

## CASE REPORT

## Surgical removal of a localised vascular hepatic hamartoma in a dog

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A 2-year-old male Labrador Retriever was presented for sudden abdominal distension. Abdominal ultrasonography revealed abundant abdominal fluid and a hepatic mass, which was removed by total lobectomy. Histologic evaluation of the mass supported a vascular hepatic hamartoma. Vascular hamartomas are rare malformations in animals and only two cases have been reported in the dog. This is the first recorded case of a canine vascular hepatic hamartoma treated surgically and having a successful long-term outcome.

**Keywords** ascites; dogs; hepatic vascular hamartoma

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A hamartoma is an excessive, focal, overgrowth of mature normal cells and tissue native to the organ or site in which it occurs.<sup>1</sup> Hamartomas are present at birth and their growth ceases with the maturity of the affected organ. The components of the hamartoma are mature, but arranged in a disorganised fashion.<sup>2,3</sup> They can develop in any organ or tissue and are regarded by some authors as a form of tissue proliferation midway between malformation and true neoplasia.<sup>4</sup> Hamartomas are seldom reported in domestic animals and most are vascular hamartomas.<sup>3</sup>

## Case report

A 2-year-old, intact male Labrador Retriever was admitted to the Small Animal Surgery Section of the University of Milan (Italy) with a history of abdominal enlargement for 1 week. The initial evaluation 2 days previously by the referring veterinarian found the dog in good general condition, but with a distended non-painful abdomen. A complete blood count showed a mildly increased haematocrit (58%, reference range 37–55%), macrocytic (mean corpuscular volume 77 fL, reference range 60–72 fL) and hypochromic (mean corpuscular haemoglobin concentration 27.29 g/dL, reference range 31–37 g/dL) red cells, microcytic platelets (mean platelet volume 8.80 fL, reference range 10.6–16 fL) and mild monocytosis (13%, reference range 3–10%). Serum biochemical profile showed a mildly increased alanine aminotransferase (121 IU/L, reference range 10–90 IU/L), low total plasma proteins (4.8 g/dL, reference range 6–7.5 g/dL) and hypoalbuminaemia (2.65 g/dL, reference range 3.1–3.5 g/dL). Serum electrophoresis revealed an increased alpha-2 protein fraction (16.20%, reference range 5.3–8.5%) and a reduced gamma fraction (4.8%, reference range 12–13%).

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Fasting and postprandial (2 h) bile acids were normal, as was the coagulation profile. Urine examination and faecal flotation were unremarkable. Right lateral abdominal radiography revealed ventral distension and diffuse opacity of the abdominal cavity with some gas-filled intestinal loops (Figure 1). Abdominal ultrasound showed an inhomogeneous area in the right hepatic lobe. The dog was referred to the University of Milan for further assessment, without prescribing any treatment.

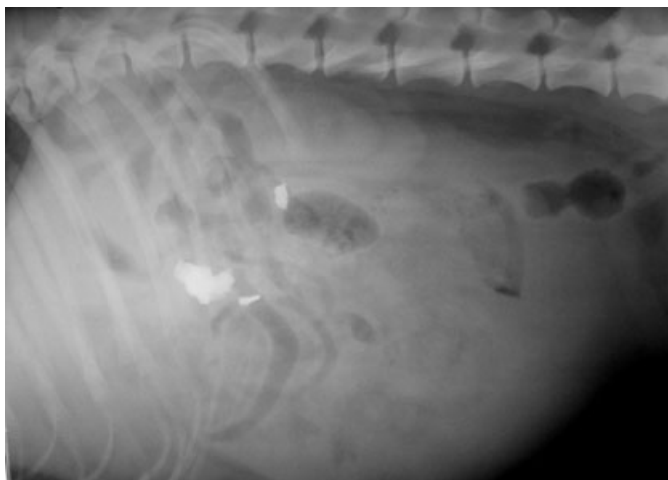
Examination on admission confirmed the referring veterinarian's physical findings.

Abdominal percussion was positive for intra-abdominal fluid. Repeat abdominal ultrasound showed abundant peritoneal effusion with clear signs of parietal peritoneal reactivity. An enlarged hyperechoic homogeneous liver was seen; the gallbladder and biliary tract were normal. A mass located in the right hepatic lobe was 34 × 42 mm in size with a complex mass aspect; no vascularisation was visible within the mass. The underlying hepatic parenchyma appeared inhomogeneous, with alternating hypoechoic and hyperechoic areas. Ultrasound differential diagnosis included a hepatic mass or haematoma associated with primary or reactive hepatopathy.

Paracentesis yielded serosanguineous, turbid fluid (≈2 L), with a specific gravity of 1.024 and protein content of 5 g/100 mL. Cytologic evaluation of the fluid revealed low cellularity with a mixed population characterised by degenerate neutrophils, small lymphocytes, mesothelial cells and reactive macrophages, which was consistent with a modified aseptic reactive transudate.

An exploratory laparotomy was performed. The dog was premedicated with atropine (0.03 mg/kg IM) and buprenorphine (10 µg/kg IM) before induction with propofol (6 mg/kg IV); anaesthesia was maintained with isoflurane in oxygen. Intravenous cephalosporin (20 mg/kg) was given before laparotomy. After midline incision and aspiration of peritoneal fluid, an enlarged thickened, fibrotic, red-brown right medial hepatic lobe could be seen; a subcapsular, 4 × 5 cm, firm, smooth, rounded mass was located on the lobe's diaphragmatic side. A total lobectomy was performed. The lobe parenchyma was bluntly dissected from the caudal vena cava using right-angle forceps. The vascular and biliary pedicle of the right lobe was isolated bluntly and doubly ligated close to the hilus with 2-0 polydioxanone. After placing Rochester-Carmalt haemostatic forceps parallel and distal to the ligatures, the right lobe was then excised with Metzenbaum scissors. After checking for haemorrhage, a segment of omentum was placed over the remaining liver stump and gently wrapped around it (omentalisation). The linea alba was sutured with 0 polyglactin 910 and the skin with 2-0 nylon.

Ringer's Lactate (2.5 mL/kg/h IV for 8 h) was administered postoperatively and buprenorphine (10 µg/kg IV) was repeated after 6 h;



**Figure 1.** Lateral abdominal radiography showing loss of serosal detail, diffuse opacity and gas-filled intestinal loops.

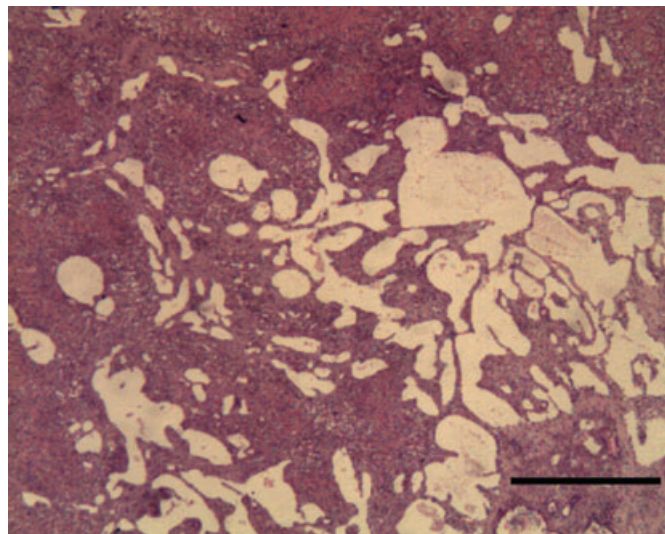
the patient was discharged on the same day with amoxicillin/clavulanic acid (12.5 mg/kg PO twice daily) for 7 days. Oral nutrition was reintroduced 12 h after surgery.

On pathologic examination, the cavitated gross aspect of the incised subcapsular hepatic mass was consistent with a large haematoma compressing the underlying parenchyma. The formalin-fixed paraffin-embedded hepatic lobe was cut into 3-micron slices and stained with haematoxylin-eosin. Microscopic examination showed that the parenchyma contained abundant connective tissue stroma of bundles of spindle mesenchymal cells, with numerous irregularly proliferating arteries and veins lined with a layer of morphologically normal endothelial cells; the lumen of the vessels contained erythrocytes. Lymph vessels and biliary ducts were also present (Figure 2a, b). The intima and elastic tunica of the vascular structures appeared thickened and mildly fibrotic. Completely degenerate hepatocytes with small, hyperchromic peripheral nuclei and a vacuolar cytoplasm were seen. Microscopically, the subcapsular mass consisted of organised haematoma with clot. Based on the microscopic findings, a vascular hepatic hamartoma was diagnosed.

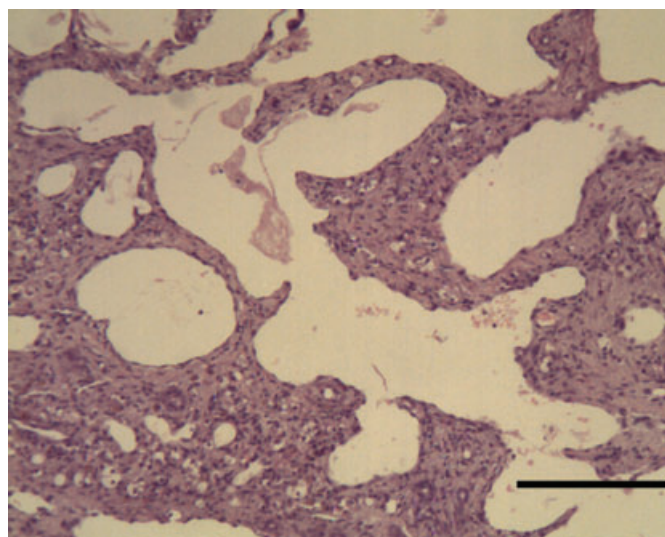
The patient was in good general condition at 1 week after surgery. No abnormalities were noted on follow-up haematology, serum biochemical profile and abdominal ultrasound performed 6 and 12 months after lobectomy. The patient was reported healthy by the owners during a follow-up phone call 24 months after surgery.

### Discussion

Hamartomas are seldom reported in domestic animals and most are vascular.<sup>3</sup> They can arise in any organ and in humans affect primarily the spleen, liver, kidney, lung and pancreas. In the liver, the most commonly reported type is the mesenchymal hamartoma, which mostly affects children less than 2 years of age; there are only sporadic reports in adults.<sup>5</sup> In dogs, most of the reported vascular hamartomas affected the brain<sup>6</sup> and only two cases of liver involvement have been reported.<sup>7,8</sup>



**Figure 2a.** Photograph of histological slide of the liver parenchyma, showing diffuse proliferation of irregular and tortuous vessels that are lined with endothelial cells that have regular euchromatic nuclei. Occasional red cells are seen in the vessel lumens. Hepatic macrovesicular lipidosis and minimal interstitial fibrosis is also present. (H&E,  $\times 10$ ; bar = 50  $\mu\text{m}$ ).



**Figure 2b.** Higher magnification of (a) ( $\times 40$ ; bar = 20  $\mu\text{m}$ ).

Vascular hamartomas are characterised by disorganised, excessive proliferation of normal vascular tissue and blood vessels. Although uncommon, they have been described in various species and can occur in several organs. A few cases of vascular hepatic, gingival, ovarian and testicular hamartomas are described in cattle.<sup>9</sup> A vascular hamartoma of the hindlimb is reported in a goat.<sup>9</sup> Three subcutaneous carpal and one ovarian vascular hamartoma are described in the horse.<sup>9,10</sup>

In dogs, vascular hamartomas have been reported in nine cases: hepatic ( $n = 2$ ),<sup>7,8</sup> pulmonary ( $n = 1$ ),<sup>11</sup> cerebral ( $n = 5$ ),<sup>6</sup> and associated with the flexor muscles of the carpus ( $n = 1$ ).<sup>12</sup> One gingival vascular hamartoma is described in a cat.<sup>13</sup>

In the only two cases of hepatic vascular hamartoma reported in the dog, one was confined to the right central lobe and the other affected the whole liver. Both cases occurred in young individuals that presented with marked abdominal enlargement. Both dogs died from surgical complications.

In the present report the differential diagnosis, based on the gross and histopathologic findings, included peliosis hepatitis, haemangioma and vascular hamartoma.

Peliosis hepatitis is a rare vasculoproliferative disorder characterised by the presence of cystic, blood-filled spaces in all lobes of the liver. On microscopic examination the cysts are partially lined with a single layer of normal endothelium. In dogs, peliosis hepatitis has been reported in association with *Bartonella henselae* infection.<sup>14</sup> The liver of the dog described here had no gross evidence of blood-filled cysts within the parenchyma and the only grossly visible lesion was a large subcapsular haematoma localised in the right medial lobe. On the basis of these macroscopic and microscopic findings peliosis hepatitis was ruled out.

The morphologic differentiation between haemangioma and vascular hamartoma can be challenging and has generated some degree of confusion in the terminology. Unlike haemangioma, in which the vessel walls consist of endothelial cells and connective tissue, but lack muscle tissue, the blood vessels in hamartoma have a normal structure.<sup>15</sup> In the present case, the evidence of well-differentiated, irregularly proliferating vascular structures (arteries, veins, lymph vessels) in the affected hepatic lobe supported a diagnosis of vascular hamartoma. In the other two cases of canine hepatic vascular hamartoma, most of the proliferating vascular structures were veins<sup>7</sup> or were characterised only as 'large blood channels' lined with mature endothelial cells.<sup>8</sup>

The dog here described was slightly older (2 years) than the other two dogs reported in the literature (2 and 15 months, respectively; in the latter dog the clinical signs had started 5 months previously). The hepatic involvement being limited to the right medial lobe and not affecting general liver function may have delayed the occurrence of clinical signs. In all three cases the hepatic lesion caused peritoneal effusion. On admission, the only abnormalities consistent with hepatic disease were mildly increased alanine aminotransferase associated with a moderate reduction in total plasma protein and hypoalbuminaemia. Normal values for bile acids indicated preserved liver function. A low total plasma protein content (4.5 g/dL), probably related to chronic haemorrhage, was also reported in one of the two dogs with liver hamartoma described in the literature.

The abundant ascitic fluid was consistent with portal hypertension, which was probably caused by arteriovenous shunting between the hepatic artery and vein within the affected lobe.<sup>16</sup>

In this dog the only organ affected was the liver and the lesion was limited to the right medial lobe, which enabled an uncomplicated total

lobectomy to be performed, followed by a successful long-term outcome. In the other two dogs described, the multiple lobar involvement or the presence of adhesions ruled out surgical treatment and complications during surgery caused the death of the patient in both cases.

The positive long-term follow-up (24 months postoperatively) of the dog described here confirms the benign nature of the lesion in the present case.

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