Caval foramen hernia in a dog: Preoperative diagnosis and surgical treatment

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ABSTRACT. A 13-year-old, 5.6-kg castrated-male Maltese was presented for reverse sneezing. A dome-shaped round mass abutting diaphragm was incidentally found ventral to caudal vena cava, which had the same echogenicity and density as that of the liver during ultrasonography and computed tomography, showing isoattenuation with a contrast study. Vascular distribution was identified throughout the mass. A caval foramen hernia (CFH) was diagnosed tentatively, followed by a herniorrhaphy and splenectomy of the chronically congested spleen. The patient had been doing well for 5-month postoperative but died because of aspiration pneumonia. CFH is an extremely rare condition, requiring surgery due to compression of the vena cava. It should be considered as a differential diagnosis when intrathoracic, mass-like lesions are identified near the diaphragm.

KEY WORDS: caval compression, caval foramen hernia, dog, intrathoracic mass

Diaphragmatic hernias (DHs) present with intrathoracic soft tissue mass-like opacity in the vicinity of the diaphragm and are often misdiagnosed as pulmonary, mediastinal, or cardiac lesions such as abscesses, cysts, granulomas, consolidations, tumors, or diaphragmatic masses or eventration (DE) [8, 9, 13]. DHs can be congenital and acquired, and traumatic causes account for 77–85%, while congenital DHs occupy only 5–10% [8, 14]. Due to its anatomical proximity, the liver is the most frequently herniated organ, identified in 88% of traumatic DHs, followed by the small intestine, stomach, spleen, and omentum [4, 8]. Clinical signs are usually non-pathognomonic, depending on which organ is herniated and whether it is undergoing inflammation, incarceration, obstruction, or strangulation [8].

There are three apertures in the diaphragm: aortic hiatus, esophageal hiatus, and caval foramen, and hiatal hernias have been reported abundantly in veterinary medicine [9, 14]. However, a caval foramen hernia (CFH), a herniation of visceral organs through a caval foramen which is a pathway of caudal vena cava (CVC), is an extremely rare condition even in human medicine with poorly understood etiology [2, 9, 13, 14]. It has not been investigated whether a CFH is congenital or acquired, and it can be challenging to make a definitive preoperative diagnosis. A CFH was first reported in human medicine masquerading as a thoracic mass in 2006 [13]. CFHs can be found incidentally but are more often misdiagnosed due to their sparsity [9].

A 13-year-old, castrated-male Maltese weighing 5.6-kg was presented for screening with reverse sneezing. The patient was in good body condition (BCS: 5/9) without traumatic history. There was no specific finding in CBC, serum chemistry, electrolytes, and urinalysis, other than mild leukocytosis and increases in liver enzyme panels: WBC: 21.01 × 10^9/l (reference range (RR): 5.2–13.9), alanine transaminase (ALT): 172 U/l (RR: 19–70), alkaline phosphatase (ALKP): 259 U/l (RR: 15–127), and gamma-glutamyl transferase (GGT): 16 U/l (RR: 0–6).

Plain thoracic radiography revealed mild tracheal collapse and a broad-based, round dome-shaped solitary mass with soft tissue opacity caudal to the carina in the right hemithorax abutting diaphragm (Fig. 1A and 1B). The intrathoracic lesion showed the same echogenicity as liver parenchyma during ultrasonography (Fig. 2). A hyperechoic septum was identified between the lesion and the liver, and the CVC was running dorsally to the mass. On suspicion of DH, ectopic liver (EL), or DE, a triple-phase computed tomography (CT) was performed (Fig. 3). An intrathoracic mass (6.5 × 3.9 × 3.5 cm) was identified ventral to the CVC, connected to the liver, showing isoattenuation of the liver before and after contrast. There was a hypodense line separating the mass from the liver, which was suspected to be a diaphragm. The mass was considered displaced liver tissue into the thoracic cavity along with the CVC through a caval foramen. The herniation deviated the CVC dorsally, resulting in caval compression. Contrast-enhanced running vessels of the hepatic artery, portal vein, and hepatic vein were clearly identified throughout the herniated liver in all three phases. There was a hepatic venous dilation caudal to the caval compression on the delayed phase. Considering these findings, it was diagnosed as a CFH of the right lateral liver lobe.
A herniorrhaphy was performed under general anesthesia of isoflurane inhalation combined with a continuous rate infusion of remifentanil (Fig. 4). D-dimer and coagulation panels were within the normal range. Remifentanil was maintained (6–11 µg/kg/hr) after initial loading (3 µg/kg), and midazolam (0.1 mg/kg) and propofol (4 mg/kg) were administered intravenously for premedication and induction. Ampicillin (20 mg/kg) was also injected. When a midline celiotomy was performed, intra-abdominal vena cava and renal veins were plump. Only the right medial liver lobe was observed beneath the right-side of the diaphragm (Fig. 4A and 4B). When the liver lobes and gall bladder were retracted caudomedially, there was a passage on the tendinous part of the diaphragm (2 cm in width approximately), through which liver tissue protruded into the thoracic cavity (Fig. 4C and 4D). The herniation was irreducible, and a right paracostal incision was added. There was a gap between the ventral rim of hernia ring and herniation, through which a Senn retractor was introduced. When the ring was opened with stay sutures (Fig. 4E), a thin membranous tissue emerged between the diaphragm and herniated liver, isolating the lesion from the thoracic cavity (Fig. 4F-white arrow). The membrane was thoroughly and circumferentially covering the herniated liver like a hernia sac (Fig. 4G-white arrow). It was tough, semitransparent, and vascularized, which was difficult to dissect from the lesion. As the mass was large and stuck inside the thoracic cavity, the diaphragmatic incision was elongated. There was a dense adhesion on the dorsal and medial side. The mass was carefully released with blunt/sharp dissections after making a circular incision of the diaphragm along the rim of hernia ring (Fig. 4G-white dashed line); lung lobes and pericardium were not involved. After complete dissection, the herniated liver was flipped over and relocated into the abdominal cavity. It was swollen, with a grossly blunt margin, but had no other specific abnormalities except for a partially uneven, dark red surface. It was considered the right lateral lobe or hilar root portion between the right lateral and medial lobes. The membranous tissue was also identified as being attached continuously to the ventral surface of the CVC at the level of hernia (Fig. 4H-white arrow). A chest tube was placed after lavage, and the diaphragmatic defect was closed using a simple continuous suture from a dorsal to the ventral direction (Fig. 4I). The caval foramen was reconstructed with its opening so as not to compromise the CVC. The discolored part of the herniated liver restored with time. During abdominal exploration, the spleen was mildly firm, swollen, showing a mottled irregular surface of dark red to violet, and was removed (Fig. 4K).

The patient recovered uneventfully and was maintained with a fentanyl patch (12 mcg/hr) and 2-week oral medication; ampicillin (20 mg/kg, tid), metronidazole (10 mg/kg, bid), famotidine (0.5 mg/kg, bid), and liver protectants (ursodeoxycholic acid, silymarin). Remifentanil was tapered and weaned off on the postoperative day (POD) 2. The patient showed good appetite, activity,
and was discharged on POD 3. Histopathological diagnosis of severe chronic splenic congestion was made.

The liver enzymes were elevated on POD 1 (ALT: 508 U/l, ALKP: 297 U/l, GGT: 8 U/l) and POD 3 (ALT: 161 U/l, ALKP: 322 U/l, GGT: 7 U/l) but restored on POD 62 recheck (ALT: 62 U/l, ALKP: 102 U/l, GGT: 3 U/l). A diaphragmatic silhouette was normally curved on thoracic radiography since POD 1 (Fig. 1C and 1D: POD 62) and ultrasonography on POD 62 showed normal echogenicity in the liver parenchyma, and CVC flow velocity was 33.5 cm/sec (RR: 20–35 cm/sec). The patient had been doing well for 5 months postoperative but died because of a non-herniorrhaphy-associated cause on POD 153.

This is a case of an intrathoracic mass-like lesion showing echogenicity of the liver parenchyma during ultrasonography. A triple-phase CT angiography determined a CFH preoperatively by clarifying its anatomical relation to abutting structures (diaphragm, CVC, and abdominal liver) and its vascularity extending into the herniated liver. An intrathoracic liver is probably associated with a DH, CFH, or intrathoracic EL [3, 5, 6, 9, 11, 13, 14]. Occasionally, it can be difficult to differentiate them from each other preoperatively [3, 5], one CFH was diagnosed intraoperatively [3].

There were only two reports of canine CFH, including eight small breed dogs [9, 14]. Only two patients underwent herniorrhaphy (one thoracotomy, no information for the other). This is the first detailed report of surgery in the English literature. In the present case, herniorrhaphy was accomplished through a midline celiotomy with a paracostal incision and the enlarging of the caval foramen. It allowed for fastidious inspection of the herniation, including the caval hernia ring constricting the liver, the membranous lining enclosing the lesion, its anatomical relation to other liver lobes, and it provided easy anatomical reconstruction. A thorough exploration of the abdominal viscera was also possible, leading to additional splenectomy. A caudal median sternotomy could have been performed instead of paracostal elongation. Most cases of CFH and intrathoracic EL have been approached using thoracotomy in both humans and dogs [3, 4, 6, 7, 14]. It allows for inspection of the convex pleural surfaces and good visual exposure of herniated organs and diaphragmatic tears, which permits easy breakdown of adhesion [3, 8]. However, in some patients, it was because of misdiagnosis as pulmonary malignancy [3, 4, 7].

Lesions in CFHs are connected to the abdominal liver through the caval foramen, occupying the space ventral to CVC with various degrees of CVC compression [3, 9, 13, 14]. In this patient, a caval compression resulted in hepatic venous dilation caudal to the compression, as in previous reports [2, 9, 13, 14]. Despite no evidence for severe liver damage and ascites at the time of diagnosis, surgical repair was attempted because spontaneous recovery could not be expected, and it could induce Budd-Chiari-like syndrome if left untreated. Although preoperative portal hypertension was not evaluated by measuring the direct portal pressure or flow velocity, the swollen herniated liver, plump intra-abdominal CVC/renal veins, and chronically congested spleen in histopathology supported abnormal blood flow and hemodynamic alterations secondary to the CVC compression. Moreover, after herniorrhaphy, the immediate restoration of GGT implies a possibility of biliary tract involvement that the CFH could have been disturbing bile flow even though a preoperative CT showed no abnormal biliary findings, such as cystic duct hernia or ductal dilation [9]. There was a human case of asymptomatic CFH of perihepatic fat with a caval obstruction that led to a pulmonary embolism [2].

The herniated tissue was not resected, just repositioned, and the patient recovered well without clinical signs, suggesting reperfusion syndrome or pulmonary re-expansion syndrome. A biopsy was not obtained because there were no malignant changes.
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or suspicious findings in preoperative CT angiography and gross assessments during surgery. A herniated liver is commonly congested or swollen and can induce hepatic venous stasis, hepatic necrosis, biliary tract obstruction, and jaundice, but it usually remains viable, which leads to a lobectomy that is rarely necessary [8]. According to previous reports of CFH or intrathoracic EL, most cases revealed normal hepatic tissue without malignancy in histopathology [1, 4–7, 11, 12, 14]. As ischemia-, necrosis-, or incarceration-associated proliferation of endogenous Clostridia and toxin release into the bloodstream after repositioning might be concerned, prophylactic and therapeutic antibiotics such as penicillin can be administered [8].

Human patients and one dog with CFHs in five reports underwent CT scans on suspicion of pulmonary nodules [9, 13, 14], cardiac mass [2], tumor of the mediastinum or pleural cavity [3], DHs [9, 14], and incidental findings [9]. The herniated lesion was the liver in all cases except the one that was perihepatic fat [2]. As an initial differential diagnosis, intrathoracic ELs are rare. However, they have been reported more than CFHs in both human and veterinary medicine, although the majority of ELs are found in the abdominal cavity near the liver (gallbladder, spleen, pancreas, omentum, adrenal gland) [1, 3, 10–12]. Intrathoracic ELs can be congenitally associated with a defect during embryological development, secondary acquisition due to trauma or previous repair of DH, or hematogenous dissemination of the native liver during transplantation surgery [1, 3, 6, 12]. It occurs with or without diaphragmatic defects, and it may or may not have a connecting pedicle to the liver [1, 3, 6]. DE occurs when diaphragmatic muscles are malpositioned with subtotal diaphragmatic tears but intact serosa of the thoracic surface of the diaphragm [9]. It does not show a pericaval pattern, and there is no actual prolapse of the abdominal organ [8, 9].

No criteria for differentiation between CFH and intrathoracic EL have been established. One could categorize CFHs into

![Fig. 4. Surgical views during herniorrhaphy (A–K) and preoperative 3-D reconstruction image (L: dorso-ventral view) of a CFH in a dog. RML: right medial lobe, GB: gall bladder, QL: quadrate lobe, LML: left medial lobe, Dt: tendinous part of diaphragm, black arrowheads: ventral rim of hernia ring, Dm: muscular part of diaphragm, white arrow: membranous tissue enveloping the herniated liver, *: herniated liver, white dashed line: rim of hernia ring converting from diaphragm to membranous sac, white arrowheads: constricted neck of hernia, L: lung, black arrow: caudal vena cava, SP: spleen.](image-url)
intrathoracic ELs based on only the gross finding of liver tissue in the thoracic cavity. However, CFHs should be distinguished from intrathoracic ELs because of the following reason: 1) a CFH has apparent parenchymal continuity to the liver with anatomically functional running vessels, 2) it uses a caval foramen as a definitive passage for herniation, and 3) the consequence of CVC compression in CFHs can be fatal without surgery. Intrathoracic ELs have been shown to adhere to the CVC or the diaphragm but are rarely associated with CVC compression [6]. Most ELs have compromised arterial supply and venous/biliary drainage, which might predispose an EL to malignant transformation [11, 12]. However, some authors suggest non-imperative surgery because benign diagnoses have recently become more common owing to an increase in incidentally found ELs [3, 12].

As the herniated liver tissue in a CFH is part of the continued liver but just displaced, 4) it always appears in contact with the diaphragm, while intrathoracic ELs could be found elsewhere in the thoracic cavity as a mediastinal, pleural, intrapericardial, or intrapulmonary mass [1, 7, 10, 12]. In this case, hepatic tissue was herniated through a caval foramen ventral to the CVC, maintaining its definitive running vessels inside. It was connected to the abdominal liver, which decisively differentiates it from two other differential diagnoses. Moreover, the membrane covering the herniated liver, continuous to the rim of the hernia ring, looked similar to a hernia sac in a true hernia. There was a case of trans-diaphragmatic liver herniation covered by a smooth layer of the peritoneum [5], and another hepatic CFH also had a hernia sac [3].

In conclusion, CFHs should be included in the differential diagnosis of dome-shaped caudal thoracic mass lesions abutting diaphragm. CT angiography is a useful tool for the preoperative diagnosis of CFHs. The severity of CVC compression, hepatic venous dilation, or biliary tract involvement should be considered in surgical planning. Further data are needed to investigate the etiology and pathophysiology of CFHs.

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