Successful outcome of hepatectomy as treatment for liver lobe torsion in four domestic rabbits

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Case Description—4 rabbits (1.5 to 6 years old) were evaluated at the Angell Animal Medical Center from June 2007 to March 2009 because of nonspecific clinical signs including anorexia, lethargy, and decreased fecal output.

Clinical Findings—Physical examination revealed signs of pain in the cranial portion of the abdomen, gas distention of the gastrointestinal tract, and diminished borborygmi. Serum biochemical analyses and CBCs revealed moderately to markedly high alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase activities and mild to moderate anemia with polychromasia. Abdominal radiographic findings were nonspecific. Three of the 4 rabbits underwent abdominal ultrasonography; abnormalities in shape, size, echogenicity, and blood flow of the liver, indicative of liver lobe torsion, were detected.

Treatment and Outcome—All 4 rabbits underwent surgery, during which liver lobe torsion was confirmed and the affected liver lobe was resected. Histologic examination of sections of the excised lobe obtained from 3 of the 4 rabbits revealed severe, diffuse, acute to subacute hepatic ischemic necrosis. All rabbits recovered from surgery; owners reported that the rabbits were doing well 22 to 43 months after surgery.

Clinical Relevance—Liver lobe torsions in any species are rarely reported, yet 4 cases of liver lobe torsion in domestic rabbits were treated at 1 referral center in a 2-year period. In rabbits, clinical signs of this condition are nonspecific and results of additional tests, including abdominal ultrasonography and serum biochemical analysis, are necessary for diagnosis. Prompt diagnosis and hepatectomy of the affected lobe are recommended and appear to be associated with an excellent prognosis. (*J Am Vet Med Assoc* 2011;238:1176–1183)

A 1.5-year-old 3.0-kg (6.6-lb) spayed female Lop rabbit was evaluated at the Angell Animal Medical Center because of anorexia and hiding behavior. On physical examination, the rabbit had a single dental point on the lingual aspect of a left lower molar; the abdomen was mildly tense on palpation, borborygmi were considered decreased from normal, and there was diarrheic fecal staining on the perineal hair. The rabbit was premedicated with midazolam hydrochloride (0.33 mg/kg [0.15 mg/lb], IM) and butorphanol tartrate (0.33 mg/kg, IM), and anesthesia was induced with isoflurane gas delivered via a nasal mask to perform a dental procedure to remove the lingual point. Upon recovery from anesthesia, the rabbit was given meloxicam (0.5 mg/kg [0.23 mg/lb], SC). A blood sample was obtained at the time of the dental procedure. Serum biochemical analyses revealed moderately to markedly high liver enzyme activities (AST, 2,935 U/L [reference range, 14 to 113 U/L[a]]; ALT, 655 U/L [reference range, 48 to 80 U/L[a]]; and ALP, 133 U/L [reference range, 4 to 16 U/L[a]]), mildly low phosphorus concentration (3.4 mg/dL; reference range, 4.0 to 6.9 mg/dL[a]), and mildly high cholesterol concentration (121 mg/dL; reference range, 10 to 80 mg/dL[a]). A CBC revealed mild leukopenia (4.2 × 10^3 WBCs/µL; reference range, 5.2 × 10^3 WBCs/µL to 12.5 × 10^3 WBCs/µL[a]), specifically lymphopenia (1.7 × 10^3 lymphocytes/µL; reference range, 3.4 × 10^3 lymphocytes/µL to 7.0 × 10^3 lymphocytes/µL[1]), which was likely evidence of a stress response in this species. Additionally, a mild normocytic, normochromic anemia (Hct, 28%; reference range, 33% to 50%[a]) was noted. Assessment of the RBC morphology revealed greater than normal polychromasia and mild anisocytosis, acanthocytosis, and schistocytosis, consistent with fragmentation anemia. The rabbit was administered LRS (4 mL/kg [1.8 mL/lb], IV, q 1 h and then 33 mL/kg [15 mL/lb], SC, q 8 h), buprenorphine (0.04 mg/kg [0.02 mg/lb], SC, q 8 h), enrofloxacin (10 mg/kg [4.5 mg/lb], PO, q 12 h), and metronidazole (20 mg/kg [9 mg/lb], PO, q 12 h). Syringe feeding[b] (15 mL/kg [6.8 mL/lb], PO, q 8 h) was performed. The following day,

Abbreviations

ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
LRS	Lactated Ringer's solution

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abdominal ultrasonography revealed a single abnormal right-sided liver lobe that had a rounded contour and decreased echogenicity of the liver parenchyma, compared with surrounding liver lobes; color flow Doppler evaluation revealed an absence of blood flow within the abnormal lobe (**Figure 1**). Additionally, increased echogenicity of the surrounding adipose tissue was noted, but there was no free fluid within the peritoneal cavity. Ultrasonographic assessment of the gastrointestinal tract was difficult because of the presence of a large volume of gas in the stomach and small intestines. On the basis of the abdominal ultrasonographic findings, a diagnosis of liver lobe torsion was made.

The rabbit was premedicated with midazolam (0.5 mg/kg, IM), buprenorphine hydrochloride (0.04 mg/kg, IM), and meloxicam (0.4 mg/kg [0.18 mg/lb], SC). Isoflurane with oxygen was delivered by use of an anesthetic chamber followed by mask induction until the rabbit's anesthetic plane allowed intubation; anesthesia was maintained with isoflurane and oxygen delivered via endotracheal tube. With the rabbit positioned in dorsal recumbency, hair on the rabbit's abdomen was clipped and the skin was aseptically prepared. An exploratory celiotomy was performed, and torsion of the right lateral liver lobe at its hilus was detected. The parenchyma of this lobe was congested and friable. The liver lobe was occluded at the hilus with 2 circumferential ligatures of 2-0 polydioxanone suture and removed via transection distal to the ligatures. Closure of the abdomen was routine, and tissue adhesive was used to close the skin incision. The treatment regimen was continued, and administration of metoclopramide hydrochloride (0.5 mg/kg, PO, q 8 h) was commenced 2 days following surgery to increase gastrointestinal motility. Histologic examination of sections of the excised liver lobe revealed severe, diffuse, subacute hepatic coagulative necrosis with marked vascular congestion, fibrin thrombi, and fibrinoheterophilic capsulitis, all of which were consistent with ischemic necrosis secondary to liver lobe torsion. The rabbit steadily improved and was discharged from the hospital 4 days after surgery. Recheck clinicopathologic analyses were performed approximately 6 weeks after surgery. Serum biochemical analyses revealed improvement in liver enzyme activities (AST, 55 U/L; ALT, 90 U/L; and ALP, 40 U/L), persistent mild hypophosphatemia (3.0 mg/dL), and persistent mild hypercholesterolemia (103 mg/dL). Hematologic variables were within reference intervals; however, RBCs had persistent mild schistocytosis, moderate acanthocytosis, and mildly increased polychromasia. Follow-up information received from the owner 43 months after surgery indicated that the rabbit was doing well and had no additional problems.

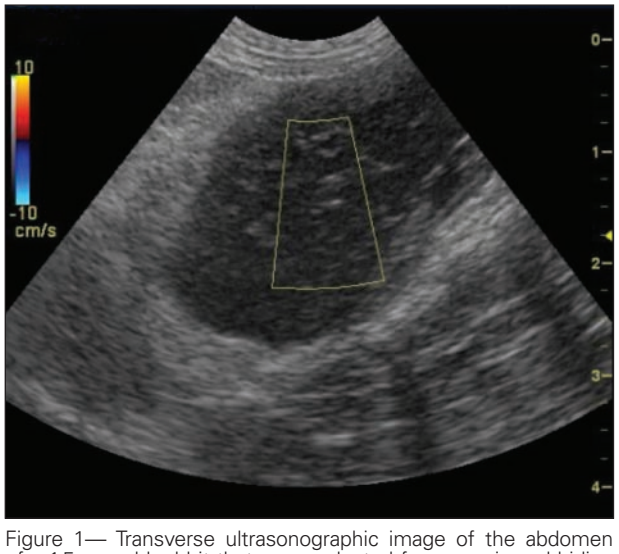

Figure 1— Transverse ultrasonographic image of the abdomen of a 1.5-year-old rabbit that was evaluated for anorexia and hiding behavior; physical examination revealed that the abdomen was mildly tense on palpation and borborygmi were diminished, and there was diarrheic fecal staining on the perineal hair. In this view, a right-sided liver lobe displays rounded margins, severely hypoechoic parenchyma (compared with nearby unaffected hepatic parenchyma), and adjacent mildly hyperechoic adipose tissue. The rectangular outline denotes the area of color flow Doppler imaging, which did not reveal any blood flow within the affected lobe. On the basis of the abdominal ultrasonographic findings, a diagnosis of liver lobe torsion was made. Numbers at the right side of the image represent depth in centimeters.

An approximately 3-year-old 2.4-kg (5.3-lb) castrated male Lop rabbit was evaluated by the Angell Animal Medical Center Emergency Service because of sudden onset of recumbency, lethargy, and anorexia. No abnormalities were detected via physical examination, but the rabbit was admitted to the hospital for supportive care and consultation with the Avian and Exotic Animal Medicine Service. Overnight, the rabbit received LRS (33 mL/kg, SC, q 8 h) and buprenorphine (0.04 mg/kg, SC, q 8 h); syringe feeding[b] (15 mL/kg, PO, q 8 h) was performed. The following morning, physical examination revealed signs of moderate pain on palpation of the abdomen, a moderately high body condition score (7/9), and lack of borborygmi. Gastrointestinal ileus was suspected. Thoracic and abdominal right lateral and ventrodorsal radiographic views revealed a diffusely distended gastrointestinal tract. Assessment of serum electrolytes and blood gas analysis revealed acidemia (peripheral venous blood pH, 7.283) and mild hyperlactatemia (11.4 mmol/L; reference range, 4.4 to 9.6 mmol/L[c]), consistent with lactic acidosis attributable to decreased perfusion and increased anaerobic metabolism. All other values were within reference intervals, including BUN and creatinine concentrations. Unfortunately, additional diagnostic tests including serum biochemical analyses and abdominal ultrasonography could not be performed on that day because of a lack of ultrasonography and laboratory services. A peripheral catheter was placed, and LRS (4 mL/kg, IV, q 1 h) was administered. On the basis of physical examination findings, the available clinicopathologic abnormalities, and patient clinical status, an exploratory celiotomy was performed to identify a potential source of acute abdominal pain and a reason for decreased perfusion.

The rabbit was premedicated with midazolam (0.7 mg/kg [0.32 mg/lb], IV) and butorphanol (0.2 mg/kg [0.09 mg/lb], IV). Anesthesia was induced with isoflurane and oxygen delivered by use of an anesthetic chamber followed by mask induction until the rabbit's anesthetic plane allowed intubation; anesthesia was maintained with isoflurane and oxygen delivered via endotracheal tube. With the rabbit positioned in dorsal recumbency, hair on the rabbit's abdomen was clipped

and the skin was aseptically prepared. During abdominal surgery, torsion and congestion of the right lateral liver lobe were detected. Two circumferential ligatures of 3-0 polydioxanone suture were placed around the hilus of the liver lobe, which was removed via transection distal to the ligatures. Closure of the abdomen was routine, and tissue adhesive was used to close the skin incision. Following surgery, the rabbit was administered LRS (4 mL/kg, IV, q 1 h and then 33 mL/kg, SC, q 8 h), buprenorphine (0.04 mg/kg, SC, q 8 h), enrofloxacin (10 mg/kg, PO, q 12 h), metoclopramide (0.5 mg/kg, PO, q 8 h), and meloxicam (0.3 mg/kg [0.14 mg/lb], PO, q 12 h). Syringe feeding[b] (15 mL/kg, PO, q 8 h) was performed. The day after surgery, a CBC and serum biochemical analyses were performed. Clinicopathologic abnormalities included moderately to markedly high liver enzyme activities (AST, 961 U/L; ALT, 587 U/L; and ALP, 45 U/L) and mild normocytic, normochromic anemia (Hct, 26%). Assessment of RBC morphology revealed greater than normal polychromasia, mild anisocytosis, and mild metarubricytosis (6 nucleated RBCs/100 WBCs). Histologic examination of sections of the excised liver lobe revealed severe, diffuse, acute hepatic necrosis with diffuse marked congestion and mild diffuse fibrinoheterophilic capsulitis, all of which were consistent with ischemic hepatic necrosis secondary to liver lobe torsion. Aerobic and anaerobic bacterial cultures of resected liver tissue specimens yielded no growth. The rabbit was discharged from the hospital 3 days following surgery. Follow-up information received from the owner 41 months after surgery indicated that the rabbit was doing well and had no additional problems.

A 6-year-old 2.7-kg (5.9-lb) spayed female rabbit of unknown breed was evaluated by the Angell Animal Medical Center Emergency Service because of sudden onset of hunched posture, lethargy, and urination and defecation outside of the litter box. Initial physical examination revealed hypothermia (rectal temperature, 37°C [98.6°F]), diminished borborygmi, and lethargy. No signs of pain were elicited during abdominal palpation. Blood sample analysis revealed moderate hyperlactatemia (17.5 mmol/L; reference range, 4.4 to 9.6 mmol/L[c]) and mild hyperglycemia (236 mg/dL; reference range, 75 to 155 mg/dL[a]). The rabbit was admitted to the hospital and given LRS (4 mL/kg, IV, q 1 h) with concurrent heat support overnight; the rabbit was transferred to the Avian and Exotic Animal Medicine Service the following morning. Repeated physical examination revealed a moderately high body condition score (7/9) and a mild amount of thick white material in the right otic canal; moderate signs of pain were elicited via palpation of the cranial portion of the abdomen. At this time, only a limited sample of blood could be obtained. Serum biochemical analysis revealed moderately high liver enzyme activities (AST, 406 U/L; ALT, 312 U/L; and ALP, 34 U/L) and persistent, albeit decreased, hyperglycemia (177 mg/dL). Abnormalities detected on thoracic and abdominal right lateral and ventrodorsal radiographic views included a mildly distended stomach filled with ingesta and gas, a notably empty intestinal tract, and slight hepatomegaly. Abdominal ultrasonography revealed a markedly enlarged right-sided liver lobe. The lobe parenchyma was heterogeneous with anechoic to hypoechoic striations throughout; no visible blood flow was evident via color flow Doppler examination. A moderate volume of nonechoic, free peritoneal fluid was present; however, no fluid analysis was performed. The adipose tissue surrounding the liver was markedly hyperechoic and hyperattenuated, consistent with severe inflammation. On the basis of the ultrasonographic findings, a diagnosis of liver lobe torsion was made.

The rabbit was premedicated with midazolam (0.3 mg/kg, IM), buprenorphine (0.04 mg/kg, IM), and glycopyrrolate (0.02 mg/kg [0.01 mg/lb], SC). Anesthesia was induced with isoflurane and oxygen delivered by use of an anesthetic chamber followed by mask induction until the rabbit's anesthetic plane allowed intubation; anesthesia was maintained with isoflurane and oxygen delivered via an endotracheal tube. With the rabbit positioned in dorsal recumbency, hair on the rabbit's abdomen was clipped and the skin was aseptically prepared. An exploratory celiotomy revealed torsion of the caudate liver lobe. Three circumferential ligatures of 3-0 polypropylene suture were placed around the hilus of the caudate lobe, and 2 stainless steel ligation clips were placed distal to the ligatures; the lobe was transected between the clips. Closure of the abdomen was routine, and tissue adhesive was used to close the skin incision. Following surgery, the rabbit was administered LRS (4 mL/kg, IV, q 1 h and then 33 mL/kg, SC, q 8 h), buprenorphine (0.04 mg/kg, SC, q 8 h), enrofloxacin (10 mg/kg, PO, q 12 h), metronidazole (20 mg/kg, PO, q 12 h), meloxicam (0.3 mg/kg, PO, q 12 h), and cisapride (0.5 mg/kg, PO, q 8 h). Syringe feeding[b] (15 mL/kg, PO, q 8 h) was performed. Three days after surgery, a CBC revealed moderately severe macrocytic, normochromic anemia (Hct, 18%); blood film evaluation revealed mild metarubricytosis (5 nucleated RBCs/100 WBCs) and moderately increased polychromasia, consistent with a regenerative or partially regenerative anemia. Histologic examination of sections of the excised liver lobe revealed severe, diffuse, acute hepatic necrosis with diffuse marked congestion, consistent with liver lobe torsion. The rabbit was discharged from the hospital 3 days after surgery. At a 2-week recheck examination, the abdominal incision was healing and the rabbit had normal appetite and fecal production. Follow-up information received from the owner 25 months after surgery indicated that the rabbit was doing well and had no additional problems.

A 3.7-year-old 3.6-kg (7.9-lb) castrated male rabbit of unknown breed was evaluated at the Angell Animal Medical Center because of anorexia and lack of fecal production during the preceding day. Past pertinent history included 1 episode of gastrointestinal ileus that had occurred 2 years previously and had resolved with supportive care. Results of clinicopathologic analyses that had been performed annually during the 2-year period prior to evaluation were unremarkable, except that the rabbit had persistent mildly high serum ALP activity (30 and 47 U/L on the 2 occasions). Palpation of the cranial portion of the abdomen elicited signs of pain, and gas was palpable in the stomach. The rabbit was admitted to the hospital and administered LRS (4 mL/kg, IV, q 1 h and then 33 mL/kg, SC, q 8 h), bu-

prenorphine (0.04 mg/kg, SC, q 8 h), enrofloxacin (10 mg/kg, PO, q 12 h), metoclopramide (0.5 mg/kg, PO, q 8 h), and meloxicam (0.3 mg/kg, PO, q 12 h). Syringe feeding[b] (15 mL/kg, PO, q 8 h) was performed. A small sample of blood was obtained for analysis; BUN and serum creatinine concentrations were within reference intervals, but the serum ALT activity was markedly high (809 U/L). On thoracic and abdominal right lateral and ventrodorsal radiographic views, there was evidence of gastric distention with ingesta and gas, as well as a dilated mid-abdominal intestinal loop that was suggestive of an obstructive ileus. Abdominal ultrasonography revealed an enlarged caudate liver lobe with striations of mixed echogenicity; no blood flow was detected during color Doppler assessment. Additionally, adipose tissue surrounding the affected liver lobe was hyperechoic, consistent with inflammatory changes (**Figure 2**). The gastrointestinal tract was distended and contained gas, ingesta, and feces. On the basis of the ultrasonographic findings, a diagnosis of liver lobe torsion was made.

The rabbit was premedicated with midazolam (0.5 mg/kg, SC), buprenorphine (0.04 mg/kg, SC), and glycopyrrolate (0.02 mg/kg, SC). Anesthesia was induced with isoflurane and oxygen delivered by use of an anesthetic chamber followed by mask induction until the rabbit's anesthetic plane allowed intubation; anesthesia was maintained with isoflurane and oxygen delivered via an endotracheal tube. With the rabbit positioned in dorsal recumbency, hair on the rabbit's abdomen was clipped and the skin was aseptically prepared. During an exploratory celiotomy, torsion of the caudate liver lobe was confirmed; the lobe appeared dark red to black and was friable on manipulation. Three circumferential sutures of 3-0 nylon suture were placed around the hilus of the caudate lobe, which was removed via transection distal to the sutures. Closure of the abdomen was routine, and tissue adhesive was used to close the skin incision. Histologic examination of sections of the excised liver lobe was not performed. The rabbit was discharged from the hospital 2 days after surgery. At a 2-week recheck examination, the abdominal incision was healing and the rabbit had normal appetite and fecal production. Follow-up information received from the owner 22 months after surgery indicated that the rabbit was doing well and had no additional problems.

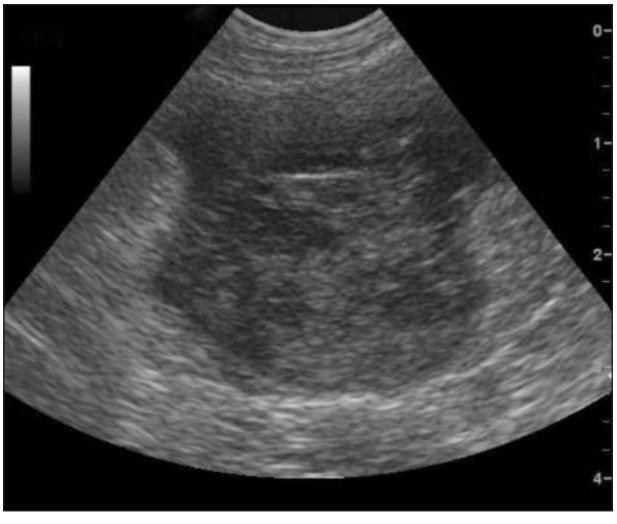

Figure 2—Transverse ultrasonographic image of the abdomen of a 3.7-year-old rabbit that was evaluated because of anorexia and lack of fecal production during the preceding day; on physical examination, palpation of the cranial portion of the abdomen elicited signs of pain and gas was palpable in the stomach. In this view, the caudate liver lobe is enlarged with rounded margins and marked heterogeneity of the parenchyma. Surrounding hyperechoic adipose tissue is also visible. On the basis of the abdominal ultrasonographic findings, a diagnosis of liver lobe torsion was made. Numbers at the right side of the image represent depth in centimeters.

Discussion

Although rarely reported, liver lobe torsions may occur in any mammalian species and have been identified in humans, dogs, pigs, horses, otters, rats, mice, and rabbits.[2–24] Liver lobe torsion leads to occlusion of the venous return prior to occlusion of the arterial supply, which causes acute congestion within the affected liver lobe. Subsequent vascular and cellular compromise causes necrosis, increases in circulating liver enzyme activities, effusion, hemoabdomen, shock, and death. Additionally, shock and death may result from disseminated intravascular coagulation secondary to the effects of bacterial toxin and ischemic by-product release into the peripheral circulation.

Although the underlying cause of liver lobe torsions is unknown, it has been hypothesized that congenital absence of supporting ligaments or their laxity secondary to trauma or dilation of other abdominal organs may be contributing factors.[10,15,17,24] Alternatively, torsion may occur secondary to variations in hepatic shape and size as a result of anatomic differences among liver lobes or disease processes that alter lobe architecture, such as neoplasia, infection, or parasitic infestation.[4,15,25] It was previously thought that the left lateral liver lobe was more prone to torsion in multiple species because of its large size, level of mobility, and anatomic separation from other lobes.[8,15] In a previous report, it was stated that of 19 published cases of animals of various species with liver lobe torsion, the left lateral lobe was the affected lobe in 9 cases.[15] A recent literature search revealed 52 case reports of liver lobe torsion in various species (23 dogs, 17 rabbits, 8 pigs, 2 horses, 1 cat, and 1 otter).[2–24] In these 52 cases, the left lateral lobe was the liver lobe that most commonly underwent torsion. This lobe was solely affected in 19% (10/52) of cases and was involved in combination with torsion of the left medial or papillary process of the caudate lobe in an additional 10% (5/52) of cases.[2–24]

In rabbits, 17 cases of liver lobe torsion have been reported, including the 4 rabbits of this report; of those 17 cases, the specific liver lobe affected was noted in 15.[12,16,21,22] It is thought that the caudate lobe of rabbits is predisposed to torsion because of its narrow attachment at the hilus that acts as a stalk about which it can rotate.[25] Indeed, of the 15 rabbits for which the affected lobe was reported, 10 (67%) had torsion of the caudate lobe. However, explanations for the torsion of the right lateral lobe (n = 2 rabbits), right medial and right lateral lobes (1), quadrate lobe (1), and left medial hepatic lobe (1) remain unknown.[22,23] The proper anatomic distinction of individual liver lobes in rabbits is inconsistent in the published literature. Wenger et al[22] stated

that the rabbit liver is divided into left and right lobes, each lobe contains anterior and posterior lobules, and the right lobe also gives rise to the quadrate lobe and the caudate lobe. For purposes of this report and others published previously, the traditional terminology used for canine liver lobes (ie, right lateral, right medial, left lateral, left medial, quadrate, and caudate) was used to identify distinct lobes.

Weisbroth[21] first reported 3 liver lobe torsions as incidental findings during necropsy of a laboratory population of 984 rabbits. Each of those rabbits had died of pasteurellosis without clinical signs attributable to abdominal disease. Two rabbits had atrophied, nonvital liver lobes without notable secondary consequences. The third rabbit appeared to have recent torsion of a liver lobe, with swelling and fresh infarcts on cut surface of the affected lobe. The findings in the third rabbit were thought to be early pathological changes that would have likely progressed to the state of the first 2 rabbits had the third rabbit not died as a result of pasteurellosis. These findings suggest that liver lobe torsions in rabbits occur rarely, but that this species can survive after complete torsion of a liver lobe. The mechanism of survival following liver lobe torsion in rabbits remains unknown. Conversely, torsion of the posterior lobule of the left liver lobe was the only abnormality identified on necropsy, and the presumptive cause of death, in a case report of a rabbit found dead in its cage.[23] To date, there are no reports of rabbits diagnosed with liver lobe torsion that subsequently survived without surgical intervention, to our knowledge.

Successful surgical removal of a liver lobe because of torsion (which was diagnosed during exploratory celiotomy) was first described by Taylor and Staff.[16] Subsequently, case reports[12,22] of 6 additional rabbits that had liver lobe torsion were published. Of those 6 rabbits, only 2 animals underwent hepatectomy and only 1 survived the immediate postoperative period.[12] Additionally, although 3 of the 6 rabbits had ultrasonographically detectable hepatic changes,[22] a definitive diagnosis of liver lobe torsion was not made in any of the rabbits before surgery or prior to death. A definitive diagnosis of liver lobe torsion was made on the basis of ultrasonographic findings before surgery in 3 of the 4 rabbits in the present report. Additionally, to date, no other single publication has reported survival of multiple rabbits after hepatectomy (as was the outcome for the 4 rabbits in the present report), to our knowledge.

Each rabbit in the present report had nonspecific clinical signs, including anorexia and lethargy. Physical examinations revealed signs of abdominal pain in 3 rabbits and abdominal tension on palpation in 1 rabbit. Additional physical examination findings included diminished borborygmi, palpable gas in portions of the gastrointestinal tract, and moderately high body condition score. The rabbits in the present report were generally small and ranged in body weight from 2.4 to 3.6 kg, with a moderately high body condition score in 2 of the 4 rabbits. Additional studies with larger numbers of rabbits would be necessary to determine whether these physiologic characteristics are associated with liver lobe torsion in this species.

Each rabbit in the present report had clinicopathologic abnormalities consistent with, but not specific for, liver lobe torsion. Complete blood counts were performed in 3 of the 4 rabbits, and all 3 had evidence of a mild to moderate anemia with greater than normal polychromasia. The polychromasia was suggestive of a regenerative or partially regenerative anemia; however, this was not confirmed by findings of a reticulocyte count. Potential causes of anemia in these rabbits included hemorrhage into the affected liver lobe, peritoneal hemorrhagic effusion, and erythrocyte fragmentation. Schistocytosis and acanthocytosis, as detected in one of the rabbits, were suggestive of erythrocyte fragmentation that could be caused by microangiopathy within the affected liver lobe or by disseminated intravascular coagulopathy.

The serum biochemical findings for the rabbits in the present report consistently included moderately to markedly high ALT, AST, and ALP activities. Moderately to markedly high activities of liver enzymes are consistent with liver lobe torsion; however, other causes of liver disease or damage must be ruled out. In the rabbits of this report, no concurrent alterations in BUN or serum creatinine concentrations were evident, but prerenal azotemia could be expected in clinically dehydrated patients with liver lobe torsion. Additionally, mild to moderate hyperlactatemia was evident in the 2 rabbits in which blood gas analysis was performed. The reference range for lactate concentration in rabbits is higher than the corresponding ranges used for dogs and cats. It is important to note that the rabbit reference range was obtained from analyses of blood samples of 20 clinically normal rabbits, and a reference range derived from a large sample population has not been published to date. Regardless, high lactate concentration is consistent with alteration in perfusion secondary to liver lobe torsion. Similarly, mild acidemia was detected in 1 rabbit in which peripheral venous pH was measured. Mild to moderate hyperglycemia was identified in 1 rabbit in this series; this finding most likely indicates a stressed state in this species.

Radiography was performed in 3 of the 4 rabbits in the present report and revealed signs of gas accumulation in the gastrointestinal tract, but was not helpful in achieving a diagnosis of liver lobe torsion. Results of ultrasonographic examination of the abdomen were diagnostic for liver lobe torsion for the 3 rabbits in which it was performed. These findings contradict a previous report[12] suggesting that abdominal ultrasonographic images are of limited diagnostic value because of interference from gas in the gastrointestinal tract. In our experience, gas did not obscure assessment of the liver, but may complicate complete assessment of the gastrointestinal tract. Upon examination of the liver, findings consistent with liver lobe torsion included abnormally large lobe size, round lobar margins, and mixed echogenicity of liver parenchyma. Color flow Doppler ultrasonography was used to confirm a lack of blood flow within the affected lobes and to achieve a definitive diagnosis of liver lobe torsion in the 3 rabbits.

Of the 4 rabbits of the present report, 1 underwent surgery on the day of the initial evaluation and 3 underwent surgery the day following the initial evaluation. The clinical consequence of a delay before completion of hepatectomy is unknown, given that all rabbits made a full recovery. Surgical correction of liver lobe torsion

in the 4 rabbits involved circumferential ligature occlusion (with sutures or sutures and metal clips) at the hilus of the lobe and transection distal to the ligatures. Histologic examination of sections of the excised liver lobe was performed in 3 of the 4 cases, and findings included severe, diffuse, acute to subacute ischemic necrosis. No evidence of underlying liver disease, such as bacterial or fungal infection, parasitism, or neoplasia, was identified. The histopathologic findings were consistent with previously published histopathologic findings in affected lobes of rabbits with liver lobe torsion.[21–23] Aerobic and anaerobic bacterial cultures were performed on the resected liver lobe tissue from 1 of the 4 rabbits and yielded no growth.

The mean duration of postoperative hospitalization for the rabbits in the present report was 3 days (range, 2 to 4). A blood sample from 1 rabbit was analyzed approximately 6 weeks after surgery, and findings indicated that there had been a considerable decrease in serum liver enzyme activities (values were just slightly greater than the upper reference limits). All 4 rabbits were alive and reported to be doing well 22 to 43 months after surgery (mean interval, 32.8 months).

For a rabbit with nonspecific clinical signs, the ideal diagnostic approach includes a thorough physical examination, blood sample analyses including a CBC and serum biochemical panel, and abdominal radiography and ultrasonography. Additionally, results of an electrolyte assessment and blood gas analysis may be helpful in identifying altered perfusion, although limited availability of reference ranges for variables evaluated by such tests in rabbits may make interpretation difficult. However, financial considerations, difficulty of obtaining adequate samples in this species, and laboratory or imaging availability may make prioritization of certain diagnostic tests necessary. Although a strong clinical suspicion for liver lobe torsion may entice a clinician to perform abdominal ultrasonography prior to other diagnostic tests, clinicopathologic analyses and radiographic assessment are still advised to assess patient status prior to anesthesia and to investigate for concurrent disease.

Results of a thorough physical examination are critical to clinical decision making. Rabbits with liver lobe torsion may have nonspecific clinical signs, including anorexia and lethargy, which could be attributed to the ill-defined but common occurrence of gastrointestinal ileus in this species. Ileus may be idiopathic and responsive to supportive and nursing care, but it may also be a result of a more serious underlying condition. Although rabbits with liver lobe torsion may have secondary ileus with a gas-distended gastrointestinal tract and diminished borborygmi, a clinician must continue to investigate and rule out underlying causes prior to accepting idiopathic ileus as a diagnosis. The primary physical examination finding that differentiates liver lobe torsion from ileus is signs of pain in the cranial portion of the abdomen. This finding may be subtle and difficult to detect depending on the individual examiner's experience with this species. Signs of pain may include bruxism, grunting, or abdominal splinting on palpation.

Ideally, a CBC and serum biochemical analysis should be performed at the time of evaluation for all rabbits with nonspecific clinical signs. Unfortunately, many factors may limit the quantity of blood that can be collected from a rabbit, including patient deterioration with the stress of restraint and difficulty in sample collection from small peripheral veins. When only a limited amount of blood may be collected from an individual rabbit, careful consideration of which tests to perform is necessary. If blood volume is limited, assessment of total solids concentration, Hct, and microscopic examination of RBC morphology in a blood smear could be performed in lieu of a CBC. If anemia is suspected and a sufficient volume of blood is available, a reticulocyte count is recommended to determine RBC regeneration status. If serum volume limits completion of a full biochemical profile, the priority should be measurement of liver and renal variables.

Assessment of serum liver enzyme activities is advised, especially in rabbits with appreciable signs of pain in the cranial portion of the abdomen, because if values are not abnormally high, liver lobe torsion can be ruled out. The findings for the rabbits in the present report suggest that serum ALT, AST, or ALP activity can be markedly elevated in association with liver lobe torsion. However, results are best evaluated in conjunction with one another. Although AST and ALT are hepatocellular leakage enzymes, ALT is more liver specific than AST. However, ALT activity may also be increased secondary to muscle damage. Alkaline phosphatase is an induced hepatic enzyme, and its activity increases during cholestasis, although other isoenzymes are found in bones and intestines. Assessment of BUN and serum creatinine concentrations is important to rule out azotemia that would alter the course of diagnostic testing and treatment. Urine specific gravity should be evaluated in conjunction with renal variables to determine whether azotemia is prerenal or renal in origin. Prerenal azotemia may be expected in dehydrated patients with liver lobe torsion. Fluid support of dehydrated patients is absolutely indicated prior to anesthesia. If samples can be obtained, results of serial blood analyses may be used to monitor for resolution of azotemia.

Thoracic and abdominal radiography are recommended during evaluation of rabbits with nonspecific clinical signs. Although radiographic findings may not be of benefit for diagnosis of liver lobe torsion, they are necessary to investigate for anomalies that might change the patient's prognosis or course of treatment. Complete radiographic assessment should be performed, including evaluation for bony injuries, urinary calculi, gastrointestinal foreign body or obstruction, abdominal masses, metastases, and cardiopulmonary disease.

An abdominal ultrasonographic examination is strongly recommended in all rabbits with nonspecific clinical signs, especially those for which liver lobe torsion is suspected. This is the single test that can provide a definitive diagnosis of liver lobe torsion prior to surgical exploration. Although gas may obscure complete assessment of the gastrointestinal system or underlying structures, attempted systematic ultrasonographic examination of the entire abdomen is recommended. A small volume of abdominal effusion may aid in the diagnosis of liver lobe torsion via ultrasonography by providing an acoustic window. In association with liver lobe torsion, free abdominal fluid may be a modified

transudate, exudate, or hemorrhagic effusion; however, the presence of a neoplastic or septic effusion may alter prognosis and treatment decisions. For this reason, collection of a sample of free abdominal fluid for cytologic analysis is recommended. Computed tomographic imaging has been recommended as an additional diagnostic procedure for liver lobe torsion.[9] However, the duration of anesthesia, delay of surgical correction, and added expense to the client associated with computed tomographic imaging as well as the ability to make a diagnosis on the basis of abdominal ultrasonographic findings limit the usefulness of this advanced technique in rabbits.

Prompt diagnosis and surgical correction of liver lobe torsion are advised because a patient's condition is expected to deteriorate with progressive dilation and rupture of the affected liver lobe. Consequences of liver lobe torsion include hemorrhage, release of bacterial toxins and ischemic by-products that lead to shock, disseminated intravascular coagulopathy, and death. Unfortunately, given the typically nonspecific signs at initial evaluation, delay in identification and treatment of liver lobe torsion in rabbits may occur. Because the interval between liver lobe torsion and evaluation cannot be determined, it is unknown how long rabbits can tolerate liver lobe torsion without fatal consequences. It is possible that the rabbits of this report did well following surgery because they received many hours of supportive care prior to anesthesia. Although additional investigations with a larger number of rabbits would be necessary to advise the exact timing of surgery, it seems prudent to proceed with surgery as soon as the diagnosis of liver lobe torsion is made, assuming the rabbit has received appropriate supportive care. Hopefully, this report will encourage practitioners to perform prompt diagnostic testing to facilitate early diagnosis of liver lobe torsion.

Supportive care provided at the time of initial evaluation and throughout the diagnostic process should include administration of fluids, pain medications, and antimicrobials as determined by use of patient clinical status and available diagnostic test results. If delays in diagnosis are likely to occur, aggressive patient support and stabilization should be performed in the interim. Depending on the interval from initial evaluation to anesthesia and surgery, supplemental feedings of anorectic rabbits are critical because of the delicate nature of rabbit gastrointestinal tracts. Prokinetic agents may also be useful when managing rabbits with nonobstructive ileus.

Once a definitive diagnosis or strong clinical suspicion of liver lobe torsion based on available diagnostic test results has been achieved, an exploratory celiotomy should be performed. At surgery, there are multiple potential methods of resecting the affected liver lobe. It is important to note that detorsion of the lobe is not performed during surgical manipulation to prevent bacterial toxin release and ischemic reperfusion injury. Ligation of the affected lobe's vascular pedicle with multiple sutures and transection distal to the sutures is 1 method of resection that can be easily performed. Occlusion with multiple stainless steel ligation clips, solely or in combination with ligatures, is another potential method of resection, depending on the thickness of tissue to be occluded. Stapling devices have been used in other species to aid in the removal of affected liver lobes and may work well in rabbits, although patient size may limit their usefulness. Additionally, electrothermal bipolar vascular sealing devices may prove useful in rabbit hepatectomy. Histologic examination and aerobic and anaerobic bacterial culture of excised liver lobe tissue are recommended in all cases to rule out underlying causes of liver lobe torsion that may affect postoperative treatment and prognosis.

The unique, delicate gastrointestinal tract of rabbits must also be accounted for in their postoperative care. Nutritional support is crucial to patient recovery. Additionally, care must be taken with antimicrobial choice to avoid damaging the native gastrointestinal flora, which is necessary for normal digestion in rabbits. In general, rabbits should be hospitalized until resolution of clinical signs and return of appetite. If the patient is otherwise stable and persistently anorectic, owners may be taught to administer syringe feedings along with oral medications after discharge from the hospital. Postoperative recheck evaluation of incisional healing at 1 to 2 weeks after surgery is advised in all cases. When possible, repeated blood sample analysis should be performed 1 to 2 months after surgery to confirm resolution of anemia and abnormalities in liver enzyme activities and to rule out persistent or concurrent disease.

To our knowledge, this is the first report in which liver lobe torsion was successfully diagnosed preoperatively in 3 rabbits and in which 4 rabbits were successfully treated via hepatectomy. Occurrence of 4 cases of liver lobe torsion in rabbits in a 2-year period at 1 referral institution suggests that this condition may be more common than previously thought. Additional studies with a larger population of rabbits would be necessary to determine true prevalence of this disease process. Given the nonspecific clinical signs and risk of sudden death, it is likely that liver lobe torsion has been misdiagnosed or not diagnosed in many affected rabbits. Prompt diagnosis and preoperative stabilization are recommended, after which hepatectomy should be performed as soon as possible. As illustrated by the 4 rabbits in this report, hepatectomy as a treatment for liver lobe torsion appears to have an excellent prognosis for a complete recovery.

a. Reference range for in-house tests at AAMC developed from rabbit data available upon request from IDEXX Laboratories Inc, Westbrook, Me.
b. Critical Care, Oxbow Animal Health, Murdock, Neb.
c. Schwartz Z, Lichtenberger MT, Thamm DH, et al. Lactate normals in healthy rabbits comparing three different analyzers (abstr), in *Proceedings*. Int Vet Emerg Crit Care Symp 2006;1023.

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