Caval foramen hernia in a cat

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Abstract
Case summary A 3-year-old neutered female domestic shorthair cat presented for a 2-week history of hyporexia, lethargy and weight loss. Aspartate aminotransferase, alanine aminotransferase and cholesterol were mildly elevated. Thoracic radiographs identified a lobulated soft tissue opacity in the caudal thorax to the right of midline, with the border effacing the caudal vena cava and broad-based towards the diaphragm. The broad base was suggestive of diaphragmatic hernia, with the other radiographic features and location suggestive of caval foramen hernia. Ultrasound confirmed diaphragmatic hernia with liver herniation. CT showed the herniation of multiple liver lobes and the gallbladder through a defect at the caval foramen. Herniorrhaphy was performed via ventral midline coeliotomy. Following this procedure, the cat’s clinical signs resolved and its weight has been regained.

Relevance and novel information To our knowledge, this is the first report of successful caval foramen herniorrhaphy in a cat. Caval foramen hernia is a type of congenital diaphragmatic hernia. The authors suggest that its embryopathology involves defective septum transversum development. The case was detected during the standard diagnostic investigation of non-specific clinical signs. Its radiographic findings may easily be mistaken for a pulmonary mass. Although not seen in our case, caval foramen hernia is commonly associated with caudal vena cava obstruction, which can potentially result in Budd–Chiari-like syndrome.

Keywords: Congenital hernia; diaphragmatic defect; caval foramen; computed tomography

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Case description
A 3-year-old neutered female domestic shorthair cat weighing 4.33 kg presented for a 2-week history of hyporexia, lethargy and weight loss. It had also lost 130 g of body weight in the past 9 days. There was an incidental chronic history of intermittent feline acne. It had been housed indoors since being in the owner’s possession from 2 months of age. Other than scabs under the rostral chin and left ear base, clinical examination was unremarkable. Complete blood count, serum biochemistry, urinalysis, and feline leukaemia virus and feline immunodeficiency virus serology were performed. Aspartate aminotransferase (77 IU/l; reference interval [RI] 2–62 IU/l), alanine aminotransferase (214 IU/l; RI 19–100 IU/l) and cholesterol (7.5 mmol/l; RI 2.2–5.5 mmol/l) were mildly increased. Urinalysis showed proteinuria and bacteriuria, which was considered to be clinically insignificant. All other results were unremarkable.

Thoracic radiographs and abdominal ultrasound were requested for further investigation of the non-specific clinical findings.

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(unlikely) pulmonary mass or caudal mediastinal mass (including granuloma, neoplasia).

Abdominal ultrasound (EPIQ 5G [Philips Medical Systems] using microconvex 8–5 MHz and linear 12–5 MHz transducers) showed a loss of continuity in the mid-diaphragmatic interface with herniation of hepatic parenchyma (Figure 2). Ultrasound was otherwise unremarkable. CT was then performed under general anaesthesia for surgical planning.

The cat was premedicated with 0.9 mg butorphanol and 21.5 µg medetomidine intravenously (IV), induced with 4 mg alfaxalone IV, intubated with a 4.5 mm endotracheal tube and maintained on isoflurane and oxygen. Multiphase contrast-enhanced CT images (Brilliance 16; Philips Medical Systems) of the thorax and abdomen were acquired in the transverse plane in sternal recumbency. Intravenous contrast (2 ml/kg Omnipaque 350 mg/ml [General Electric Healthcare]) was administered via the left cephalic vein using a
power injector (EmpowerCTA; E-Z-EM). CT images were viewed using smoothing and edge enhancement algorithms on a dedicated workstation (RadiForce GS220; Eizo) using Osirix (v10.0.5; Pixmeo SARL).

Just ventral to and including the caval foramen, there was a loss of continuity in the diaphragm through which multiple liver lobes (identified vascularly as likely the quadrate lobe and part of the right medial and left medial liver lobes) and most of the gallbladder were herniated, contacting and displacing the heart to the left side (Figure 3). Some hepatic parenchyma was in apposition with, but not compressing, the intrathoracic caudal vena cava. From these findings, the cat was diagnosed with caval foramen hernia.

Forty-eight hours prior to midline exploratory coeliotomy, the cat began receiving 90 mg amoxicillin–clavulanate IV q8h and Hartmann’s IV fluid therapy at a maintenance rate. It was premedicated with 10µg dexmedetomidine and 1mg methadone IV, induced with 7.5 mg alfaxalone IV, intubated with auffed 4 mm endotracheal tube, and maintained on isoflurane and oxygen. Perioperative analgesia included a 0.5 mg morphine epidural injection and fentanyl (5–7 µg/kg/h) constant rate infusion (CRI). The cat was mechanically ventilated intraoperatively. Anaesthetic complications included mild hypotension, tachycardia and hypothermia. Intraoperatively, the cat received dopamine (5–10 µg/kg/h) CRI, 90 µg atropine IV, 100 mg amoxicillin–clavulanate IV q2h for two doses and 2.5 mg alfaxalone IV. A 3 cm defect was identified in the central tendon of the diaphragm at the caval foramen (Figure 4a). The hernial ring was extended ventrally to the sternum with sharp and blunt dissection using Steven’s tenotomy scissors and cotton tip applicators, respectively. The cranial aspect of the right lateral, right medial, quadrate along with the entire left

Figure 3 CT scan of a 3-year-old neutered female domestic shorthair cat with hyporexia, lethargy and weight loss. (a) Transverse, (b) sagittal and (c) dorsal image planes post-contrast portal phase after reconstruction with a smoothing algorithm and viewed in a soft tissue window. There is herniation of multiple liver lobes as a hepatic mass formation (arrow) and the gallbladder (asterisk) through the caval foramen.
medial liver lobe, gallbladder and omentum were herni-ated. The left and right lateral hepatic ligaments were transected to increase mobility of the liver lobes. The left medial liver lobe appeared lobulated and adhered to the pericardium. This adhesion was bluntly dissected off the pericardium to enable its reduction into the perito-neal cavity. The edges of the caval foramen defect were apposed with full-thickness simple interrupted sutures using 2/0 polydioxanone (PDS II; Ethicon) (Figure 4b). Laxity in the right component of the central tendon was addressed with horizontal mattress sutures using 2/0 polydioxanone following scalpel scarification on either side. Prior to its closure, a thoracostomy tube (MILA International) was placed through the left muscular component of the diaphragm and exited through the ventral abdomen. A 12 F oesophagostomy tube (MILA International) was also placed.

The cat recovered uneventfully. Postoperatively, it received Hartmann’s IV fluid therapy, fentanyl (2–7 µg/kg/h) CRI and 4 mg meloxicam PO for 2 days. It received 90 mg amoxicillin–clavulanate q12h IV for 2 days and PO for 3 days. The thoracostomy tube was removed 24 h postoperatively. Four days postoperatively, the cat began eating voluntarily with a good appetite and the oesophageal tube was removed. It remained under boarding care for 3 weeks, during which time it was bright and had a good appetite. At the referring veterinarian 4 months later, the cat’s body weight had increased to 4.5 kg. The cat is currently being managed with prednisolone and a hypoallergenic diet for a skin condition.

**Discussion**

In the present case, the caval foramen hernia was considered congenital owing to the absence of any history of trauma and the cat being housed indoors. Caval foramen hernia has been rarely reported in dogs and humans.1–5

Embryologically, the septum transversum is closely involved in the development of the diaphragm, liver and caudal vena cava. The septum transversum forms the central tendon of the diaphragm.6,7 It also supports the growth and proliferation of the liver and formation of the hepatic connective tissue, hepatic capsule and its peritoneal covering.8 The mature liver remains attached to the central tendon of the diaphragm via the coronary ligament.8 The caudal vena cava develops from the cranial segment of the right vitelline vein, which initially traverses through the septum transversum into the sinus venosus of the developing embryo.9 When fully developed, the adventitial layer of the caudal vena cava fuses with the central tendon of the diaphragm.10

The authors of a human case report suggested that the embryopathology of caval foramen hernia may involve overgrowth of hepatic cords through the septum transversum or late digression of hepatocyte precursors during the formation of the intrahepatic inferior vena cava.4 However, this does not explain the occurrence of omental
fat herniation, nor in the present case, herniation of the
gallbladder.3

We propose that defective septum transversum develop-
ment can result in caval foramen hernia. This is sup-
ported by the diaphragmatic defect involving the central
tendon in the present case.

The cat’s non-specific clinical signs were attributed to
the hernia owing to their resolution following hernior-
rhaphy. It is unclear what may have caused the cat to
develop these signs. Its presentation is similar to two
canine cases, which developed anorexia and vomiting.1

However, another canine case reported lethargy since
infancy and most cases were found incidentally in older
patients.1–5 These presentations are similar to another
canine case, which developed anorexia and vomiting.1

In other cases of caval foramen hernia, only a small por-
tion of omental fat or a single liver lobe were herniated.1–5

In the present case, the diaphragmatic defect
was relatively large, allowing herniation of multiple
organisms. There was a mild elevation in liver enzymes
and left medial liver lobe adhesions. These are not uncommon
in other diaphragmatic hernias involving liver hernia-

tion.11–14 Perhaps in our case, because more structures were
herniated, there was an increased risk of organ entrapment
causing reversible disease. The herniation may also have
resulted in relatively recent adhesion formation, causing
secondary lobulation, irritation and hepatocellular dam-
age, resulting in clinical signs.11

Another cause for the lob-
ulated liver lobe could be defective septum transversum
development given its role in the formation of the hepatic
connective tissue and capsule.8

In the present case, thoracic radiographs and abdomi-
nal ultrasound were the initial imaging modalities used
for the investigation of non-specific clinical signs. These
modalities are inexpensive and relatively widely availa-
ble. Radiographically, caval foramen hernias most often
appear as a caudal dome-shaped soft tissue opacity
broad-based to the diaphragm.1 These can be confused for
pulmonary masses.1,5 In this case, the broad base
helped prioritise diaphragmatic hernia over other ori-
gins. Caval foramen hernia was specifically considered
because of the location of the soft tissue opacity and bor-
der effacement of the caudal vena cava. Ultrasound may
be used if diaphragmatic hernia cannot be confirmed on
survey radiographs.15

CT enables assessment of organs in multiple planes
without superimposition.16 CT is commonly used to con-
firm caval foramen hernia.1,5 In this case, it also enabled
assessment of vasculature and identification of herni-
ated structures, aiding anaesthetic and surgical plan-
ing. If there is no access to CT or if the client is financially
constrained, the combined clinical, radiographic and
ultrasonographic findings would provide indication for
surgical intervention.

Caval foramen hernias are commonly associated with
caudal vena cava obstruction, and may be associated
with hepatic vein dilation and pulmonary embolism.1,3

Interestingly, the majority of these patients remained
asymptomatic.1,3 In the present case, multiple organs
were herniated without caudal vena cava obstruction.
This is likely attributable to the relatively large diaphrag-
matic defect allowing space for structures to herniate
around the caudal vena cava. Nevertheless, caudal vena
cava obstruction must be considered in cases of caval
foramen hernia, and clinicians should be aware of the
potential clinical signs. Caudal vena cava obstruction
result in Budd–Chiari-like syndrome. There is hepatic venous outflow obstruction and portal hyper-
tension, leading to hepatomegaly and ascites.1,17,18

Additionally, a dog with intrathoracic caudal vena cava
obstruction also had impaired venous return to the heart,
reduced cardiac output and renal blood flow, leading to
secondary azotaemia.19

This dog presented with more
clinical findings including ascites, diarrhoea, lethargy,
tacky mucous membranes, prolonged capillary refill
time, a non-pulsatile left jugular vein and pitting oedema
in the hindlimbs.19

Conclusions

To our knowledge, this is the first report of successful
caval foramen hernorrhaphy in a cat. The hernia was
detected on routine investigation of non-specific clinical
signs. Radiographic features suggestive of caval foramen
hernia were its positioning at the mid-height of the caudal
thorax to the right of midline, border effacement of the
caudal vena cava and broad based towards the diaphragm.
These hernias have been radiographically mistaken for
pulmonary masses. Therefore, recognition of these fea-
tures can potentially significantly alter case management.
CT confirmed caval foramen hernia and enabled assessment of the caudal vena cava to aid anaesthetic and surgical planning. Caudal vena cava obstruction is commonly associated with caval foramen hernia. Therefore, it may be considered as a differential diagnosis in cats presenting with clinical signs of Budd–Chiari-like syndrome.

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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 recognised high standards (‘best practice’) of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not necessarily required.

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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