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Acute liver lobe torsion in a kitten

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Abstract

Case summary A 10-week-old intact male domestic shorthair kitten presented for an acute onset of lethargy, vomiting and anorexia. An abdominal mass effect was palpable on presentation. Blood work, abdominal radiographs and point-of-care abdominal ultrasound showed severe anemia, decreased serosal detail and abdominal effusion, respectively. Based on the concern for an abdominal organ torsion or ruptured mass, an emergency abdominal exploratory surgery was performed. Torsion of the entire caudate liver lobe was discovered with a secondary hemoabdomen, and a liver lobectomy was performed. The kitten was stabilized and discharged 3 days after surgery. At the recheck examination, 15 days postoperatively, the patient was reported to be doing well.

Relevance and novel information Liver lobe torsion is a rare condition previously reported in six other cats; however, this is the first peer-reviewed report in a kitten successfully treated with surgery with no identifiable underlying cause.

Keywords: Hepatic disease; small animal critical care; shock; abdominal surgery

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Introduction

An acute abdomen is a common presentation seen at veterinary emergency hospitals caused by various etiologies. An acute abdomen secondary to a liver lobe torsion (LLT) is rarely reported in dogs, cats, horses and humans, but is relatively frequent in the rabbit.^{1–26} The exact cause of LLT is still unknown. There are several potential causes of LLT in dogs and cats, such as secondary to trauma, neoplasia, gastric dilatation and volvulus in dogs, abnormal hepatic ligaments or a diaphragmatic hernia.^{1–18} The purpose of this case report is to document the first peer-reviewed report of an acute LLT in a kitten and its successful management.

Case description

A 10-week-old intact male domestic shorthair kitten was referred to an emergency animal hospital for an abdominal exploratory surgery for a suspected foreign body. The kitten initially presented to the referring veterinarian for an acute onset of lethargy, vomiting and anorexia of <12h duration. Initial diagnostics performed by the referring veterinarian included a complete blood count and serum biochemical panel (CBC/CHEM; IDEXX Laboratories), feline leukemia virus and feline immunodeficiency virus point-of-care ELISA test (FeLV/FIV;

IDEXX Laboratories) and abdominal radiographs. The CBC/CHEM abnormalities were a marked non-regenerative anemia with a hematocrit of 9.8% (reference interval [RI] 30.3–52.3%) and reticulocyte count of 34.5 K/μl (RI 3.0–50.0 K/μl), toxic neutrophils with suspected bands, mild lymphocytosis of 7.23 K/μl (RI 0.92–6.88 K/μl), moderate thrombocytopenia of 54 K/μl (RI 151–600 K/μl), mild hyperglycemia of 186 mg/dl (RI 77–153 mg/dl), increased blood urea nitrogen (BUN) of 57 mg/dl (RI 16–33 mg/dl), decreased creatinine of 0.3 mg/dl (RI 0.6–1.6 mg/dl), decreased total protein of 4.9 g/dl (RI 5.2–8.2 g/dl) and decreased globulin of 2.6 g/dl (RI 2.8–4.8 g/dl). The kitten's liver values were normal (alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase, gamma-glutamyl transferase and total bilirubin). FeLV/FIV tests were both negative. The primary veterinarian's interpretation of the abdominal radiographs was abdominal distension

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with decreased serosal detail and radio-opaque grit-like material in the stomach. Given the severity of the kitten's condition, the kitten was referred to an emergency facility for further care.

On physical examination at the hospital, the kitten was laterally recumbent with a temperature of 94.1°F (34.5°C), a heart rate of 190 beats/min, a respiratory rate of 40 breaths/min with increased respiratory effort, pale white mucous membranes, non-palpable femoral pulses, abdominal pain, and palpable cranial abdominal organomegaly or mass effect. An indirect systolic blood pressure of 138 mmHg was obtained via Doppler (Biomed Technologies). A point-of-care abdominal ultrasound (SonoScape: Universal Medical Systems) showed a mild amount of ascites in the cranial abdomen. An abdominocentesis on presentation was unsuccessful, but a sample was successfully obtained an hour later. There was only sufficient amount of fluid obtained to perform a microscopic evaluation. In-house cytological evaluation of the ascites revealed only red blood cells without evidence of microorganisms or white blood cells.

The kitten was started on lactated Ringer's solution (120 ml/kg/day [Hospira]), ampicillin sodium/sulbactam sodium (30 mg/kg IV [Unaysn; Roerig Division of Pfizer]), hydromorphone (0.05 mg/kg IV [West-Ward]) and flow by oxygen supplementation (2l/h). A venous blood gas analysis (Biomedical Stat Profile pHox; Nova Biomedical Ultra) and packed cell volume (PCV) revealed a metabolic acidosis with compensatory respiratory alkalosis (pH 7.408 [RI 7.344–7.441], pCO₂ 19.2 mmHg [RI 32.6–38.4 mmHg], pO₂ 164.7 mmHg [RI 38.0–65.6 mmHg], base excess extracellular fluid –12.6 mmol/l [RI –10.7 to 0.7 mmol/l], HCO₃⁻ 12.3 mmol/l [RI 15.4–23.4 mmol/l]), anemia (PCV 9% and hematocrit 11% [RI 35–55%]), hyponatremia (138.6 mmol/l [RI 142.0–150.0 mmol/l]), hypermagnesemia (0.87 mmol/l [RI 0.30–0.50 mmol/l]), hyperglycemia (152 mg/dl [RI 75–116 mg/dl]), hyperlactatemia (4.6 mmol/l [RI 0.7–2.8 mmol/l]) and an elevated BUN (54 mg/dl [RI 8–30 mg/dl]). Blood type was also evaluated as type A (Rapid Vet-H; Dmslaboratories). Further diagnostic imaging, such as an abdominal ultrasound, were not available at the time of presentation. A packed red blood cell transfusion (10 ml/kg IV over 2 h [Animal Blood Resources]) was given to address the kitten's anemia. Based on the clinical presentation and concern for an abdominal organ torsion, an abdominal exploratory surgery was pursued.

Within 4 h of presentation, the kitten was taken to surgery. An additional dose of hydromorphone (0.05 mg/kg IV [West-Ward]) was used as premedication. General anesthesia was induced with propofol (3.5 mg/kg IV given to effect [Zoetis]). A size 3 mm cuffed endotracheal tube was used for intubation, and the kitten was maintained on 2% sevoflurane (Zoetis) in oxygen at 1.5l/min.

The kitten was aseptically prepared for surgery. A ventral midline incision was made from the xiphoid process extending caudally to the pubis, approximately 7 cm in length. A moderate amount of hemorrhagic abdominal effusion was present. The entire caudate liver lobe was twisted on its axis and appeared congested, enlarged and necrotic. A hemoclip (Teleflex Medical) liver lobectomy of the caudate liver lobe was performed. The left lateral liver lobe appeared slightly congested, and a biopsy was obtained. The caudate liver lobe and left lateral liver lobe biopsy were submitted for histopathology review (Antech Diagnostics). The remainder of the abdomen was unremarkable, and no cause for this torsion was identified. The abdomen was lavaged copiously with warmed saline. The body wall was closed with a continuous pattern using 3-0 nylon suture (Nylon; J&J Healthcare Systems). The subcutaneous tissue was closed with a continuous pattern using 4-0 polydioxanone suture (PDS; J&J Healthcare Systems). The skin was closed using surgical staples.

Owing to the amount of blood loss during surgery, a PCV/total solids (TS) was performed 2 h postoperatively. The PCV and TS were 14% and 4.6 g/dl, respectively. The kitten was also exhibiting clinical signs of anemia (pale-pink mucous membranes, slight increased to increased respiratory effort, snappy femoral pulses, bradycardia and hypotension). Therefore, a second packed red blood cell transfusion (12.5 ml/kg IV over 4 h [Animal Blood Resources]) was administered. One dose of diphenhydramine hydrochloride (2.2 mg/kg IM [West-Ward]) was given towards the end of the transfusion owing to concern over transfusion reaction (tachycardia). The following day the kitten was cardiovascularly stable and eating well. The kitten was discharged 3 days postoperatively on buprenorphine (0.015 mg/kg PO [Roadrunner Pharmacy MFG]), amoxicillin trihydrate/clavulanate potassium (Clavamox 13.9 mg/kg [Zoetis]), and a probiotic (Purina Pro Plan Veterinary Fortiflora; Nestlé Purina PetCare). Antibiotic therapy was continued postoperatively owing to concerns about a secondary infection from hypoxic injury given the changes viewed in the left lateral liver lobe.

At follow-up examination, 15 days postoperatively, the kitten was reported to be doing well. The recheck PCV/TS was 22%/6.4 g/dl. A fecal specimen for intestinal parasite screening was submitted, and results were positive for *Toxocara cati*. A 5-day course of fenbendazole was prescribed (50 mg/kg/day PO [Panacur; Intervet, Merck Animal Health]).

The histopathology findings (Antech Diagnostics) of the caudate liver lobe revealed mild sinusoidal and vascular dilation and congestion, mild diffuse coagulative necrosis with a variable infiltrate of degenerated neutrophils throughout the parenchyma, subcapsular hemorrhage and a multifocal capsular fibrinohemorrhagic

exudate. The overall findings were diffuse, subacute, hepatic congestion with necrosis, hemorrhage and hemorrhagic exudate. The left lateral liver lobe biopsy revealed mostly diffuse panlobular coagulative necrosis with scattered degenerate neutrophils and lesser macrophages, dilated bile ductules with inspissated luminal bile pigment, a small amount of portal scattered inflammatory and hematolymphoid cells, and dilated portal lymphatic vessels and portal hemorrhage. There was no evidence of infectious organisms or neoplasia. The overall findings were marked subacute panlobular hepatic necrosis with bile duct ectasia and cholestasis. No culture or special stains were performed on the samples submitted.

Discussion

To our knowledge, there are six published feline case reports of LLT (11-month-old, 5-, 6-, 10-, 13- and 15.7-year-old cats).¹⁻⁶ This is the first peer-reviewed report of LLT in a kitten. In the six previous reports, five were diagnosed at the time of surgery, while the remaining cat was diagnosed post mortem.^{1-3,5,6} The suspected causes for LLT in these cats were a pseudocyst, neoplasia, diaphragmatic hernia secondary to a motor vehicle accident, pectus excavatum and unknown etiology.^{1-3,5,6} A report of an 11-month-old kitten diagnosed with torsion of the right medial and quadrate liver lobes along with the gallbladder was presented at the ECVS Annual Scientific Meeting in 2019.⁴ A pseudocyst and hemorrhagic infarction associated with a mucocele was discovered on histopathologic examination of the liver segments and gallbladder in this kitten.⁴ In the present case, the cause for LLT is unknown. There was no history of trauma, neoplasia, diaphragmatic hernia or congenital abnormalities predisposing to LLT. This kitten was later diagnosed with *T. cati*, which is known to migrate from the gastrointestinal tract to the liver after ingestion of infected eggs.²⁷ A parasitic infiltration of the liver resulting in alterations in liver architecture has been a proposed cause for torsion.^{19,20} However, no microorganisms were observed on histopathology.

It has been postulated that the ligaments supporting the liver lobes have a role in LLT.^{1-3,5-15,19,22,23} Increased laxity, aplasia, stretching or rupture of these ligaments can result in increased mobility of the liver lobe and possible torsion.^{1-3,5-15,19,22,23} There were no abnormalities reported at the time of surgery in these ligaments. For most cases, the cause for LLT is not identified, as in the case reported here.^{3,6-8,10-13,16,17}

The clinical signs and physical examination findings in this case were of acute onset and non-specific for LLT, but were consistent with previous reports.¹⁻²⁶ This kitten exhibited lethargy, vomiting and anorexia. Previous cases in dogs and cats have reported abdominal pain, lethargy, vomiting, anorexia, collapse and diarrhea, all of which are non-specific for LLT.¹⁻¹⁸ The most significant

physical examination finding in this case suggestive of LLT was a palpable abdominal mass effect and abdominal pain. In previous cases in dogs and cats, abdominal pain, abdominal distension and a palpable abdominal mass were reported.^{1-4,6,7,9,10,12-18}

This kitten exhibited signs of hypovolemic shock and severe decompensation on presentation. It is suggested that the severity of presentation depends on the degree of torsion and occlusion of hepatic blood flow, leading to hepatic congestion, arterial and venous thrombosis, and subsequent necrosis.^{3,7,8,10,12,22} A complete LLT resulting in complete occlusion or avulsion of hepatic blood flow results in a more severe and acute presentation.^{2,7} A partial or intermittent LLT resulting in partial or intermittent complete occlusion results in a less severe and chronic presentation.^{2,7} This kitten had a complete LLT diagnosed at the time of surgery with subsequent hemorrhage and hypovolemic shock, resulting in a severe, acute presentation. Although a PCV/TS on the ascites was not performed, the cytological and gross examination of the ascites, histopathologic findings of the liver and low peripheral PCV/TS were suggestive of a hemoabdomen. The ascites may have also developed from increased hydrostatic pressure secondary to torsion or thrombosis of the hepatic vessels.^{7,8,10-14}

In this case report, LLT was diagnosed at the time of surgery. The abdominal radiographs performed prior to surgery showed non-specific changes of abdominal distension with decreased serosal detail. Further diagnostics, such as an abdominal ultrasound with Doppler flow or CT scan, were not available at the time of presentation. Abdominal radiographs may reveal decreased serosal detail due to peritoneal effusion, mass effect in the cranial abdomen or displacement of the abdominal organs.^{1,2,7,9,11,13,17,19,20,22,23} Abdominal ultrasound may reveal changes in echogenicity, decreased hepatic blood flow with color-flow Doppler, peritoneal effusion, liver mass and/or segmental hepatomegaly.^{1-11,13,15,17-20,22-24} In a previous case series study in dogs, LLT was diagnosed on Doppler abdominal ultrasound findings in five cases.⁷ CT may reveal enlargement or abnormal positioning of the liver lobe or a mass adjacent to the liver with low attenuation and reduced to absent contrast enhancement.^{2,6,10,11,13,16,24-26} There has also been a report of a 'whirl sign' on CT findings of a dog with LLT and secondary disseminated intravascular coagulation and multiorgan infarction.¹³ A 'whirl sign' is a CT finding described as the rotation of vessels around the point of torsion.¹³ Changes on imaging are usually non-specific, which creates a challenge for definitively diagnosing LLT.^{1,2,11,12}

In this case, hematological and biochemical changes were also non-specific for LLT. Laboratory abnormalities reported in the veterinary literature include anemia, leukocytosis, elevated liver enzymes, thrombocytopenia, azotemia, hypoalbuminemia, hypoproteinemia, hyperlactatemia and elevated amylase and lipase.¹⁻²³

The significant laboratory findings in this kitten were a non-regenerative anemia, thrombocytopenia, leukocytosis, increased BUN, and decreased total protein and globulins. The non-regenerative anemia, thrombocytopenia and decrease in globulins were most likely due to acute hemorrhage. Owing to the acute nature of hemorrhage, the bone marrow had not yet generated a regenerative response.²⁸ The leukocytosis was most likely a result of the kitten's inflammatory response to LLT. The elevated BUN was most likely pre-renal secondary to hypovolemia and dehydration. The liver values in this patient were normal at the time of presentation. It can take up to 12h for alanine aminotransferase activity to peak after injury.²⁸ Blood was obtained from this patient within 12h of presentation, so it is possible that this may have changed if another chemistry panel was checked at a later time.

LLT has been reported in all liver lobes in the veterinary literature, with the left lateral lobe most commonly affected. It has been suggested that the left liver lobe is more mobile owing to its large size and location relative to the other liver lobes.^{1-3,6,7,13,16,17,19} In reported cases in dogs and cats from 2001 to 2020 (38 LLTs in 32 dogs and cats [in two cases, affected liver lobes were not recorded]), the left lateral liver lobe was most commonly affected (12/32 cases), with two of these cases also having the left medial lobe affected.¹⁻¹⁸ The left medial liver lobe (7/32 cases: two of these cases also had the left lateral lobe affected), caudate liver lobe (3/32 cases), papillary process of caudate lobe (3/32 cases), right lateral lobe (1/32 cases), right medial liver lobe (5/32 cases, with four of these cases also having the quadrate liver lobe affected) and quadrate liver lobe (5/32 cases, with four of these cases also having the right medial liver lobe) have also been reported.^{3-12,14,16,17} In the cat, the right medial liver lobe was most frequently reported (two right medial and quadrate liver lobes, one right medial liver lobe, two left lateral liver lobes, one papillary process of the caudate lobe).¹⁻⁶ In this study, the entire caudate liver lobe was affected, which, to our knowledge, has not previously been reported in the cat.

For an increased chance of survival, the treatment choice for both chronic and acute LLT for dogs and cats is prompt surgical intervention.^{1-11,15,17} In this case report the entire affected liver lobe was resected without untwisting the lobe back to its normal position. Untwisting the affected liver lobe can lead to ischemia-reperfusion injury, thromboembolism and endotoxin release, which can lead to disseminated intravascular coagulation and septic shock.^{2,8,10} Therefore, it is not recommended to untwist the liver lobe prior to resection.

The histopathologic findings seen in this kitten are consistent with the histopathologic findings reported in previous cases of LLT.⁶⁻¹³ The histopathology examination of the caudate liver lobe showed necrosis, congestion, inflammation and hemorrhage. The histopathology examination

of the left lateral lobe that was biopsied also showed significant changes of necrosis, inflammation, hemorrhage, and bile duct dilation and stasis. Although the reasons for these changes are unknown, we speculate that they may have been secondary to hypoxic injury from decreased perfusion secondary to shock and hypovolemia, or could have been evidence of intermittent LLT.

Conclusions

This is the first peer-reviewed report of LLT in a kitten. The kitten described in this case report presented in acute hypovolemic shock with clinical signs consistent with acute LLT. An exploratory abdominal surgery was used to diagnose and successfully treat this kitten. It is up to the clinician to recommend surgery when diagnostic imaging is not available. LLT can carry a good prognosis if diagnosed and treated in a timely fashion.

Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval This work involved the use of non-experimental animals only (including owned or unowned animals and data from prospective or retrospective studies). Established internationally recognized high standards ('best practice') of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not specifically required for publication in *JFMS Open Reports*.

Informed consent Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (either experimental or non-experimental animals) for the procedure(s) undertaken (either prospective or retrospective studies). No animals or humans are identifiable within this publication, and therefore additional informed consent for publication was not required.

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