

Case Report

Pneumatosis of the intestines, colon and liver in a young cat

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Abstract

To describe a case of naturally occurring pneumatosis intestinalis, pneumatosis coli and emphysematous hepatitis in a cat. A 9-month-old, indoors-only, female spayed, domestic medium hair cat presented for vomiting, open-mouth breathing and acute collapse. The initial physical examination identified moderate to severe hypothermia [35°C (95°F)], obtunded mentation, weak femoral pulses, tachycardia (heart rate 240 beats per min), pale pink mucous membranes and significant splenomegaly on abdominal palpation. Immediate diagnostics performed [packed cell volume and total solids (PCV, TS), venous blood gas and electrolytes] revealed severe anaemia (PCV 12%), hypoproteinaemia (TS = 2.2 g/dl), and severe metabolic acidosis (pH 6.956). Additional diagnostics performed included Feline Leukaemia Virus and Feline Immunodeficiency Virus testing (FeLV/FIV), complete blood count (CBC) with pathology review, serum biochemistry profile, prothrombin time (PT) and partial thromboplastin time (PTT), urinalysis, and abdominal radiographs. Abdominal radiographs were consistent with gas within hepatic and splenic veins and parenchyma, small intestinal walls and colonic wall. Due to the guarded prognosis, euthanasia was elected. Necropsy was performed and the most significant gross and histopathological findings included intra-luminal and intra-mural intestinal haemorrhage and vascular congestion with mild neutrophilic hepatitis, and marked hepatic periportal emphysema. *Clostridium perfringens* and *Escherichia coli* were cultured from the bowel wall; no bacterial growth from the liver or spleen was identified. This case report describes idiopathic emphysematous hepatitis, with concurrent emphysema of the spleen and intestinal wall and intestinal haemorrhage. To the authors' knowledge, this type of pathology in a feline patient has not been previously described.

Keywords: Pneumatosis, emphysematous, hepatic portal venous gas (HPVG).

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Introduction

Pneumatosis, the presence of “air or gas in abnormal locations in the body (Studdert *et al.* 2012),” is an uncommon but well-documented occurrence in people. Pneumatosis most commonly involves the hepatic and portal venous system, but can also involve other organs such as the bowel. It was first documented in children suffering from necrotizing enteritis in 1955 (Wolfe & Evans 1955). Pneumatosis intestinalis was also reported in adults that year and was described as “abdominal gas cysts.” (Kukral *et al.* 1955) Hepatic portal venous gas (HPVG) is a form of pneumatosis and refers to the presence of gas in the portal vein. Even though uncommon, it should

be considered in people presenting to the emergency room with signs of acute abdomen as this condition has historically carried a high mortality rate in humans (Chan *et al.* 2005). The first published case in an adult patient was identified in 1960 (Susman & Senturia 1960), in a patient who suffered from gas embolization of the portal venous system. The diagnosis is made with radiography, ultrasound or computed tomography (CT), with CT being the gold standard. The pathogenesis is unclear. Diseases commonly associated with HPVG in humans include: necrotic bowel (72%), ulcerative colitis (8%), intra-abdominal abscess (6%), small bowel obstruction (3%) and gastric ulcer (3%) (Liebman *et al.* 1978). It most commonly occurs in older patients and those

who suffer from other co-morbidities. This case report describes a unique disease process and clinically severe presentation in a young cat with idiopathic emphysematous hepatitis, with concurrent emphysema of the intestinal wall, spleen, and small intestinal and colonic haemorrhage.

Case summary

A 9-month-old, female spayed, domestic medium hair presented to The University of Minnesota Veterinary Medical Center Emergency Service for acute collapse and an episode of vomiting. The patient was indoors-only and no prior health concerns were reported. Upon physical examination at presentation, the cat was hypothermic at 35°C (95°F), tachycardic (240 beats per min) and tachypneic (50 breaths per min) with open-mouth breathing. She was obtunded, had weak femoral pulses, and pale pink mucous membranes. A large and irregular spleen was palpated. Soon after presentation an intravenous catheter was placed while flow-by oxygen was provided. The patient was hypotensive (systolic blood pressure, Doppler, 60 mmHg. Hypotension defined as systolic <80 mmHg). An intravenous fluid bolus (10 mL/kg) of an isotonic, balanced electrolyte solution, Normosol-R,¹ was initiated followed by heat support, which was provided with a warm water blanket and Bair Hugger² once the patient was more haemodynamically stable. Immediate diagnostics performed included a venous blood gas, packed cell volume and total solids (PCV, TS). Blood gas analysis performed using the iSTAT³ identified a severe metabolic acidosis pH 6.956 (reference interval 7.3–7.4), a negative base excess –23 mmol/L (reference interval –2 mmol/L to +2 mmol/L), and hyperlactataemia 15.41 mmol/L (reference interval 0.3–2.5 mmol/L). PCV/TS were 12% and 2.2 g/dl, respectively. Additional diagnostics performed included a complete blood count (CBC), serum biochemistry panel, prothrombin time (PT) and partial thromboplastin time (PTT), Feline

Leukaemia Virus and Feline Immunodeficiency Virus testing (FeLV/FIV Antibody/Antigen SNAP⁴), urinalysis, and abdominal radiographs.

The CBC (see Table 1) revealed a severe non-regenerative anaemia (hematocrit 13.3%; reference interval 29.5–47% and reticulocyte count $0.027 \times 10^6/\mu\text{L}$; reference interval $0.004\text{--}0.066 \times 10^6/\mu\text{L}$), a mild lymphocytosis ($10.28 \times 10^9/\text{L}$ [$10.28 \times 10^3/\mu\text{L}$]; reference interval $0.2\text{--}9.4 \times 10^9/\text{L}$ [$0.2\text{--}9.4 \times 10^3/\mu\text{L}$]) and mild thrombocytopenia ($91 \times 10^9/\text{L}$ [$91 \times 10^3/\mu\text{L}$]; reference range $110\text{--}413 \times 10^9/\text{L}$ [$110\text{--}413 \times 10^3/\mu\text{L}$]). Metarubricytosis, rare atypical lymphocytes, large platelets and a mild left shift (Table 1) were all noted on the pathology report. A normal neutrophil band count was present on the CBC ($0.14 \times 10^9/\text{L}$ [$0.14 \times 10^3/\mu\text{L}$]; reference range $0\text{--}0.16 \times 10^9/\text{L}$ [$0\text{--}0.16 \times 10^3/\mu\text{L}$]). The anaemia was classified as a marked normocytic, hypochromic non-regenerative anaemia.

Serum biochemistry analysis (Table 2) showed marked hypoalbuminaemia (11 g/L [1.1 g/dL]; reference range 24–41 g/L [2.4–4.1 g/dL]), hypoglobulinaemia (11 g/L [1.1 g/dL]; reference range 25–53 g/L [2.5–5.3 g/dL]), mild hypernatraemia (159 mmol/L [159 mEq/L]; reference interval 147–158 mmol/L [147–158 mEq/L]), hyperchloraemia (130 mmol/L [130 mEq/L]; reference interval 113–123 mmol/L [113–123 mEq/L]), hypermagnesaemia (1.2 mmol/L [2.8 mg/dL]; reference range 0.7–1.0 mmol/L [1.6–2.4 mg/dL]), mild hypokalaemia (3.6 mmol/L [3.6 mEq/L]; reference range 3.9–5.3 mmol/L [3.9–5.3 mEq/L]), markedly elevated alanine aminotransferase (ALT) (3785 U/L; [reference interval 16–12 U/L]), aspartate aminotransferase (AST) (4884 U/L; [reference range 14–42 U/L]) and creatine kinase (1054 U/L; [reference range 54–744 U/L]), and marked hypocholesterolaemia (0.67 mmol/L [26 mg/dL]; reference interval 1.4–5.8 mmol/L [56–226 mg/dL]). PT/PTT showed marked elevation, with a PT of 30.0 s (reference range 7.4–12.8 s) and a PTT of 57.9 s (reference 11.1–16.4 s). The patient was negative for FeLV/FIV. Urinalysis (free catch) identified an elevated specific gravity (1.062), 1 + bilirubin, 1 + blood, 2 + protein, many rod-shaped

¹Normosol®- R, Pfizer (Hospira, Inc), NY, NY

²Bair Hugger, 3M, Maplewood, MN (Minnesota)

³iSTAT, Abbott Point of Care, Princeton, NJ

⁴SNAP® IDEXX Laboratories, Inc. Westbrook, ME (Maine)

Table 1. Complete blood count

Test result	Patient, conventional	Reference interval, conventional	Patient, SI units	Reference interval, SI units
WBC	13.52	1.83–16.27 × 10 ³ /μL	13.52	1.83–16.27 × 10 ⁹ /L
Neutrophil Segs	2.84	1.2–13.2 × 10 ³ /μL	2.84	1.2–13.2 × 10 ⁹ /L
Neutrophil Bands	0.14	0–0.16 × 10 ³ /μL	0.14	0–0.16 × 10 ⁹ /L
Lymphocytes	10.28	0.2–9.4 × 10 ³ /μL	10.28	0.2–9.4 × 10 ⁹ /L
Monocytes	0.00	0–0.8 × 10 ³ /μL	0.00	0–0.8 × 10 ⁹ /L
Eosinophils	0.27	0–1.9 × 10 ³ /μL	0.27	0–1.9 × 10 ⁹ /L
Basophils	0.00	0–0.3 × 10 ³ /μL	0.00	0–0.3 × 10 ⁹ /L
RBC	2.70	6.44–10.36 × 10 ⁶ /μL	2.7	6.44–10.36 × 10 ¹² /L
Hematocrit	13.3	29.5–47	13.3	29.5–47
MCV	49.0	37.4–50.4 fL	49.0	37.4–50.4 fL
MCH	14.9	14–18 pg	14.9	14–18 pg
MCHC	30.3	32.1–39.7 g/dL	303	321–397 × 10 g/L
RDW	15.4	%; No range	15.4	%; No range
Retic #	0.027	0.004–0.066 × 10 ⁶ /uL	0.027	0.004–0.066 × 10 ⁹ /L
Retic %	0.99	0.1–0.8%	0.99	0.1–0.8%
Platelet	91	110–413 × 10 ³ /μL	91	110–413 × 10 ⁹ /L
MPV	14.4	9.9–21.5 fL	14.4	9.9–21.5 fL
Nucleated red blood cells	11	/100 WBC; No range	11	/100 WBC; No range

Conventional units are used in the United States. Smear comment: RBC Morphology WBC Morphology Platelet Morphology 1+ Anisocytosis, 1+ echinocytes, 1+ polychromasia, rare RBC precursors 1+ reactive lymphocytes, 1+ smudged WBCs, rare large platelets.

bacteria per high powered field (HPF), moderate coccoid bacteria per HPF, occasional yeast per HPF and 0–5 WBC per HPF. No intracellular bacteria were identified. Two-view abdominal radiographs (Fig. 1a,b) were obtained and revealed gas opacity throughout the parenchyma of the liver and spleen and gas within the walls of gas-dilated small intestines and colon. Gas was also suspected to be in the portal vein and splenic vessels. The visible thorax may have had an excessive bronchial pattern.

Shortly after presentation the patient was humanely euthanized due to the severity of clinical signs, diagnostic results, and a suspected guarded prognosis. Necropsy findings included a diffusely pale brown to tan-coloured liver with a prominent reticular pattern and markedly reddened small and large intestines distended with gas. Throughout the small intestines and colon, red mucoid material, consistent with haemorrhage, was found. The mucosa of the small and large bowel was also thickened and congested (dark red to purple). The spleen was dark red and diffusely enlarged with rounded edges, and the mesenteric lymph were prominent and mottled tan and dark red in colour.

Histopathology identified that 40% of total hepatic parenchymal surface area was replaced by variably

sized (30–500 μm diameter) circular clear areas, which contained small amounts of smooth to slightly granular pale eosinophilic material. These areas were interpreted as gas with small amount of proteinaceous fluids and although mainly centred around portal areas, they were also randomly distributed in mid-zonal regions. There was also a small to moderate number of non-degenerate and poorly preserved neutrophils within the sinusoids surrounding a few of the gas bubbles. The abdominal lymph nodes contained haemorrhage and the pancreatic vessels were dilated and congested. The small and large intestinal serosa was moderately reddened and the intestines were distended with gas. The vessels in the stomach, duodenum, jejunum, ileum and colon were diffusely dilated and congested, the intestines contained intraluminal haemorrhage and there was extensive extravasation of erythrocytes. A small number of basophilic intralesional spiriform bacteria presumed to be *Helicobacter* were present within the superficial gastric pits. A moderate number of basophilic 1 μm x 6 μm bacilli (most consistent with Enterobacteriaceae-family bacteria) and a small number of basophilic 2 μm x 5 μm bacilli (most consistent with *Clostridium spp.*) were identified within the crypts of the jejunum and ileum. Bacteriology

Table 2. Biochemistry profile

Test	Patient, conventional units	Reference interval, conventional units	Patient, SI units	Reference interval, SI units
Blood Urea Nitrogen	25	12–39 mg/dL	8.9	4.3–13.9 mmol/L
Creatinine	1.1	0.5–2.1 mg/dL	97.2	44–185.6 μ mol/L
Phosphorus	5.4	3.3–7.8 mg/dL	1.9	1–2.5 mmol/L
Magnesium	2.8	1.6–2.4 mg/dL	1.2	0.7–1 mmol/L
Total protein	2.2	5.9–8.2 g/dL	22	59–82 g/L
Albumin	1.1	2.4–4.1 g/dL	11	24–41 g/L
Globulin	1.1	2.5–5.3 g/dL	11	25–53 g/L
Sodium	159	147–158 mmol/L	159	147–158 mEq/L
Chloride	130	113–123 mmol/L	130	113–123 mEq/L
Potassium	3.6	3.9–5.3 mmol/L	3.6	3.9–5.3 mEq/L
Bicarbonate	10.4	12–20 mmol/L	10.4	12–20 mEq/L
Osmolality	321	298–319 mOsm/kg	321	298–319 mmol/L
Anion gap	22	19–30	22	19–30
Bilirubin, total	0.1	0–0.3 mg/dL	1.7	0.0–5.1 μ mol/L
Alkaline phosphatase	62	2–88 U/L	62	2–88 U/L
Gamma-glutamyl transferase	<3	0–3 U/L	<3	0–3 U/L
Alanine transferase	3785	16–127 U/L	3785	16–127 U/L
Aspartate transferase	4884	14–42 U/L	4884	14–42 U/L
Creatine kinase	1054	54–744 U/L	1054	54–744 U/L
Glucose	127	74–143 mg/dL	7.0	4.1–7.9 mmol/L
Cholesterol	26	56–226 mg/dL	0.67	1.4–5.8 mmol/L
Amylase	190	555–1600 U/L	190	555–1600 U/L

identified *Clostridium perfringens* amongst cellular debris in the autolytic luminal epithelium of the small intestine and colon on anaerobic culture. Beta-hemolytic *E. coli* were found within the intestinal crypts and were often found closely associated with crypt epithelium on aerobic culture. No pathogens were identified in the liver macroscopically or on aerobic and anaerobic culture. The patient's lungs were soft, mottled red and pink and floated in formalin. There were no significant microscopic lesions present and there was no growth on aerobic and anaerobic culture. Unfortunately, the luminal aspect of the intestinal walls were autolytic and uninterpretable, with desquamation of the luminal fourth of the duodenal villi, and marked postmortem autolysis of the jejunal and ileal villi.

Discussion

The pathogenesis of pneumatosis remains unclear. Two theories have been proposed based on known predisposing factors, including intestinal wall alteration, bowel distention, ischaemia and sepsis

(Algahtani *et al.* 2007). The first theory, The Mechanical Theory, suggests that the presence of gas in organs and/or in the portal venous system is secondary to gas diffusion from abnormal or disrupted bowel mucosa or from an intestinal abscess (Liebman *et al.* 1978; Yumamuro & Ponsky 2000). Extension of intraluminal gas into the portomesenteric venous system can occur with any disease process that damages the underlying intestinal mucosa; however, this is more common in human patients with underlying intestinal ischaemia (Kinoshita *et al.* 2001; Fujii *et al.* 2003; Algahtani *et al.* 2007). This theory is supported by the fact that approximately 85% of human patients diagnosed with the HPV form of pneumatosis also suffer from concurrent bowel ulcerations (infarcted bowel, ulcerative colitis, peptic ulcer disease), bowel distention and bowel infection (Liebman *et al.* 1978). Interestingly, there have been isolated case reports of people with respiratory disease, such as chronic obstruction pulmonary disease), who have developed HPV secondary to dyspnoea and aerophagia (Wong & Lien 2014). The patient in this report also presented

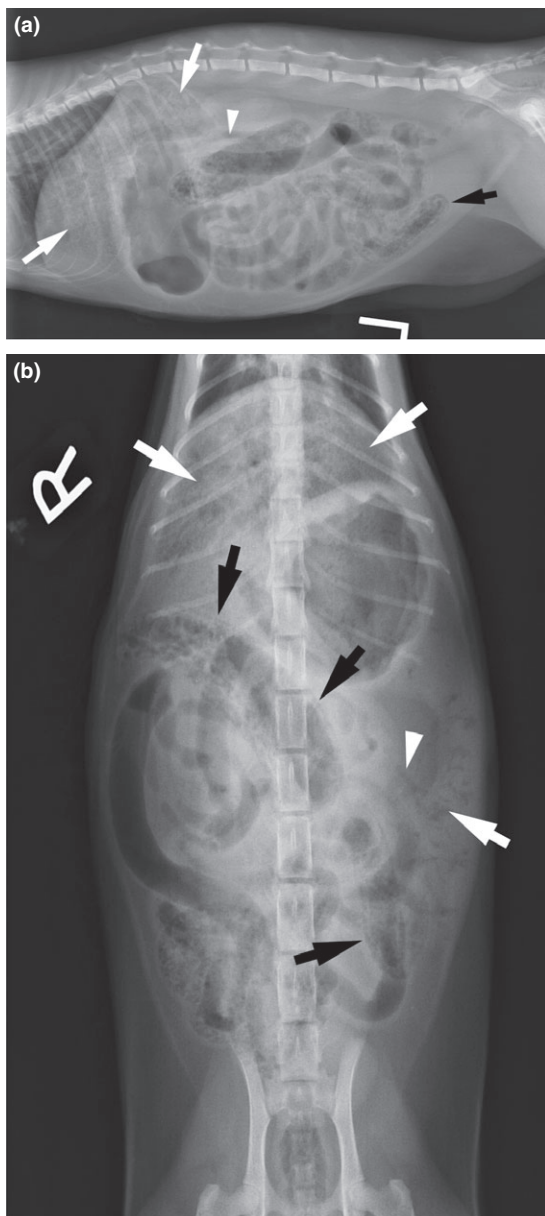


Fig. 1. Left lateral (a) and ventrodorsal (b) abdominal radiographs of a 9-month-old cat with gas opacity present throughout the parenchyma of the liver and portions of the spleen (white arrows), gas suspected within the walls of gas-dilated small intestines and colon (black arrows) and gas suspected within the portal and splenic veins (arrow heads).

tachypneic and dyspneic. The part of the lungs visualized on the abdominal radiographs show that the cat may have had an excessive bronchial pattern and underlying disease process, however, since thoracic

radiographs were not obtained, it is impossible to comment additionally on this. The patient's lungs were grossly normal and no abnormalities were documented histopathologically, however, the contribution of the dyspnoea to the development of the pneumatosis remains unclear. The second theory, The Bacterial Theory, proposes that abnormal gas production in the intestines is secondary to gas-producing bacteria that migrate through the submucosa to the mucosa (Liebman *et al.* 1978; Yumamuro & Ponsky 2000). Patients predisposed to this are usually those suffering from the same illnesses listed above (e.g., bowel ischaemia, bowel distention and mucosal damage).

Pneumatosis is not a primary disease process, but is secondarily caused by a variety of illnesses ranging from fairly benign processes to lethal ones; the prognosis is tied to the underlying disease process. Pneumatosis of intra-abdominal organs and the portal venous system has been described in human patients suffering from a variety of non-gastrointestinal illnesses as well; any condition that may lead to ileus and intestinal dilation secondary to decreased blood flow and perfusion can possibly lead to intestinal mucosal damage and bacterial replication. It is common for patients with HPVG to concurrently suffer from pneumatosis intestinalis and the prognosis for this subset of patients is grave (Wu & Liang 2008). The most common pathogens associated with emphysematous disease are *Clostridium spp.* and *E. coli*; however, *Streptococcal spp.*, *Enterobacter*, and *Staphylococcus aureus* have been all been identified (1970). Most human patients suffer from concurrent underlying diseases which predispose them to the development of HPVG, including chronic renal disease, hypertension, immunosuppression, diabetes mellitus, recent gastrointestinal surgery, alcoholism, and ischaemic heart disease (Chan *et al.* 2005). Iatrogenic causes include blunt abdominal trauma (Stuijvenberg *et al.* 2009), complications following intestinal surgery (Sicard *et al.* 1976), barium enema (Cho *et al.* 1990) and enteric tube placement (Knechtle *et al.* 1990).

Diagnosis of pneumatosis can be made with survey abdominal radiographs, abdominal ultrasound (with colour flow Doppler), and abdominal CT. Most

patients will undergo abdominal radiographs as part of the initial diagnostic work-up, followed by ultrasound and then CT if needed. Radiographically, HPVG can be identified as branching gas radiolucencies present near the liver capsule. Gas present in the intestinal wall is commonly found radiographically as crescent-shaped gas bubbles in the bowel wall (Liebman *et al.* 1978). On ultrasound, an experienced radiologist may be able to identify echogenic particles within the hepatic portal vein or the non-dependent portion of the parenchyma (Abboud *et al.* 2009). However, CT remains the gold-standard and the findings mirror those on radiographs. CT carries a higher sensitivity for detecting gas lucencies and is more able to identify any additional abnormalities.

Treatment is aimed towards addressing the primary disease process. Mortality rates have been reported to be as high as 75–80%; (Cambria & Margolies 1982) however, recent reports document a much lower mortality rate of 25–35% (Faberman & Mayo-Smith 1997). The highest survival rate may be seen in patients suffering from iatrogenic pneumatosis. Historically, treatment recommendations involved early exploratory laparotomy and aggressive management; however, this is currently only recommended in cases where necrotic bowel is suspected or identified. Instead, aggressive medical management is warranted in non-surgical cases and includes hospitalization with intravenous fluids, antibiotics, analgesia and close monitoring. If the patient deteriorates during medical management, re-imaging and exploratory laparotomy should be considered.

Pneumatosis has rarely been reported in veterinary patients. Similar to diabetic humans, pneumatosis has also been documented in diabetic dogs; specifically, emphysematous cystitis (Root & Scott 1971) and emphysematous cholecystitis (Armstrong *et al.* 2000). Pneumatosis is suspected to occur in diabetes secondary to glucose fermentation (Lord & Wilkins 1972) and overgrowth of bacteria. The most commonly cultured bacteria include *Clostridium perfringens* and *E. coli* (Lord & Wilkins 1972; Loar & Deluca 1988; Avgeris & Hoskinson 1992; Alvarez *et al.* 1994; Gill *et al.* 1997). Additional cases of pneumatosis have been documented in other

scenarios such as: emphysematous hepatitis in a case of liver lobe torsion in a dog (Sato & Solano 1998), portal venous gas in a dog secondary to hydrogen peroxide administration (Faverzani *et al.* 2009), gastric pneumatosis with gastric dilation and volvulus (Fischetti *et al.* 2004) and pneumatosis coli (Morris 1992). Pneumatosis has also been documented in two feline patients; in the stomach in one (Lang *et al.* 2011) and the orbit in the other (Meomartino *et al.* 2015). In both cases, the pneumatosis occurred as a post-operative complication. In the first mentioned case, a 9-year-old Siamese cat was diagnosed and treated for post-operative gastric pneumatosis following gastrotomy and enterotomy for multiple gastric foreign bodies (Lang *et al.* 2011). In the second case, a 2-year-old neutered male domestic short-haired cat was diagnosed with orbital pneumatosis secondary to a patent nasolacrimal duct following enucleation (Meomartino *et al.* 2015). Interestingly, pneumatosis appears to be most common in human adults whereas in veterinary medicine, young patients, such as the one presented in this case report, are most commonly affected.

A peripheral blood smear of the patient presented here was evaluated by a pathologist and consisted of a markedly decreased RBC density, adequate leukocyte density, and mildly decreased platelet density. The leukocyte population identified in this cat was composed of a mildly increased number of lymphocytes, neutrophils and eosinophils. Even though a normal band count was estimated on the automated analyser, the pathologist identified a mild increase in band neutrophils. Pathology review described the lymphocytes as generally appearing small and well differentiated with scant cytoplasm and condensed chromatin, however, there were rare intermediate lymphocytes that were larger than neutrophils, with smoother chromatin and undulating nuclear margins. Lymphocytosis may have been associated with chronic inflammation or, given the few circulating atypical lymphocytes, an emerging neoplastic process. Infectious diseases such as Toxoplasmosis and FeLV are other considerations for a lymphocytosis.

The red blood cell population was characterized by minimal anisocytosis and polychromasia, occasional metarubricytes and scattered echinocytes. The

red blood cell population was classified as a marked normocytic hypochromic non-regenerative anaemia. Causes of non-regenerative anaemia can be extra-marrow and intra-marrow in origin. Possible extra-marrow causes for this patient include blood loss and iron deficiency given the hypochromasia present; microcytosis was not noted, making iron deficiency less likely. Intra-marrow causes for this patient must also be considered given the bicytopenia present, and include neoplasia, fibrosis, necrosis and infectious diseases such as FeLV and FIV, which is considered less likely since the patient tested negative for these viral agents. Additionally, an immune mediated anaemia at the level of the bone marrow cannot be excluded. The metarubricytosis may be secondary to hypoxic marrow endothelial insult or bone marrow pathology. A pre-regenerative anaemia must also be considered as a differential for the apparently non-regenerative anaemia, especially with the clinical finding of haemorrhage in this patient. The platelet density appeared decreased and occasional macroplatelets are noted. The mild thrombocytopenia is likely secondary to a consumptive/inflammatory process. An intra-marrow cause of thrombocytopenia, similar to those discussed with regards to anaemia, cannot be eliminated.

Biochemically, there were significant alterations in the patient's liver enzymes (ALT, AST), as well as elevations in creatine kinase and a decreased cholesterol value. ALT and AST are leakage enzymes that can markedly increase with decreased blood flow and oxygen delivery to the liver. This patient presented in a state of shock, with a marked anaemia, both of which can contribute to an increase in ALT. Infection, neoplasia and other primary hepatic diseases can cause ALT elevations. ALT can also be affected by underlying cardiac, renal, skeletal muscle and brain disease. AST is also affected by cardiac and skeletal muscle dysfunction as well as red cell haemolysis. Histopathology of the liver in this patient identified a neutrophilic hepatitis and significant parenchymal alterations, with 40% of the liver parenchyma replaced by gas and proteinaceous fluid. Adjacent hepatocytes were compressed and frequently contained lacy pale eosinophilic material (water/glycogen). There were multifocal, linear

arborizing areas of loss of hepatocyte cohesion, loss of hepatocytes, dilated and congested sinusoids and moderate postmortem autolysis.

This patient also suffered from a coagulopathy, identified by a marked elevation in PT/PTT as described previously (elevation of >30% high-end of normal). The coagulopathy present may have been the primary cause of haemorrhage and anaemia or may have developed as a consequence of another primary disease process, such as the patient's severe hepatopathy. The ultimate correlation between and significance of the pneumatosis and abnormal coagulation is unknown at this time.

Limitations in this case presentation include the delay between euthanasia and necropsy (i.e. the cat was euthanized at approximately 5:00 PM on 5/19/14 and the necropsy was performed on 5/20/14 between 10:00 AM and 12:00 PM). This delay makes interpretation of the gas within the organs difficult as considerable intra-organ and intra-vascular gas can accumulate within a short time frame post-mortem. Gas was not present histologically within the spleen or intestinal walls, however, barbiturate euthanasia often results in massive splenic congestion which may have obliterated splenic gas pockets.

In summary, this cat suffered from an acute and clinically severe presentation of intestinal haemorrhage and vascular congestion, with mild neutrophilic hepatitis and marked hepatic periportal emphysema. Although *C. perfringens* and *E. coli* were cultured from the bowel, no pathogens were identified in the liver, biliary system, lung or spleen. Additionally, no viral (negative stain electron microscopy on faeces) or fungal organisms were seen. A definitive cause of the gas formation in this patient was not identified. *C. perfringens* and *E. coli* were isolated from the intestines and it is possible that these gas-forming pathogens contributed to the intestinal haemorrhage. However, these pathogens are also part of the normal gastrointestinal flora, so it is equally plausible that they did not play a role in this cat's illness. Furthermore, since the culture samples were collected at a similar delay post-mortem, the interpretation of these organisms is very limited as bacterial overgrowth is expected in the post-mortem period. It is suspected that the severe anaemia and hypoproteinaemia of this

patient was secondary to gastrointestinal blood loss as documented on necropsy. Intestinal necrosis cannot be ruled out as a primary cause of this patient's disease due to the postmortem autolysis of the gastrointestinal tract as mentioned previously. To the authors' knowledge, there are no similar published reports of this disease occurring in a cat.

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Conflict of interest

The authors have no conflict of interest to declare.

Ethics statement

No animals were used in this study and no outside funding was provided.

Contribution

There are no additional contributors to this manuscript.

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