Liver lobe torsion in a dog

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Abstract — Abdominal radiographs of a dog presented for anorexia and vomiting revealed an ill-defined increase in opacity caudal to the stomach and caudal displacement of the small intestines. Ultrasonographs revealed an enlarged liver lobe with vascular thrombosis. Left medial liver lobe torsion was confirmed at postmortem.

Résumé — Torsion d’un lobe hépatique chez un chien. Les radiographies abdominales d’un chien présenté pour anorexie et vomissement montraient une zone mal définie avec opacité augmentée située caudalement à l’estomac et un déplacement caudal du petit intestin. L’examen échographique a révélé une augmentation de volume d’un lobe hépatique avec thrombose vasculaire. Une torsion du lobe hépatique médial gauche a été confirmée lors de la nécropsie.


A 7-year-old, spayed female golden retriever was presented to the Veterinary Teaching Hospital at the Western College of Veterinary Medicine with a 24-hour history of anorexia and 2 episodes of vomiting. Vaccinations were current and there was no history of acute trauma. According to the owners, the dog might have consumed garbage.

On physical examination, the dog was lethargic and had a temperature of 37.7˚C, heart rate of 160 beats/min, and respiratory rate of 40 breaths/min. The capillary refill time was 3 s and the mucus membranes were pale pink, perhaps indicating dehydration. The dog was tense and grunted when pressure was applied to the abdomen. The elevated heart rate was likely due to stress and the acute abdominal condition. Differential diagnoses considered for this dog were gastric or intestinal foreign body, gastric ulceration, pancreatitis, and splenic hemangiosarcoma.

A complete blood (cell) count (CBC), serum biochemical analyses, and urine analysis following cystocentesis were conducted. Radiographs of the abdomen and thorax revealed a generalized loss of serosa detail in the mid- to cranioventral abdominal area. The small intestines were displaced caudally and an ill-defined increase in opacity was observed in the region between the stomach and the displaced small intestines, suggesting a cranial abdominal mass and peritoneal effusion (Figure 1). Thoracic radiographs revealed multiple sites of thoracolumbar spondylosis deformsans, which is common in older dogs (1).

Abdominal ultrasonographs showed a moderate volume of hypoechoicogenic peritoneal effusion, predominantly in the cranial part of the abdomen, and separating the liver lobes. One liver lobe was abnormally rounded, markedly enlarged, extended...
caudal beyond the costal arch, and was creating the mass effect in the cranial part of the abdomen. The large blood vessels within this liver lobe appeared thrombosed, with echogenic blood clots, suggestive of liver lobe torsion, a hepatic infarct, or both (Figure 2).

Ultrasound guided abdominocentesis yielded hemorrhagic fluid. The fluid contained nucleated cells (3.5 × 10^3 cells/L), red blood cells (5.05 × 10^3 cells/L), total solids (51 g/L), and had a specific gravity of 1.033. On microscopic examination of a direct smear of the fluid, an abundance of erythrocytes and few leukocytes were seen. On microscopic examination of a lysed cytocentrifuge preparation, a population of nondegenerate neutrophils predominated, with fewer small lymphocytes, monocytes, eosinophils, and the occasional macrophage. The cytological interpretation was hemorrhagic effusion. The absence of erythrophagia or hemoglobin degradation products (hem siderin or hemotoidin), or both, indicated that the hemorrhage was acute.

The CBC indicated the presence of a mild to moderate leukocytosis (20.2 × 10^3 cells/L; reference range, 4.80 to 13.9 × 10^3 cells/L), moderate neutrophilia (18.180 × 10^3 cells/L; reference range, 3.0 to 10 × 10^3 cells/L), mild monocytosis (1.414 × 10^3 cells/L; reference range, 0.08 to 1.0 × 10^3 cells/L), and mild lymphopenia (0.606 × 10^3 cells/L; reference range, 1.2 to 5.0 × 10^3 cells/L). These results were interpreted as a stress response. The thrombocyte count, erythrocyte parameters, and cell morphology were all within normal reference ranges. The serum biochemical analysis revealed a mild increase in alanine aminotransferase (ALT) (226 U/L; reference range, 19 to 59 U/L), glutamate dehydrogenase (GLDH) (19 U/L; reference range, 0 to 7 U/L), and cholesterol (8.13 mmol/L; reference range, 2.70 to 5.94 mmol/L). Serum sorbitol dehydrogenase (SDH) was markedly increased (180.0 U/L; reference range, 0.0 to 4.0 U/L).

The owners were advised that surgical intervention was required immediately; however, they opted for euthanasia.

On postmortem examination, approximately 1 L of unclotted blood was within the peritoneal cavity. The left medial liver lobe was enlarged, severely congested, and friable. The lobe was twisted counterclockwise on its axis and there was a tear at its base with associated hemorrhage. All other liver lobes appeared normal grossly. There was mild thickening of the left ventricle and left atrio-ventricular valve. The morphological diagnoses were as follows: 1) left medial liver lobe torsion — acute, severe, and hemorrhagic; 2) he-moabdomen — acute and severe; 3) left ventricular hypertrophy — mild and chronic; and 4) left atrio-ventricular valvular endocardiosis — mild and chronic. Mild cardiac disease is not uncommon in older dogs and is associated with degeneration of valvular collagen (2).

Histopathologic examination of the left medial liver lobe showed diffuse congestion; severely dilated, congested sinusoids; and a thinning of hepatic cords containing hepatocytes, indicating pressure atrophy. The central veins and portal areas were severely congested with dilated vessels. The remaining liver lobes were also congested, especially at the periacinar and periportal areas, and the sinusoids were dilated moderately. There were also multifocal areas of vacuolation of hepatocytes, which was marked at the periacinar areas.

Hepatic lobe torsion is uncommon in veterinary medicine, but it has been reported in dogs, cats, rabbits, pigs, and horses (3–9). The presenting clinical signs are often nonspecific and can include lethargy, vomiting, inappetence, polyuria/polydipsia, colic, abdominal pain, and abdominal distention (4). Swann and Brown (4) reported nonspecific hematologic and chemical parameters in liver lobe torsions; however, in this case, blood analysis revealed hepatic involvement. The elevation in ALT without an increase in creatinine kinase (CK) and the elevations in SDH and GLDH indicate hepatocellular damage (10). An elevation in cholesterol can occur with hepatic disease; cellular swelling often accompanies hepatocellular damage, which constricts bile canaliculi and induces cholesterol (10). A urinalysis showed an elevation in ketones, which indicated increased lipolysis. Ketone production occurs in times of negative energy balance (deficiency in carbohydrate metabolism) and ketonuria is usually detected before ketonemia, due to the low renal threshold for ketones (10). The absence of ketonemia and the presence of ketonuria in this case could be explained by the anorexia (10).

Torsion of the left lateral liver lobe seems to be more common, as it represents nearly 50% of clinical cases reported (4). Spatial support is provided to the liver by the left and right triangular ligaments, the left and right coronary ligaments, and the falciform ligament (4). The left and right triangular ligaments provide support of the left and right lateral liver lobes to the muscular portion of the diaphragm, while the left and right coronary ligaments provide attachment of these lobes to the tendinous portion of the diaphragm (4). Damage or absence of any of these support mechanisms can predispose an animal to liver lobe torsion. Traumatic disruption of the left triangular ligament in its association with left lateral liver lobe torsion has been described in both the horse and dog (6,9). It has also been suggested that chronic bouts of gastric dilatation may be a predisposing factor to hepatic lobe torsion as the ligamentous support structures may be compromised (5). Congenital defects could also play a role in predisposition; however, most often the cause remains unknown (3).

This dog had been hit by a vehicle about 5 y previously. No complications were noted at that time, and the dog was simply monitored overnight in a veterinary clinic. This traumatic incident possibly weakened the ligamentous support structures of the left medial lobe, predisposing it to torsion. The sequella of a hepatic lobe torsion is venous infarction, increased hydrostatic pressure, effusion, arterial and venous thrombosis, and eventually necrosis (4), making prompt diagnosis and surgical intervention critical to patient survival. Surgical removal or repositioning of the affected liver lobe can result in a successful outcome (4). If treatment is instituted early, the prognosis for hepatic lobe torsion is good. Histopathological examination of resected tissues is important as the venous congestion associated with hepatic lobe torsion can have a gross appearance that mimics neoplasia (4).
References