = 0.18$
Peritoneal TP (g/L)	< 20	$n = 22$	34 ± 16	$n = 60$	23 ± 13	$P = 0.003$

PCV — packed cell volume; TP — total protein; WBC — white cell count; TNCC — total nucleated cell count.

Table 2. Comparison of categorical variables for signalment, historical findings and colic examination findings for horses with confirmed gastric rupture compared to contemporaneous colic cases

Variable	Gastric rupture	Colic control	Significance
Gender	Gelding — 21 Mare — 19 Stallion — 7	Gelding — 48 Mare — 38 Stallion — 6	$P = 0.26$
Duration	< 12 hours — 32 ≥ 12 hours — 11	< 12 hours — 52 ≥ 12 hours — 38	$P = 0.06$
Intestinal borborygmi	Absent — 23 Decreased — 9 Normal — 1	Absent — 14 Decreased — 46 Normal — 13	$P < 0.0001$
Mucous membranes	Toxic/injected — 32 Pale pink/normal — 10	Toxic/injected — 20 Pale pink/normal — 58	$P < 0.0001$
Nasogastric tube findings	Reflux — 8 None — 16	Reflux — 9 None — 57	$P = 0.03$
Rectal findings[a]	Rupture — 16 Not rupture — 17	Rupture — 0 Not rupture — 69	$P < 0.0001$
SIRS criteria	Yes — 30 No — 5 Incomplete data[b] — 12	Yes — 13 No — 62 Incomplete data — 17	$P < 0.0001$

SIRS — systemic inflammatory response syndrome (2 or more of the following abnormalities: temperature > 38.6°C; heart rate > 60 beats/min; respiratory rate > 30 breaths/min; white blood cell count > 12.5×10^9 or < 4.5×10^9 cells/L or > 10% band neutrophils).

[a] Rectal palpation findings suggestive of rupture include collapsed rectum within freely movable peritoneal space and detection of gritty film on serosal surface.

[b] Incomplete data refers to horses that did not have information available for ≥ 1 of the SIRS categories; therefore, the presence or absence of SIRS could not be determined.

were under-represented (Bonferroni adjusted $P = 0.02$) and Friesians and draft breeds were over-represented (Bonferroni adjusted $P = 0.049$, and $P = 0.007$, respectively) in the gastric rupture cases compared to the colic population. Age and gender were not significantly different between the gastric rupture and colic population. Compared to the seasonal pattern of colic presentations to the VMTH (20% Winter, 26% Spring, 29% Summer, 26% Fall), season was significantly different in the overall comparison ($P = 0.03$), with more cases of primary gastric rupture presenting in Winter, although this was not significant after Bonferroni adjustment ($P = 0.14$).

Colic duration and admission clinical and clinicopathological findings are presented in Tables 1 and 2. In general, horses diagnosed with gastric rupture were severely tachycardic and tachypneic, with abnormal mucous membranes and reduced to absent intestinal borborygmi. Clinicopathologic findings included marked hemoconcentration, hyperlactatemia, and leukopenia. Forty-one gastric rupture horses had abdominocentesis performed, 2 of which did not have any fluid collected. Peritoneal fluid was recorded as feed in 16 horses, bloody/serosanguinous in 8 horses, light brown in 7 horses, and dark brown in 8 horses. Peritoneal fluid samples from 22 horses (Table 1) were examined cytologically and all had evidence of bacteria and/or plant material present. Abdominal radiographs were evaluated in 5 gastric rupture cases and were suggestive of rupture in 3 cases (radiographic identification of pneumoperitoneum).

Ninety-two case-controls were collected for comparison. Two primary gastric rupture cases presented sequentially, which reduced the number of available case-controls. Gastric rupture cases had significantly higher heart rate, respiratory rate, PCV, TP, blood lactate and peritoneal TP and lower peripheral WBC compared to case-controls (Table 1). Horses with gastric rupture

Table 3. Comparison of breed distributions for horses with idiopathic gastric rupture compared to horses with gastric rupture secondary to confirmed or suspected gastric impaction

Breed	Idiopathic gastric rupture (%)	Gastric impaction (%)	Overall *P*-value	Bonferroni-adjusted *P*-value
Thoroughbred *n* = 12	11/12; 92%	1/12; 8%	0.02	0.02
Warmblood *n* = 8	5/8; 62.5%	3/8; 37.5%		0.62
[a]Arabian *n* = 7	4/7; 57%	3/7; 43%		not tested
[a]Quarter horse *n* = 5	2/5; 40%	3/5; 60%		not tested
Draft horse *n* = 5	1/5; 20%	4/5; 80%		0.69
[a]Friesian *n* = 2	1/2; 50%	1/2; 50%		not tested
Other *n* = 5	0/5; 0%	5/5; 100%		0.07

[a] One horse from each of these breed groups had a perforating gastric ulcer and these horses were not included in this analysis.

were significantly more likely than case-controls to have absent borborygmi, abnormal mucous membranes, gastric reflux, rectal findings supportive of intestinal rupture (collapsed rectum within freely movable peritoneal space and detection of gritty film on serosal surface), and fulfill SIRS criteria (Table 2).

None of the horses with gastric rupture survived. Two horses were agonal at presentation. Eleven horses had gastric rupture confirmed during surgical exploration. Necropsy was performed in all cases. Cases were categorized as either idiopathic (*n* = 24) or having a known cause (*n* = 23) of the gastric rupture. The known causes were gastric impaction in 20 cases and perforating gastric ulcer in 3 cases. Non-perforating gastric ulcers were an incidental finding in 14 cases (7 gastric impaction and 7 idiopathic causes). Forty horses ruptured in the greater curvature of the stomach, 1 at the fundus, 1 at the cardia, 1 at the pylorus, and the location of rupture was not recorded in 4 cases.

The 20 gastric impaction causes for gastric rupture were compared to the 24 idiopathic gastric rupture cases. Number of perforating ulcer etiologies was insufficient for analysis. There was a significant breed effect on etiology ($P = 0.02$) with the Thoroughbred breed being significantly over-represented in the idiopathic gastric rupture group (Bonferroni adjusted $P = 0.02$; Table 3). There was no apparent effect of season, duration, gender, or age on primary gastric rupture etiology.

Discussion

This study identified important breed-associations for primary gastric rupture as a whole and idiopathic primary gastric rupture individually. An overall effect of season was significant, although the *post hoc* pairwise comparisons lacked power to attain statistical significance. Duration of clinical signs was not significantly different between known and idiopathic causes of gastric rupture. The presentation of gastric rupture cases was markedly different from case-control colic cases for most clinical and clinicopathological variables, but not for rectal temperature and peritoneal TNCC.

The most remarkable finding of this study was the apparent breed-associated predisposition for development of primary gastric rupture. When gastric rupture cases were compared to the overall hospital colic population, Quarter horses were significantly under-represented and both Friesians and Draft horse breeds were over-represented in the primary gastric rupture population. The apparent breed associations seen in this study may be related to breed-related management differences or, less likely, to owner willingness to refer. More specifically, the apparent under-representation of Quarter horses in the gastric rupture population may reflect different management practices among breeds or disciplines, with some management systems predisposing horses to gastric rupture as suggested by Kiper et al (8) or it may be an unrepeatable finding. The apparent breed predisposition of Friesians and Draft breeds to gastric rupture may be related to breed associated differences in pain tolerance or the presence of underlying connective tissue defects or gastrointestinal motility dysfunction. Subjectively, Draft horses and Friesians are more stoic breeds and may not manifest colic pain until severe, possibly delaying treatment and predisposing them to gastric rupture. It is also possible that both Draft horses and Friesians have an underlying structural or functional defect in their stomach, making them more likely to rupture compared with other breeds. The Friesian breed suffers from several soft tissue disorders, including megaesophagus and aortic rupture (11–18), and it is conceivable that other disorders could affect the stomach (19,20).

Most cases (51%) in this study did not have an etiology identified for the cause of the gastric rupture, which is comparable to the percentages of idiopathic cases reported in other studies (6,8). The large number of idiopathic primary gastric rupture cases indicates that causes of gastric rupture may not be readily evident on necropsy examination and our understanding of this disease remains incomplete. Sixty-eight percent of horses in this study showed clinical signs of colic for less than 12 h, including 17/20 (85%) of horses with confirmed or probable gastric impaction. These findings concur with a study describing gastric impaction in horses (21) and suggest that events leading to gastric rupture, even gastric impaction, may not be long standing or may not be associated with recognizable clinical signs prior to rupture.

It is the authors' subjective impression that draft breeds are predisposed to gastric impaction, which if untreated, may progress to gastric rupture. However, there was no apparent breed predisposition to gastric impaction and rupture when different etiologies were compared. The power for this analysis was limited due to low numbers in each breed group. The apparent predisposition of Thoroughbreds to idiopathic gastric rupture was unexpected and we do not have a good explanation for this apparent predisposition. Details regarding management-associated risk factors were limited in this study, because the severe clinical presentation often directed attention toward diagnosis and management of the patient and away from history-taking. Histopathology was not routinely performed as part of

the necropsy examination due to the advanced gross pathology lesions; however, subsequent to this study, we have been requesting histopathology for these cases. Histopathologic examination of human neonatal idiopathic gastric rupture has suggested that these cases may be related to motility disorders and reduced gastrointestinal pacemaker cells (22,23). Reduced density of these same pacemaker cells has been associated with colic of large intestine origin in horses (24,25). The potential breed predispositions seen in this study warrants further confirmation and investigation of these potential associations, including consideration of the role of the gastrointestinal pacemaker cells.

One horse (a Thoroughbred) in this study was reported to have fallen prior to presentation and presented with a complaint of neck pain and possible colic. This horse was diagnosed with idiopathic gastric rupture due to the absence of other gastric pathology. Trauma is an uncommon, but described, cause of gastric rupture in humans (26). Trauma is a common cause of various injuries in horses, although only a fraction of these events are witnessed and documented; therefore, we speculate that abdominal trauma may have been a cause for some of the idiopathic gastric rupture cases.

Not surprisingly, horses presenting with gastric rupture had more severe clinical and hematological derangements compared with case-controls (contemporaneous horses presenting for colic), reflecting the fact that horses with gastric rupture are more critically ill on presentation than horses with colic not associated with gastrointestinal compromise (9). Interestingly, rectal temperature was not significantly different between the gastric rupture and case-controls, although temperature derangements would be expected in the gastric rupture group. When the gastric rupture horses were compared to case-controls, there were more hypothermic and febrile horses in the gastric rupture group although several gastric rupture horses had normal rectal temperatures. This was attributed to horses presenting at varying times following rupture and the effect of prior treatments. The gastric rupture cases had significantly higher peritoneal TP compared to the case-controls, but no difference in peritoneal TNCC was noted, despite cytological evidence of septic peritonitis. This apparent discrepancy in peritoneal TNCC in the presence of septic peritonitis has been previously reported in gastric rupture cases (8,9) and is attributed to clumping and fragmentation of the nucleated cells in the low pH of gastric fluid (8). Horses with gastric rupture were also significantly more likely to have a complete lack of gastrointestinal motility on auscultation and were more likely to have gastric reflux than case-controls. Both of these findings likely reflect severe ileus, secondary to the overwhelming sepsis seen with gastrointestinal contamination of the abdomen. While it may seem counterintuitive to have gastric reflux when the stomach is ruptured, gastric contents are often partially contained within the stomach, omentum, and cranial abdomen, which may allow for gastric reflux.

Most of the horses with gastric rupture had clinical signs indicative of gastrointestinal rupture; however, 5 horses did not fulfill the SIRS criteria and had relatively unremarkable physical examination findings on presentation despite confirmed gastric rupture based on concurrent cytological examination of the peritoneal fluid or on surgical exploration. The difference in clinical presentation between these horses and those with signs compatible with gastrointestinal rupture may be attributed to factors such as prior treatment in the field, duration of gastric rupture, and degree of peritoneal contamination. These horses may have ruptured more recently, allowing less time for a systemic response. There may have been different degrees of peritoneal contamination among horses in this study which may have also influenced the degree of inflammation and clinical signs on presentation. Peritoneal fluid did not always have gross evidence of gastrointestinal contamination, and cytological evaluation was essential for early identification of peritoneal contamination. Finally, there are variations in individual responses to endotoxic insults (27), suggested to be due to circulating antibodies against endotoxin or genetic polymorphisms in the inflammatory response pathways (28,29). These findings suggest that in some cases overt clinical signs of gastric rupture may not be obvious on initial clinical examination and that early diagnosis is facilitated by cytological examination of peritoneal fluid.

A strength of this study was the varied breed distribution, which may have highlighted potential breed predispositions that were not evident in previous studies. Despite the detail of the medical records, the dramatic clinical presentation for many of these horses limited the collection of historical and management information that may have aided in identifying other risk factors for gastric rupture. Weaknesses of this study include those inherent with retrospective studies including missing data, incomplete historical information, and the small number of cases which limited the statistical power of some comparisons. The large number of idiopathic cases would also suggest more data need to be collected both pre- and post-mortem to find an underlying etiology for the gastric rupture.

This study demonstrates that gastric rupture continues to be an unpredictable and frustrating diagnosis. There may be breed predispositions to the development of gastric rupture, with draft horses and Friesian horses being over-represented in primary gastric rupture cases and Thoroughbred horses being over-represented in the subgroup of idiopathic gastric rupture. Further investigation is needed to determine if the breed predispositions are associated with management factors, which may be modifiable, or with breed phenotype or genotype, which may require specific interventions in susceptible individuals. CVJ

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