Case Series: Diagnostic investigation and treatment of peritonitis in six horses

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The diagnosis of peritonitis is based on an elevated abdominal fluid white blood cell count (>10 x 10^9/l) (Dyson 1983). Clinical signs frequently associated with peritonitis include anorexia, signs of depression, pyrexia, colic and diarrhoea (Mair 2002). Primary peritonitis is uncommon in adult horses but does occur associated with *Actinobacillus equuli* infection which has a higher prevalence in Australia than other geographical areas (Golland et al. 1994; Matthews et al. 2001). Secondary peritonitis has many potential aetiologies including surgical interference, gastrointestinal rupture, intestinal ischaemia, external trauma, rectal tears, breeding and foaling injuries, cholangiohepatitis (Mair 2002; Javiscus et al. 2010), parasitism (Dyson 1983), penetrating foreign bodies (Ramirez et al. 1997), abdominal abscessation (Pusterla et al 2007), and neoplasia (Taylor et al. 2006). Reported short-term survival rates for peritonitis have varied [53 to 86 per cent (Dyson 1983; Mair et al. 1990; Feige et al. 1997; Henderson et al. 2008)] and long-term complications such as recurrent colic, adhesion or abscess formation may further reduce survival rates (Henderson et al. 2008). This case series describes the clinical findings, diagnostic investigation, treatment and outcome of 6 horses with peritonitis.

Case history and clinical examination

The signalment, history and presenting complaints for all cases are outlined in Table 1. All cases had been treated by referring veterinarians for colic with non-steroidal anti-inflammatory drugs. Clinical findings at admission, diagnosis and outcome are summarised in Table 2.

Diagnostic Investigations

All 6 horses had the following diagnostic tests performed at admission; haematology and biochemical analysis, faecal egg count, peritoneal fluid cytology and culture, rectal examination, transabdominal ultrasound and gastroscopy (24 hours after admission). The findings are summarised in Tables 2-4.

Laboratory Findings

Abdominocentesis, performed at admission, yielded turbid samples in all cases, with an abnormal colour [cream (case 1), orange (case 5), and serosanguineous (cases 2, 3, 4, 6)], an elevated protein concentration (median 47.5 g/l, range 34.0-71.8g/l, normal<25g/l) and elevated total nucleated cell count [median 144.6x10^9/l, range 21.2-243.0x10^9/l, normal<10x10^9/l) (Table 3)]. Neutrophils predominated in all samples (median 84%, range 80-90%, normal 20-90%) and were degenerate in 2 samples (cases 1 and 6). Gram-negative rods were identified on a peritoneal fluid smear from case 1. Aerobic and anaerobic culture of peritoneal fluid collected into plain tubes was negative in all cases.

The initial haematological and biochemical analyses (Table 4) revealed haemoconcentration (cases 1 and 6), leukopaenia (cases 1, 2 and 6) and leukocytosis (case 3). Cases 1 and 2 developed a neutrophilic leukocytosis within 5 days of therapy while case 6 remained persistently leukopaenic. Azotaemia that resolved following 48 hours of fluid therapy was noted in cases 1 and 6. Four cases were hypoproteinaemic (total protein <58g/l) and cases 2 and 5 were also hypoalbuminaemic (albumin<23g/l).

Faecal egg count was performed in all 6 horses and was elevated in case 2 (800 epg); all other cases were negative. Five day sequential faecal samples were collected from the horse with diarrhoea (case 6) and submitted for bacterial culture; these samples were negative for Salmonella organisms. An ELISA for detection of Clostridium difficile toxin A in faeces was also negative (Clostridium difficile Tox A ELISA,Techlab, Blacksburg, Virginia, U.S.A.).

Table 1: Subject details, history, clinical signs and treatment prior to referral of 6 horses with peritonitis.

	Age (years)	Sex	Breed	Duration clinical signs prior to referral	Clinical signs	Treatment prior to referral	Other
Case 1	10	Mare	Thoroughbred	72 hours	Inappetance, dullness, mild abdominal pain, pyrexia	Phenylbutazone	Naturally mated 4 days prior to referral
Case 2	9	Mare	Thoroughbred	24 hours	Inappetance, dullness, moderate abdominal pain	Flunixin	
Case 3	7	Stallion	Warmblood	12 hours	Inappetance, dullness, moderate abdominal pain	Flunixin	
Case 4	7	Gelding	Thoroughbred	48 hours	Inappetance, dullness, mild abdominal pain, pyrexia, muscle fasciculations	Flunixin	
Case 5	19	Mare	Thoroughbred	2 months	Partial inappetance, dullness, recurrent mild abdominal pain, weight loss	Phenylbutazone, anthelmintics	Chronic weight loss and recurrent colic preceding 2 months
Case 6	28	Gelding	Pony	24 hours	Inappetance, dullness, mild abdominal pain, diarrhoea	Flunixin	

Table 2: Clinical findings, diagnosis and outcome of 6 horses with peritonitis

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Temp (°C)	40.1	37.6	38.3	39.5	38.0	39.3
Heart rate (beats/min)	80	44	48	40	48	90
Respiratory rate (breaths/min)	24	25	28	16	16	30
Mucous membranes, capillary refill time	Hyperaemic, 3 seconds	Pink, 2 seconds	Jaundiced, 2 seconds	Jaundiced, 2 seconds	Pink, 2 seconds	Hyperaemic, 3 seconds
Hydration status	Marked dehydration	Normal	Mild dehydration	Normal	Normal	Marked dehydration
Intestinal sounds	Absent	Hypomotile	Hypomotile	Hypomotile	Hypomotile	Absent
Rectal examination	Secondary impaction of the large colon	>100 adult cyathostomes on rectal sleeve	Secondary impaction of the large colon	No abnormalities detected	Abnormal hard lobulated mass in ventral abdomen (circa 20 cm x 25 cm)	Gaseous distension of caecum
Nasogastric intubation	3litres reflux	3litres reflux	None	None	None	None
Other clinical findings	Vaginal tear, mild colic, profoundly dull	Moderate colic	Mild colic	Mild colic, muscle fasciculations	Ventral oedema, poor body condition (2/5)	Profuse diarrhoea, mild colic, profoundly dull
Diagnosis	Peritonitis, vaginal tear	Peritonitis, larval cyathostominosis	Idiopathic peritonitis	Glandular gastric ulceration, peritonitis	Peritonitis, alimentary lymphosarcoma	Peritonitis, gastric diverticulum
Outcome	Recovered	Recovered	Recovered	Recovered	Euthanasia	Euthanasia

Table 3: Results of sequential peritoneal fluid samples in Cases 1-6.

Sequential samples	Parameter	Units	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
1	WBCC	*x10⁹/l*	167.3	243.0	224.0	122.0	21.2	77.6
	TP	*g/l*	71.8	48.9	39.2	53.2	34.0	46.1
2	WBCC	*x10⁹/l*	85.3	39.2	92.0	126.4		177.6
	TP	*g/l*	31.1	32.1	38.3	46.2		46.9
3	WBCC	*x10⁹/l*	89.2	14.3	8.4	17.0		137.4
	TP	*g/l*	37.0	19.7	25.0	18.6		43.9
4	WBCC	*x10⁹/l*	9.6	5.6		13.0		258.4
	TP	*g/l*	39.9	18.7		30.6		46.1
5	WBCC	*x10⁹/l*	6.8			7.9		
	TP	*g/l*	24.9			25.6		

WBCC-white blood cell count, TP-total protein

Table 4: Relevant haematology and clinical chemistry results from initial blood sample in all cases.

Measurement	Units	Normal values	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6
Total white blood cells	*x10⁹/l*	5.5 - 12.5	**4.4**	**4.8**	**14.8**	9.7	8.7	**4.6**
Neutrophils (segmented)	*x10⁹/l*	2.7 - 6.8	3.25	3.26	**13.32**	**7.66**	**7.39**	3.91
Neutrophils (band)	*x10⁹/l*	0 - 0	**0.48**	0	0	0	0	0
Lymphocytes	*x10⁹/l*	1.5 - 5.5	**0.62**	**1.20**	**1.33**	**1.07**	**0.87**	**0.41**
Packed cell volume	*l/l*	0.24 - 0.42	**0.55**	0.33	0.36	0.30	0.34	**0.59**
Total protein	*g/l*	58 - 75	**55.4**	**55.8**	71.8	66.9	**47.5**	**55.2**
Albumin	*g/l*	23 - 35	27.2	**20.1**	31.6	31.4	**16.5**	23.6
Urea	*mmol/l*	2.5 - 8.3	**10.3**	8.0	5.0	2.8	2.9	**14.3**
Creatinine	*µmol/l*	40 - 150	**160.3**	149.0	102.0	99.8	119.0	**186.0**
Fibrinogen	*g/l*	2 - 4	3.1	1.9	3.5	3.6	3.6	2.1

Values lying outside the normal range are in bold.

Ultrasonographic examination findings

Transabdominal ultrasonographic examination showed a mild increase in peritoneal fluid in cases 2-5 whereas cases 1 and 6 (figure 1) had large volumes of echogenic peritoneal fluid. Multiple moderately distended loops of small intestine (circa 4-5cm in diameter) were identified in cases 1, 2 and 3. Increased small intestinal wall thickness (circa 0.8cm, normal<0.4cm) was present in case 5. Transrectal ultrasonography allowed visualisation of the mass palpated transrectally in case 5, showing it to be of mixed echogenicity with several small hypoechoic areas.

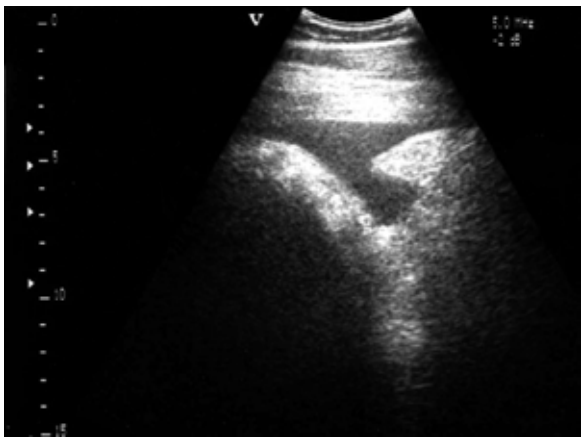

Figure 1: Transabdominal ultrasonography in case 6 demonstrating excess echogenic peritoneal fluid.

Gastroscopic examination findings

Gastroscopy was unremarkable in cases 1, 2, 3, and 6. Case 4 had several areas of deep ulceration in the glandular mucosa, particularly severe in the pyloric region (grade 3) (Vastistas et al. 1999). Superficial squamous mucosal ulcerations adjacent to the margo plicatus on the greater curvature of the stomach were also detected in case 5 (grade 1) (Vastistas et al. 1999).

Vaginal examination findings

This revealed a large (10cm) dorsal vaginal tear extending from the 7 o'clock to 11 o'clock position 5cm caudal to the cervix in case 1; endoscopy confirmed direct communication of the tear with the peritoneal cavity.

Laparoscopic and histopathologic examination findings

Laparoscopy was performed in case 5, as described by Ragle (2002). Briefly, the mare was sedated, the abdominal instrument portal sites were aseptically prepared and infiltrated with mepivacaine (Intra-Epicaine; Arnolds Veterinary Products). A dark-red lobulated mass (20cmx20cm) was visualised laparoscopically adherent to the jejunum. Several biopsy samples were obtained and fixed in formalin. Results of histopathologic examination were consistent with intestinal lymphosarcoma and the mare was subsequently euthanized 3 days after surgery. Necropsy confirmed a large mass adherent to the proximal jejunum (figure 2) and partial stenosis of the intestinal lumen at this point. Multiple focal areas of neoplastic infiltration were identified throughout the small intestine. The mesenteric and caeco-colic lymph nodes were enlarged. Histopathologic examination of the intestinal mass and mesenteric lymph nodes revealed the presence of neoplastic lymphoid cells.

Treatment

Medical therapy was attempted in all cases except case 5. Feed was withheld from all horses for at least the first 24 hours until resolution of abdominal pain and ileus associated with peritonitis and then food was gradually re-introduced.

Broad-spectrum antimicrobial therapy was initiated in all horses and included penicillin (15mg/kg IV TID Crystapen; Schering-Plough), gentamicin (6.6mg/kg IV SID Gentaject; Franklin Pharmaceuticals) and metronidazole (20mg/kg PO BID Metronex; Pharmacia Animal Health). The duration of IV antibiotic therapy varied between the cases and was continued until a clinical response and improvement in the laboratory data were achieved (median 7 days, range 6-18 days).

Cases 1 and 6 required IV replacement fluid therapy, consisting of hypertonic saline (7.2% NaCl) (Ivex Pharmaceuticals), followed by compound Ringer's lactate solution (Isolec; Ivex Pharmaceuticals) and 6 litres of fresh plasma. Flunixin meglumine was administered to all cases. Fenbendazole (7.5mg/kg PO SID Panacur Equine Guard; Intervet) was administered to case 2 for 5 consecutive days followed by moxidectin (0.4mg/kg PO Equest; Fort Dodge) on day 6. Case 4 received omeprazole (4mg/kg PO SID Gastrogard; Merial Animal Health) for 30 days.

Vaginal surgery and peritoneal lavage

Following sedation (romifidine and butorphanol) and epidural anaesthesia [lignocaine (0.33mg/kg Lidocaine Injection 2% Solution; Hameln Pharmaceuticals)]; the vaginal tear was repaired in case 1 with a continuous loop suture using 3 metric polyglycolic acid (Dexon; Ethicon). Peritoneal drains were inserted in the left paralumbar fossa and the ventral midline, after aseptic preparation and local anaesthesia (Figure 3). Peritoneal lavage was performed with 10 litres of warmed compound Ringer's lactate solution twice daily for 4 days.

Clinical progression

Cases 2, 3 and 4 showed a marked clinical improvement within 48 hours of admission, with cessation of colic signs and return of appetite, normal gastrointestinal activity and faecal output. Case 1 remained tachycardic (range 68-80 bpm) and pyrexic for 3 days after admission, but the mare's demeanour improved and her appetite returned and intravenous fluids were discontinued on day 7. Vaginal examination confirmed the vaginal tear to be healing satisfactorily on day 12. The total white cell count for all of the above cases showed a decline in sequential peritoneal fluid samples (Table 3).

In contrast case 6, despite a temporary improvement following fluid resuscitation and supportive treatment, showed continuing deterioration in clinical parameters and an increasing peritoneal total white cell count (Table 3). Furthermore, repeat transabdominal ultrasonography showed diffuse fibrin tags on the peritoneal surfaces on day 5. An exploratory laparotomy was declined by the owner and euthanasia was performed 6 days after admission. Necropsy showed diffuse fibrinous peritonitis and a closed diverticulum with separation of the muscular layers on the greater curvature of the stomach (25cmx25cm). No direct communication between the diverticulum and the gastric lumen was evident.

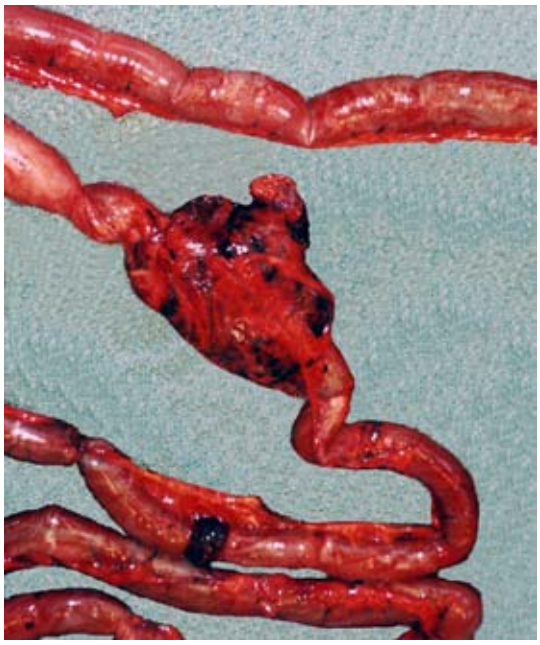

Figure 2: The small intestine at necropsy from case 5 showing a large red-black mass in the proximal jejunum and a second small annular mass.

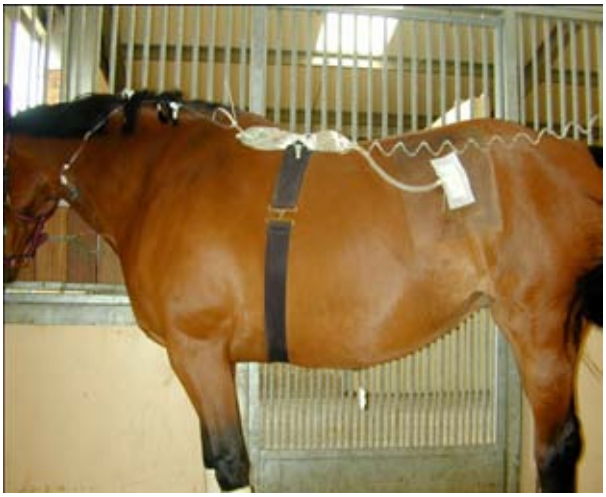

Figure 3: Case 1, peritoneal lavage system with an ingress catheter in the left paralumbar fossa and a ventral midline egress catheter.
Photograph courtesy of Dr. Todd Booth, Morphettville Equine Clinic

Follow-up

Four of the 6 cases survived to discharge (median 10.5 days of hospitalisation, range 8-16 days). Repeat examinations 1 month after discharge, consisting of clinical examination, transabdominal ultrasonography and abdominocentesis, were within normal limits. Additional diagnostic investigations at these repeat examinations included a faecal egg count (negative) in Case 2 and gastroscopy in Case 4. Repeat gastroscopy in case 4 showed complete healing of the previously noted ulcerated glandular mucosa. Telephone updates from the owners 6-12 months after discharge confirmed that the four surviving cases returned to their previous use.

Discussion

This case series describes the clinical presentation, diagnostic investigation and management of 6 cases of peritonitis. Although the diagnosis of peritonitis was relatively straightforward based on clinical signs and peritoneal fluid analysis in these cases, treatment was complicated by the difficulty in determining a definitive aetiology without surgical exploration and in giving an accurate prognosis based on the initial assessment. Dyson (1983) reviewed the clinical findings in 30 cases of peritonitis but the primary cause was rarely recognised. In other studies (Mair et al. 1990; Henderson et al. 2008), the aetiology was identified in 50% and 23% of cases respectively.

The cause of the peritonitis in case 1 was clear given the history of recent breeding and the identification of a vaginal tear communicating directly with the peritoneal cavity. In case 2, it was uncertain whether the larval cyathostominosis was the primary disease process with transmural extension of colonic inflammation to the peritoneum or whether the mass larval emergence was stimulated by an endogenous cortisol rise secondary to peritonitis. This horse did have mild hypoalbuminaemia (serum albumin 20.1 g/l), which is commonly seen (albeit as a non-specific laboratory finding) in larval cyathastominosis. Other common presenting signs of larval cyathostominosis (diarrhoea, weight loss, ventral oedema) were not evident (Lyons et al. 2000).

Although the inciting cause was not determined in case 3, possibilities would include localised perforation of intestine secondary to ulceration or foreign body penetration (Ramirez et al. 1997), abdominal abscessation (Pusterla et al. 2007), inflammatory bowel disease (Schumacher et al. 2000), blunt abdominal trauma, parasitism (Mair 2002) and primary peritonitis due to *Actinobacillus equuli* infection (Golland et al. 1994; Matthews et al. 2001). Exclusion of an ischaemic intestinal lesion was important in this case. However, in light of the stable clinical signs and diminishing peritoneal white cell count, further investigation using an exploratory laparotomy was considered unnecessary.

Gastric glandular ulceration was detected in case 4 and was considered as a potential cause of peritonitis. However it is unusual for even severe gastric glandular ulceration to result in extension of inflammation to the peritoneum or to result in gastric perforation in adult horses (Murray 2002). Furthermore, deep, perforating gastric ulcers would release large numbers of mixed bacteria into the peritoneal cavity and would have been associated with a much poorer prognosis than observed in case 4. The stress of concurrent illness (e.g. peritonitis) may result in an endogenous cortisol rise that inhibits mucosal prostaglandin production allowing glandular ulceration to occur (Nadeau and Andrews 2003). The ulceration was most likely secondary to the peritonitis in this case.

The peritonitis in case 5 with alimentary lymphosarcoma can be explained by either necrosis around the margins of the tumours or release of inflammatory mediators secondary to tumour infiltration (East and Savage 1998). Taylor et al. (2006) reported 34 horses with intestinal neoplasia and found that many intestinal tumours were associated with mucosal ulcers, which may lead to bacterial translocation and peritonitis; this mechanism was also a potential cause of the peritonitis in Case 5. Exclusion of more common causes of weight loss, such as dental abnormalities, inadequate nutrition, parasites, a chronic inflammatory focus, or liver disease (Taylor 1997), was important in this case. The weight loss in case 5 was likely due to a combination of factors including reduced intake as a result of inappetance, recurrent abdominal pain associated with partial stenosis of the jejunal lumen with neoplastic tissue, altered energy requirements due to the neoplasm, decreased utilisation of nutrients (e.g. through malabsorption secondary to infiltration of the small intestinal mucosa with neoplastic cells), and increased protein loss into the peritoneal cavity and/or intestinal tract.

Possible aetiologies for the gastric diverticulum in case 6 could have included a previous penetrating foreign body or prior episode of severe gastric distension (e.g. small intestinal obstruction, gastric impaction, pyloric stenosis). Gastric impactions may be primary (Vainio et al. 2011) or secondary to pyrrolizidine alkaloid toxicosis (Milne et al. 1990). There was no supporting evidence of liver disease in this case and it is unlikely that a primary gastric impaction would have spontaneously resolved without treatment. Pyloric stenosis in adult horses is rare (Venner 2004), particularly in the older horse, and was not identified on case 6's post-mortem examination. Possible pathogenic mechanisms for the severe diarrhoea in case 6 may have included disturbed motility, mural inflammation secondary to peritonitis (Dyson 1983), clostridial colitis (Baverud et al. 1998), salmonellosis (Gibbons 1980) and idiopathic colitis.

Transabdominal ultrasound examination was used extensively in this report both diagnostically and for monitoring purposes. Identifying the volume and character of the peritoneal fluid can be useful (Reef 1998; Southwood and Russell 2007); the echogenicity of peritoneal fluid will increase with increasing cellular content (Reef 1998). As a result of third spacing of fluid within the peritoneal cavity secondary to increased capillary permeability caused by acute, diffuse peritonitis, cases 1 and 6 had large volumes of abnormal (echogenic) peritoneal fluid visible on ultrasound examination. Transabdominal ultrasonography was additionally useful to identify moderately distended small intestinal loops in cases 1, 2, and 3 consistent with ileus that were not palpable transrectally; it also identified small intestinal mural thickening in case 5. Mural oedema or inflammatory cell infiltration was considered possible; however taken in conjunction with the large abnormal mass imaged by transrectal ultrasonography; this raised the clinical suspicion of a neoplastic process.

The absolute value of peritoneal fluid total white blood cell count in previous studies of peritonitis appeared to be of little prognostic value (Dyson 1983;

Mair et al. 1990). In this case series, case 2 had the highest white blood cell count (243.0 x 109/l) initially but a good clinical outcome while case 6 which was subsequently euthanised had an initial white blood cell count of 77.6 x 109/l, the lowest value of the cases selected for medical management. However this value did increase concurrently with the clinical deterioration of this case. No statistically significant association has been found previously between the identification of bacteria in peritoneal fluid smears and case outcome (Mair et al. 1990); case 1 had a positive Gram-stain of peritoneal fluid but a good clinical outcome. In agreement with findings of Dyson (1983) and Mair et al. (1990), sequential peritoneal fluid evaluations were found to be useful to monitor response to therapy in this case report. Bacterial culture of peritoneal fluid from all 6 cases was negative. Prior antimicrobial therapy may reduce culture rates (Mair et al. 1990); however this was not a causative factor for any of the horses in this case series. In previous studies, positive culture results were obtained from 59.7% (Hawkins et al. 1993), 26.7% (Dyson 1983) and 14.3% (Mair et al. 1990) of horses.

The main objectives of treatment for peritonitis in horses include elimination of the cause of inflammation and infection (if present), restoration of fluid and electrolyte balance, and administration of appropriate antimicrobial,analgesic and anti-endotoxic therapy (Davis 2003). The decision for medical versus surgical therapy in these cases was based on several factors including response to therapy, degree of abdominal pain and ileus, resolution of pyrexia, absence of persistent tachycardia. Successful medical therapy has the advantage of reduced cost and recovery time, and avoids the risks associated with general anaesthesia. Surgical exploration was indicated in case 6 based on the poor response to medical management, but it was unlikely, given the limited surgical options for treatment of a gastric diverticulum, that this would have altered the outcome.

In acute septic peritonitis, the inflamed peritoneum becomes a freely diffusible membrane, allowing a large influx of fluid and plasma proteins from the intravascular space into the peritoneal cavity resulting commonly in concurrent hypovolaemia, dehydration and hypoproteinaemia (Browning 2005); this was observed in cases 1 and 6. The severity of the hypoproteinaemia may be masked by accompanying dehydration. Hypertonic saline was administered to cases 1 and 6 for its rapid, although temporary, expansion of the intravascular volume (Bertone et al. 1990; Schmall et al. 1990). This was followed by large volume isotonic fluid and plasma administration. The concurrent administration of plasma with crystalloid therapy may help maintain intravascular oncotic pressure (thereby reducing extravasation of crystalloids into the interstitial space) and provides additional beneficial factors, such as coagulation factors, anti-coagulants (e.g. antithrombin, protein C) and anti-endotoxin antibodies.

Although some cases of peritonitis may be aseptic, antibiotic therapy was initiated in all cases. Preferably therapeutic choices are based on the results of culture and sensitivity testing of peritoneal fluid, although bacterial culture results were negative in all 6 cases. Prior antimicrobial therapy may reduce culture rates (Mair et al. 1990); however this was not a causative factor for any of the cases reported. Ancillary medications, including NSAIDS, anthelmintics and omeprazole, were used as indicated in individual cases. Peritoneal lavage has been reported to be difficult to perform effectively in the large equine abdomen because the direction of fluid flow cannot be regulated (Dyson 1983; Mair et al. 1990); nonetheless in case 1 it appeared efficacious.

In conclusion, although peritonitis in the horse is a potentially life-threatening disease, the diversity of potential aetiologies for peritonitis confounds the clinician's ability to accurately predict outcome from the initial clinical assessment and laboratory results. This case series supports a favourable outcome for the medical management of peritonitis in those cases where the primary cause is not identified, or is amenable to treatment. Peritonitis should be considered in any horse showing signs of abdominal pain and/or pyrexia and prompt intervention is crucial to the successful management of these cases.

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