Received: 21 April 2023 | Accepted: 28 April 2023

DOI: 10.1002/ccr3.7352

CASE REPORT

Clinical Case Reports Open Access WILEY

Medical management of feline emphysematous hepatitis—A case report

Corinna Palko | **Samuel Durkan**

Pittsburgh Veterinary Specialty and Emergency Center, Pittsburgh, Pennsylvania, USA

Correspondence
Corinna Palko, Pittsburgh Veterinary Specialty and Emergency Center, Pittsburgh, Pennsylvania, USA.
Email: corinna.palko@bluepearlvet.com

Key Clinical Message

Case report summary: A 9–year-old male castrated domestic shorthair feline was presented to the emergency department with a chief complaint of constipation of 3-day duration, decreased urination of 1-day duration, vomiting, and hind limb weakness. Physical examination abnormalities included hypothermia, dehydration, and generalized paresis with inability to stand for a prolonged period of time. Abdominal ultrasonography showed pinpoint hyperechoic foci throughout the hepatic parenchyma, small gas foci circulating within the portal vasculature consistent with emphysematous hepatitis, and mild volume of ascites. Cytology of the ascites was consistent with inflammatory effusion. Hepatic cytology was consistent with mixed inflammation/hepatitis with no apparent cause for inflammation noted. Urine culture yielded a negative result. Surgical liver biopsy and culture were declined by the patient's family. Ultrasound changes were presumed to be most likely secondary to an ascending infection.

KEYWORDS

diabetes mellitus, emphysema, emphysematous hepatitis, emphysematous, hepatic emphysema, hepatitis

1 | INTRODUCTION

Few reports of emphysematous hepatitis exist in the human literature. Emphysematous hepatitis is a rapidly progressive and often fatal liver infection that can occur with uncontrolled diabetes mellitus (DM), hepatic disease, or following recent abdominal surgery.[1–6] It results in the replacement of hepatic parenchyma with gas causing acute liver failure.[1–6] Diagnosis requires diagnostic imaging, predominantly computed tomography (CT), but can include abdominal ultrasound, in human medicine where they characteristically show air bubbles within the liver parenchyma. Treatment consists of aggressive antibiotic administration and supportive therapy. Ultimately, many of the reported cases resulted in fatality secondary to septic shock.[3–6]

Emphysematous infections are rarely reported in veterinary medicine. Reported cases include emphysematous cystitis, gastritis, intestinalis/colitis, and splenitis.[7–14] Diagnostic imaging including radiographs, ultrasound, and/or CT is required for diagnosis. Pathogenesis is not well understood; however, a combination of elevated glucose levels in the tissue and immune system impairment have been implicated in facilitating glucose-fermenting infections resulting in emphysematous cystitis in canines.[15]

2 | CASE DESCRIPTION

A 9-year-old castrated male domestic shorthair feline was presented to Pittsburgh Veterinary Specialty and Emergency Center (PVSEC) for evaluation of a 3-day history of constipation, decreased urination of 1-day duration, vomiting twice over 2 days, and hind limb weakness. He was treated at home with oral lactulose twice daily for 2 days prior to presentation. He had a known history of diabetes mellitus managed with his primary care veterinarian with 3 units of glargine insulin subcutaneously every 12 h. His family continued to administer his insulin as prescribed with his last dose given approximately 10 h prior to presentation.

On presentation, the patient weighed 6.4 kg with a body condition score of 4/9. Rectal temperature (T) was consistent with hypothermia (T 97.6 °F). Heart rate and respiratory rate remained within normal limits. Abnormalities on physical examination included moderate periodontal disease, 7%–10% dehydration with tacky mucous membranes and prolonged skin tent, dull hair coat with generalized flaking, generalized muscle atrophy most prominent in the hind limbs and epaxial muscles, and reluctance to stand. Blood work, urinalysis, abdominal focused assessment with sonography for trauma (AFAST), and thoracic and abdominal radiography were performed on presentation.

Blood work revealed mild hemoconcentration (hematocrit 47.7%, [reference interval 25.0%–45.0%]) and thrombocytopenia (35,000/μL, [RI 200,000–500,000/μL]). Manual blood smear evaluation revealed a pseudothrombocytopenia given 3+ platelet clumping noted. Abnormalities noted on serum biochemistry included hypochloremia (113.9 mmol/L, [RI 114.0–127.0 mmol/L]), hypercholesterolemia (250 mg/dL, [RI 95–200 mg/dL]), hypophosphatemia (2.1 mg/dL, [RI 2.6–5.7 mg/dL]), hypoalbuminemia (2.5 g/dL, [RI 2.8–4.0 g/dL]), hyperglobulinemia (5.1, [RI 2.0–5.0]), elevated BUN (41 mg/dL, [RI 10-40 mg/dL]), hyperglycemia (310 mg/dL, [RI 70-160 mg/dL]), hypocalcemia (6.2 mg/dL, [RI 8.2–10.6 mg/dL]), elevated creatinine kinase (831 U/L, [RI 90–330 U/L]), and hypermagnesemia (3.3 mg/dL, [RI 1.9–2.6 mg/dL]). Urinalysis showed urine specific gravity (USG) 1.056, cloudy pale urine, proteinuria (100 mg/dL), glucosuria (1000 mg/dL), and hematuria (10Ery/μL). Urine culture did not identify any microorganisms. AFAST showed abdominal fluid score (AFS) 0 (Table 1). Thoracic and abdominal radiography were concerning for trace peritoneal effusion or mesenteric edema possibly a result of nonspecific gastroenteritis and/or pancreatitis, hypovolemia, and chronic lumbosacral degeneration (Figure 1). Abdominal ultrasound was recommended when available.

The patient was hospitalized for treatment with buffered crystalloid solution intravenously, lactated ringers solution (LRS, no additives; 7 mL/kg bolus over 15 min, then 75 mL/kg/day), maropitant (1 mg/kg IV q24hr), pantoprazole (1 mg/kg IV q12hr), humulin R (regular insulin) (1.5 U IM, once), and calcium gluconate 10% (49.4 mg/kg IV), diluted 1:1 with 0.9% NaCl, slow over 15 min while monitoring electrocardiogram (ECG). On day 2 of hospitalization, the patient was febrile (T 103.6 °F) with generalized weakness and remained 5% dehydrated. Recheck blood work showed worsening hypophosphatemia (1.5 mg/dL) and hypoalbuminemia (2.0 g/dL) and persistent total hypocalcemia (6.4 mg/dL) with decreased ionized calcium (iCa) (0.85 mmol/L, [RI 1.25–1.5 mmol/L]). Serum ketones were within normal limits (0.6 mmol/L, [RI <2.0 mmol/L]). Clotting times showed a mildly prolonged aPTT (127 s, [RI 65.0–119.0 s]). Additional medical therapy was implemented including ondansetron (0.3 mg/kg IV q8hr), mirtazapine (0.3 mg/kg PO q24hr), enrofloxacin (4.8 mg/kg IV q24hr), metoclopramide (0.47 mg/kg IV once), and calcium gluconate 10% was repeated as previously described. Crystalloid therapy was transitioned to Normosol-R at the previous rate of 75 mL/kg/day (potassium phosphate additive, 0.1 mmol/kg/hr). Abdominal ultrasound revealed a diffusely hypoechoic and heterogeneous hepatic parenchyma with numerous pinpoint hyperechoic foci throughout the parenchyma, small gas foci circulating in the portal vasculature, generalized gastric wall thickening, thickened and heterogeneous pancreas with irregular margins, and mild volume of peritoneal fluid (Figure 2).

A sample of the serosanguinous peritoneal effusion was obtained under ultrasound guidance without complication. Fluid analysis and cytology from a reference laboratory revealed an elevated leukocyte count (4230 per μL, [RI 0–3000 per μL]) consistent with an inflammatory effusion with a low number of immature lymphocytes and no bacteria or neoplastic cells observed (including atypical lymphocytes). Serial blood work showed a persistent hypocalcemia (iCa 0.87 mmol/L, [RI 1.25–1.5 mmol/L]). A third dose of calcium gluconate 10% was administered at the aforementioned dose with the previously outlined

TABLE 1 Adapted from Lisciandro, et al.[21]

AFAST fluid scoring system: 4 point scale		
AFS 0	Negative in 4 quadrants	AFS 1 and 2—considered minor injury or pathology, small volume hemorrhage
AFS 1	Positive in 1 quadrant	
AFS 2	Positive in 2 quadrants	AFS 3 and 3—considered major injury or pathology, large volume hemorrhage
AFS 3	Positive in 3 quadrants	
AFS 4	Positive in all 4 quadrants	

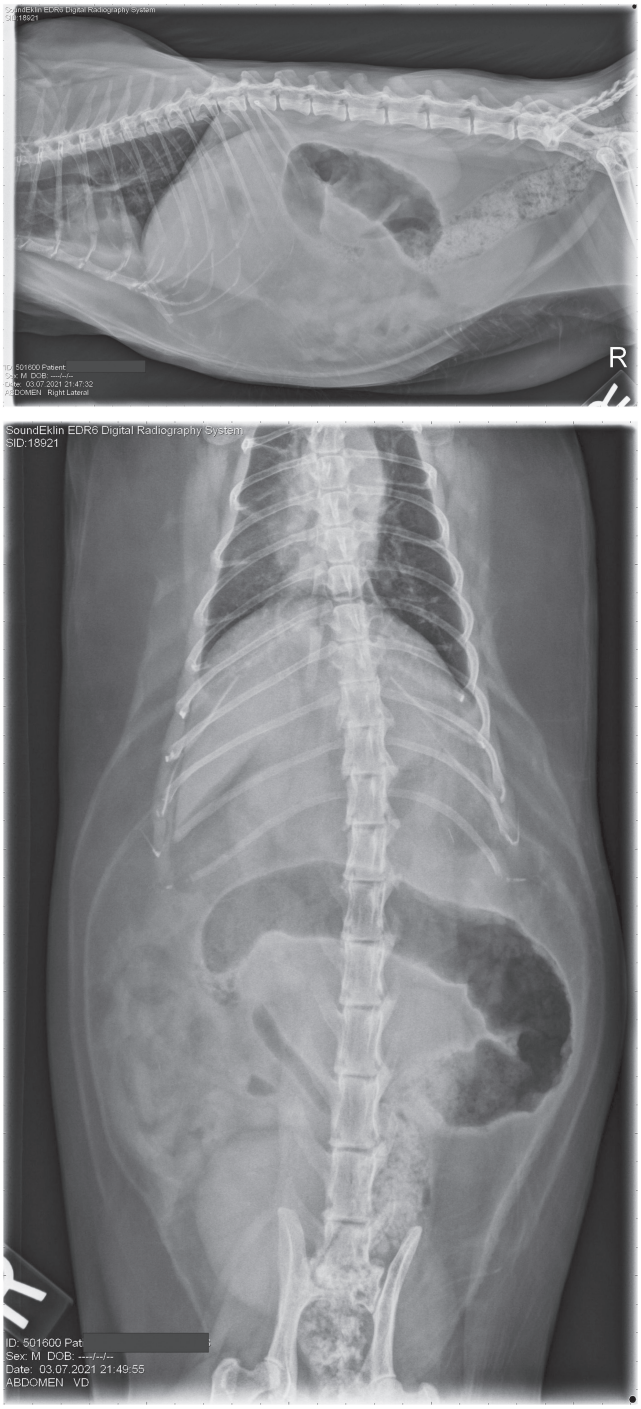

FIGURE 1 Two view whole-body radiographs: ventral dorsal and right lateral views. Intrahepatic gas foci not visible at presentation.

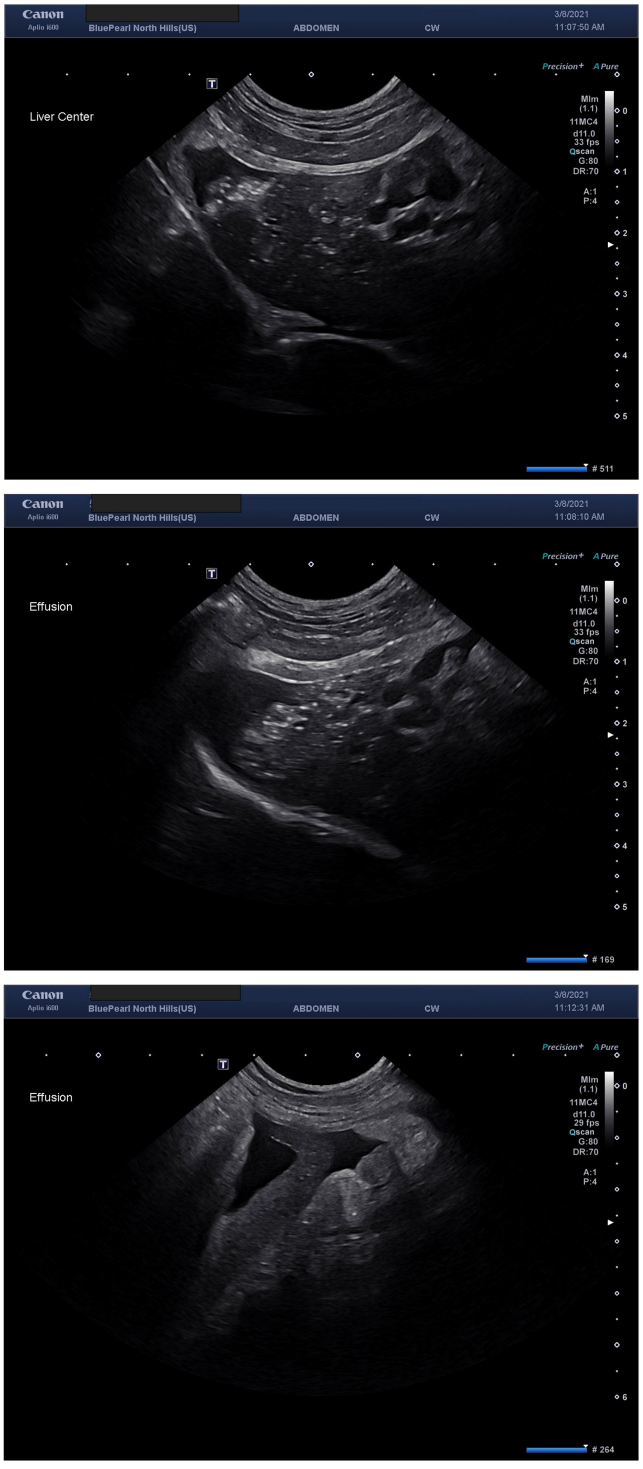

FIGURE 2 Ultrasonographic images of the liver and peritoneal effusion.

protocol. Pyrexia resolved a few hours after implementing antibiotic therapy. Liver aspirate via ultrasound guidance was performed without complication, and the samples were submitted for pathology review. Current therapy was continued with addition of buprenorphine (0.019 mg/kg transmucosally q8hr) for pain and discomfort.

On day 3 of hospitalization, the patient's weakness was improving, and he began eating well when offered. Glargine (Lantus, 1.5 units SQ q12hr) administration was started. Serial blood work showed a persistent total hypocalcemia (5.9 mg/dL [RI 8.2–10.6 mg/dL]), progressive hypoalbuminemia (1.8 g/dL, [RI 2.8–4.0 g/dL]), and hyponatremia (144.5 mmol/L, [RI 148.0–159.0 mmol/L]). Liver cytology showed mixed inflammation and hepatitis, mild hepatic lipid vacuolar

change, and hepatocellular hyperplasia. No cause for the inflammation was found via cytology, with no neoplastic or infectious etiologic agents seen. Livery biopsy and culture were declined by the patient's family as clinically the patient was improving and eating well. Oral calcium supplementation was started (Tums, calcium carbonate, 187.5 mg PO q12hr), and the patient was transitioned to omeprazole (1 mg/kg PO q12hr) from pantoprazole. By day 4 of hospitalization, the patient was eating well and tolerating oral medications. Recheck blood work showed static hyponatremia (143.3 mmol/L, [RI 148.0–159.0 mmol/L]) and hypoalbuminemia (1.8 g/dL, [RI 2.8–4.0 g/dL]) and mild ionized hypocalcemia (1.08 mmol/L, [RI 1.25–1.50 mmol/L]). An additional dose of calcium gluconate was administered intravenously as previously outlined. The patient was discharged home.

At follow-up 1 month later, the cat was reportedly doing well clinically following discontinuation of enrofloxacin and calcium carbonate supplementation. Recheck ultrasound showed no evidence of emphysematous hepatitis and resolving pancreatitis. Two months following discharge, the cat had a recheck abdominal ultrasound which showed nearly resolved pancreatitis with no evidence of emphysematous hepatitis. The cat was clinically doing well; therefore, only long-acting insulin therapy was continued. At last known recheck 3 months post discharge, the patient was reportedly doing well at home on insulin therapy with no recurrence of clinical signs. At the time of submission of this paper, the patient was reportedly alive and doing well.

3 | DISCUSSION

Emphysematous hepatitis (EH) is a condition that is rarely reported in both the human and veterinary literature. EH is characterized by hepatic parenchyma replacement by gas followed by inflammation of the liver which often progresses to fulminant acute liver failure.[6] To the best of our knowledge, only 12 cases are reported in the human literature with two patients treated successfully: one via urgent laparotomy with surgical debridement and supportive therapy and one via peritoneal lavage and supportive therapy. In the remaining human reports, 10 patients succumbed to severe multiple organ failure and septic shock within 3 days of hospital admission (Table 2).[3,4,16]

In the human literature, EH is reported predominantly in women with diabetes mellitus and/or liver disease as a predisposing comorbidity.[3,6] Other cases are reported in patients with neoplasia: cholangiocarcinoma and metastatic pancreatic neoplasia.[13,16] Clinical manifestation is nonspecific with abdominal pain and fever most commonly reported. Diagnosis is confirmed via the presence of hepatic parenchymal gas on CT without evidence of intrahepatic fluid.[1–6] This differs in the veterinary literature where abdominal ultrasound is the modality used most commonly for diagnosis followed by abdominal radiography. This may be due to the need for sedation in our veterinary patients to complete abdominal or thoracic CTs or due to financial limitations. Combined mortality rates around 41% have been seen in veterinary patients that have hepatic portal venous gas and hepatic parenchymal emphysema.[17] When this is subdivided, hepatic parenchymal gas as a diagnostic finding is a negative prognostic indicator with a mortality rate of 90%, similar to human reports.[17]

Human emphysematous abdominal infections have also been reported in the stomach, pancreas, gallbladder, uterus, and urinary tract and have been shown to share features with emphysematous pyelonephritis.[3,13,16] Bacterial infections cultured in emphysematous pyelonephritis have also been found in EH and include *Eschericia coli*, and genera *Clostridium*, *Enterobacter*, *Klebsiella*, *Proteus*, *Pseudomonas*, and *Streptococcus*.[3,7–9,11,13,14] *Aerobacter aerogenes* has been isolated in cases of emphysematous pyelonephritis and cystitis.[14] Emphysematous infection pathophysiology is thought to be a result of bacterial tissue fermentation. Bacterial tissue fermentation results in the production of nitrogen (60%), hydrogen (15%), carbon dioxide (5%), and oxygen (5%).[3,15] This theory differs from the mechanical theory which suggests that gas present in an organ or portal venous system is secondary to abnormal or disrupted intestinal mucosa, increased intraluminal pressure, or intestinal abscess resulting in gas diffusion.[10–12] Diabetes mellitus, by providing a high level of glucose substrate for organisms, has been known to predispose to emphysematous infections.[1–4] Diabetes mellitus has also been implemented as a risk factor for microangiopathy which can lead to accumulation of gas products given slow catabolic product transport.[3,14] Despite impairments noted with diabetes mellitus, Francois, et al. reported successful treatment of EH in a diabetic human patient with a well-controlled glycemic state.[3]

Sato and Solano (1998) reported on one of two veterinary cases of a patient with emphysematous hepatitis diagnosed in a 12-year-old male intact malamute.[18] The dog presented in a clinically similar state as the cat reported above: for vomiting, lethargy, unwillingness to rise on physical examination, and pyrexia. On abdominal radiographs the dog was noted to have a 12 cm gas-filled mass in the region of the right liver lobes. Differential diagnosis consisted of hepatic abscess, necrotic hepatic neoplasia, and emphysematous cholecystitis. The dog was humanely euthanized given guarded prognosis, and necropsy was performed. Necropsy revealed serosanguinous peritoneal

TABLE 2 Emphysematous hepatitis case reports in human literature.

Reference	Year	Age/sex	Comorbidities	Treatment	Pathogen(s)	Outcome
Blachar et al.[22]	2002	43y/F	Diabetes mellitus, hyperlipidemia, short gut syndrome, and peripheral vascular disease	IV antibiotics, radiographic guided drainage	Blood and liver aspirate: *Klebsiella pneumoniae*	Died 3 days post admission
Lopez, et al[23]	2006	72y/F	Diabetes mellitus	NA	Postmortem liver lesion culture: *Klebsiella oxytoca*	Died 24 h post admission
Létourneau-Guillon et al[24]	2010	53y/M	3 months prior to admission—left hepatectomy with hepaticojejunostomy for hilar cholangiocarcinoma; No adjuvant chemotherapy 1 week before admission: Cellulitis at surgical incision treated with cephalexin	IV antibiotics	Blood culture: *Enterobacter cloacae*, *Clostridium perfringens*	Died 36 h post admission
Chauhan et al[1]	2012	77y/F	Diabetes mellitus	IV antibiotics, radiographic guided drainage	NA: lack of postmortem analysis consent	Died 3 days post admission
Kim et al[8]	2012	80y/F	Hilar cholangiocarcinoma; ERCP with stenting was performed 3 months prior to admission followed by radiotherapy for 17 days after admission	IV antibiotics, radiographic guided drainage	Blood culture: *Clostridium perfringens*, *Escherichia coli*	Died 3 days post admission
Dimitriou et al[2]	2014	72y/M	Diabetes mellitus	IV antibiotics	NA	Died within hours post admission
Nada et al[4]	2017	73y/F	Pancreatic adenocarcinoma: Whipple performed 8 months prior to admission. Lung and liver metastasis diagnosed 6 weeks prior to admission. COPD, hypertension, chronic hepatitis C, pulmonary embolism	IV antibiotics	Blood culture: *Streptococcus mutans, Enterococcus faecalis*	Died within 24 h post admission
Ghosn et al[13]	2019	38y/F	Diabetes mellitus, cholecystectomy	IV antibiotics, emergent laparotomy	Perioperative fluid: *Escherichia coli*, *Enterococcus faecium*	Survived. Discharged 13 days post admission
Calderon et al[5]	2020	80y/F	Hypertension, diabetes mellitus, and chronic renal disease	IV antibiotics	Blood culture: *Clostridium perfringens*	Died within 16 h post admission
Azri et al[25]	2020	75y/F	Hilar cholangiocarcinoma; ERCP with stenting 14 months prior to admission followed by stereotactic radiotherapy until 4 months prior to admission	NA	Blood culture: *Klebsiella pneumoniae*, *Escherichia coli*, *Enterococcus faecalis*, *Clostridium perfringens*, *Aeromonas ichtiosmia*	Died
Miranda et al[6]	2020	74y/M	Hypertension, gastroesophageal reflux	IV antibiotics	Blood culture: *Escherichia coli*	Died 3 days post admission
Francois et al[3]	2022	70y/F	Diabetes mellitus, cholecystectomy, heterozygote alpha-1 antitrypsin deficiency	IV antibiotics, CT—guided drainage	Blood and fluid cultures: *Escherichia coli*, *Streptococcus anginosus*, *Klebsiella oxytoca*	Survived. Discharged 1 month post admission

Note: Table adapted from Francois, et al.[3]

Abbreviations: COPD, Congestive obstructive pulmonary disease; D, days; ERCP, Endoscopic retrograde cholangiopancreatography; F, Female; H, hours; NA: Not available; M, Male;Y, year.

effusion and quadrate liver lobe torsion with capsular and parenchymal tears. Peritoneal effusion cytology revealed rod shaped bacteria consistent with *Clostridium* or *Bacillus* species. The patient's final diagnosis was "acute torsion and entrapment of the quadrate lobe resulting in hepatic rupture and peritonitis".[18] With no hepatic abscess found, it was speculated that bacterial hepatitis was secondary to vascular compromise.[18]

The other reported case of canine emphysematous hepatitis occurred in a 16–year-old mixed breed female canine with neurologic signs (ataxia, circling, head tilt, abnormal vocalization, mental depression, and strabismus of the left eye) and elevated liver enzymes.[19] The attending clinician was concerned for hepatic encephalopathy necessitating an abdominal ultrasound. The abdominal ultrasound revealed a liver with heterogeneous echotexture and focal areas of hyperechogenicity with posterior reverberation but lacking twinkle artifact, which are typical ultrasound finding with emphysematous disease of other organs including but not limited to the gallbladder, kidney, and urinary bladder.[19] These findings are consistent with the abdominal ultrasound findings in our reported case. The author did note that CT is considered the most specific and sensitive modality to evaluate emphysematous changes.[19]

Other emphysematous infections reported in the veterinary literature include emphysematous cystitis, gastritis, intestinalis/colitis, pyelonephritis, and splenitis.[7–15,18] Emphysematous gastritis (EG) is another life-threatening disease caused by gastric wall infiltration by gas-forming bacteria.[8,9] Predisposing factors in the human field include pancreatitis, recent gastrointestinal surgery, immunosuppression, ingestion of caustic substances, alcoholism, recent nonsteroidal anti-inflammatory ingestion, diabetes mellitus, renal failure, neoplasia, and gastroenteritis.[8,9] Gastric mucosal erosion and gastric wall necrosis are common endoscopic findings. The ischemic findings are thought to be secondary to vascular occlusion, gastric infarction, or gastric volvulus. Isolated organisms were similar to those isolated in human EH: *Streptococci* sp., *Escherichia coli*, *Enterobacter* spp., *Clostridium welchii*, *Staphylococcus aureus*, and *Candida* spp. In a retrospective review of all adult cases, they found that in 42.2% of cases an organism was not identified and in 20.3% of cases a polymicrobial infection was isolated.[8] In the cases where an infectious agent was identified, *Clostridium* was the most common single isolate (17.6%). This may suggest that polymicrobial infections may be more commonly seen in emphysematous diseases such as EH. All the animals in Theirry, et al were euthanized due to clinical deterioration which correlates to the high mortality rate in human medicine (40%–60% due to sepsis).[8]

Emphysematous splenitis (ES) has also been reported in the veterinary and human literature. ES was reported in three golden retriever dogs by Battiato, et al.[7] It is also rarely reported in the human literature but is known to also be a life-threatening disease process. Emphysematous splenitis has been reported secondary to immunosuppression and diabetes mellitus, leading to bacterial translocation from the mesenteric to splenic vein, or idiopathic. Etiologies remain similar to other emphysematous disease processes. The patients were treated with total splenectomy and peritoneal lavage. All three cases had splenic culture performed which yielded *Clostridium* spp. growth. One dog's hepatic biopsy and culture found growth of *Staphylococcus pseudintermedius* further showing similarities between etiologies of emphysematous infections. Two of the dogs were treated successfully with IV fluids and antibiotics (not further specified) and discharged within a few days. The dog diagnosed with the polymicrobial infection/severe hepatitis succumbed to septicemia in the perioperative period.[7]

Emphysematous cystitis (EC) and emphysematous pyelonephritis (EPN) have been reported in both the human and veterinary literature. Similar to the previously discussed emphysematous diseases, EC is characterized by the presence of gas within the wall of the urinary bladder, bladder lumen, or both. EPN is characterized by the presence of gas extending into the renal collecting system or parenchyma and perirenal tissues. Definitive diagnosis is made via abdominal radiographs and/or abdominal ultrasound. In over 50% of the EC cases published in the veterinary literature, diabetes mellitus was a concurrent diagnosis.[20] Alternatively, a recent review of 27 dogs with emphysematous cystitis found diabetes mellitus in 33% of those dogs.[15] The prevalence of EC and EPN in diabetic patients is also thought to be secondary to glycosuria and impaired immune function as suspected in other emphysematous diseases. Abdominal radiographs were diagnostic in a few cases of EC and EPN. Of those that were not confirmatory, abdominal ultrasound showed similar hyperechoic foci with reverberation artifacts seen in EH and other emphysematous disease processes. The predominant bacterial isolate noted in EC was *E. coli*, which is similar to other emphysematous diseases.[15,20] The other isolates were reported less than approximately 11% of the time and included *Proteus mirabilis*, *Enterococcus faecalis*, *Staphylococcus aureus*, *Enterobacter cloacae*, *Streptococcus* spp., and *Klebsiella pneumoniae*.[15,20] Given that many nondiabetic patients were reported by Lippi, et al. and a single case reported by Moon, et al., they suspected urinary lactulose or albumin may also act as substrates for gas formation, unlike in other reported emphysematous diseases.[15,20]

Accordingly, EC may occur more frequently with chronic urinary tract infections or any disease process that results in immune system impairment rather than diabetes mellitus.[15,20]

4 | CONCLUSIONS

In conclusion, emphysematous hepatitis is a potentially life-threatening liver infection as reported in the human medical literature. Although few case reports exist on emphysematous disease in veterinary patients with no previously published reports of feline emphysematous hepatitis, this case reports the successful treatment of emphysematous hepatitis in a feline patient.

AUTHOR CONTRIBUTIONS

Corinna Palko: Conceptualization; formal analysis; resources; writing – original draft. **Samuel Durkan:** Supervision; writing – review and editing.

FUNDING INFORMATION

The authors received no grant from any funding agency in the public, commercial, or not-for-profit sectors for preparation of this case report.

CONFLICT OF INTEREST STATEMENT

The authors do not have potential conflicts of interest to declare.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

ETHICS STATEMENT

The work described in this manuscript involved the use of nonexperimental (owned or unowned) animals. Established internationally recognized high standards ("best practice") of veterinary clinical care for the individual patient were always followed. Ethical approval from a committee was therefore not specifically required for publication.

CONSENT

Informed consent (verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work for all procedure(s) undertaken. No animals or people are identifiable within this publication, and therefore additional informed consent for publication was not required.

ORCID

Corinna Palko https://orcid.org/0009-0000-4990-5968

REFERENCES

1. Chauhan U, Prabhu SM, Shetty GS, Solanki RS, Udiya AK, Singh A. Emphysematous hepatitis—a fatal infection in diabetic patients: case report. *Clin Res Hepatol Gastroenterol*. 2012;6(6):e114-e116. doi:10.1016/j.clinre.2012.05.018
2. Dimitriou C, Arvaniti M, Kaitartzis C, Papadimitriou A, Nalmpantidou C, Katsiba D. Emphysematous hepatitis in a diabetic patient. *Eur Secur*. 2014. doi:10.1594/EURORAD/CASE.12307
3. Francois S, Aerts M, Reynaert H, et al. Step-up approach in emphysematous hepatitis: a case report. *World J Hepatol*. 2022;14(2):464-470. doi:10.4254/wjh.v14.i2.464
4. Nada KM, El Husseini I, Abu Hishmeh ME, Shah NS, Ibragimova N, Basir R. A rare case of septic shock secondary to emphysematous hepatitis. *Case Rep Crit Care*. 2017;2017:1-3. doi:10.1155/2017/3020845
5. Calderon H, Serfin J. 13-hour progression of emphysematous hepatitis as depicted on repeat computerized tomography. *J Surg Case Rep*. 2020;2020:rjaa089. doi:10.1093/jscr/rjaa08
6. Miranda G, Dionísio AC, Azevedo C, et al. Fulminant emphysematous hepatitis—a rare cause of septic shock. *Eur J Case Rep Intern Med*. 2020;7(5):001539. doi:10.12890/2020_001539
7. Battiato P, Fernandez Y, Salgüero R, et al. Spontaneous emphysematous splenitis: radiographic and ultrasonographic findings in three golden retriever dogs. *Vet Radiol Ultrasound*. 2020;62(3):309-315. doi:10.1111/vru.12941
8. Rhue KE, Biddick AA, Foltz KM, Cole RC, Newton JC, Bacek LA. Emphysematous gastritis in a cat. *J Vet Emerg Crit Care*. 2018;28(6):596-602. doi:10.1111/vec.12779
9. Thierry F, Ferreira MF, Paterson GK, Liuti T, del Pozo J. Canine and feline emphysematous gastritis may be differentiated from gastric emphysema based on clinical and imaging characteristics: five cases. *Vet Radiol Ultrasound*. 2018;60(2):136-144. doi:10.1111/vru.12691
10. Silveira C, Benigni L, Gugich K, McClaran JK. Feline gastric pneumatosis. *JFMS Open Reports*. 2018;4(2):205511691878277. doi:10.1177/2055116918782779
11. Walczak R, Paek M, Suran J, Amory JT, Specchi S, Sanchez M. Radiography and ultrasonography of pneumatosis intestinalis in a cat. *Vet Radiol Ultrasound*. 2018;61(3):E26-E30. doi:10.1111/vru.12635
12. Hutchinson KM, Tart K, Anderson KL, Powell LL. Pneumatosis of the intestines, colon, and liver in a young cat. *Vet Med Sci*. 2018;4(2):150-158. doi:10.1002/vms3.96
13. Ghosn Y, Abdallah A, Hussein Kamareddine M, et al. Gas-forming liver abscess versus emphysematous hepatitis: a radiologic diagnostic dilemma—a case report and review of the literature. *Case Reports in Hepatol*. 2019;1–4:5274525. doi:10.1155/2019/5274525
14. Gould EN, Cohen TA, Trivedi SR, Kim JY. Emphysematous pyelonephritis in a domestic shorthair cat. *JFMS Open Report*. 2019;18(4):357-363. doi:10.1177/1098612x15600481
15. Lippi I, Mannucci T, Santa DD, Barella G, Oranges M, Citi S. Emphysematous cystitis: retrospective evaluation of predisposing factors and ultrasound features in 36 dogs and 2 cats. *Can Vet J*. 2019;60:514-518.
16. Kim JH, Jung ES, Jeong SH, et al. A case of emphysematous hepatitis with spontaneous pneumoperitoneum in a patient with hilar cholangiocarcinoma. *Korean J Hepatol*. 2012;18(1):94-97. doi:10.3350/kjhep.2012.18.1.94

17. Manfredi S, Fabbi M, Bonazzi M, et al. Ultrasonographic differentiation between portal venous and parenchymal gas may be important for the prognosis of canine and feline hepatic emphysema: 37 cases. *Vet Radiol Ultrasound*. 2019;60(6):734-744. doi:10.1111/vru.12797
18. Sato AF, Solano M. Radiographic diagnosis: liver lobe entrapment and associated emphysematous hepatitis. *Vet Radiol Ultrasound*. 1998;39(2):123-124. doi:10.1111/j.1740-8261.1998.tb01977.x
19. Babicsak VR, Santos DR, Zardo KM, et al. Ultrasonographic findings of emphysematous hepatitis in a dog. *Vet Radiol Ultrasound*. 2013;54(4):412.
20. Moon R, Biller DS, Smee NM. Emphysematous cystitis and pyelonephritis in a nondiabetic dog and a diabetic cat. *J Am Anim Hosp Assoc*. 2014;50(2):124-129. doi:10.5326/jaaha-ms-5972
21. Lisciandor GR, Lagutchick MS, Mann KA, et al. Evaluation of an abdominal fluid scoring system determined using abdominal focused assessment with sonography for trauma in 101 dogs with motor trauma. *J Vet Emerg Crit Care*. 2009;19(5):426-437. doi:10.1111/j.1476-4431.2009.00459.x
22. Blachar A, Federle MP, Brancatelli G. Acute fulminant hepatic infection causing fatal "emphysematous hepatitis". *Abdom Imaging*. 2002;27:188-190. doi:10.1007/s00261-001-0067-y
23. Lopez Zarraga F, Aisa P, Saenz de Ormijana J, et al. Fulminant infection with emphysematous changes in the biliary tract and air-filled liver abscesses. *Abdom Imaging*. 2006;31:90-93. doi:10.1007/s00261-005-0346-0
24. Letourneau-Guillon L, Audet P, Plasse M, Lepanto L. Answer to case of the month #162. Emphysematous infection of the liver parenchyma. *Can Assoc Radiol J*. 2012;61:117-119. doi:10.1016/j.carj.2009.10.003
25. Azri A, Pichon J, Fartoukha M, Djibré M. Fatal emphysematous hepatitis with spontaneous pneumoperitoneum. *Liver Int*. 2020;40:1224. doi:10.1111/liv.14450

How to cite this article: Palko C, Durkan S. Medical management of feline emphysematous hepatitis—A case report. *Clin Case Rep*. 2023;11:e7352. doi:10.1002/ccr3.7352