Liver lobe torsion in dogs: 13 cases (1995–2004)

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Objective—To determine history, results of diagnostic testing, surgical findings, complications, and outcome for dogs with liver lobe torsion (LLT).

Design—Retrospective case series.

Animals—12 dogs (1 with 2 episodes).

Procedure—Signalment, clinical signs, clinicopathologic findings, radiographic and ultrasonographic findings, surgical and histologic findings, complications, and hospitalization time were evaluated.

Results—The most common clinical signs were nonspecific abnormalities (eg, vomiting, lethargy, and anorexia) of acute or chronic duration. All dogs were large-breed dogs (median body weight, 37.2 kg [82 lb]). Biochemical abnormalities included high alanine aminotransferase (n = 12) and aspartate aminotransferase (11) activities. Results of abdominal ultrasonography were supportive of the diagnosis in 5 of 8 cases. Affected lobes included the left medial lobe (n = 4), left lateral lobe (3), papillary process of the caudate lobe (2), caudate lobe (1), and right lateral lobe (1). Exploratory celiotomy and liver lobectomy were performed in 12 of 13 cases, and in 11 of those 12 cases, the dog survived.

Conclusions and Clinical Relevance—Results suggest that development of nonspecific clinical signs of vomiting, lethargy, and anorexia in conjunction with high serum hepatic enzyme activities and mature neutrophilia in a medium-sized or large-breed dog should increase the index of suspicion for LLT. Abdominal ultrasonography with Doppler assessment may be useful in establishing the diagnosis. The long-term outcome for dogs that survive the hospitalization period is excellent. (*J Am Vet Med Assoc* 2006;228:242–247)

Liver lobe torsion is an uncommon condition of unknown etiology that has been identified in dogs, cats, horses, sows, rabbits, otters, and humans.[1-23] In dogs, clinical signs are typically nonspecific and surgical findings and outcome are variable.[2,3,11,16-18,23] Clinical signs in affected dogs may be acute or chronic and most often consist of signs of abdominal pain, vomiting, anorexia, lethargy, collapse, and sudden death.

A search of the veterinary literature yielded reports of only 25 cases of LLT involving various species[1-23] and only 11 cases involving dogs.[2,3,11,14-17,23] Dogs with LLT in these previous reports were typically middle-aged to older and of large or medium body size, with no sex predilection evident. An inciting cause for LLT has not been determined, nor have predisposing factors been identified. Despite anatomic variations in the affected species, the left lateral lobe is reported as the most commonly torsed liver lobe.[8,14,17,19] The predisposition of this lobe to torsion is attributed to its relatively large size, greater mobility, and relative separation from the other lobes.[17]

Clinical signs, results of diagnostic testing, treatment, and outcome of dogs with LLT described in previous reports have varied substantially. For this reason, more information about affected dogs would be useful. The purpose of the study reported here was to determine history, results of diagnostic testing, surgical findings, complications, and outcome for dogs with LLT.

Criteria for Selection of Cases

Dogs examined at the Foster Hospital for Small Animals at Tufts University's Cummings School of Veterinary Medicine or at the Angell Animal Medical Center in Boston between January 1995 and December 2004 in which LLT was diagnosed were eligible for inclusion in the study. Dogs were included in the study only if the diagnosis had been confirmed at surgery or during a postmortem evaluation and results of histologic evaluation were consistent with the diagnosis.

Procedures

Data obtained from the medical records included signalment, nature and duration of clinical signs, clinicopathologic findings, treatments, and outcome. Results of CBCs; serum biochemical profiles; determinations of PCV and TS concentration; and, when available, clotting profiles were recorded. Specific details of the history, including duration of illness and the chief complaint at the time of examination, were also recorded. Reports of radiographic and ultrasonographic examinations were reviewed. Surgery and anesthesia reports were reviewed to determine the timing of surgical intervention (ie, whether surgery was performed on an emergency or elective basis), surgical technique, liver lobe involved, and intraoperative complications. Histologic findings, postoperative complications, and duration of hospitalization were recorded.

Long-term follow-up information was obtained by telephone interviews with referring veterinarians or owners or through reexamination at one of the institutions included in the study. If the dog was no longer alive, the cause of death, if known, was recorded.

Statistical analysis—Data were examined graphically. Normally distributed data were expressed as mean ± SD; data that were not normally distributed were expressed as median and range. When possible, nonparametric data were transformed. Independent *t* tests or Mann-Whitney *U* tests were used to compare continuous

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LLT Liver lobe torsion
TS Total solids

variables between groups, depending on data distribution. Relationships between categorical parameters were determined via the Fisher exact test. Pearson or Spearman correlations were performed to determine relationships between variables, depending on data distribution. All analyses were performed with standard software.[a] Values of $P \leq 0.05$ were considered significant.

Results

Twelve dogs met the criteria for inclusion in the study. However, one of these dogs had 2 separate episodes of LLT 2 months apart. Therefore, data were collected for 13 cases of LLT. Median age of the dogs at the time LLT was diagnosed was 10 years (range, 0.3 to 13 years). Median body weight was 37.2 kg (82 lb; range 8.3 to 82 kg [18.3 to 180 lb]). Four dogs were castrated males, 3 were spayed females, and 5 were sexually intact males. Although most affected dogs were male, no sex predilection was identified. There were 3 Golden Retrievers, 3 Akitas, a Siberian Husky, a Weimaraner, an Irish Setter, a German Shepherd Dog, a Rottweiler, and a Saint Bernard.

Median duration of clinical signs was 3 days (range, 1 to 22 days). In 10 of the 13 cases of LLT, dogs had a history of acute onset (< 3 days) of illness. In the 3 cases in which dogs had a history of chronic (≥ 3 days) signs, signs had been present for 3 to 22 days.

Nine of the 12 dogs did not have any relevant medical problems prior to the onset of LLT. One dog had a history of colitis and had undergone belt-loop gastropexy because of gastric dilatation-volvulus 5 years earlier. The dog that had 2 episodes of LLT had a history of excessive gastric and intestinal gas. One dog, a 4-month-old male Weimaraner, had an abdominal hernia that was scheduled for repair at the time of castration.

Clinical and physical examination findings at the time of hospital admission were recorded for all 13 cases of LLT. The most common medical complaints were vomiting (n = 10), lethargy (7), anorexia (6), collapse (3), diarrhea (2), and signs of abdominal pain (2). Tachycardia (heart rate > 120 beats/min; n = 7), lethargy (6), dehydration (4), signs of abdominal pain (4), abdominal fluid wave (3), weak femoral pulses (3), and cardiac arrhythmias (2) were the most common physical examination findings. Other physical examination findings included a palpable abdominal mass, pale mucous membranes, and hyperthermia (rectal temperature > 39.4°C [103°F]).

For 12 of the 13 cases of LLT, a CBC and serum biochemical profile were performed at the time of admission. A clotting profile was performed at the time of admission for 11 of the 13 cases. The most common hematologic abnormalities were mature neutrophilia (n = 11) and leukocytosis (10). Median WBC count was 24.0 × 10^3 cells/µL (range, 9.2 to 31.3 × 10^3 cells/µL; reference range, 4.9 to 16.9 × 10^3 cells/µL), and median neutrophil count was 18.0 × 10^3 cells/µL (range, 5.6 to 29.6 × 10^3 cells/µL; reference range, 2.8 to 11.5 × 10^3 cells/µL). One dog had mild lymphopenia. Anemia was present in 2 dogs. Median PCV was 41.5% (range, 9.0% to 55.0%; reference range, 39% to 55%).

The most common biochemical abnormalities were high alanine aminotransferase (n = 12 cases), aspartate aminotransferase (11), and alkaline phosphatase (6) activities; low albumin concentration (5); high total bilirubin concentration (3); and low PCV (2). Median alanine aminotransferase activity was 908 U/L (range, 272 to 5,926 U/L; reference range, 18 to 86 U/L), median aspartate aminotransferase activity was 554 U/L (range, 45 to 5,441 U/L; reference range, 16 to 54 U/L), median alkaline phosphatase activity was 328 U/L (range, 71 to 2,924 U/L; reference range, 12 to 121 U/L), mean ± SD albumin concentration was 3.0 ± 0.6 g/dL (reference range, 3.0 to 4.0 g/dL), mean total protein concentration was 5.9 ± 1.4 g/dL (reference range, 5.5 to 7.8 g/dL), median total bilirubin concentration was 0.5 mg/dL (range, 0.21 to 6.80 mg/dL; reference range, 0.1 to 0.3 mg/dL), and median PCV was 41.5% (range, 9.0% to 55.0%; reference range, 39% to 55%). Other biochemical abnormalities included low sodium and chloride concentrations (n = 3 cases), high BUN and creatinine concentrations (2), high cholesterol concentration (1), and high phosphorus concentration (1).

Results of clotting profiles were within reference limits in 9 of 11 cases. In 1 dog, prothrombin time was mildly prolonged (8.9 seconds; reference range, 6.0 to 8.6 seconds), although activated partial thromboplastin time was normal. In another dog, the activated partial thromboplastin time was prolonged (27.5 seconds; reference range, 12 to 20 seconds), although prothrombin time was normal. Problems with coagulation during or after surgery were not reported in either of these dogs.

Abdominal radiographs were obtained in 8 of the 13 cases of LLT. An abdominal mass effect was seen in 6 of the 8 cases, and gas distension of the intestines and stomach was seen in 2. Abdominal ultrasonography was performed in 9 of the 13 cases. In 1 of these 9 cases, ultrasonography was performed by the referring veterinarian and the ultrasonography report described a noncavitated abdominal mass of unknown origin that was later found at surgery to be a torsed liver lobe. In 5 of the remaining 8 cases, Doppler ultrasonographic assessment of the hepatic vessels revealed decreased blood flow, supporting a diagnosis of LLT (**Figure 1**). In 5 of 8 cases, the affect-

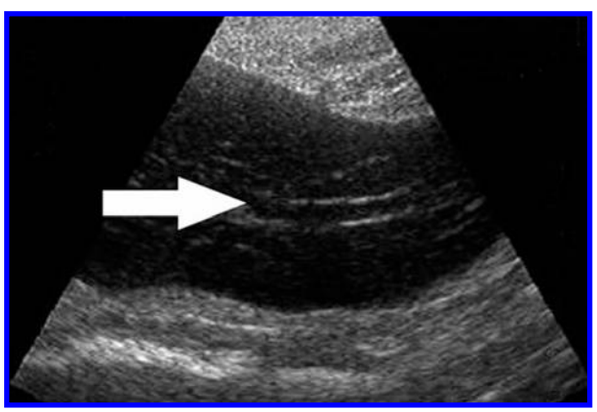

Figure 1—Longitudinal ultrasonographic view of the liver in a dog with LLT. Color-flow Doppler echocardiography did not reveal any blood flow through large vessels in the affected liver lobe, and echogenic material within these vessels was a result of thrombosis (arrow). Notice that the wall of the portal vessel is prominent because surrounding liver parenchyma is hypoechoic.

ed liver lobe was hypoechoic, and in the remaining 3, the affected lobe was heterogenous. Abdominal effusion was detected in 6 of 9 cases. The presence of abdominal effusion was not related to any other variable, including duration of hospitalization or development of postoperative complications.

Exploratory celiotomy and liver lobectomy were performed in 12 of the 13 cases of LLT. Median time between hospital admission and surgery was 4 hours (range, 1 to 72 hours). In the remaining case of LLT, the dog was euthanatized and the diagnosis was confirmed during a postmortem examination. No intraoperative surgical complications were recorded, although 1 dog received a packed RBC transfusion and 2 dogs received hetastarch during surgery. In 8 of the 12 cases that underwent surgery, the affected liver lobe was resected with a stapling device,[b] and in 1 case, the affected lobe was resected by means of a circumferential strangulation ligature. Method of lobe resection was not reported for the remaining 3 cases. Other surgical procedures were performed in 4 dogs and included gastrotomy for gastric foreign body removal (1), intestinal biopsy (1), incisional gastropexy (1), and lymph node biopsy (1).

In 11 of the 13 cases of LLT, the affected liver lobe was recorded in the medical record. Affected lobes included the left medial lobe (n = 4), left lateral lobe (3), papillary process of the caudate lobe (2), caudate lobe (1), and right lateral lobe (1). A predilection for a particular lobe could not be demonstrated.

In 6 of 13 cases, samples from the liver or abdominal cavity were submitted for aerobic bacterial culture, and in 4 cases, samples were submitted for anaerobic bacterial culture. Results of aerobic bacterial culture were positive in 3 of 6 cases; bacteria that were isolated included *Klebsiella pneumoniae*, *Pasteurella* spp, *Escherichia coli*, and gram-negative nonfermenting rods. In 1 case, cytologic examination of a smear of material submitted for anaerobic bacterial culture revealed rods with variable gram-staining characteristics. However, for all 4 cases, anaerobic bacterial culture did not yield any growth.

Results of histologic examination of affected liver lobes were reviewed by one of the authors (JHK), and in all instances, results were consistent with acute or chronic LLT. All affected liver lobes had histologic evidence of acute inflammation, extensive coagulative hepatocellular necrosis, and vascular and sinusoidal ectasia and thrombosis. Some liver lobes had areas with variable degrees of fibrosis and chronic inflammation.

In 11 of the 13 cases of LLT, the dog survived the perioperative period. One dog was found to have ultrasonographic evidence of peritoneal effusion, in addition to a possible LLT, and radiographic evidence of pleural effusion. A cause for the bicavitary effusions could not be determined, and the owners elected euthanasia. At postmortem examination, LLT was confirmed, but no other pathologic disease processes were identified. The 4-month-old Weimaraner died of cardiac arrest approximately 12 hours after surgery secondary to postoperative abdominal hemorrhage. The cause of the abdominal hemorrhage was suspected to be a failed ligature, but the dog died before exploratory celiotomy could be performed to determine the cause of the hemorrhage, and a postmortem examination was not performed.

Postoperative complications occurred in 6 of the 12 cases of LLT in which surgery was performed. Complications included development of anemia, arrhythmia, worsening of hypoalbuminemia, and vomiting followed by aspiration pneumonia. The 4-month-old Weimaraner with postoperative abdominal hemorrhage received a transfusion of packed RBCs. The dog that developed aspiration pneumonia was treated with antimicrobials IV, antiemetics, and IV fluid therapy. Two dogs with severe hypoalbuminemia received hetastarch IV, and one of these dogs received a fresh-frozen plasma transfusion. Arrhythmias were identified in 4 of 12 cases; in 2 of these dogs, arrhythmias were only identified postoperatively. Ventricular arrhythmias were treated with lidocaine boluses, followed by constant rate infusions of lidocaine. Median duration of hospitalization for the 11 cases that underwent surgery and were discharged was 4 days (range, 1.5 to 8.0 days). The duration of hospitalization was not significantly related to any other variable.

The dog that had 2 episodes of LLT recovered completely following the first surgery and did not develop any postoperative complications. The owner reported that the dog was healthy between episodes, although it continued to have episodes of gas bloating and excessive flatulence. The dog also recovered without complications from the second episode of LLT but was euthanatized 1 year later because of gastric dilatation-volvulus.

For 6 of the 10 dogs that survived, a follow-up CBC and serum biochemical profile were performed between 2 weeks and 1 year after surgery. In all instances, serum hepatic enzyme activities, total bilirubin concentration, and neutrophil count were within reference limits or had substantially improved, compared with preoperative values.

Follow-up information was obtained for all 11 cases in which the dog was discharged from the hospital. Median follow-up time was 14 months (range, 1.5 to 43 months). In all instances, owners reported that preoperative clinical signs resolved after surgery.

Four of the 10 dogs for which follow-up information was available were still alive at the time of final follow-up. Of the remaining 6 dogs, 2 died of gastric dilatation-volvulus 1 month and 12 months, respectively, after undergoing liver lobectomy and 4 were euthanatized because of uncontrollable seizures (1), multiple liver masses evident on abdominal ultrasonograms (1), an abdominal mass evident on abdominal ultrasonograms (1), and an unknown cause (1). The dog with the abdominal mass and the dog with multiple liver masses did not undergo biopsy or necropsy.

Discussion

The veterinary literature provides little information regarding LLT in dogs, but reports[8,14,17,19] that have been published suggest that the left lateral lobe is the most commonly affected lobe and that prompt surgical

intervention can result in a successful outcome. As LLT is a rare condition in all species, including dogs, there is little information regarding etiology, signalment, diagnosis, surgical findings, and short- and long-term outcomes.

In the present study, most dogs were evaluated because of acute or chronic, nonspecific clinical signs, including vomiting, anorexia, lethargy, diarrhea, weight loss, and collapse. Most cases in the present study were examined because of an acute worsening of nonspecific clinical signs, but 3 cases had a chronic clinical course. Acute decompensation is likely a result of partial or intermittent LLT developing into complete LLT, with complete venous obstruction and resultant hepatic necrosis.[24] Chronic signs were likely a result of partial torsion or intermittent torsion that did not completely occlude hepatic blood flow.[24]

Previous reports of LLT in dogs have involved mainly large-breed dogs and a few medium-sized dogs. In the present study, all affected dogs were large-breed dogs, with half of the dogs being Akitas or Golden Retrievers. The clinical importance of this information is unknown because of the low number of cases, and it is unclear whether the size of the dog is associated with the pathogenesis of LLT. However, the risk for gastric dilatation-volvulus is higher in large- and giant-breed dogs,[25,26] as is the risk for splenic torsion.[24,27,28]

The most common hematologic abnormalities in the present study were mature neutrophilia and leukocytosis. Increases in WBC count were consistent with endogenous glucocorticoid-associated neutrophilia or neutrophilia associated with inflammation and infection.[29] Occlusion of the hepatic vessels during LLT results in congestion and subsequent necrosis of the lobe, likely explaining the neutrophilia.[3] Neutrophilia could also result from survival of organisms usually phagocytized by hepatic sinusoid macrophages.[30]

In a previous report[17] of dogs with LLT, laboratory abnormalities were nonspecific, whereas in the present study, we consistently identified high serum hepatic enzyme activities compatible with hepatic injury. As the liver lobe twists, venous obstruction causes increased hydrostatic pressure; ascites; arterial and venous thrombosis; and, eventually, tissue necrosis.[17] Increases in alanine aminotransferase and aspartate aminotransferase activities are consistent with hepatocellular injury and altered cell membrane permeability secondary to hepatic necrosis. In contrast, serum alkaline phosphatase activity is less frequently affected by hepatocellular damage but is still routinely increased with severe hepatic tissue damage.[30] Although increases in serum hepatic enzyme activities are not specific for LLT, abnormal values in conjunction with clinical signs of acute abdomen should lead to the inclusion of LLT in the list of differential diagnoses.

In contrast to findings in a previous report,[15] results of the present study suggest that abdominal ultrasonography may be a useful imaging modality for detection of LLT. Radiographic evidence of a cranial abdominal mass is not specific for a diagnosis of LLT but does suggest that additional interventions, such as abdominal ultrasonography or exploratory surgery, are indicated. Abdominal ultrasonography with Doppler assessment of the hepatic vessels revealed decreased blood flow and supported the diagnosis of LLT in 5 of 8 cases in the present study. In people, ultrasonography, computed tomography, and magnetic resonance imaging are all used to diagnose LLT, but no method is definitive for the diagnosis of LLT.[1,4,7,10,13] In the future, other imaging modalities, such as nuclear scintigraphy or hepatic vascular contrast studies, may be used to diagnose LLT, but because LLT is a surgical emergency, performing such diagnostic tests may be clinically inappropriate.[9,13,19]

Abdominal effusion was detected ultrasonographically or radiographically in 6 of 9 cases in the present study, but pleural effusion was detected in only 1 case. The abdominal effusion was suspected to have resulted from the increase in hydrostatic pressure associated with hepatic vein obstruction.

In the present study, the left medial and left lateral liver lobes were most commonly involved, but caudate lobe and right lateral lobe involvement were also documented. In previous reports,[8,17,19] the left lateral lobe was the most commonly affected lobe. The frequent involvement of the left lateral lobe is thought to be related to laxity of the hepatogastric ligament or the large size and mobility of the lobe and its anatomic separation from the other lobes. However, these anatomic characteristics fail to explain torsion of the other liver lobes. Traumatic disruption and an absence of supporting hepatic ligaments have been described as potential causes for LLT,[14,17] but cases included in the present study did not have a history of trauma, and none were found to be obviously lacking the normal supporting hepatic ligaments. Although an abdominal hernia was suspected as the precipitating cause for the 4-month-old Weimaraner, the medical record provided incomplete details concerning the hernia and the cause of the LLT could not be confirmed.

Intermittent gastric gas distension leading to stretching of the left triangular ligament has been suggested as a potential risk factor for LLT in dogs.[17,18] One dog in the present study had a history of gastric dilatation-volvulus prior to development of LLT, and 2 dogs reportedly died of gastric dilatation-volvulus after recovering from LLT. Previous reports[15,18] describe torsion of the left hepatic lobe and torsion of the right lateral hepatic lobe in 2 dogs with gastric dilatation-volvulus. In the dog with torsion of the left hepatic lobe, gastric dilatation-volvulus was believed to have resulted in laxity of the left triangular ligament,[15] but in the dog with torsion of the right lateral hepatic lobe, LLT was not confirmed to be related to the gastric dilatation-volvulus.[18] It is possible that potential space created by laxity of the hepatic ligaments during LLT or as a result of liver lobectomy could predispose high-risk, large-breed dogs to develop gastric dilatation-volvulus. Although there is no evidence that prophylactic gastropexy will prevent a first occurrence of gastric dilatation-volvulus, gastropexy has been shown to prevent recurrences of gastric dilatation-volvulus

following an acute episode and may prevent occurrences following liver lobectomy.[31] The authors recommend considering that prophylactic gastropexy be performed in surgically stable dogs at the time of liver lobectomy.

There are many similarities between previously reported cases of splenic torsion[26-28] and cases of LLT in the present study, including signalment and clinical signs. Additionally, many dogs in both case populations had a history of excessive flatulence, intestinal gas distension, or gastric dilatation-volvulus. A relationship between splenic torsion and gastric dilatation-volvulus has been proposed but has not been fully characterized, and the nature of the relationship appears unclear.[26] One theory proposes that repeated episodes of gastric dilatation stretch the gastrosplenic and splenocolic ligaments, causing increased splenic mobility.[32] Thus, previous authors have recommended that prophylactic gastropexy be performed at the time of splenectomy in dogs with splenic torsions.[26]

Two dogs in the present study did not survive the perioperative period. One was a 4-month-old Weimaraner with an abdominal hernia that died of cardiopulmonary arrest secondary to acute abdominal hemorrhage 5 hours after surgery, and the other was a dog with bicavitary effusions for which the owner elected euthanasia because of the uncertain prognosis. Bicavitary effusions are most frequently observed in conjunction with neoplasia, followed by cardiovascular disease, infectious disease, sepsis, pancreatitis, and nonneoplastic liver disease.[33] Postmortem examination did not demonstrate any of the diseases previously associated with bicavitary effusions; therefore, LLT was suspected to be related to the formation of bicavitary effusions.

In conclusion, our results indicate that development of nonspecific clinical signs of vomiting, lethargy, and anorexia in conjunction with high serum alanine aminotransferase and aspartate aminotransferase activities and mature neutrophilia in a medium-sized or large-breed dog should increase the index of suspicion for LLT. No particular historical, physical, or biochemical findings were found to be diagnostic for LLT, but abdominal ultrasonography with Doppler assessment of the hepatic vessels may be an effective imaging modality.

Further, findings from the present study suggest that the prognosis for dogs with LLT that undergo liver lobectomy is excellent. Life-threatening complications, such as hemorrhage and aspiration pneumonia, can occur, but the long-term outcome for dogs with LLT that survive the hospitalization period appears to be excellent. Although time between admission and surgery was not significantly associated with outcome in the present study, we recommend that surgical intervention be performed promptly to prevent further hepatic necrosis and, potentially, formation of abscesses in the affected lobe.[3]

a. SPSS, version 10.1, SPSS, Chicago, Ill.
b. TA Premium 30-3.5, 55-3.5, 90-3.5, Kendall Healthcare Products, Mansfield, Mass.

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