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IMAGING DIAGNOSIS

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Left medial liver lobe torsion and postoperative acute gastric rupture in a 2.5-year-old male-castrated Flemish Giant rabbit (*Oryctolagus cuniculus*)

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Abstract

A 2.5-year-old male-castrated rabbit presented with acute abdominal pain, lethargy, and anorexia. Digital radiography revealed increased left-sided hepatomegaly, gastric dilation, and decreased peritoneal serosal detail. Abdominal ultrasonography identified a torsed left liver lobe, gastric dilation, and peritoneal effusion. Surgery confirmed a left medial liver lobe torsion, with subsequent lobectomy and seven days of hospitalization. The patient re-presented 2 days after discharge and suddenly died while hospitalized, with acute gastric rupture, fulminant peritonitis, and multifocal hepatic infarcts diagnosed on necropsy. We believe this is the first recorded imaging diagnosis of a left medial liver lobe torsion in a rabbit.

KEYWORDS
gastrointestinal, hepatic, imaging, lagomorph

1 | SIGNALMENT, HISTORY, AND CLINICAL FINDINGS

A 2.5-year-old, BCS 6/9, male-castrated Flemish Giant rabbit presented for acute onset of hunched posture, lethargy, anorexia, and decreased defecation since the prior evening. On abdominal palpation, the patient had a tense, painful cranial abdomen, distended stomach, and no palpable feces within the colon. Other significant findings were pale mucous membranes and ~7% dehydration. Hematology revealed a severe normocytic, normochromic, regenerative anemia with moderate hypoproteinemia. Biochemistry identified a severe elevation in liver enzymes and CK, mild hypokalemia, and creatinine at the upper end of reference limits. These clinical findings were considered indicative of liver lobe torsion (LLT) with secondary peritoneal effusion (e.g., hemoabdomen, serosanguinous), so abdominal imaging was pursued.

2 | IMAGING, DIAGNOSIS, AND OUTCOME

Abdominal digital radiography (DX) revealed moderate left-sided hepatic enlargement, which raised concern for an underlying acute hepatopathy (Figure 1A–C.). There was decreased serosal detail within the cranial abdomen, likely reflecting peritoneal effusion and/or peritonitis. The stomach was moderately dilated with soft tissue opaque ingesta, fluid, and scant gas. (Figure 1A–C.) The sum of gastric height and length on the lateral projections was greater than the length of L1 to the coxofemoral joints. There was no pathologically dilated small intestine or mineral-opaque foreign material. The cecum contained a large volume of gas and was largely devoid of ingesta, with no fecal balls within the colon (Figure. 1A–C). Abdominal ultrasonography (AUS) identified a moderately enlarged liver lobe with a diffusely coarse echotexture in the left cranial abdomen. The abnormal left liver lobe was peripherally markedly hypoechoic with a stellate echotexture

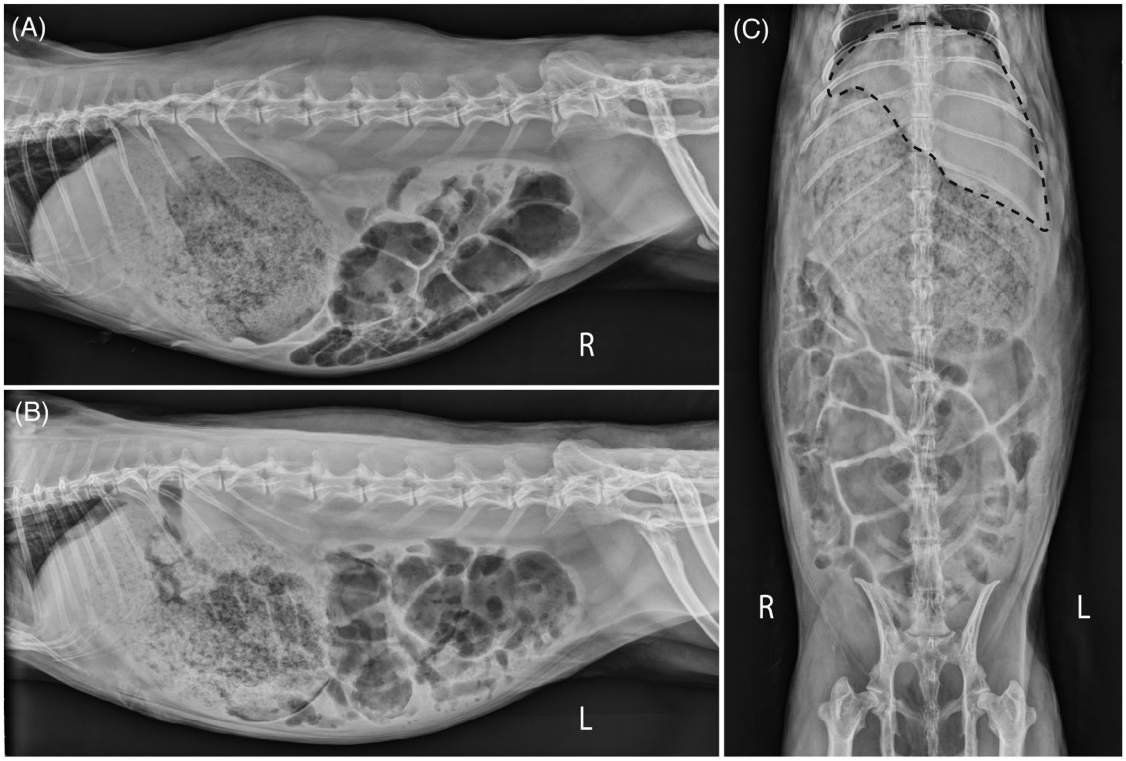

FIGURE 1 Abdominal radiography demonstrating hepatomegaly and gastric dilation secondary to left-medial LLT in a rabbit. A, Right lateral abdominal radiograph. B, Left lateral abdominal radiograph. C, Ventro-dorsal abdominal radiograph. Left-sided hepatic enlargement (dashed line), decreased serosal detail within the cranial abdomen, and a moderately dilated stomach. Acquired using Summit-Toshiba Rotanode with LX125 collimator, Canon CXDI-50G plate. CXDI postprocessing algorithm. kVp 70, mAs 5.0.

(Figure 2A, B) and markedly decreased to absent blood flow on Power Doppler (Figure 2A). The enlarged left liver lobe had a stalk-like base from which multiple dilated vessels with no flow on Power Doppler could be traced into the left hepatic parenchyma, suspected to represent a site of torsion (Figure 3A–C). Normal Power Doppler flow was visualized in the hepatic vessels proximally adjacent to this stalk-like region (Figure 3B). There was scant blood flow on superb microvascular imaging (SMI) across the stalk-like region synchronous with the patient's pulse, suspected to likely reflect scant persistent arterial flow to the affected lobe (Figure 3C). The right and caudate liver lobes maintained a normal echogenicity, with normal blood flow on Power Doppler (Figure 2C–D). There was moderate mildly echogenic peritoneal effusion, primarily located in the left cranial abdomen around the liver (Figure 2B). The falciform fat was diffusely hyperechoic and beam attenuating (Figure 2B). The stomach was moderately dilated with gas and echogenic ingesta, and no abnormalities of the visible gastric wall were found. The colon was predominantly empty, with concurrent subjectively decreased cecal fill. Based on the AUS and DX findings, a diagnosis of left-sided liver lobe torsion with secondary peritonitis, peritoneal effusion, and gastric dilation (GD) was made. The patient was then transferred to surgery, which confirmed a diagnosis of a left medial LLT and a small volume of hemorrhagic peritoneal effusion. The torsed left medial liver lobe was surgically ligated and removed without complication.

Immediate postoperative medical management consisted of multimodal analgesia, broad-spectrum antibiotic therapy, a norepinephrine continuous rate infusion (CRI), intravenous fluid therapy (IVFT), and a cross-matched blood transfusion (due to anemia and hypotension). Postoperative rabbit gastrointestinal syndrome (RGIS) was also managed with diet via syringe-fed OXBOW Critical Care. Two days postoperatively, the patient developed increased respiratory effort due to pleural effusion, which resolved with thoracocentesis and cessation of IVFT. The patient was discharged after seven days with antibiotics, analgesia, and nutritional support.

Two days after discharge, the rabbit re-presented due to acute hyporexia and lethargy, having reared on his hind legs while fighting the other rabbit in the household the prior evening. The patient was moderately pyrexic, but clinical parameters were otherwise within normal limits. Later that day, while hospitalized, the patient was found dead and nonresponsive to CPR in lateral recumbency. Necropsy revealed severe focally extensive gastric perforation with transmural edema, severe acute peritonitis, fibrinous polyserositis with abundant free feed material, multifocal left lateral/caudal liver lobe necrosis/infarction, surgically absent left medial liver lobe, mild serosanguinous pleural/pericardial effusion, and minimal focal chronic pododermatitis of the third digit of the right thoracic limb.

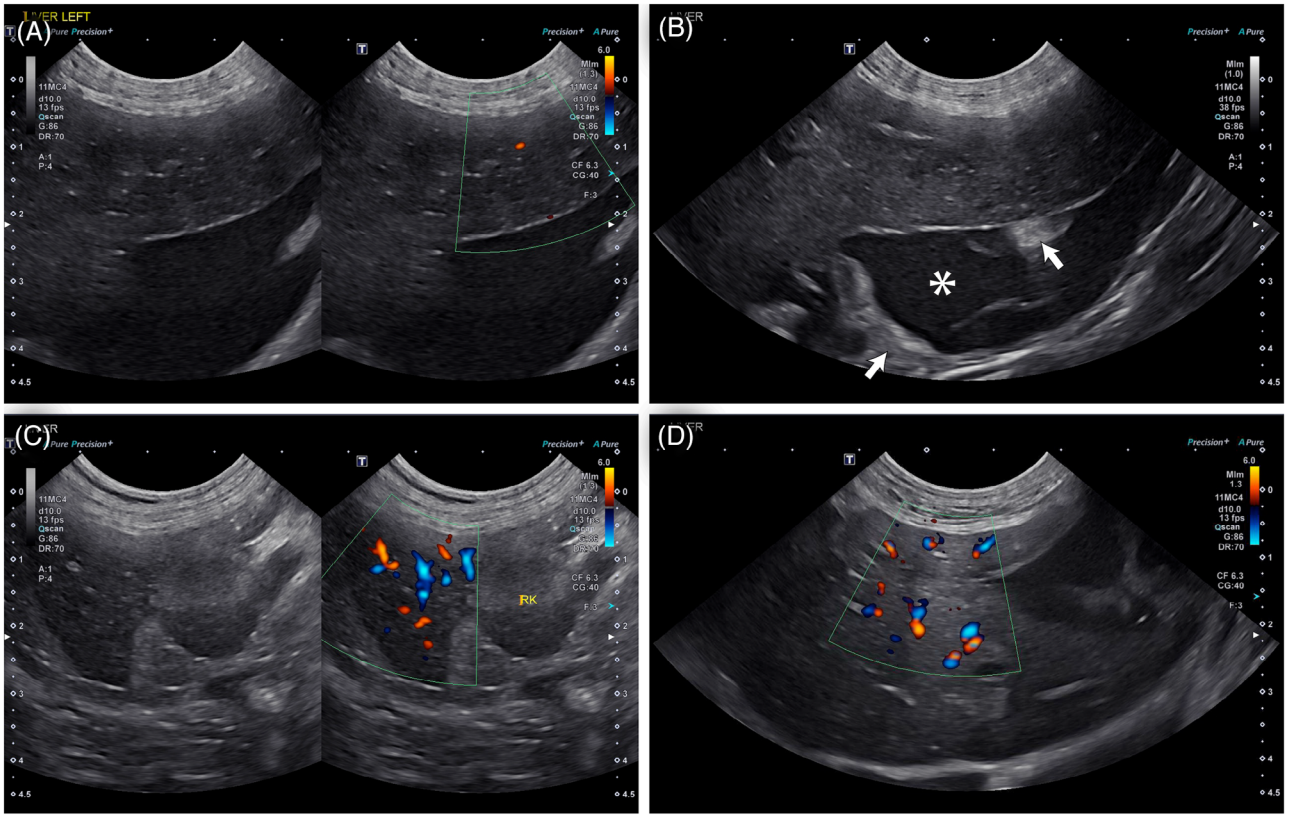

FIGURE 2 Hepatic ultrasonography demonstrating decreased blood flow within the torsed left liver lobe and normal blood flow within the right liver in a rabbit (transverse view). A, Left-sided hepatic parenchyma with minimal to absent blood flow on directional Power Doppler, B, Left liver lobe with adjacent faintly echogenic peritoneal effusion (asterisk) and hyperechoic peritoneal fat (arrow). C, Caudate lobe with normal blood flow on Power Doppler. D, Right-sided hepatic parenchyma with normal blood flow on directional Power Doppler. B-mode images acquired on Canon Aplio i700 using a microconvex electronic 7 MHz transducer. Patient in dorsal recumbency with abdominal fur clipped and warmed ultrasound coupling gel.

3 | DISCUSSION

LLT has been well documented as a significant cause of morbidity and mortality in rabbits, with both acute[2–10] and chronic[3,6] presentations. LLT occurs when a liver lobe rotates at the level of its hilar attachment, resulting in venous occlusion and compromised arterial supply, leading to hepatic congestion and necrosis.[2,3,4] The exact etiology of LLT has not been definitively confirmed, with speculated mechanisms including trauma, gastrointestinal dilation, congenital hepatic ligament dysplasia/aplasia, hepatic ligament laxity, and an underlying hepatopathy.[4,6] Lop breeds are over-represented, but there is no statistically significant correlation between signalment and incidence of LLT.[4,6] The caudate lobe of the liver is the most common site of LLT in rabbits, likely due to the narrow stalk-like morphology of the attachment of the caudate lobe.[2–8,10] Right lateral, right medial, left lateral, and LLT affecting multiple liver lobes (right lateral/left lateral, right lateral/caudate) have been described as occurring more infrequently.[2–8,10] However, no cases of left medial LLT have been described within the literature. Acute LLT is a medical emergency, with death reported to occur as rapidly as 12–72 h from onset of clinical signs in some cases,[3] with common complications including hemoabdomen, systemic inflammatory response syndrome (SIRS), disseminated intravascular coagulopathy (DIC) and acute-death.[2,4,7] Chronic LLT have also been reported, often discovered incidentally at necropsy, with clinically affected individuals being asymptomatic or showing nonspecific low-grade signs of RGIS and/or abdominal pain.[3,6]

Common clinical signs and clinical examination findings reported with LLT include lethargy,[2,3,5,9,10] altered mentation,[3,5,7] symptoms of RGIS[3,4,6] (e.g., hypo-/anorexia,[2,3,5,7,9,10] decreased fecal output,[2,5] doughy distended stomach,[4] diarrhea[5]), cranial abdominal pain,[2–7,9,10] cranial abdominal mass effect,[2,6,7] jaundice,[9,10] pale mucous membranes,[3,4,7,10] tachycardia,[7] tachypnoea,[7] decreased skin turgor,[4] and hypothermia.[3,4] Hematological and biochemical abnormalities associated with LLT in the literature include elevated liver enzymes[4–6,8,10] (i.e., ALT,[2,3,7,9] AST,[2,3,7,9] ALP,[2,7,9] GGT[3,7,9]), anemia,[2,3,5–9,10] azotemia,[4–6,8,10] hyperglycemia,[4,5] thrombocytopenia,[8] elevated creatine kinase,[10] and rarely mild electrolyte abnormalities.[5] Therefore, the presenting clinical signs and hematological/biochemical changes of the patient in this case report are consistent with what has been previously described.

The diagnosis of LLT in rabbits using imaging has been described utilizing radiography, ultrasonography, and CT. Radiographic findings are typically nonspecific, including hepatomegaly,[2,5,9,10] rounded liver margins,[2,5] increased gastrointestinal gas/dilation[2,5,9,10], and

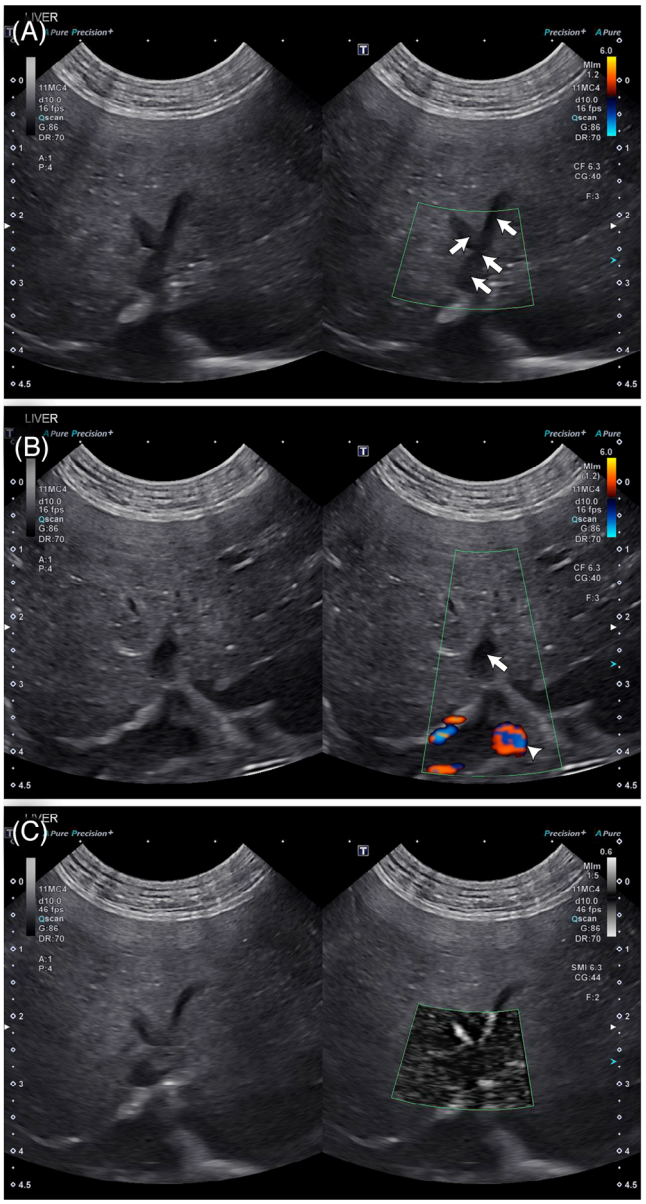

FIGURE 3 Hepatic ultrasonography demonstrating obstruction of blood flow at the site of LLT in a rabbit (transverse view). A, Absent blood flow within dilated vessels in the left liver adjacent to the stalk-like region on Doppler (arrow). B, Blood flow present on directional Power Doppler in vessels proximal to the stalk-like region (arrowhead) but not within dilated vessels within the left liver (arrow). C, Scant blood flow within the left liver on SMI synchronous with the patient's pulse. B-mode images acquired on Canon Aplio i700 using a microconvex electronic 7 MHz transducer. Patient in dorsal recumbency with abdominal fur clipped and warmed ultrasound coupling gel.

decreased peritoneal serosal detail.[2,3,5,10] Ultrasonography is considered the gold standard imaging modality for diagnosing LLT in rabbits.[2] Commonly described findings include hyperechoic falciform fat,[2,4–6] focal lobar/generalized hepatomegaly,[2,3,5,6] rounded hepatic lobar margins,[2,3,4,6] and decreased/mixed hepatic parenchymal echogenicity.[2–6,9,10] Disruption of the vasculature of the affected liver lobe can also be identified sonographically, with decreased blood flow on Colour Doppler.[2,3,5,6] Common additional findings include free peritoneal fluid,[2–6,9,10] gastrointestinal dilation,[2] and decreased gastrointestinal peristalsis.[1,9] CT is used for diagnosing LLT in humans and dogs,[2] but less frequently in rabbits due to cost, anesthetic risk, and the minimal advantages relative to AUS.[6] CT findings of LLT include hypoattenuating enlarged liver lobe(s), minimal to absent postcontrast enhancement of the affected lobe(s), and peritoneal effusion.[2,4] Hounsfield Unit torsed:normal liver ratio has been described as an objective measurement to diagnose LLT on CT, with 50% less contrast enhancement of the torsed liver lobe compared to normal liver on average.[2] Therefore, the radiographic and ultrasound findings were consistent with the published literature. CT was not performed since, with the clear AUS diagnosis, it was thought unlikely to provide further diagnostic value.

Hepatic lobectomy with appropriate perioperative supportive care has been widely described as the treatment of choice for LLT with good prognosis, infrequent postoperative complications, and a longer survival time versus medical management alone.[2–9] However, in several studies, there was no statistically significant difference in overall outcome between surgical and medical management of LLT in rabbits.[4,8] The most common complication of LLT is recurrent episodes of RGIS,[2,6] which is reported more frequently in medically managed cases than surgically.[6] The moderate postoperative pleural effusion, which occurred as a postoperative complication in this case, was presumed to reflect fluid overload, supported by the marked improvement following cessation of IVFT and thoracocentesis. An alternative differential is increased vascular permeability, for example, secondary to SIRS or thromboembolic disease.[4] Reported less common perioperative complications include fatal hemorrhage, cardiopulmonary arrest, hypotension, hypothermia, and arrhythmias.[8] No cases of acute gastric rupture have been recorded as a perioperative complication associated with hepatic lobectomy in rabbits.

This patient presented with nonspecific GI signs and was diagnosed with RGIS both pre- and postoperatively. Concurrent RGIS is frequently found in cases of LLT,[3,5] with cases of LLT reported to have presented as suspected gastrointestinal obstruction.[3] Stress, illness, and pain are common causes of decreased GI motility in rabbits.[3] Gastric contents have been previously described as a method of determining between GI stasis and obstruction in rabbits, with GI stasis typically resulting in GD with "doughy" or "desiccated" ingesta and obstruction typically resulting in GD due to fluid and gas.[1,3] Additional findings in RGIS include variable small intestinal and cecal gas dilation, with decreased colonic fecal content.[3] It has been hypothesized that in cases of caudate LLT, the enlarged lobe may obstruct the adjacent duodenum.[4] However, this is an unlikely etiology in left-sided LLT. The cause of death in this patient was gastric perforation with associated fulminant peritonitis. Gastric rupture has been described in rabbits with gastrointestinal mechanical obstruction,[3] with the ratio of gastric height+length:length of L1 to the coxofemoral joints demonstrated to be suggestive of obstruction.[1] Given the increased ratio in this case, gastric rupture secondary to functional gastrointestinal obstruction was considered a possible etiology. This patient also re-presented with

a history of a recent fight, so abdominal trauma was another possible etiology.

Another important necropsy finding was the multifocal hepatic infarcts. Multisystemic inflammatory changes and infarcts have been described on necropsy in cases of LLT in rabbits, indicating SIRS and DIC are potential sequelae of LLT.[4,8] Another proposed etiology for hepatic multilobar infarcts outside the torsed lobe is cardiogenic shock, resulting in impaired hepatic perfusion.[8]

This patient is the first case of a left medial LLT published in a rabbit, highlighting the requirement to scrutinize all portions of the liver in rabbits presenting with nonspecific signs of RGIS or suspected LLT, not just the previously described predilection sites (e.g., caudate lobe). Given the poor outcome in this case, further studies could investigate the etiology of left medial LLT and whether it is associated with a worse prognosis than more common locations (e.g., caudate lobe). The cause of death in this patient was acute gastric rupture and secondary fulminant peritonitis; further research into risk factors that could predispose to gastric rupture and potential etiologies is also warranted due to the association between GD and LLT. SMI has been described within the human literature as an adjunctive tool to support deficits in blood flow identified on other color Doppler modalities.[11] This case report demonstrates the utility of SMI as a novel modality to evaluate the persistence of arterial flow and venous drainage in LLT as rabbits.

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Category 2

(a) Drafting the article: Chapple
(b) Revising article for intellectual content: Chapple, Mikoni, Dutton, Brust

Category 3

(a) Final approval of the completed article: Chapple, Mikoni, Dutton, Brust

Category 4

(a) Agreement to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: Chapple, Mikoni, Dutton, Brust

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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REFERENCES

1. Debenha JJ, Brinchmann T, Sheen J, Vella D. Radiographic diagnosis of small intestinal obstruction in pet rabbits (*Oryctolagus cuniculus*): 63 cases. *J Small Anim Pract*. 2019; 60:691-696.
2. Dagget A, Loeber S, Le Roux AB Beaufrere H, Doss G. Computed tomography with Hounsfield unit assessment is useful in the diagnosis of liver lobe torsion in pet rabbits (*Oryctolagus cuniculus*). *Vet Radiol Ultrasound*. 2021; 62:210-217.
3. Oglesbee BL, Lord B. Gastrointestinal Diseases of Rabbits. *Ferrets, Rabbits, and Rodents*. Elsevier; 2020;174-187.
4. Sheen JC, Vella D, Hung L. Retrospective analysis of liver lobe torsion in pet rabbits: 40 cases (2016–2021). *Vet Rec*. 2022; 191. Available at: https://onlinelibrary.wiley.com/doi/10.1002/vetr.1971
5. Graham J, Basseches J. Liver lobe torsion in pet rabbits. *Vet Clin N Am Exotic Anim Pract*. 2014; 17:195-202.
6. Graham JE, Orcutt CJ, Casale SA, Ewing PJ, Basseches J. Liver lobe torsion in rabbits: 16 cases (2007 to 2012). *J Exot Pet Med*. 2014; 23:258-265.
7. Pignon C, Donnelly TM, Mayer J. Hepatic lobe torsion in a rabbit (*Oryctolagus cuniculus*). *Pratique Médicale et Chirurgicale de l'Animal de Compagnie*. 2013; 48:91-98.
8. Ozawa SM, Graham JE, Guzman DS-M, et al. Clinicopathological findings in and prognostic factors for domestic rabbits with liver lobe torsion: 82 cases (2010–2020). *JAVMA*. 2022; 1-9.
9. Varga M. Digestive disorders. *Textbook of Rabbit Medicine*. Elsevier; 2014;303-349.
10. Wenger S, Barrett EL, Pearson GR, Sayers I, Blakey C, Redrobe S. Liver lobe torsion in three adult rabbits. *J Small Anim Pract*. 2009; 50:301-305.
11. Artul S, Nseir W, Armaly Z, Soudack M. Superb microvascular imaging: added value and novel applications. *J Clin Imag Sci*. 2017; 7:45.

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