The importance of ultrasonographic pneumatosis intestinalis in equine neonatal gastrointestinal disease

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Summary

Reasons for performing study: Recognising the presence of a necrotising component of the gastrointestinal disease may be clinically useful in ill equine neonates.
Objectives: To study the importance of abdominal sonograms in neonatal foals suffering from gastrointestinal conditions and to describe the clinical features of necrotising gastrointestinal disease.
Hypothesis: There is a subgroup of neonates with sonographically detectable pneumatosis intestinalis (PI), reflecting a necrotising disease.
Methods: Records of foals aged ≤7 days hospitalised from 2005 to 2009 with signs of gastrointestinal disease were evaluated (n = 89). The association of sonographic, clinical and clinicopathological signs with necrotising gastrointestinal disease and outcome was determined.
Results: PI was imaged in 19 foals. Twenty-seven foals were classified as having necrotising gastrointestinal disease based on the presence of gastrointestinal signs (colic, diarrhoea, gastric reflux or abdominal distension) and sonographic PI (n = 19), surgical (n = 2) or pathological (n = 6) evidence of gastrointestinal necrosis. There was a difference between survival rate in foals with and without necrotising disease (33.3 and 69.4%, respectively, P = 0.005) or foals with and without PI detected sonographically (36.8 and 72.1%, respectively, P = 0.023). PI was the only sonographic finding associated with outcome. Prematurity, the presence of blood in the faeces, gastric reflux, abdominal distension, abnormal echogenicity of the colon and the lowest white blood cell count during hospitalisation were associated with necrotising gastrointestinal disease (P<0.05).
Conclusions and potential relevance: Abdominal sonograms have prognostic value in neonatal gastrointestinal disease. PI and the presence of necrotising gastrointestinal disease were common and associated with a poor prognosis.

Keywords: horse; pneumatosis intestinalis; necrotising gastrointestinal disease; neonate; abdominal ultrasonography

Introduction

Gastrointestinal (GI) disease is common in equine neonates. Foals presented for abdominal pain or diarrhoea may be easily identified as suffering from GI disease. However, neonates with other clinical presentations, such as sepsis, prematurity or perinatal asphyxia syndrome, may show nonspecific signs, with GI disease being one of many components of the process. Furthermore, clinical and clinicopathological signs overlap between these other disease conditions. Case management varies between various diseases and prognoses differ. Abdominal ultrasonography has proved useful in the assessment of human and veterinary cases with different conditions. In human critical care units, the time from presentation to operative care, length of hospitalisation, complications and cost decrease when abdominal sonograms are performed during emergency evaluations, potentially due to a more rapid diagnosis (Levy and Noble 2008). In paediatric colitis (Baud et al. 2004) and GI emergencies (Puylaert 2001; Pastore et al. 2007) sonographic findings correlate with anatomical distribution of the disease or diagnosis and often help patient management. Abdominal ultrasonography is well described in equine neonates (Reef 1998; McAuliffe 2004; Reef 2004; Porter and Ramirez 2005); however, studies that quantify its usefulness, accuracy and limitations are scarce.

Necrotising enterocolitis (NEC) is a disease process of the GI tract of neonates that results in inflammation and bacterial invasion of the bowel wall. Necrotising enterocolitis occurs frequently in human newborn intensive care units (Babcock 2002; Thompson and Bizzarro 2008), primarily in premature neonates (Lee and Polin 2003; Obladen 2009). Necrotising enterocolitis has been described in foals in isolated case reports (Cudd and Pauly 1987; Yager 1987; Jones et al. 1988; East et al. 1998) and several texts have suggested that it may be an under recognised syndrome (Slovis 2003; Magdesian 2005; Vaala 2009; Wilkins 2010). The diagnosis of NEC in human infants is based on clinical and imaging findings. The presence of intramural gas (pneumatosis intestinalis, PI) is considered virtually pathognomonic for necrotising enterocolitis in neonates with a compatible clinical picture (Walsh and Kliegman 1986; St. Peter et al. 2003; Epelman et al. 2007; Kim et al. 2007). Radiographic documentation of PI is the current standard to confirm NEC diagnosis in man, but the sonographic detection of PI has been shown to be both sensitive and specific (Kim et al.

2007) and to have a growing and evolving role in the evaluation of these patients (Epelman et al. 2007).

This retrospective study was designed to describe the abnormalities present in abdominal sonograms of equine neonates with signs of GI disease and the clinical and clinicopathological findings in neonates with necrotising GI disease. The data were analysed to study the association of sonographic, clinical and clinicopathological abnormalities with the presence of sonographic PI, necrotising GI disease and outcome. We hypothesised that the sonographic signs in foals with GI disease are varied but there is a subgroup of neonates with characteristic sonographic findings (including PI) and a clinical presentation suggestive of a necrotising component of the GI disease. We also hypothesised that foals with necrotising GI disease and PI have a poor prognosis.

Materials and methods

Case selection

Records of foals aged ≤7 days presented between 31 December 2005 and 31 December 2009 with clinical signs of GI disease (colic, diarrhoea, gastric reflux or abdominal distension) or that developed these signs during hospitalisation were studied retrospectively. Clinical, clinicopathological and sonographic data were obtained from medical records. Videotapes of the sonographic examinations were reviewed blindly by one of the authors (C.N.S.).

Ultrasonographic interpretation

Categorical sonographic variables included in the analysis were: abdominal sonogram performed during hospitalisation; presence and location of PI within the GI tract; presence of abnormalities in echogenicity of the stomach, duodenum, jejunum, ileum, colon and caecum; presence of ileus within the GI tract; presence of abnormal stomach contents; presence of functional abnormalities in the duodenum, jejunum, ileum, colon or caecum, presence of meconium; presence and distribution of abnormal peritoneal fluid; and presence of abnormalities in the liver. PI was identified
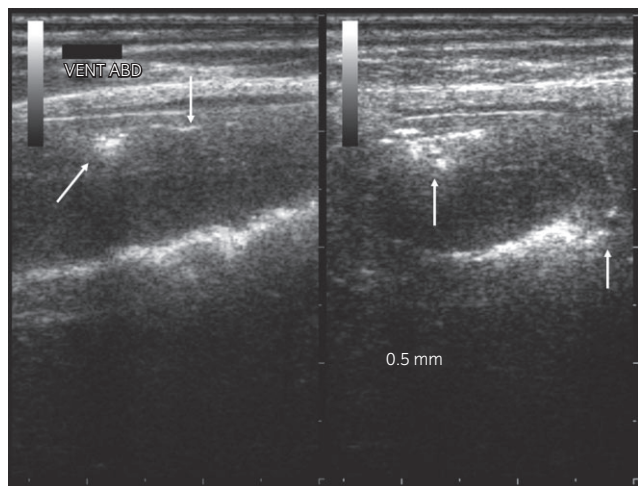
Fig 1: Sonogram of the ventral abdomen obtained from a foal with enterocolitis. Notice the hyperechoic intramural gas echoes (arrows) within a loop of thickened small intestine (5 mm). The gas echoes are imaged in mutually perpendicular longitudinal (left) and transverse (right) planes.

when hyperechoic echoes casting a dirty shadow were imaged within the bowel wall consistent with intramural gas (Fig 1 and Supporting information). Abnormalities in bowel wall echogenicity were areas of increased, decreased or heterogeneous echogenicity within the intestinal wall (Fig 2). Functional abnormalities of the gastrointestinal tract were defined as abnormalities in the contents or motility of the affected portion of the GI tract. Motility was considered normal if there were frequent, rhythmic peristaltic waves. Subjectively increased, decreased or arrhythmic contractions were considered abnormal. The stomach was not evaluated for motility disorders. Ileus was defined as diffuse hypomotility and distension of the gastrointestinal viscera (Fig 3). Abnormal peritoneal fluid was defined as fluid that is hypoechoic to echoic or increased in amount (Reef 2004; Porter and Ramirez 2005) (Fig 4). The presence of focally abnormal peritoneal fluid (vs. diffusely) was analysed as a separate variable.

Data recorded

Categorical clinical and clinicopathological variables obtained from records were outcome, presenting complaint, prematurity, feeding
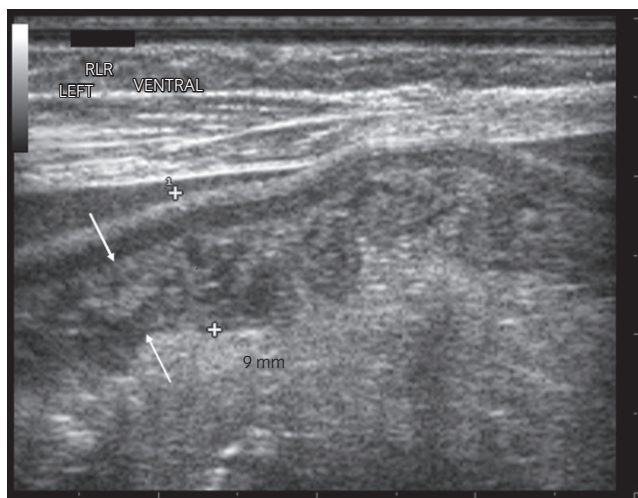

Fig 2: Sonogram of the large intestine obtained from a foal with enterocolitis. Notice the increased thickness of the wall (9 mm) and the thickened heteroechoic submucosa (arrow).
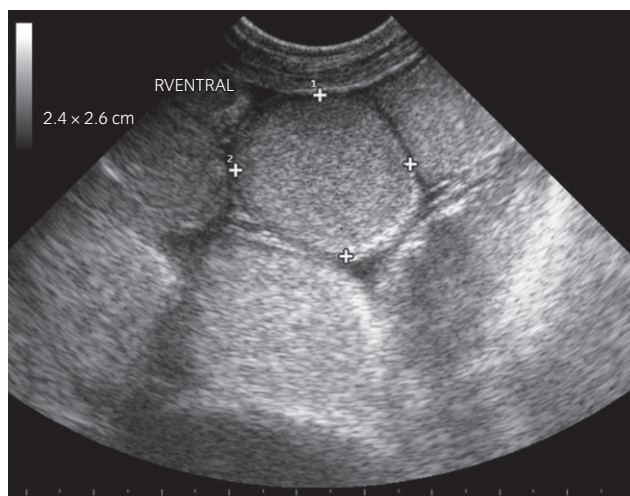

Fig 3: Sonogram of the abdomen obtained from a foal with enterocolitis. Notice the distended loops of small intestine consistent with ileus.

strategy, presence of necrotising gastrointestinal disease, presence of colic, diarrhoea, gastric reflux, blood in faeces, abdominal distension, pneumonia, respiratory distress, bacteraemia, clostridiosis, salmonellosis and congenital heart disease. The presenting complaint was categorised as colic, diarrhoea or other. If there were multiple complaints, the problem prompting hospitalisation was chosen for analysis. Foals were considered premature or to have respiratory distress if clinicians recorded these in the medical records. Foals were considered to have pneumonia if recorded by the clinician or pathologist. Necrotising gastrointestinal disease was defined as signs of GI disease and sonographic evidence of PI or surgical or pathological evidence of necrosis of the intestinal wall.

Continuous sonographic variables recorded were the thickness of stomach, duodenum, jejunum, ileum, colon and caecum. The continuous clinicopathological variables recorded were highest white blood cell count (WBCC), lowest WBCC, lowest total protein (TP), highest fibrinogen, plasma IgG concentration (DVM Rapid Test)[a] at presentation, highest lactate concentration, lowest PaO_2 and highest $PaCO_2$.

Data analysis

For the statistical analysis, the uniqueness of each record was checked. Fisher's exact test was used to detect the association between pairs of
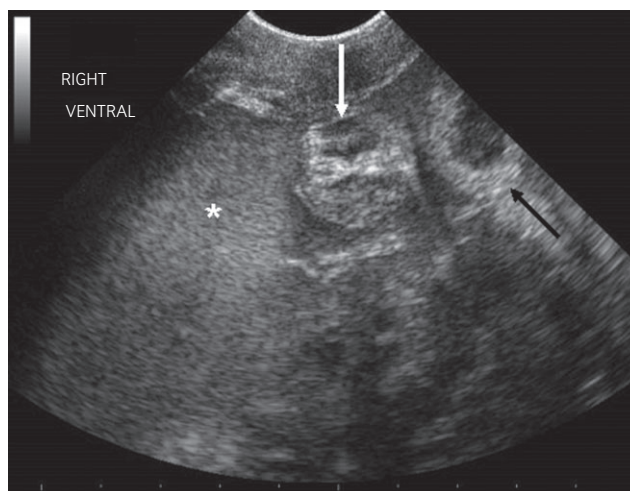

Fig 4: Sonogram of the ventral abdomen of a foal with enterocolitis. Notice the increased amount of echoic peritoneal fluid (asterisk), surrounding loops of ileum (white arrow) and jejunum (black arrow).

categorical variables and to assess the significance of such associations. Logistic regression was used to quantify the associations between dichotomous outcomes and plausible metabolic drivers. For polychotomous outcomes, associations were detected by the Kruskall-Wallis procedure and the strength, sense and significance of the associations additionally confirmed with the aid of Somer's D procedure. Differences were reported as observed. The statistical software Stata 11.1[b] was used for the analysis. A P value of 0.05 was used to distinguish significant from nonsignificant associations. Only foreshadowed associations were explored, in keeping with the study design.

Results

The inclusion criteria were met by 115 foals. Twenty-six foals were eliminated because of insufficient information in the medical records; therefore data from 89 foals were analysed. Variables related to the sonographic appearance of the ileum were excluded from the analysis owing to insufficient data. Variables related to feeding strategies were excluded owing to heterogeneity of feedings before presentation and changes during hospitalisation. There were 48 males and 41 females. There were 58 Thoroughbreds, 15 Standardbreds, 6 Warmbloods, 4 Arabians and 6 foals of other breeds. Twenty-four foals presented for colic, 8 for diarrhoea (4 had colic and diarrhoea as presenting complaints but abdominal pain prompted referral) and 57 presented with other complaints. These included weakness/depression (n = 15), the result of a high risk pregnancy (n = 7), dystocia (n = 6), neonatal encephalopathy (n = 6), recumbency (n=4), straining to urinate (n=4), failure to nurse (n=3), premature placental separation (n = 3), respiratory distress (n = 2), prematurity (n = 2), ruptured bladder (n = 1), septic shock (n = 1), anaemia (n = 1), flexural deformity (n = 1) and haemorrhage (n = 1). Forty-seven neonates did not have signs of gastrointestinal disease recorded at presentation and developed them during hospitalisation. The mean ± s.d. age at presentation was 1.9 ± 1.4 days. Abdominal ultrasound examinations were performed stall-side in 62 neonates. PI was most frequently imaged as scattered hyperechoic foci within the intestinal wall, although large clusters of hyperechoic foci within the bowel wall were also imaged in some foals. The intramural gas was imaged more frequently in the small intestine (57.9%, 11/19) than in the large intestine (31.6%, 6/19), and infrequently (10.5%, 2/19) in both the small and the large intestine. Twenty-seven foals were classified as having necrotising disease based on the clinical signs of GI disease and sonographic evidence of PI (19), and/or surgical (2) or pathological (6) evidence of necrosis. *Post mortem* examinations were performed in 4 foals with sonographic PI and, in 2 cases, histology was consistent with necrotising disease. Another foal with sonographic PI had an exploratory laparotomy and showed extensive macroscopic abnormalities most consistent with necrotising GI disease, including intramural gas.

Summaries of the clinical/clinicopathological and sonographic abnormalities of all foals and foals having necrotising GI disease are shown in Tables 1 and 2, respectively. Exploration of associations of clinical and clinicopathological abnormalities with the presence of PI revealed that the lowest WBCC (P = 0.029) and lowest TP (P = 0.032) were associated with the presence of PI. Exploration of associations of sonographic, clinical and clinicopathological abnormalities with the presence of necrotising GI disease and outcome revealed that PI was the only sonographic change associated with survival (P = 0.023). The survival rate was 36.8% (7/19) if PI was imaged sonographically vs. 72.1% (31/43) in foals that had abdominal scans and PI was not imaged. There was also an association between outcome and necrotising GI disease (P = 0.005). Only 33.3% (9/27) of foals with necrotising disease survived vs. 69.4% (43/62) of foals without evidence of necrotising disease. The presence of necrotising GI disease was associated with prematurity (P = 0.03, odds ratio [OR] 5.1), the presence of blood in the faeces (P = 0.009), gastric reflux (P = 0.003), abdominal distension (P = 0.009) and the lowest WBCC (P<0.001). Detection of abnormal echogenicity of the colon was associated with the presence of necrotising disease (P = 0.015).

Clinical or clinicopathological variables associated with outcome were presence of pneumonia (P = 0.01), respiratory distress (P = 0.008), lactate concentration (P<0.001), highest WBCC (P = 0.034), lowest WBCC (P = 0.025), highest fibrinogen (P = 0.02), and highest PaCO$_2$ (P = 0.014).

TABLE 1: Clinical and clinicopathological variables in foals with GI disease

	All cases	Cases with necrotising GI
Complaint	24 colic, 8 diarrhoea and 57 other	7 colic, 3 diarrhoea, 17 other
Prematurity	13/46 (28.2%)	7/13 (53.8%)*
Survived to discharge	52/89 (58.4%)	9/27 (33.3%)*
Colic	59/81 (72.8%)	20/24 (83.3%)
Reflux	20/80 (25%)	11/23 (47.8%)*
Abdominal distension	52/78 (66.6%)	19/21 (90.4%)*
Diarrhoea	27/78 (34.6%)	10/23 (43.5%)
Blood in faeces	12/79 (15.2%)	8/23 (34.8%)*
Congenital heart disease	3/83 (3.6%)	2/26 (7.7%)
Pneumonia	22/86 (25.6%)	9/26 (34.6%)
Respiratory distress	17/85 (20%)	4/25 (16%)
TP (g/l)	51 ± 11	50 ± 15
Lactate (mmol/l)	6.7 ± 4.9	6.7 ± 4.7
Fibrinogen (g/l)	5.73 ± 2.00	5.53 ± 2.57
IgG	688.3 ± 654.4	619.3 ± 646.5
Surgery	7/89 (7.9%)	2/27 (7.4%)
Highest WBCC (10^9 cells/l)	8.73 ± 4.66	7.35 ± 4.47
Lowest WBCC (10^9 cells/l)	4.84 ± 3.29	3.76 ± 3.14*
Clostridium toxins	8/31 (25.8%)	3/16 (18.7%)
Salmonella PCR	7/47 (14.9%)	2/14 (14.3%)
Bacteraemia	32/66 (48.5%)	12/22 (54.6%)
Lowest PaO$_2$ (mmHg)	59.0 ± 18.6	62.3 ± 17.4
Highest PaCO$_2$ (mmHg)	57.4 ± 24.3	56.5 ± 16.8

Results are presented as abnormal individuals/total and percentages in parentheses. Continuous data are presented as mean ± s.d. TP = total protein. FPT = failure of passive transfer. WBCC = white blood cell count. * denotes variables for which significant differences were found between the 2 groups (P<0.05).

Prematurity was a perfect predictor of outcome (0/13 premature foals survived to discharge).

Discussion

Abnormalities in all parts of the GI tract and abdominal viscera were found in the sonograms of neonates with signs of GI disease, supporting the importance of performing comprehensive abdominal ultrasound examinations in this group. Using high-frequency ultrasound transducers, the thickness, echogenicity and layering of the wall of the GI viscera can be evaluated (Porter and Ramirez 2005). Different patterns of abnormal intestinal echogenicity, based on the loss/maintenance of stratification and folding, have been shown to help define the GI disease process in children with colitis (Baud *et al.* 2004). In this series of cases abnormalities in the GI wall, echogenicity was frequently associated with the diagnosis of necrotising GI disease. However, we did not analyse changes in wall layering and the lack of definitive diagnosis in many cases would have made pathological correlations difficult. Further studies are needed to investigate the association of different sonographic patterns with specific diagnoses in foals.

The radiographic diagnosis of PI had been reported in 2 equine neonates (Cudd and Pauly 1987). To our knowledge, this is the first report of the sonographic detection of PI in foals and evaluation of its prognostic value. The sonographic appearance of PI in the equine neonates was most frequently that of scattered hyperechoic foci, although clustered foci were imaged in some instances. Detailed images using high frequency transducers allow for the differentiation between intraluminal and intramural gas, for detection of small amounts of intramural gas and for determining the extent of the PI. The pattern or extent of PI does not correlate with the severity of the symptoms in clinical NEC in human neonates (St. Peter *et al.* 2003), but it did in an experimental model in

TABLE 2: Sonographic findings in foals with gastrointestinal disease

	All cases	Cases with necrotising GI
Sonogram performed	62/89 (69.9%)	24/27 (88.9%)
Abnormal region of GI	SI 19/52, LI 8/52, none 4/52, SI and LI 20/52	SI 8/23, LI 5/23, none 0/23, SI and LI 10/23
Ileus	15/57 (26.3%)	7/23 (30.4%)
PI	19/62 (30.6%)	19/24 (79.1%)*
Gastric echogenicity	1/45 (2.2%)	1/21 (4.7%)
Gastric contents	8/41 (19.5%)	5/19 (26.3%)
Gastric wall thickness (mm)	3.4 ± 2.1	3.4 ± 2.8
Duodenal echogenicity	1/24 (4.2%)	0/10 (0%)
Duodenal function	19/33 (57.6%)	11/17 (64.7%)
Duodenal wall thickness (mm)	2.3 ± 1.1	2.4 ± 1.0
Jejunal echogenicity	17/54 (31.4%)	12/22 (54.5%)
Jejunal function	36/54 (66.7%)	15/23 (65.2%)
Jejunal wall thickness (mm)	3.3 ± 1.7	3.5 ± 2.0
Colonic echogenicity	10/56 (17.8%)	10/22 (45.4%)*
Colonic function	25/52 (48.1%)	12/22 (54.5%)
Colonic wall thickness (mm)	2.6 ± 1.3	3.1 ± 1.6
Caecal echogenicity	3/21 (14.2.%)	2/7 (28.5%)
Caecal function	8/14 (57.1%)	6/7 (85.7%)
Caecal wall thickness (mm)	2.5 ± 1.8	2.5 ± 2.3
Peritoneal fluid	14/54 (25.9%)	5/20 (25.0%)
Focal fluid	1/54 (1.8%)	1/20 (5%)
Liver	1/57 (1.8%)	0/22 (0%)

Results are presented as abnormal individuals/total and percentages in parenthesis. Continuous data are presented as mean ± s.d. PI = pneumatosis intestinalis. SI = small intestine, LI = large intestine. * denotes variables for which significant differences were found between the 2 groups (P<0.05).

rabbits (Kim *et al*. 2007). We did not attempt to describe a pattern of PI nor to quantify or grade the severity of the PI in our foals because of the retrospective nature of the study. Intramural gas may involve any part of the GI tract in human NEC but is more commonly present in the distal small bowel and large bowel and imaged more commonly in the right lower quadrant (Kim *et al*. 2007). In our study, the intramural gas was imaged more frequently in the small intestine than in the large intestine and was detected infrequently in both small and large intestine.

Sonograms were performed stall-side without the need for anaesthesia. Other imaging modalities may be more difficult to perform in the equine neonatal intensive care unit setting. With the exception of occasional radiographs, other imaging modalities were not used in this group of neonates and we did not attempt comparison with other potentially useful imaging techniques (radiography, computed tomography or magnetic resonance imaging).

Of the many sonographic features of NEC, a recent study in human neonates showed that an adverse outcome was associated with the presence of free peritoneal gas, focal fluid collections or 3 or more of the following: increased bowel wall echogenicity, absent bowel perfusion, portal venous gas, bowel wall thinning, bowel wall thickening, free peritoneal fluid with echoes, and intramural gas (Silva *et al*. 2007). Sonographic assessment of intestinal vascularisation was not routinely performed in the foals in this study and thus bowel perfusion could not be critically evaluated. Further studies are needed to characterise intestinal vascularisation using colour flow Doppler ultrasound in equine species and to determine its value as a diagnostic tool.

In our analysis, PI was the only sonographic finding associated with a poor outcome. Pneumatosis intestinalis is not a diagnosis or a clinical problem *per se*, but a pathological or imaging finding that is a consequence of an underlying disease (St. Peter *et al*. 2003). PI has been attributed to at least 58 causes in man (King and Suckett 1992; Gurland *et al*. 1998; Silva

et al. 2007) although it is virtually pathognomonic of NEC in human neonates with compatible clinical presentation (Kim *et al*. 2007). The imaging findings can therefore only be interpreted in the context of the neonatal patient with an appropriate clinical presentation.

The precise aetiology of NEC is unknown, but its aetiopathology probably represents an interaction of factors leading to intestinal inflammation and bacterial invasion. Wall ischaemia is the final pathway and not the primary initiator of the PI (Lee and Polin 2003; Thompson and Bizzarro 2008). Prematurity is the only independent determinant of human NEC (Lee and Polin 2003), and prematurity and dystocia (Cudd and Pauly 1987) have been suggested to be associated with NEC in foals. In this retrospective study, there was a difference (P = 0.03, OR 5.1) in the presence of NEC between premature (7/13, 53.8%) and term (6/32 18.75%) foals. However, the actual breeding date was seldom recorded in the medical record, and also conclusions should be drawn with caution. Even though in this group premature foals were more likely to develop necrotising disease (5.1 times more likely as determined by the OR), foals analogous to premature very low birthweight children are not frequent. Therefore, direct extrapolation of information from human NEC to necrotising GI disease in equine neonates may not yield applicable conclusions in all instances.

Three factors are felt to be necessary for NEC to occur in human neonates: ischaemic/hypoxic mucosal injury, feeding and the presence of bacteria (Lee and Polin 2003). These factors may be present in critically ill equine neonates. Respiratory distress syndrome or congenital heart disease has been suggested to play a role in the pathogenesis of NEC in human neonates by causing hypoxic mucosal injury (Guner *et al*. 2008). We tested the association between respiratory disease, arterial blood gases values and congenital heart disease with the presence of necrotising gastrointestinal disease and no association was found. The number of neonates with congenital heart disease in this series was small (n = 3) and therefore the lack of association could be due to lack of statistical power. Bacteraemia has been documented in up to 35% of NEC cases in children (Lee and Polin 2003) and some authors have hypothesised that NEC is a contagious disease, implicating *Klebsiella*, *E. coli*, *Enterobacter*, *Salmonella*, *Pseudomona*, *Clostridia*, *Staphylococci*, coronavirus, rotavirus or enterovirus (Walsh and Kliegman 1986). There are reports associating *C. difficile* (Jones *et al*. 1988), *C. perfringens* (East *et al*. 1998) and *Rhodococcus equi* (Yager 1987) with NEC in foals. Blood cultures, polymerase chain reactions for detection of salmonella in faeces and faecal testing for the detection of clostridial toxins were performed frequently in the foals reported herein, and a positive result was not associated with a necrotising component to their GI disease.

Progression of NEC in human neonates may take a variable period, although it can be an extremely rapid process. Sonographic findings match the pathological stages and may precede clinical signs (Faingold *et al*. 2005; Epelman *et al*. 2007). The hallmark pathological finding of NEC is ischaemic or coagulative necrosis, which is characterised by full thickness necrosis, paucity of acute inflammatory cells (neutrophils) and a predominantly lymphocytic infiltrate (Guner *et al*. 2008). In our group, it was not possible to stage the disease as there were not enough serial scans performed to assess the sonographic progression. The sonographic and pathological or surgical findings were well correlated in 3 individuals that had both an abdominal ultrasound examination and surgery/necropsy. Detailed complete evaluation of the GI tract histologically may be necessary to detect focal regions of necrosis. This may be the reason for the lack of detection of a necrotising component in the pathological examination of 2 of the cases in this series. In these 2 foals in which histopathology did not find evidence of necrosis only a few areas of PI were seen sonographically, which would have been difficult to find at *post mortem* without guiding the pathologist to the area of interest.

Differentiating intraluminal from intramural gas by ultrasonography may be difficult in some cases. Rotating the transducer, changing subject position and the use of higher frequency transducers with excellent resolution help in distinguishing PI from artefacts (pseudopneumatosis intestinalis) (Kim *et al*. 2005, 2007). Only subjects in which the hyperechoic echoes of gas were confidently imaged within the wall were classified as having PI in this study. Statistical analysis of the correlation between sonographic and histological changes was not performed owing to the small number of cases that had both.

This study has all the weaknesses associated with its retrospective nature, including the presence of missing data. Therefore, a prospective study assessing the maturity of the foal, the feeding practices, clinical findings, clinicopathological findings, ultrasonographic findings and the results of surgical intervention should be performed to further study this disease affecting neonatal foals. Prospective examination of tissue samples from foals with sonographically diagnosed PI is indicated to further study the association between sonographic PI and pathological necrotising GI disease. The sonographic evaluation of the neonatal intestine should include colour Doppler assessment of intestinal blood flow, as submucosal blood flow characterisation using colour Doppler sonography correlates well with histology and outcome and allows differentiation of focal from diffuse necrosis in human neonates (Faingold *et al*. 2005). Normal descriptions of the sonographic appearance of the neonatal GI tract, including measurements of wall thicknesses and assessment of intestinal blood flow, also need to be obtained.

In conclusion, this is the first report of the detection of PI sonographically and the evaluation of its prognostic value in neonatal foals. PI and necrotising GI disease were associated with an adverse outcome in this case series. Prematurity, the presence of blood in the faeces, gastric reflux, abdominal distension, abnormal echogenicity of the colon and leucopenia were associated with a necrotising component of the disease. The prevention, detection and clinical needs of equine neonates with necrotising GI disease deserve further attention.

Conflicts of interests

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Acknowledgements

Manufacturers' addresses

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[b]Stata Corp, College Station, Texas, USA.

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Supporting information

Additional supporting information may be found in the online version of this article.

Video: Sonogram of the ventral abdomen of a foal with enterocolitis. Notice the hyperechoic intramural gas within a loop of thickened small intestine. The gas echoes are imaged extending from the mucosal surface into the submucosa and reach the limit between this and the muscularis. The submucosa is thickened and presents increased echogenicity.

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