Liver lobe torsion in six horses

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Case Description—6 horses were determined to have torsion of a liver lobe at 4 referral institutions over a 21-year period.

Clinical Findings—Clinical findings were nonspecific but often included signs of marked inflammation. Two of the 6 horses were examined because of colic, and 2 were assessed because of peritonitis that failed to respond to treatment; the remaining 2 horses were examined because of nonspecific clinical signs that included inappetence, lethargy, and weight loss. The results of laboratory tests were widely variable, and values for liver enzyme activities were typically within reference limits or only mildly increased. Most affected horses had markedly increased peritoneal nucleated cell counts.

Treatment and Outcome—Exploratory laparotomy and resection of the affected liver lobe was performed in 5 horses. Three of those patients survived to discharge.

Clinical Relevance—Results suggested that diagnosis of liver lobe torsion in horses may be difficult because clinical signs and results of laboratory testing are nonspecific and variable. Most affected horses had markedly abnormal peritoneal fluid. The prognosis for hepatic lobe torsion can be good, and early surgical correction is expected to improve outcome. (*J Am Vet Med Assoc* 2012;241:615–620)

A 3-year-old Appaloosa colt (horse 1) used for breeding was examined because of a 2-day history of inappetence, mild signs of depression, and apparent hematuria. The referring veterinarian reported persistent tachycardia (68 to 84 beats/min), intermittent tachypnea (up to 24 breaths/min), and grossly discolored urine, although rectal temperature was normal. A CBC performed by the referring veterinarian revealed a total leukocyte (9,360 cells/µL; reference interval, 5,500 to 12,000 cells/µL) and neutrophil (4,960 cells/µL; reference interval, 3,000 to 7,000 cells/µL) count within reference limits but an increased nonsegmented neutrophil count (1,123 cells/µL; reference interval, < 100 cells/L). Plasma creatinine concentration was increased on repeated sampling (2.5 to 3.7 mg/dL; reference interval, 0.8 to 2.2 mg/dL), and mild electrolyte abnormalities were also present. Results of a dipstick analysis of a free-catch urine sample were strongly positive for blood and protein; urine pH was 6.0, and specific gravity was 1.025. The colt was treated with procaine penicillin (22,000 U/kg [10,000 U/lb], IM, q 12 h), flunixin meglumine (1.1 mg/kg [0.5 mg/lb], IV, q 12 h), and IV fluid therapy for 2 days but was referred for further assessment when its condition failed to improve.

Abbreviations

ALP	Alkaline phosphatase
GGT	γ-Glutamyltransferase

On initial evaluation at the referral institution, the colt appeared comfortable but depressed. Physical examination revealed tachycardia (92 beats/min) and tachypnea (24 breaths/min), but rectal temperature (37.3°C [99.2°F]) was within normal limits. Other relevant findings included injected mucous membranes, cool extremities, decreased borborygmi, and mild dehydration. No abnormalities were detected on palpation of the abdomen per rectum, and no reflux was obtained on passage of a nasogastric tube. Samples were collected for a CBC and serum biochemical analysis at admission (**Table 1**).

No abnormalities were evident on cystoscopy, and results of urinalysis were within reference limits with the exception of an increased number of erythrocytes (20 to 30 RBCs/hpf; reference interval, 0 to 8 RBCs/hpf) evident on cytologic examination, possibly due to passage of the endoscope. Urine specific gravity was 1.018, and urine chemistries and electrolyte fractional excretions were within reference limits. Ultrasonographic evaluation of the abdomen revealed a moderate volume of swirling, echogenic fluid consistent with free blood. No other abnormalities were identified at that time, and no ultrasonographic abnormalities were appreciated within the thoracic cavity. Peritoneal fluid obtained via abdominocentesis was grossly serosanguineous, and cytologic examination revealed increases in the nucleated cell count (24,500 cells/µL; reference interval, < 5,000 cells/µL), erythrocyte count (665,000 RBCs/µL; corresponding to

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Table 1—Selected laboratory results from a 3-year-old Appaloosa colt (horse 1), a 7-year-old Quarter Horse gelding (horse 2), a 12-year-old Quarter Horse gelding (horse 3), a 14-year-old Quarter Horse mare (horse 4), a 12-year-old Thoroughbred mare (horse 5), and a 16-year-old Peruvian Paso Fino gelding (horse 6) determined to have torsion of a liver lobe at 4 referral institutions over a 21-year period.

	Horse											
	1		2	3		4	5			6		
Variable	Admission	Before surgery	Admission	Admission	Before surgery	Admission	Referral	Admission	Before surgery	Referral	Admission	Reference interval
Leukocytes (cells/L)	7,280	—	7,300	7,400	11,700*	13,400*	21,500*	8,800	—	3,000	11,200	5,500–12,000
Segmented neutrophils (%)	56	—	79	70	87	69	91	79	—	64	79	—
Band neutrophils (%)	5*	—	0	0	2*	19*	0	0	—	0	0	—
Lymphocytes (%)	29	—	21	28	11	12	3	21	—	27	21	—
PCV (%)	40	—	26	36	34	47	43	32	—	36	32	30–50
Fibrinogen (mg/dL)	500*	—	—	—	—	988*	220	600*	—	234	1,000*	100–400
Total protein (g/dL)	4.3*	3.0*	5.1*	5.9	6.5	7.3	7.9	—	—	7.8	7.0	5.5–7.5
Albumin (g/dL)	2.3*	1.7*	2.4	3.0	—	2.2*	4.0*	—	—	3.9	—	2.5–3.5
AST (U/L)	352*	287	283	228	500*	273	1,272*	—	—	418	—	160–300
CK (U/L)	772*	—	156	558*	685*	122	61	—	—	325*	—	120–350
ALP (U/L)	248*	287*	127	136	209*	931*	198	—	179	82	—	45–240
GGT (U/L)	14	14	20	15	18	144*	32*	—	91*	11	—	4–20
SDH (U/L)	—	—	—	—	9.2	7.6	—	—	—	—	—	4–13
Total bilirubin (mg/dL)	6.4*	7.3*	2.3	2.1*	6.7*	3.1*	3.3*	—	4.3*	2.2*	—	0.6–2.6
Creatinine (mg/dL)	4.7*	4.0*	1.6	2.2*	1.7	2.9*	1.8	—	—	1.3	—	0.9–2.0
Glucose (mg/dL)	121		140*	106	113	166*	112	—	—	168*	—	65–130
Lactate (mmol/L)	—	3.6*	0.6	—	—	—	—	—	—	—	—	< 1.5

Because testing was performed at several laboratories, only approximate reference intervals are presented.
*Results fell outside the testing laboratories' reference intervals.
— = Data not available. AST = Aspartate aminotransferase. CK = Creatine kinase. SDH = Sorbitol dehydrogenase.
For horse 2, a CBC was performed by the referring veterinarian on the morning of initial examination and plasma biochemistry analysis was performed at the referral institution. For horse 5, a CBC and plasma biochemistry analysis were performed by the referring veterinarian 2 days prior to initial examination. For horse 6, a CBC and plasma biochemistry analysis were performed by the referring veterinarian 1 day prior to initial examination.

a PCV of 4%), and total protein concentration (3.0 g/dL; reference interval, < 2.5 g/dL). The nucleated cells were predominantly neutrophils, consistent with suppurative inflammation. No microorganisms were observed, and culture of the peritoneal fluid yielded no growth.

Feed was withheld, and broad-spectrum antimicrobials, including potassium penicillin (22,000 U/kg, IV, q 6 h), gentamicin (6.6 mg/kg [3 mg/lb], IV, q 24 h), and metronidazole (15 mg/kg [6.8 mg/lb], PO, q 8 h); flunixin (0.25 mg/kg [0.11 mg/lb], IV, q 8 h); and polyionic fluids (4 mL/kg/h [1.8 mL/lb/h], IV) were administered. The colt remained afebrile and appeared comfortable, although its heart rate was consistently increased (72 to 94 beats/min). Although the horse was slightly brighter in attitude on day 2 of hospitalization, its respiratory rate and effort were increased. Plasma creatinine concentration had decreased on repeated serum biochemical analysis; however, both total protein and albumin concentrations had decreased. Ultrasonographic examinations of the abdomen and thorax were repeated; a moderate volume of fluid similar in appearance to that seen in the abdomen previously was apparent within the thorax, and the volume of fluid within the abdomen appeared to have increased. A mass of mixed echogenicity measuring approximately 10 to 15 cm in diameter was apparent in the cranial ventral abdomen. The origin of the mass could not be identified. Abdominocentesis was repeated; results of cytologic examination of the peritoneal fluid were largely unchanged, although the erythrocyte count had increased (2,410,000 RBCs/μL; PCV, 10%). A clotting profile was performed and revealed that prothrombin time was within reference limits (18.1 seconds; reference interval, 10.0 to 20.0 seconds); however, both partial thromboplastin time (> 60 seconds; reference interval, < 59.0 seconds) and concentration of fibrin degradation products (10 to 40 μg/mL; reference interval, < 10 μg/mL) were increased. Venous pH was within reference limits (7.40), although venous lactate concentration was increased (3.6 mmol/L; reference interval, < 1.5 mmol/L).

On the basis of a lack of clinical improvement and concerns of an intra-abdominal septic process, an exploratory laparotomy was performed. Immediately before surgery, approximately 22 L of serosanguineous fluid was drained from the thorax; the PCV and total protein concentration of this fluid were 4% and 2.0 g/dL, respectively. Upon entering the abdomen, a large volume of serosanguineous fluid was evident and a strong odor of necrotic tissue emerged. Both the large and small intestines were diffusely reddened, and the mesentery was hemorrhagic in appearance. On manual exploration, the left liver lobe was found to be very mobile and floating within the peritoneal fluid. A torsion was felt at the base of this lobe; the affected section of liver was friable on palpation with a roughened surface that was gray and tan in color. The torsion was easily corrected; however, to improve access, the abdominal incision was extended to the xiphoid process and the large colon exteriorized. This allowed insertion of a thoracoabdominal stapling device[a] and placement of a staple line of 4.8-mm staples at the level of the original torsion. A second line of staples was placed paral-

lel to the first approximately 2 to 4 cm further away from the remaining liver. The affected lobe was then resected with curved Mayo scissors cutting along the jaws of the stapling instrument and removed. The abdomen was examined closely; no other abnormalities were evident, and there was no evidence of hemorrhage from the transected hepatic stump. The abdomen was lavaged copiously with sterile saline (0.9% NaCl) solution and subsequently closed routinely. During surgery, 2 L of heparinized (20 U/L) plasma was administered IV and commercially prepared *Salmonella typhimurium* anti-endotoxin antibodies[b] were administered immediately after surgery (1.5 mL/kg [0.7 mL/lb], IV). Histologic evaluation of the resected hepatic tissue revealed a mixture of severe autolysis interposed with less severely affected areas. Necrosuppurative hepatitis with complete loss of cellular detail and normal architecture were evident in the less severely affected regions. The hepatic sinusoids were congested and infiltrated with inflammatory cells.

After a difficult recovery from anesthesia, the colt's condition deteriorated rapidly and it died approximately 3.5 hours after surgery. Gross postmortem findings included serosanguineous peritoneal and pleural fluid, moderate-to-marked hemorrhagic enteritis, and signs consistent with disseminated intravascular coagulopathy. Histologic examination revealed hemorrhagic enteritis and colitis with vasculitis and thrombus formation of the subserosal vessels. There was evidence of pulmonary congestion and changes consistent with severe systemic inflammation. Moderate growths of *Escherichia coli*, *Pseudomonas aeruginosa*, and *Acinetobacter baumannii* were obtained from cultures of lung tissue obtained at postmortem examination. Gross and histologic postmortem changes were considered consistent with overwhelming gram-negative sepsis and endotoxic shock.

A 7-year-old Quarter Horse gelding (horse 2) used for reining was evaluated because of mild but progressive colic of approximately 18 hours' duration. Eight liters of reflux were obtained on passage of a nasogastric tube by the referring veterinarian on the morning of admission. A CBC performed by the referring veterinarian revealed mild anemia (Table 1). On initial evaluation at the referral institution, the gelding was mildly sedated (0.3 mg of xylazine/kg [0.15 mg/lb], IV) and appeared comfortable, with both heart (42 beats/min) and respiratory (16 breaths/min) rates within normal limits and a low-normal rectal temperature (36.5°C [97.7°F]). Findings on palpation per rectum and on ultrasonographic examination of the abdomen were considered within normal limits. Serum biochemical analysis was performed (Table 1). Fluid obtained via abdominocentesis during the initial examination was grossly serosanguineous, and the gelding developed signs of abdominal discomfort once placed in a stall. Subsequently, an exploratory laparotomy was performed approximately 3 hours after arrival at the referral institution, during which torsion of the left medial liver lobe was diagnosed. The initial laparotomy incision was 30 cm in length beginning 20 cm caudal to the xiphoid process but was extended 15 cm cranially once torsion of the liver was diagnosed. The torsion of the affected liver lobe was corrected, and a stapling device[a] was applied twice at the most proximal aspect of the affected lobe. Mayo scissors were then used to resect the affected lobe distal to the double row of staples. The remainder of the abdominal contents was within normal limits, and no hemorrhage was appreciated at the site of the resection. The gelding recovered from surgery uneventfully. Perioperative treatment included broad-spectrum antimicrobials (potassium penicillin [22,000 U/kg, IV, q 6 h]; gentamicin [6.6 mg/kg, IV, q 24 h]; and metronidazole [15 mg/kg, PO, q 8 h]), flunixin (1.1 mg/kg, IV, q 12 h), and polyionic fluids (4 mL/kg/h, IV).

After recovery from surgery, the gelding developed intra-abdominal hemorrhage that was diagnosed on the basis of clinical signs, a decrease in PCV, and ultrasonographic findings. The hemorrhage was assumed to originate from the site of liver lobe resection, necessitating whole blood transfusion (6 L). Acute renal failure was diagnosed on day 3 of hospitalization on the basis of an increased plasma creatinine concentration (4.5 mg/dL) and isosthenuria; consequently, the dose of flunixin meglumine was decreased (0.5 mg/kg [0.23 mg/lb], IV, q 12 h) and the gentamicin was replaced with enrofloxacin (7.5 mg/kg [3.4 mg/lb], IV, q 12 h). On day 7 of hospitalization, the gelding developed a fever of unknown origin that responded to a change in antimicrobial treatment to chloramphenicol (50 mg/kg [23 mg/lb], PO, q 8 h). Subsequently, the gelding's condition steadily improved and it was discharged on day 16 of hospitalization. Twelve months after discharge, the gelding was returned to the hospital for a dental procedure; the owners reported that it was competing successfully and had had no further episodes of colic.

A 12-year-old Quarter Horse gelding (horse 3) with a history of acute onset of signs of abdominal pain was evaluated. On initial evaluation at the referral institution, the gelding appeared comfortable and physical and rectal examination findings were within normal limits. Results of a CBC were within reference limits, and plasma biochemistry analysis revealed only mild increases in creatine kinase activity and creatinine concentrations (Table 1). Peritoneal fluid analysis revealed a nucleated cell count within normal limits (952 cells/μL) and a slight increase in total protein concentration (2.6 g/dL). However, approximately 24 hours after admission, the gelding became febrile (39.4°C [103.0°F]), tachycardic (66 beats/min), and progressively more lethargic, although it did not have overt signs of colic. Abdominocentesis was repeated on day 2 of hospitalization and yielded fluid with an increased nucleated cell count (153,000 cells/μL) and total protein concentration (6.0 g/dL). The majority of peritoneal cells were nondegenerate neutrophils (93%), with rare intracellular rods seen. An exploratory laparotomy was performed approximately 24 hours after admission, and torsion of the quadrate liver lobe was diagnosed. On opening the abdomen, the peritoneal fluid was dark red and a fetid odor was noted. The affected liver lobe was black with an irregular contour. The abdominal incision was extended cranially to the xiphoid process to allow placement of a stapling device[a] once the hepatic torsion was identified. A single line of staples was placed, and the affected lobe was sharply resected by

use of Mayo scissors against the stapling instrument. An abdominal drain was placed at surgery and removed approximately 36 hours after surgery. Perioperatively, the horse was treated with potassium penicillin (22,000 U/kg, IV, q 6 h), gentamicin (6.6 mg/kg, IV, q 24 h), and metronidazole (15 mg/kg, PO, q 8 h). The horse recovered from surgery uneventfully and was discharged on day 6 of hospitalization. Culture of the resected liver tissue grew a clostridial species.

Following discharge, the gelding had recurrent colic episodes, and an exploratory laparotomy performed 4 years after the initial surgery revealed fibrous adhesions between the liver, colon, and omentum. The adhesions were manually broken down and the omentum resected. The gelding was evaluated again because of colic 7.5 years after the first surgery (3.5 years after the second surgery) and was euthanized when clinical signs failed to respond to medical management. At necropsy, there were adhesions between the right liver lobe, body wall, and duodenum. Moderate-to-severe multifocal centrilobular fibrosis of the liver was evident on histologic evaluation.

A 14-year-old Quarter Horse mare (horse 4) was evaluated because of weight loss, anorexia, tachycardia, and leukocytosis of 2 weeks' duration. Physical examination revealed tachycardia (72 beats/min), tachypnea (32 breaths/min), and hypothermia (36.2°C [97.1°F]). The mucous membranes were pale with a toxic line, and the mare was clinically judged to be mildly to moderately dehydrated. A CBC and plasma biochemistry analysis were performed (Table 1). Peritoneal fluid was grossly abnormal, with an increased nucleated cell count (231,000 cells/µL), the majority of which (97%) were degenerate neutrophils, and an increased protein concentration (6.6 g/dL). Extracellular and intracellular rods were evident on cytologic examination. On the basis of clinical findings, results of peritoneal fluid analysis, and financial constraints, the mare was euthanized. Torsion of the right liver lobe was identified at necropsy. Histologic examination of the affected liver tissues revealed findings consistent with coagulative necrosis and a bile duct carcinoma.

A 12-year-old Thoroughbred mare (horse 5) was examined because of peritonitis that had been diagnosed by the referring veterinarian 2 days before. The referring veterinarian initiated antimicrobial treatment with potassium penicillin (30,000 U/kg [13,600 U/lb], IV, q 8 h), gentamicin (2.2 mg/kg [1.0 mg/lb], IV, q 8 h), and metronidazole (15 mg/kg, PO, q 8 h) and administered phenylbutazone (2.2 mg/kg, PO, q 12 h). At admission to the referral institution, the mare was tachycardic (66 beats/min) but normothermic (37.8°C [100.0°F]). A CBC and plasma biochemistry analysis were performed. Peritoneal fluid obtained at admission was turbid with a reddish tinge; analysis revealed an increased nucleated cell count (576,695 cells/µL) and protein concentration (4.6 g/dL). By day 3 of hospitalization, the mare's condition had not improved and a standing flank laparoscopy was performed, revealing only small amounts of fibrin on the serosal surfaces of the intestines and peritoneum. An exploratory laparotomy was performed the following day via a 35-cm preumbilical median incision, and torsion of the left medial liver lobe was diagnosed. Multiple adhesions were present in the cranial abdomen between the stomach and liver, and the cecum was adhered to the ventral colon adjacent to the cecocolic fold. The adhesions were manually broken down. The left medial lobe of the liver was torsed and darkly discolored, and its capsule was covered in fibrin. A stapling device[a] was placed across the base of the twisted lobe and discharged once. The affected liver lobe was then sharply transected and removed. An abdominal drain was placed prior to closure of the abdomen; the mare recovered uneventfully from surgery, and the drain was removed 48 hours after surgery. The mare developed colitis on day 6 of hospitalization and died on day 12 of hospitalization. Histologic evaluation of the resected liver tissue revealed coagulative necrosis and congestion, with fibrosis. On postmortem examination, gross and histopathologic lesions of the colon were consistent with colitis.

A 16-year-old Peruvian Paso Fino gelding (horse 6) was evaluated because of peritonitis that had been diagnosed by the referring veterinarian 1 day before the horse was referred. Analysis of peritoneal fluid obtained by the referring veterinarian revealed an increased nucleated cell count (120,000 cells/µL) comprised predominantly (99%) of degenerate neutrophils and an increased total protein concentration (7.0 g/dL). Abdominal ultrasonography performed at the referral institution identified a heterogeneous mass measuring 10 to 15 cm in diameter with hyperechoic areas in the cranial ventral abdomen. By day 3 of hospitalization, the gelding had not responded to antimicrobial treatment with ampicillin (10 mg/kg [4.5 mg/lb], IV, q 12 h) and gentamicin (2.2 mg/kg, IV, q 8 h), and laparoscopy was performed with the horse anesthetized and positioned in dorsal recumbency. Torsion of left medial liver lobe with extensive adhesions to the large colon was identified. A ventral midline laparotomy was then performed, with the incision beginning caudal to the xiphoid process and proceeding caudally. The adhesions were broken down with blunt dissection; a stapling device[a] was placed across the base of the twisted lobe and discharged once. Intraoperative ultrasonography was used to ensure that there were no large patent vessels present in the resected tissue prior to application of the stapler. The affected liver lobe was then sharply transected and removed. Recovery from surgery was uneventful, and the gelding was discharged after 15 days of hospitalization. However, colic recurred 6 months after surgery and the gelding was euthanized without further evaluation or necropsy.

Discussion

In veterinary medicine, liver lobe torsion has most commonly been described in dogs but, even in this species, it is considered rare.[1–6] Liver lobe torsions have also been described in laboratory animals (rats, rabbits, and mice),[7] cats,[2] an otter,[8] pigs,[9,10] and 2 horses.[11,12] A handful of reports exist describing liver lobe torsions in humans, most of which involve accessory liver lobes (Reidels's lobe), which are a common and otherwise benign abnormality of the liver.[13] The liver in horses is maintained in position by multiple ligamentous at-

tachments to the diaphragm and body wall and by the pressure exerted by other abdominal organs.[14] The equine liver is anchored by 6 ligaments[14]; in humans, congenital absence, disruption, or stretching of these attachments is required to allow torsion of a lobe to occur.[15] However, only the dorsal margins of the equine left and right liver lobes are secured to the diaphragm and changes in gastrointestinal fill might allow rotation of the less restricted ventral margins. An abnormality of the securing ligaments might have allowed lobe torsion in some of the patients of the present report, although this was not apparent at surgery or necropsy. In domestic animals, hepatic lobe torsion has occasionally been associated with gastric dilatation-volvulus,[1] hepatic abscesses,[3] neoplastic masses, and trauma[2]; however, in most cases, the underlying cause is not apparent.[2] In 1 horse of the present report, bile duct carcinoma was identified at necropsy that might have predisposed to torsion. In dogs, large breeds appear to be more commonly affected and, in early reports,[2] torsion most often involved the left lateral lobe. The predilection for torsion of the left lateral lobe is less apparent in later reports,[6] and other lobes can be affected. The left medial liver lobe was affected in 3 of the 6 patients described in the present report and in 1 previous report[12] of hepatic lobe torsion in a horse; the entire left liver lobe was affected in one of the horses of the present report and likely was involved in the initial report[11] of liver lobe torsion in a horse.

Animals with liver lobe torsions can have severe clinical abnormalities, including weakness, collapse, and signs of shock.[1–6] In some cases, affected animals are found dead, probably as a result of septic or hypovolemic shock subsequent to hepatic necrosis.[1–6] Commonly reported historical complaints in affected dogs include lethargy or signs of depression, anorexia, polyuria or polydipsia, and sporadic vomiting.[1–6] The duration of disease is often chronic (days), with animals evaluated after acute worsening of nonspecific signs. Physical examination findings commonly reported for affected dogs are nonspecific but include weakness or recumbency, pyrexia, tachycardia, tachypnea, signs of hypovolemia, and abdominal distention.[1–6] Signs of pain on palpation of the cranial abdomen are not uncommon but can be absent. Clinical signs in other species are similar.[2,7,8] In the first published description of a horse with a liver lobe torsion, a 14-year-old Arab gelding was initially examined because of signs of mild abdominal pain, signs of depression, and anorexia.[11] In a second report,[12] a 4-year-old Belgian mare was examined because of anorexia and fever of unknown origin. Two of the patients described in the present report were examined because of signs of abdominal discomfort; clinical signs in the others were less specific but included signs consistent with a severe inflammatory process.

Hematologic and plasma biochemistry abnormalities in animals with liver lobe torsion are widely variable and nonspecific.[1–8] Changes in the leukogram are generally consistent with severe inflammation, although the peripheral WBC count can be increased, decreased, or within reference limits. In a report[1] describing liver lobe torsion in 13 dogs, mature neutrophilia and leukocytosis were the most common hematologic abnormalities. An increase in the number of band neutrophils is not an infrequent finding. In most cases in dogs, there is an increase in hepatocellular enzyme activity (ie, alanine transferase, aspartate aminotransferase, and sorbitol dehydrogenase) indicative of hepatocellular necrosis and enzyme leakage.[1–8] Increases in enzyme activities more consistent with cholestatic disease (ie, ALP and GGT) have also been described.[1–8] In the first report[11] of a horse with liver lobe torsion, there was an increase in sorbitol dehydrogenase activities but ALP, GGT, and aspartate aminotransferase activities were within reference limits. In that case, enzyme activities rapidly returned to within reference limits after resection of the affected liver lobe.[11] In the patients of the present report and the previously reported case in a Belgian mare, liver enzyme activities were either within reference limits or only mildly increased; the notable exception was the mare diagnosed with bile duct carcinoma, which had marked increases in GGT and ALP activities. Total bilirubin concentration was increased in many cases, although interpretation is confounded by anorexia in horses. In affected dogs, as in horse 1 of this report, there is often clinical and clinicopathologic evidence of coagulopathy.[1–6]

Peritoneal fluid in animals with liver lobe torsion is often grossly serosanguineous with an increased protein concentration.[1–6] Diapedesis of erythrocytes subsequent to venous obstruction, passive congestion, and increased hydrostatic pressure within the liver is likely, at least in part, responsible for the hemorrhagic abdominal effusion. Abnormalities of hemostasis secondary to a severe inflammatory response might also contribute to hemoabdomen in some animals. Coagulopathy is the likely explanation for effusions of similar character in both the abdominal and thoracic cavities in horse 1 of this report. In reported cases, peritoneal leukocyte counts usually exceed that which would be expected from hemorrhage alone consistent with peritoneal inflammation.[1–6] Moderately to markedly increased peritoneal nucleated cells counts were recorded in all horses of this report for which results from abdominocentesis samples were reported.

In the initial report[11] describing liver lobe torsion in a horse, intracellular and extracellular bacteria were observed in peritoneal fluid samples, and similar findings have been reported in affected dogs.[1–3] In clinically normal dogs, bacteria can be present in the liver due to migration through the portal vein from the intestine and this might also occur in clinically normal horses.[16] Clostridial organisms are present in normal bovine livers[17] and might also be present in normal equine livers.[18] It has been suggested that tissue hypoxia and necrosis subsequent to lobe torsion results in proliferation and overgrowth of resident clostridial organisms in affected dogs.[3] The presence of these and other bacterial organisms and their toxins are thought to contribute to the clinical picture of marked inflammation reported in some canine cases[3] and might have played a role in the equine cases described in the present report.

Abdominal imaging has provided supportive evidence for a diagnosis of liver lobe torsion in small animal and human medicine.[4,13] Techniques used include radiography, ultrasonography, CT, and MRI. Radio-

graphs of dogs with liver lobe torsion often reveal a mass within the cranial abdomen and can indicate the presence of necrosis, gas-forming organisms, and peritoneal effusion.[19] Ultrasonographic findings consistent with liver lobe torsion include evidence of decreased blood flow or vessel congestion, a hypoechoic or heterogenous appearance to the hepatic parenchyma, and peritoneal effusion.[4] Ultrasonography revealed a mass of mixed echogenicity in the cranial abdomen in 2 of the horses of the present report, but results of abdominal ultrasonography of the other patients of this report and the 2 previous reports[11,12] were either not reported or considered within normal limits. Intraoperative imaging of the torsed lobe was considered valuable in one of the horses of this report as previously suggested.[12] Ultrasonographic diagnosis of hepatic lobe torsion in horses is likely limited by the small available imaging windows and the difficulty in identifying individual lobes.[20] In particular, much of the ventral aspect of the left liver lobe is usually obscured by the gastrointestinal tract.[20] Standing flank laparoscopy was not useful in the 1 horse in which it was used. However, laparoscopy performed with the horse in dorsal recumbency was useful in both reaching a diagnosis and planning the subsequent surgical approach.

In the 5 horses of this report that were taken to surgery, stapled resection of the affected liver lobe was performed with a stapling device.[a] A stapled resection technique was also described in a previous report[11] of liver lobe torsion in a horse, and it was the most common technique reported in a retrospective study of liver lobe torsion in dogs.[1] Intraoperative hemorrhage is a potentially life-threatening complication of liver resection; the stapled technique has the advantage of providing hemostasis of the lobar vessels and parenchyma without requiring tissue dissection and vessel identification. A bipolar vessel sealing device[c] and harmonic scalpel have been used in human patients for hepatic resection,[21,22] and these techniques might also have applicability in horses.

This case series illustrates the difficulties in diagnosing liver lobe torsion in horses. Clinical signs are often suggestive of an inflammatory disease but are nonspecific and can be either acute or chronic. Results from laboratory tests are also nonspecific, and liver enzyme activities are only occasionally increased. The peritoneal fluid in each of the horses of the present report was abnormal, and most patients had markedly increased peritoneal nucleated cell counts. Two horses underwent laparotomy that allowed definitive diagnosis because of persistent clinical signs of abdominal pain. Surgical exploration of the abdomen of 3 horses was performed when their condition failed to respond to medical management. A recent report[23] has suggested that horses with peritonitis and persistent signs of pain are more likely to require surgery; however, overt signs of colic were not present in all the patients described in the present report. Liver lobe torsion might also be considered in horses with peritonitis that fails to respond to apparently appropriate medical treatment. On the basis of this case series, the prognosis for hepatic lobe torsion can be good. Early surgical correction is expected to improve outcome; however, this is hindered by the difficulty in reaching a definitive diagnosis.

a. TA-90 Premium Reusable Stapler, Covidien Surgical, Mansfield, Mass.
b. Endoserum, IMMVAC Inc, Columbia, Mo.
c. Ligasure, Covidien, Mansfield, Mass.

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