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CASE REPORT

Companion or pet animals

Gall bladder rupture and atrophy causing a hepato-cutaneous fistula in a dog

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Abstract

An 8-year-10-month-old, male, neutered Hungarian Vizsla presented with an unresolved discharging nodule on the right aspect of the xiphoid process. The dog was reported to experience intermittent lethargy and inappetence 15 months after 7 days of critical care hospitalisation for significant gastrointestinal clinical signs including inappetence, weight loss, abdominal pain, profuse vomiting and diarrhoea. An abdominal computed tomography scan revealed communication between the skin and the hepatic parenchyma. Exploratory celiotomy revealed extensive hepatic adhesions, absence of the gall bladder, and communication between the skin and a fibrotic tissue cavity within the right medial-quadrate interlobar space. Histopathological examination of the fistula confirmed the absence of the characteristic features of cholecystocutaneous fistulas. The surgical approach reported for cholecystocutaneous fistulas was only partially successful. Despite the relapse, the patient's quality of life improved substantially following our intervention, with a complete restoration of energy levels and appetite up to 1 year postoperatively.

KEYWORDS

abdomen, biliary peritonitis, biliary surgery, cholecystocutaneous fistula, gall bladder atrophy

BACKGROUND

Cases of fistulous tracts developing between the liver and the skin are scarce in the human literature.[1,2] In these rare cases, a connection between the liver and the skin was described without direct communication with the biliary tract and was named hepato-cutaneous fistula.[1] These fistulae are reported in people as a delayed complication of pyogenic liver abscesses[1] or as a complication after radiofrequency ablation therapy for hepatocellular carcinoma.[2] These cases are yet to be described in the veterinary literature. Similar reports in dogs describe cases where the gall bladder was directly communicating with the skin through a cholecystocutaneous fistula.[3–5] Although the latter condition has been more frequently described in the human literature, less than 100 cases have been recorded.[6,7] In people, the cause is usually linked to untreated cholangitis and cholelithiasis.[6–10] The discharge from the fistula can be purulent, serosanguinous or bile and occasionally contains choleliths[8,10–13]; a 'honey-like' appearance has been reported in canine patients.[3–5] The condition is diagnosed by radiography, ultrasound and computed tomography (CT) scan, with fistulography reported as a valuable additional modality.[4,7,11,14] Only six reports describe this condition in dogs, with one report in a cat.[3–5,14–17] Communication was found between the gall bladder and pleural space in the latter study, causing biliothorax. A seventh report describes a similar condition in a young English bulldog, thought to be a congenital malformation connecting the biliary system and the umbilicus, defined as an umbilical-biliary fistula.[18] In all the cases reported in human and canine patients, the diseased or ruptured gall bladder was identified intraoperatively.[3–5,7,15,16] Adhesions between the body wall and the gall bladder result in a fistulous tract that drains externally.[3–5,7,12,19] Interestingly, the gall bladder and common bile duct (CBD) appeared normal in one dog.[14] In the present case report, the gall bladder was macroscopically absent on exploratory celiotomy. It is important to acknowledge that in people, gall bladder atrophy is a well-recognised sequela of cholecystitis, cholelithiasis, micro-gall bladder, gall bladder volvulus and pancreatitis.[20] In dogs, only one case of gall bladder atrophy was reported to be associated with pancreatitis,[20] whereas 17 rare cases of gall bladder agenesis were described in 2016.[21] To the authors' knowledge, the absence of an identifiable gall bladder following rupture, resulting in a chronic fistulous tract between the liver and skin, has yet to be reported in the dog. This article describes unique and unusual

findings, the diagnosis of this rare condition, and its surgical management.

CASE PRESENTATION

An 8-year-10-month-old, male, neutered Hungarian Vizsla presented with a 4-year history of non-specific intermittent gastrointestinal problems, including lethargy, abdominal discomfort, vomiting, diarrhoea, anorexia, and weight loss. Transient episodes of pyrexia were reported by the referring veterinarian (RV) and were treated with 5-day courses of paracetamol (10 mg/kg every 8 hours) and amoxicillin-clavulanic acid (12.5 mg/kg every 12 hours). Upon presentation, the dog was bright, alert and responsive. The vital parameters were all within normal limits with regular heart rate (120 beats per minute), respiratory rate (28 breaths per minute) and pink mucous membranes with normal capillary refill time. The clinical examination was unremarkable, except for a 2 cm swelling with a non-discharging fistula on the right xiphoid area. Nineteen months before presentation, the patient suffered from a severe acute gastrointestinal episode, manifesting lethargy, inappetence, vomiting and abdominal discomfort, which were unresponsive to antiemetic and opioid medications, and was hospitalised by the RV for intensive care and oesophagostomy tube feeding. At that time, blood tests showed normal haematology parameters. There was a mild elevation of alanine transaminase (ALT) (158 U/L; reference range: 10–125 U/L) and lipase (1849 U/L; reference range: 200–1800 U/L), a positive canine pancreatic lipase immunoreactivity snap test, and a presumptive diagnosis of pancreatitis was made. The RV reported no structural abnormalities of the viscera on abdominal ultrasonography. The patient regained interest in food 4 days after hospitalisation and was discharged after 7 days of intensive care with the feeding tube in place, a 5-day course of oral cefalexin (20 mg/kg every 12 hours), paracetamol (10 mg/kg every 8 hours), maropitant (2 mg/kg every 24 hours) and a low-fat diet. Six days after discharge, the dog was re-presented with a recurrence of lethargy, inappetence and vomiting. Exploratory celiotomy was offered but declined, and a subcutaneous injection of dexamethasone (0.1 mg/kg as a single injection) was administered. A significant improvement was observed, and oral prednisolone (1 mg/kg every 24 hours for 7 days) was started alongside a further 7 days of oral cefalexin (20 mg/kg every 12 hours). The feeding tube was removed 1 week later, and the patient was maintained on prednisolone (starting with 1 mg/kg every 24 hours, then tapered down to 0.5 mg/kg every 24 hours), which was eventually discontinued after 6 months. No further concerns were raised, although the owner retrospectively reported that the patient never fully recovered from this event as energy levels and appetite were much more reduced.

The patient presented to the RV 4 months before presentation to us with a 3 cm swelling on the right aspect of the xiphoid process. Intermittent lethargy and inappetence were described with the absence of gastrointestinal signs. An incisional tissue biopsy performed by the RV showed a pyogranulomatous reaction consistent with trauma or a foreign body reaction. A 2-week course of oral amoxicillin-clavulanic acid (15 mg/kg every 12 hours) and meloxicam (0.1 mg/kg every 24 hours) was commenced. Although the swelling reduced in size after 2 weeks, a sinus, discharging white purulent material, had developed by the end of this course. Microbiology testing of the purulent fluid revealed *Staphylococcus pseudintermedius* sensitive to amoxicillin-clavulanic acid. After a 10-day prednisolone trial (0.5 mg/kg every 24 hours) combined with a 7-day course of amoxicillin-clavulanic acid at the same dose, the swelling visibly reduced, and the discharge became serous with reduced volume. The prednisolone was tapered down and stopped 1 month before presentation to us. The sinus tract was still present, discharging a large amount of clear serous liquid.

LEARNING POINTS/TAKE-HOME MESSAGES

- Gall bladder atrophy might be a sequela of gall bladder rupture, leading to a chronic fistula between the liver and skin in dogs.
- Given the different histopathological features, this condition may be considered separate from cholecystocutaneous fistulas.
- Clinical suspicion should be raised in dogs with a history of severe gastrointestinal signs presented with unresolved discharging nodules on the right aspect of the xiphoid process.

INVESTIGATIONS

The RV performed an abdominal CT scan to investigate further, as a penetrating foreign body was suspected. CT revealed a right parasagittal cutaneous and subcutaneous lesion, extending throughout the sternal part of the diaphragm and right transverse thoracis and abdominis muscles, which was in contact with the ventral aspect of the hepatic parenchyma and anatomical gall bladder location (Figure 1a,b). These findings raised high suspicion for a potential cholecystocutaneous fistula, and the patient was referred to our institute for exploratory celiotomy. Preoperative blood tests revealed unremarkable blood cell count. The only abnormality in biochemistry was an increased ALT (325 U/L; reference range: 10–125). The patient was sedated with dexmedetomidine (3 μg/kg intravenously [IV]) and methadone (0.3 mg/kg), and abdominal ultrasound was performed. Maropitant (1 mg/kg IV) was concurrently administered. Preoperative ultrasonography found the liver normal in size and of homogenous architecture with tapering lobes and a dilated CBD. An ill-defined heterogeneous structure in the right ventrolateral abdominal cavity, approximately 5 cm dorsal to the external fistula, was found to communicate with the abdominal wall. Ultrasound failed to identify the gall bladder.

TREATMENT

The patient was induced with alfaxalone (2 mg/kg IV given to effect) and maintained on 2% isoflurane in 100% oxygen via an endotracheal tube. Paracetamol (10 mg/kg IV) was given immediately after induction. Perioperative cefuroxime (20 mg/kg IV) was started 30 minutes before surgery and continued every 90 minutes for the duration of the procedure

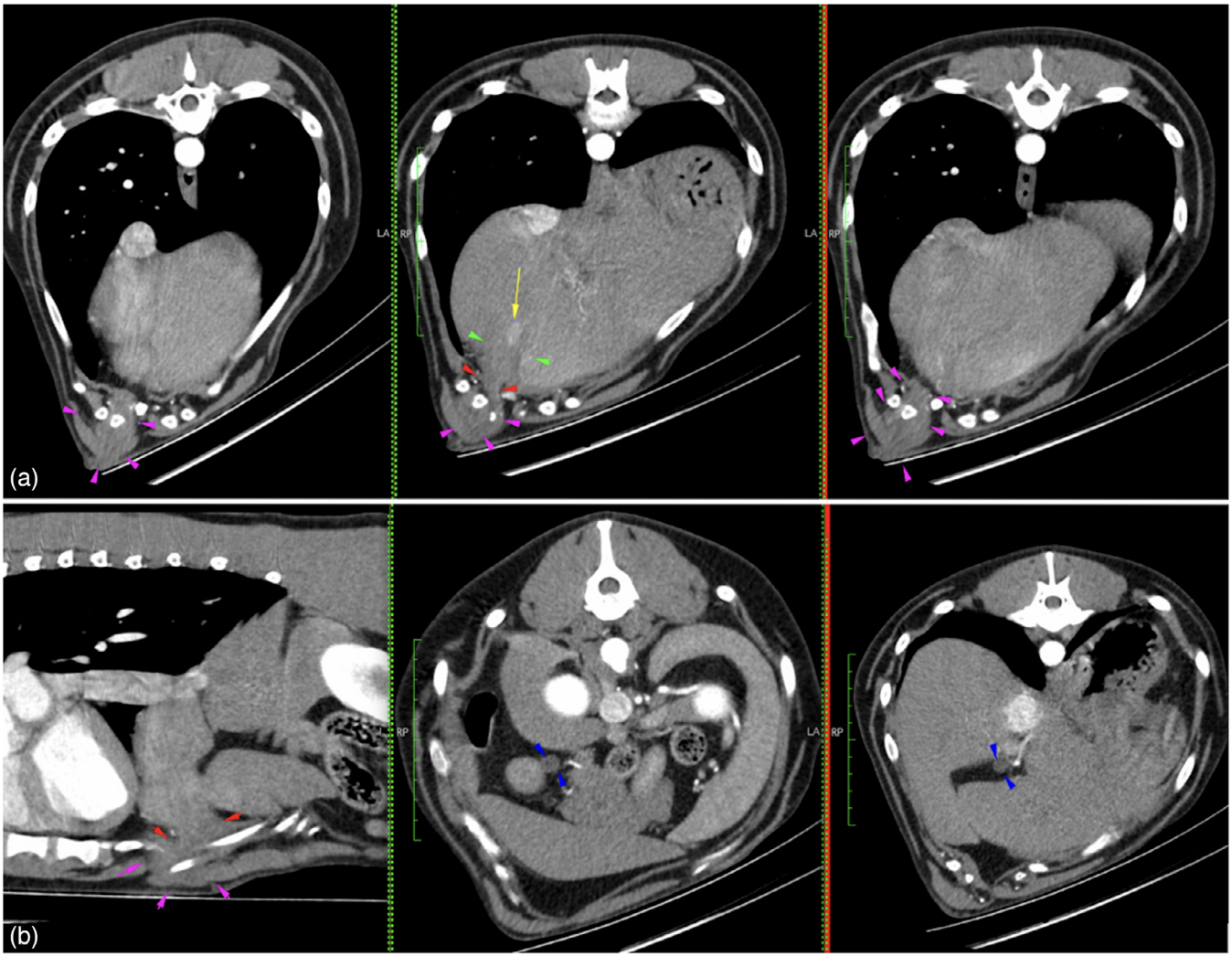

FIGURE 1 Abdominal computed tomography scan. Right parasagittal cutaneous and subcutaneous, poorly contrast-enhancing, soft-tissue attenuating lesion surrounding the xiphoid process and the distal aspect of the surrounding costal cartilages (a and b, pink arrows). This lesion measured up to 7 cm in the craniocaudal, 3.1 cm in the mediolateral and 3.7 cm in the ventrodorsal directions. It extended throughout the sternal part of the diaphragm and right transverse thoracis and abdominis muscles, and was in contact with the ventral aspect of the hepatic parenchyma and gall bladder (a and b, red arrows). The definition of the gall bladder was very poor (a, green arrows), and within the area of its lumen in the liver parenchyma, an ill-defined hyperattenuating structure was noticed (a, yellow arrow). The common bile duct was distended, measuring up to 10 mm in diameter, but no obvious mechanical obstruction was identified (b, blue arrows).

until recovery. Perioperative IV fluid therapy (5 mL/kg/h) was provided with Hartmann's solution.

An exploratory celiotomy through a standard cranial ventral midline approach was undertaken. Multiple adhesions affecting the entire diaphragmatic and visceral faces of the liver were noticed, including all the liver segments (Figure 2). Separation of the right division of the liver started ventrally using a combination of sharp and blunt dissection and Harmonic Focus technology (Ethicon) and continued along the right lateral abdominal wall and last rib dorsally until the right lateral and medial liver lobes could be mobilised. A similar approach was repeated on the left-hand side. Not all adhesions could be released due to concerns related to iatrogenic injury, and some portions of the left lateral liver lobes remained attached to the diaphragm. Adhesions were also present between the individual liver lobes (Figure 2), affecting the extrahepatic biliary tract, including CBD and hepatic ducts. Partial right medial liver lobectomy using a suture-fracture technique with a Modified Miller's knot using a 2-0 polypropylene suture material was required to provide visualisation of this area and to control parenchymatous bleeding. A cavity within the fibrotic tissue formed at the right medial-quadrate interlobar space was identified and dissected (Figure 3). The distended CBD was localised and confirmed to be patent through a duodenotomy and retrograde catheterisation. A bile sample was obtained through the same catheter for a macroscopical evaluation and was confirmed to be normal in colour and viscosity. The gall bladder was absent, with only a fibrotic stump identified at the level of the residual cystic duct. The latter was identified and initially ligated within the hepatic fossa using a single encircling 2-0 polydioxanone suture. A second identical ligation of the cystic duct was performed at its entrance into the CBD, 2 cm away from the first one. This area was then debrided and flushed. After abdominal exploration, an elliptical skin incision around the fistula was performed, and the subcutaneous tissue was dissected using a combination of blunt and sharp dissection. The fistulous tract was probed, and communication with the abdominal cavity at the right medial liver lobe level was confirmed (Figure 3). The lesion was excised en bloc from where it traversed the

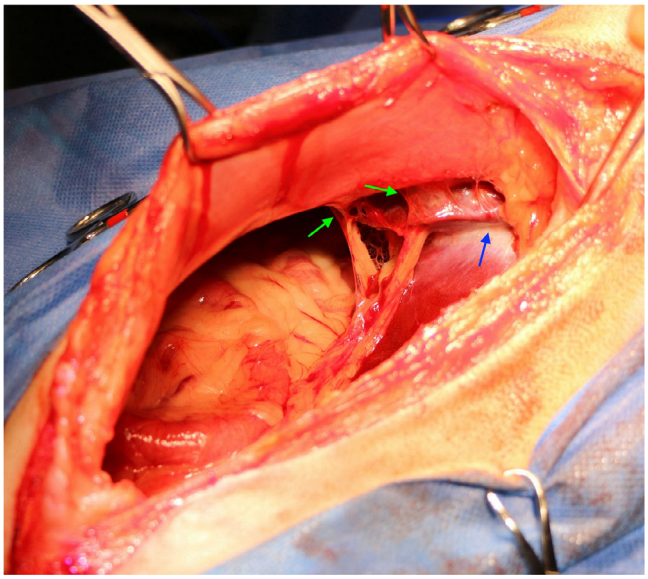

FIGURE 2 Intraoperative image—Upon entry to the abdominal cavity. Adhesions between the liver and abdominal wall (green arrows) and between two adjacent liver lobes (blue arrow).

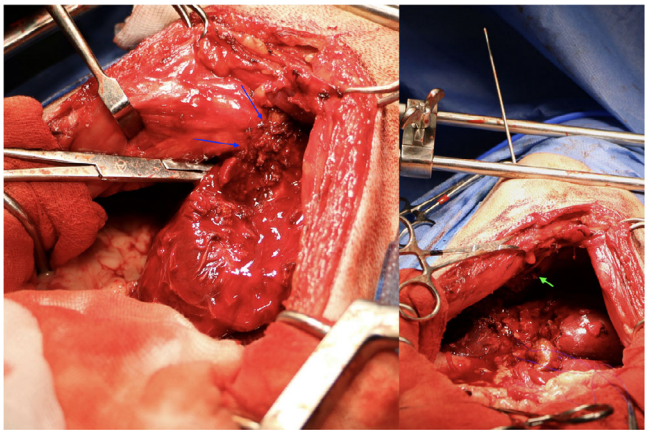

FIGURE 3 Intraoperative image—Identification of the interlobar space and fistulous tract. On the left is shown the adhesion between the right medial liver lobe and the abdominal wall containing the fistulous tract (blue arrows). Right-angled forceps are inserted in the cavity formed within the right medial-quadrate interlobar space. On the right, the fistulous tract is probed to track its trajectory and entry point within the abdominal cavity (green arrow) after the right partial liver lobectomy.

body wall. The inner muscular layer was omentalised from the abdominal side (Figure 4), and the rest of the abdominal wall was closed externally from the skin wound. Subcutis and skin were then primarily closed in two layers. The abdomen was flushed with a warm saline solution, suctioned and closed routinely. The dog recovered from anaesthesia uneventfully and was hospitalised for 24 hours on methadone (0.2 mg/kg IV every 4 hours), paracetamol (10 mg/kg IV every 8 hours) and cefuroxime (20 mg/kg IV every 8 hours). Intravenous fluid therapy with Hartmann's solution (4 mL/kg/h) was continued for 12 hours postoperatively.

OUTCOME AND FOLLOW-UP

The day after the surgery, the dog was bright, alert and responsive, and vital parameters were within normal limits. Blood

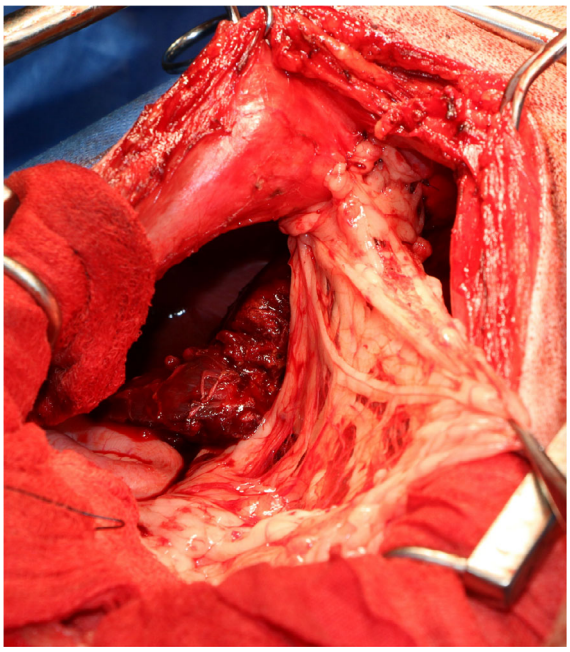

FIGURE 4 Intraoperative image—Omentalisation of the fistulous tract. The abdominal aspect of the fistulous tract was debrided and omentalised.

tests repeated the day after the surgery showed increased ALT levels (795 U/L; reference range: 10–125). Results were otherwise unremarkable. The dog was discharged 24 hours after the surgery with oral cefalexin (22 mg/kg every 12 hours for 7 days), paracetamol and codeine (15 mg/kg every 8 hours for 5 days) and meloxicam (0.1 mg/kg every 24 hours for 14 days).

A histopathological examination of the resected lobe and tract was performed. The fistula comprised a central sinus tract surrounded by a fibrous capsule blended with granulation tissue infiltrated by pyogranulomatous and lymphoplasmacytic inflammation (Figure 5A–C). The classical lining of hyperplastic cells resembling the biliary epithelium, previously described in the veterinary literature for cholecystocutaneous fistulas,[2,12] was absent in this case. No bile was observed within the fistula in an additional section stained with a Fouchet's histochemical stain (Figure 5B). The observed rarefaction of midzonal to centrilobular hepatocytes was considered secondary to the steroid administration. There was no evidence of irreversible liver disease or a biliary obstruction (Figure 5d), nor evidence of infectious agents or neoplasia within the fistula or liver biopsy.

Two weeks postoperatively, the client reported a substantial reduction of the xiphoid swelling and no discharge. There were no abnormalities on clinical examination. Both surgical wounds fully healed, and the skin sutures were removed.

At the 2-month follow-up, the patient's activity level had substantially increased, restoring its usual demeanour and normal appetite as before the intensive care hospitalisation. There were no abnormalities on clinical examination, and the swelling in the xiphoid region was absent. The repeated blood test revealed unremarkable blood cell count. ALT was only mildly increased (198 U/L; reference range: 10–125) but markedly reduced compared to the measurement taken the day after the surgery (795 U/L), and the remaining biochemical parameters were unremarkable.

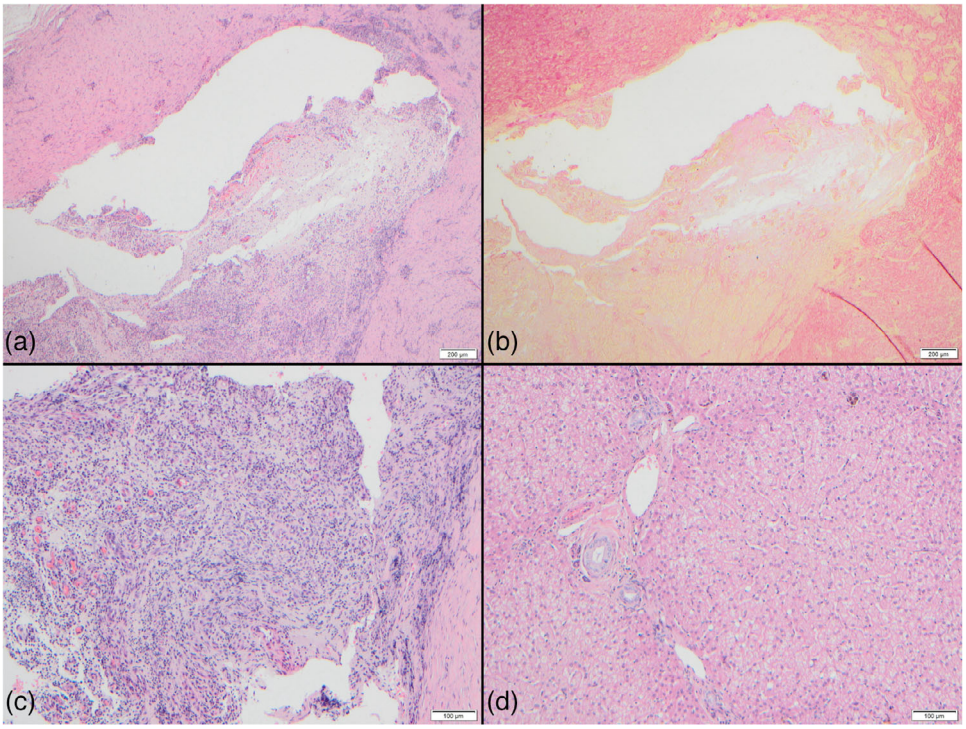

FIGURE 5 Histological images—Sections through the fistula and excised liver lobe. (a) Haematoxylin–eosin (H&E). Low magnification of the fistulous tract that is composed of a central lumen surrounded by a fibrous capsule. There is no evidence of biliary epithelium lining the sinus tract. (b) Fouchet's stain. Low magnification of the fistulous tract, revealing no positive staining for bile within the central lumen (bile should stain green). (c) H&E. Higher magnification of the edge of the fistulous tract, which is lined by granulation tissue infiltrated by large numbers of neutrophils, macrophages, lymphocytes and plasma cells. (d) H&E. High power of the excised liver lobe, demonstrating the normal appearance and number of biliary profiles within the portal triad as well as the midzonal to centrilobular hepatocellular rarefaction.

The patient re-presented 4 months postoperatively because of the recurrence of a subcutaneous swelling in the same area, which burst, discharging serous exudate for 3 weeks. The owner reported that the discharge had stopped, and the wound healed a week before the presentation. The dog was doing clinically well with normal energy levels and appetite. No abnormalities were detected on clinical examination, and only a dry crust was present at the same level as the former fistula. The dog was comfortable at palpation, but an ultrasound scan of the area raised the suspicion for sinus tract recurrence (Figure 6). Blood tests revealed a mild elevation of ALT, which was still trending towards the normal range compared to the previous measurement (164 U/L; reference range: 10–125). Total protein was mildly increased (87 g/L; reference range: 52–82) as well as globulin (60 g/L; reference range: 25–45) and alkaline phosphatase (ALP) (324 U/L; reference range: 23–212). The rest of the blood parameters were unremarkable. Fistulography confirmed an open communication between the skin and the abdominal cavity (Figure 7). Revision surgery was recommended to the owner, but this was declined as the dog was doing very well, and the fistula was not impairing its quality of life.

Eight months postoperatively, demeanour and appetite were still optimal. There were no abnormalities on physical examination, except the fistula, which was fresh with dried stains of serous-like discharge in the surrounding skin. Collection of the fluid for analysis was not possible as the fistula was not actively discharging. The owner reported that the fistula was mainly dry and clean. It only occasionally discharged a small amount of clear serous-like fluid, especially after excessive exercise, but this did not bother the patient. On blood tests, ALT was still mildly elevated (189 U/L; reference range: 10–125). Total protein, globulin and ALP were still mildly elevated (89 g/L; reference range: 52–82: 62 g/L; reference range: 25–45; 271 U/L; reference range: 23–212, respectively). The patient was clinically doing very well; for this reason, the owner declined further surgical intervention. Instructions were given to clean the area when discharge was present, but no additional medical management was deemed necessary. To the authors' knowledge, no deterioration of the clinical signs was observed up to 1 year postoperatively.

DISCUSSION

Fistulas between the liver and the skin are rare and only reported in a limited number of cases in human literature.[1,2] In the six known canine cases, the condition was classified as a spontaneous cholecystocutaneous fistula.[3–5,14–16] In fact, as described in people,[6,8] all reported dogs presented with a few-month history of a subcutaneous, non-painful nodule,[15] discharging mass[4,5,14,16] or wound[3] on the right caudo-ventral aspect of the thorax.

None of the previously reported cases showed signs of remarkable systemic illness before presentation.[3–5,14–16] Galliano reports vague signs of abdominal discomfort, which were more noticeable when the owner picked up the dog.[5] Kligman reports normal appetite and activity levels.[14] In the human literature, patients' histories can be unremarkable[9] or present multiple episodes of colicky, transient, but

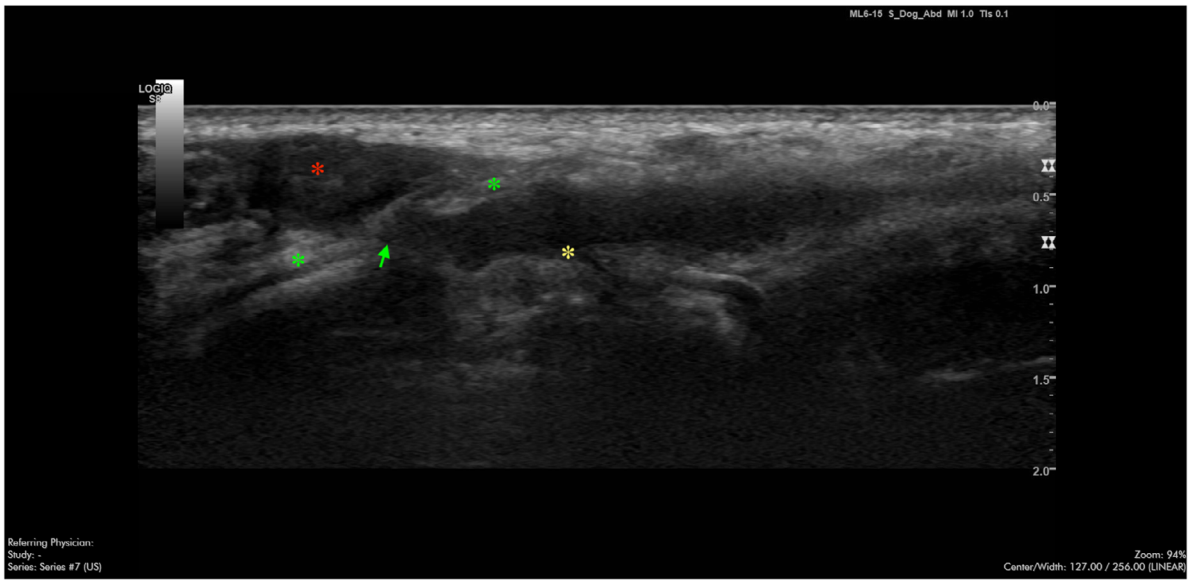

FIGURE 6 Ultrasound scan of the lesion—The abdominal wall (blue asterisks) presented a gap (green arrow) connecting the subcutaneous layer underneath the lesion (red asterisk) and the abdominal cavity (yellow asterisk).

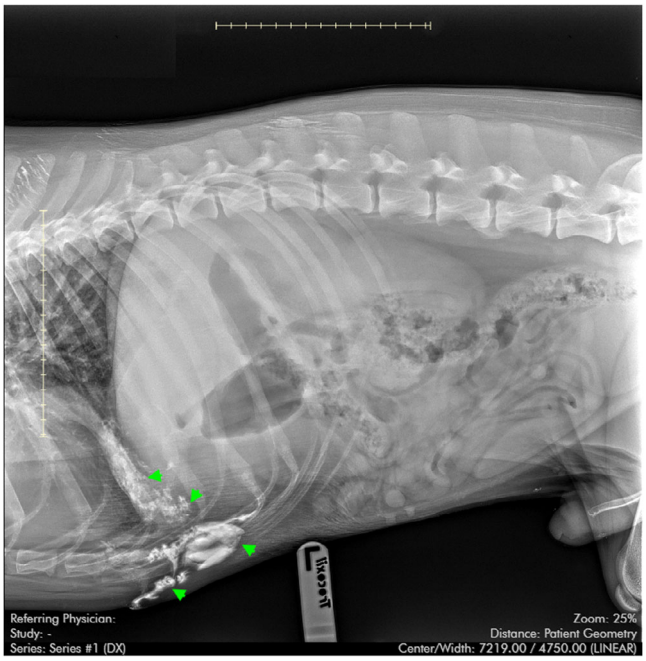

FIGURE 7 Fistulography—Diffusion of the contrast from the subcutaneous tissue into the abdominal cavity (green arrowheads) confirming the communication between the two.

tolerable upper abdominal pain associated with heavy meals.[7] In our case, the patient was hospitalised for 7 days in critical care, 19 months before the presentation. At that time, no final diagnosis was achieved, and euthanasia was taken into consideration. The RV performed an ultrasound scan, but no comments were made on the state of the gall bladder or free peritoneal fluid. While the scan was not conducted by a specialist ultrasonographer, free peritoneal fluid is a relatively easily detectable sign. The absence of free fluid in case of biliary rupture and consequent peritonitis would be unusual. This might be associated with a technical error of the operator, but it is also possible that the gall bladder was not ruptured when the scan was performed. Unfortunately, we do not have enough information to clarify our doubts. The owner reported that the dog never fully recovered from that event. We postulate that the dog suffered from an acute biliary rupture and undiagnosed biliary peritonitis, followed by chronic fibrotic repair. This theory could explain the large number of adhesions found during the exploratory celiotomy (Figure 2).

This process differs from a spontaneous cholecystocutaneous fistula for several reasons. First, the pathogenesis of the sinus tract appears different. In spontaneous cholecystocutaneous fistula, an adhesion initially forms between the gall bladder and the abdominal wall, and then, a draining tract connecting the skin and the biliary system develops.[3–5,7] In a canine case, the fistula was formed between a dilated bile duct and the diaphragm, but the gall bladder and the rest of the liver were grossly normal.[14] Three cases describe a process affecting the gall bladder and some of the adjacent liver lobes with normal appearance of the rest of the organ.[5,15,16] In the two remaining reported canine cases, the process was only localised to the gall bladder, and the liver was macroscopically unremarkable with no adhesions.[3,4] In the case reported here, the adhesions were extensive over the Glisson's liver capsule.

Furthermore, the gall bladder was not found macroscopically, with a fibrotic remnant being the only structure that could be anatomically located. The tract developed within the adhesions between the right medial liver lobe and the abdominal wall (Figure 3). The fistula connected the skin, and an abscess formed within a fibrotic tissue cavity at the right medial-quadrate interlobar space (Figure 3), which was not directly connected to the biliary system.

This theory is supported by histopathological examination of the fistulous tract and the right medial liver lobe (Figure 5). In the sections of the examined liver, there was no evidence of biliary obstruction (no biliary ductule hyperplasia or bile plugs within hepatic canaliculi) (Figure 5D). There was no evidence of hyperplastic epithelium resembling biliary epithelium lining the fistulous tract, characteristic of cholecystocutaneous fistula[4,14] (Figure 5A). In this case, the fistulous tract was instead associated with a pyogranulomatous and lymphoplasmacytic inflammatory response more

characteristic of an abscess (Figure 5C). Unfortunately, Marquardt and Galliano do not describe the histopathological appearance of the fistula, but the 'honey-like' discharge was very similar to what was described by Fabbi.[3–5] Kligman describes a purulent discharge as was observed in our case, but with the characteristic microscopic appearance of the cholecystocutaneous fistula.[14] Although the histopathological appearance of the fistulous tract was not explicitly described in the two most recent cases, their diagnosis of an external biliary fistula based on the imaging and intraoperative findings suggests that the fistula would have been lined by biliary epithelium, similar to that described by Fabbi and Kligman.[4,14–16] In these two cases, gall bladder involvement and direct communication between the skin and the biliary system were confirmed intraoperatively, similar to the rest of the reported external biliary fistulas.[3–5,14–16] Cholecystectomy was needed in all the cases mentioned above, but it was not required in our patient, as the gall bladder was macroscopically absent. Instead, ligation of the residual cystic duct was performed.

Ideally, a liver and fistulous tract culture should have been performed.[3–5,14,15] Bacteriological culture was recommended but declined due to financial constraints. Given the previous culture at the RV and the fact that the purulent discharge turned serous-haemorrhagic with medical management before presentation to us, a prophylactic course of postoperative antibiotics was chosen instead.

Unlike other cases, we could not submit the gall bladder for histopathology and determine the cause of the potential rupture and subsequent atrophy. In people, destructive inflammation and fibrosis of the gall bladder wall caused by rare xanthogranulomatous cholecystitis was reported to be the cause of fistula formation in a 75-year-old Saudi Arabian man.[7] However, the gall bladder was found and removed in that case.[7] We can only speculate that an acute biliary rupture and subsequent peritonitis caused the severe illness reported previously in this dog. The subsequent adhesions surrounding the right medial-quadrate interlobar space led to the chronic fibrotic cystic structure within the right quadrant of the liver. Suppurative changes of the gall bladder followed, with abscessation and subsequent fistula formation. This event's timeline could explain why the dog never fully recovered after medical management but was reported to return to the usual demeanour after surgical intervention and definitive cystic duct ligation.

Our surgical approach was extrapolated from what was already reported for cholecystocutaneous fistulas. Although the fistula recurred 3 months after surgical intervention, the discharge was occasional, and the patient's quality of life improved substantially. In focal processes such as cholecystocutaneous fistulas, excision with or without omentalisation of the tract, followed by cholecystectomy, was reported to be successful in achieving full resolution.[3–5,14–16] Given the diffuse nature of the process, not all the abnormal tissue was potentially resected in our case, leading to the persistence of chronic inflammation and relapse a few months down the line. This could explain why postoperative ALT never fully normalised despite a general improvement compared to the preoperative values. Equally, a perpetual chronic inflammation would explain the persistent increase of inflammatory markers such as ALP, total protein and globulin after the surgery. Unfortunately, further surgical exploration was not possible, and we cannot say what caused the relapse. Ultrasound scan and fistulography suggested further communication between the skin and abdominal cavity. With these modalities, we were not able to confirm the exact location of the new communication, but the contrast appears to travel further cranially towards the diaphragm. Advanced imaging was declined and a more detailed assessment of the area was not possible. The improvement of the systemic clinical signs in our case could be partially attributed to the cystic duct ligation, which stopped chronic bile leakage within the abdominal cavity, reducing tissue irritation. On the other hand, we do not have any proof that chronic bile leakage was the definitive cause of the problem, and it is possible that the surgical debridement of the diseased tissue could have also contributed substantially to the improvement of the clinical signs.

This report describes the first documented case of gall bladder rupture and atrophy, resulting in chronic abscessation and fistulation between the liver and skin. Notably, this condition has yet to be reported in human or veterinary literature, likely due to the low survival of patients without surgical intervention. The authors hypothesise that in rare cases of survival, gall bladder atrophy may occur, leading to a chronic hepatocutaneous fistula in dogs. Therefore, clinical suspicion should be raised in dogs with a history of severe gastrointestinal signs presented with unresolved discharging nodules on the right aspect of the xiphoid process.

Our case's clinical history and surgical findings differ from those already reported for cholecystocutaneous fistula.[3–5,14–16] Also, histopathology was consistent with an abscess as there was no evidence of hyperplastic epithelium resembling biliary epithelium lining the tract as previously described,[4,14] so the term 'cholecystocutaneous' might be inappropriate. This finding suggests no direct communication with the biliary tract and allows us to consider this a separate entity, more similar to the human hepato-cutaneous fistula.[1,2] The latter term may be, in fact, more appropriate. In our case, the surgical approach reported for cholecystocutaneous fistulas was only partially successful, as the fistula recurred a few months after the surgery. Despite the relapse, the patient's quality of life substantially improved following the intervention.

AUTHOR CONTRIBUTIONS

Renato Miloro conceived and designed the paper. Renato Miloro and Barbora Mala acquired the data. Barbora Mala and Michal Vlasin critically reviewed the manuscript. Rachel Garty and Dylan Yaffy performed histopathological interpretation and critical revision of the histopathological description. Dylan Yaffy prepared the histological images. Michal Vlasin was the primary surgeon on the case and was responsible for the final approval of the manuscript. Renato Miloro wrote the manuscript.

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Emilie Fauchon DVM, Dip. ECVDI (VetCT) was responsible for the computer tomography report, and Callum Sharp MA, VetMB BSc was responsible for a critical review of the manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

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ETHICS STATEMENT

All the actions described in this case report were entirely undertaken in the best interest of the patient, basing the decision-making on the low number of relevant published evidence. An informed client consent was obtained before intervention. No ethical approval was needed for this case report.

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IMAGE QUIZ

The communication between the subcutaneous tissue and the abdominal cavity was highlighted by the diffusion of the contrast medium between the two compartments (Figure 7).

MULTIPLE-CHOICE QUESTION

Which of the following is not a characteristic feature of a hepato-cutaneous fistula in the dog?

POSSIBLE ANSWERS TO MULTIPLE-CHOICE QUESTION

a) The presence of granulation tissue infiltrated by large numbers of neutrophils, macrophages, lymphocytes and plasma cells lining the fistulous tract.
b) A swelling in the right aspect of the xyphoid process discharging pus or serous-haemorrhagic fluid.
c) The presence of classical lining of hyperplastic cells resembling the biliary epithelium lining the fistulous tract.
d) History of severe gastrointestinal signs attributable to biliary peritonitis.

CORRECT ANSWER

c) The presence of classical lining of hyperplastic cells resembling the biliary epithelium lining the fistulous tract.

This is the only feature that is typically associated with a cholecystocutaneous fistula and not with a hepato-cutaneous fistula.