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Extrahepatic biliary duct obstruction as a result of involuntary transcavitary implantation of hair in a cat

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Abstract

Case summary A 4-year-old male neutered domestic shorthair cat was referred for investigation of jaundice. The cat had a recent history of a skin laceration repair following trauma. Sequential serum biochemistry demonstrated increasing plasma bilirubin concentrations; abdominal ultrasonography revealed ongoing pancreatitis and apparent extrahepatic obstruction of the common bile duct. Exploratory laparotomy identified constriction of the common bile duct with foreign material (cat hair). The constricting band of hair was removed surgically; cholecystoduodenostomy was performed. Postsurgical quality of life is excellent with chronic treatment of tylosin, omeprazole and ursodeoxycholic acid.

Relevance and novel information To our knowledge, this is the first reported case of extrahepatic biliary duct obstruction resulting from the intra-abdominal migration of a foreign body, in this case, hair shafts. The mechanism by which this occurred was likely a combination of physical constriction by the hair shafts and subsequent foreign body reaction surrounding this. This should be included in the differential diagnosis of a cat with jaundice.

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Case description

A 4-year-old male neutered domestic shorthair cat, weighing 5.7 kg was referred for investigation and management of jaundice. One week prior to presentation at Eastside Veterinary Emergency and Specialists, the cat presented to the referring veterinarian when the owner discovered a 2–3 cm skin laceration adjacent to the xyphoid process of the sternum (cause unknown). The cat was allowed to roam freely outdoors; it had been involved in territorial aggression in the past. The laceration was explored under general anaesthesia. Following flushing with sterile 0.9% NaCl the laceration was closed and the cat treated with amoxicillin-clavulanate (Amoxyclav; Apex Laboratories) 17.5 mg/kg, PO, q12h for 1 week. The cat received tri-annual intestinal parasitic prophylaxis and core vaccines annually. It was fed commercial wet and dry cat food.

On physical examination the cat was normothermic (37.9°C). The laceration suture site appeared to be healing well. The cat's mucous membranes were jaundiced and there was abdominal discomfort noted on palpation. Marked bruising was identified on the right abdominal wall at the level of the caudal rib margins.

Differentials for prehepatic, hepatic or posthepatic causes of jaundice were considered. Immune-mediated haemolysis (secondary to tissue damage or medications) and traumatic pancreatitis leading to extrahepatic biliary duct obstruction (EHBDO) were considered the most likely differential diagnoses. Serum biochemistry, haematology and an abdominal ultrasound were undertaken to help differentiate between these causes.

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Table 1 Serial serum biochemistry observations

Analyte	Day 1	Day 2	Day 3	Day 4	Day 7	Day 10	Day 12	Day 67	RI
Sodium (mmol/l)	141.0			161.0	162.0	162.0	157.0		144.0–158.0
Potassium (mmol/l)	4.1			3.9	3.6	3.2	4.7		3.7–5.4
Chloride (mmol/l)	124.0			123.0	120.0	116.0	116.0		106.0–123.0
Glucose (mmol/l)	6.6			6.0	9.3			6.07	4.1–8.8
Urea (mmol/l)	6.1			3.9	4.9			7.6	5.7–12.9
Creatinine (µmol/l)	148.0			116.0	144.0			184.0	71–212
Calcium (mmol/l)	2.4			2.6	2.4			2.5	1.95–2.83
Phosphate (mmol/l)	1.4			2.0	1.0			1.3	1.0–2.42
Protein (total) (g/l)	80.0			81.0	78.0			80.0	57.0–89.0
Albumin (g/l)	34.0			34.0	30.0			31.0	22.0–40.0
Globulin (g/l)	46.0			47.0	48.0			49.0	28.0–51.0
Bilirubin (µmol/l)	108.0	17.0	21.0	56.0	35.0	15.0	15.0	8.0	0–15.0
ALP (IU/l)	131.0			117.0	227.0			70.0	14.0–111.0
ALT (IU/l)	>1000.0	1301.0	3000.0	2197.0	>1000.0	845.0	395.0	137.0	12.0–130.0
Cholesterol (mmol/l)	5.3			5.8	7.8			7.3	1.7–5.8
GGT (IU/l)	2			6	12			4	0–9.0

RI = reference interval; ALP = alkaline phosphatase; ALT = alanine transaminase; GGT = γ-glutamyl transpeptidase

The results of serum biochemistry analysis (IDEXX Catalyst Dx Chemistry Analyser) are presented in Table 1. Abnormalities included a marked elevation in alanine transaminase (ALT), a moderate hyperbilirubinaemia and mild elevations in alkaline phosphatase and γ-glutamyl transpeptidase (GGT). Haematology results were within the reference intervals (RI; IDEXX ProCyte Dx Haematology Analyser); specifically, the haematocrit was 32.1% (RI 30.3–52.3%). Citrated prothrombin time was 18 s (Coag Dx Analyzer) and within the RI (12–22 s). Feline immunodeficiency virus and feline leukaemia virus serology was negative (IDEXX SNAP FIV and FeLV Combo Test). Feline pancreas-specific lipase was positive (IDEXX SNAP fPL Test). Urine concentration was adequate (>1.050) and bilirubin crystalluria the only abnormality noted on urine sediment examination.

Abdominal ultrasound identified a hypoechoic pancreas surrounded by hyperechoic mesentery. The gall bladder was moderately distended, as was the proximal common bile duct (Figure 1). No free fluid was identified. Survey radiographs of the abdomen and thorax revealed no abnormalities. The clinical assessment was EHBDO secondary to pancreatitis.

Supportive treatment included intravenous fluids (5 ml/kg/h, Hartmann's Compound Sodium Lactate; Baxter Viaflex), maropitant citrate (1.0 mg/kg SC q24h, Cerenia; Zoetis), buprenorphine (10 µg/kg SC q8h, Temgesic; Rackitt Benckiser), mirtazapine (0.7 mg/kg PO q3d, Axit; Alphapharm) and amoxicillin–clavulanate (17.5 mg/kg PO q12h) was continued.

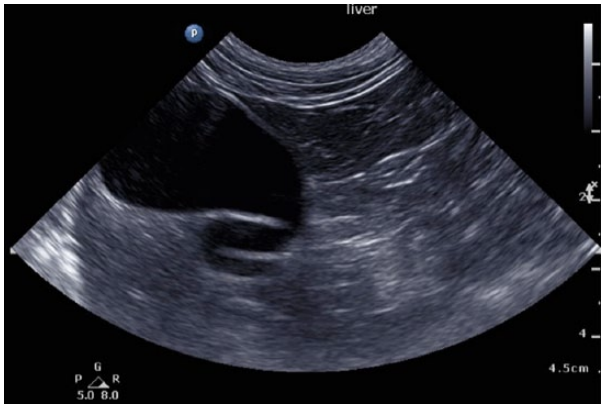

Figure 1 Ultrasound image of the liver, gall bladder and common bile duct (4.3 mm). Sonographically, a diagnosis of extrahepatic biliary duct obstruction can be made in cats when the bile duct diameter exceeds 5 mm[2]

Initial improvement in demeanour in the first 24 h was attributed to correction of dehydration, as anorexia, vomiting and abdominal pain persisted. Serum biochemistry revealed an increase in ALT and bilirubin (Table 1). Repeat abdominal ultrasonography showed no worsening of biliary duct or gall bladder distension. Mesentary around the pancreas remained hyperechoic. The treatment regimen was continued except the amoxicillin–clavulanate was substituted for a combination of clindamycin (8.7 mg/kg PO q12h, Clinacin; Intervet) and marbofloxacin (4.4 mg/kg PO q24h, Zeniquin;

Zoetis). A lincosamide and fluoroquinolone were chosen based on the high frequency of *Escherichia coli* and/or *Streptococcus* species (35%) identified using fluorescence in situ hybridisation from pancreatic sections of confirmed cases of pancreatitis in one report.[1] Prednisolone (0.4 mg/kg, PO, q24h, Microlone; Mavlab) was administered to reduce pancreatic inflammation.

By the following day, there was marked improvement, the cat was eating well and appeared comfortable. The cat was discharged from hospital with the above therapeutic regimen. It did well for 7 days at which time its appetite reduced rapidly and the owner noticed that it was icteric. Repeat abdominal ultrasound revealed marked dilation of the gall bladder and distension of the entire common bile duct (Figure 2). A small amount of free fluid was present around the common bile duct (Figure 3).

The dilation of the common bile duct suggested biliary obstruction.[2] Free fluid in the region raised a concern of biliary rupture. Owing to the cat's rapidly deteriorating clinical state, an exploratory laparotomy was performed.

During surgical exploration of the abdomen an omental adhesion was identified that surrounded the common bile duct at the level of the duodenal papilla and right pancreatic lobe. The gall bladder was unable to be expressed. The common bile duct was distended. The omental adhesion was carefully dissected away revealing foreign material. This material was a mass of what appeared to be the cat's own fur. The hair (a solid 3 mm wide band of individual hair shafts) that wrapped around the common bile duct was also removed until the common bile duct could be completely visualised.

Free peritoneal fluid was localised around the pancreas and common bile duct. It was serosanguinous in appearance. In-house microscopy of the peritoneal fluid showed a scant number of non-degenerative neutrophils. No bacteria were identified (Diff-Quik). Comparative glucose concentration between the free peritoneal fluid and peripheral venous blood (9.0 mmol/l and 6.0 mmol/l, respectively) made septic peritonitis unlikely.[3] Therefore, the non-septic peritoneal effusion was most likely the result of acute and ongoing pancreatic inflammation and/or inflammation secondary to a foreign body. This was consistent with the gross findings at exploratory laparotomy – specifically, no gastrointestinal perforation was identified. Neoplasm such as teratoma was considered unlikely to be the origin of these hair shafts given that the hair shafts did not appear to germinate from the bile duct itself.

Patency of the common bile duct was evaluated by making a 2.5 cm anti-mesenteric incision opposite the duodenal papilla. A 22 G intravenous catheter was introduced into the duodenal papilla but could not be fed more than 2 cm. Flushing with sterile saline was unsuccessful in achieving patency of the common bile duct.

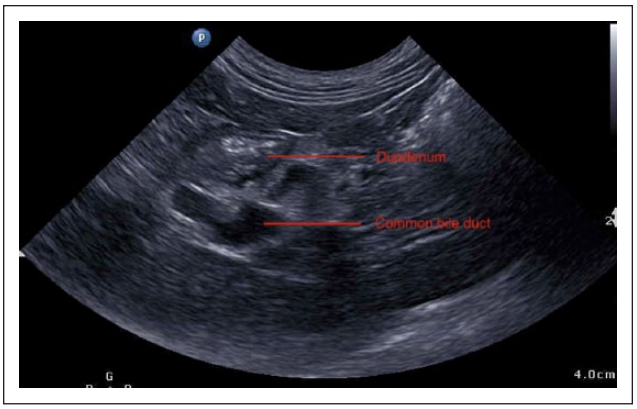

Figure 2 Distension of the common bile duct through to the duodenum

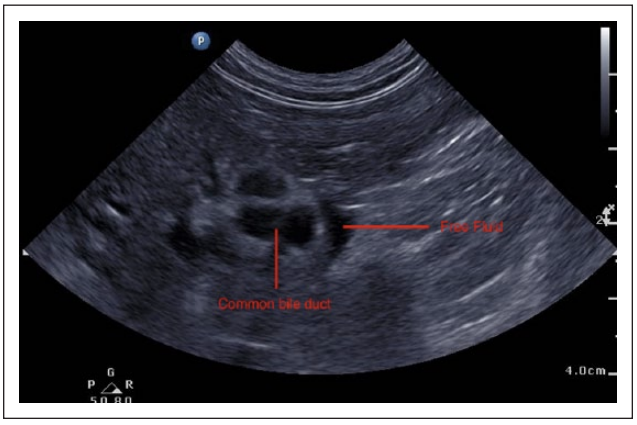

Figure 3 Ultrasound image shows free fluid around the common bile duct. Free fluid in this region raised a concern of biliary rupture

A 5 mm incision into the gall bladder was made and a 5 Fr catheter was passed from the gall bladder into the common bile duct but would not pass the obstructed area. To establish biliary patency a cholecystoduodenostomy was performed. The gall bladder was loosened from the hepatic fossa with blunt dissection. A 2.5 cm incision in the distal end of the gall bladder corresponding to the incision in the duodenum opposite the duodenal papilla was made. The two mucosal surfaces were approximated and sutured in the form of a stoma with continuous 4/0 polydioxanone (PDS; Ethicon Johnson & Johnson). There was no tension noted at the anastomosis site. The anastomosis was pressure tested. The abdomen was lavaged with saline. The linea alba, subcutaneous tissue and skin was closed in a routine manner.

A 14 Fr oesophagostomy feeding tube (Canine Esophagostomy Feeding Tube; Mila International) was placed prior to recovery from anaesthesia.

A diagnosis of EHBDO secondary to migration and entrapment of the common bile duct by autologous hair shafts was made. It was thought that the hair shafts

induced adhesions (secondary to a foreign body reaction), which contributed to biliary duct obstruction. It was presumed that hair shafts were introduced into the abdomen via the penetrating wound present at the time of presentation to the referring veterinarian.

Postsurgical recovery was rapid; serum bilirubin had normalised and ALT concentration had decreased significantly by day 10 (Table 1). Resting energy requirement was calculated at 258 kcal per 24 h, and this was achieved by gradually increasing tube feeding volumes over a 4 day period to reach 290 kcal of Hill's a/d.

The cat was discharged 5 days postsurgery on clindamycin (8.7 mg/kg PO q12h), marbofloxacin (2.2 mg/kg PO q24h), transdermal fentanyl (12.5 µg, Duragesic; Janssen-Cilag), mirtazapine (0.7 mg/kg PO every 3 days as required), ondansetron (0.2 mg/kg, PO, q12h for 10 days, Zofran Wafer; Aspen Pharmacare), omeprazole (1.8 mg/kg PO q24h, Losec; AstraZeneca) indefinitely and metoclopramide (0.4 mg/kg PO q6–8h for 7 days, Metomide; Ceva).

Nine days postoperatively, administration of marbofloxacin and clindamycin ceased. Tylosin (13.2 mg/kg, PO, q12h, Bova Compounding) was introduced to the cat's treatment regimen. Tylosin's beneficial mechanism of action in enteric disease remains unknown.[4] Despite this, the rationale of its use in this case is that, anecdotally, tylosin may help modify enteric bacterial populations favourably to reduce the likelihood of ascending bacterial cholangitis, a common complication of cholecystoduodenostomy in cats.[5]

Two months following initial presentation the patient was eating well and had gained weight but was having frequent episodes of vomiting. Bilirubin was within RI and ALT and GGT were only slightly elevated (Table 1). Ultrasound identified moderate intrahepatic biliary distention. Ursodeoxycholic acid (8.7 mg/kg PO q24h) was added to the treatment regimen (omeprazole and tylosin) to improve bile flow and reduce cholesterol saturation of bile. Following this, the owner reported a reduced frequency of vomiting. Intrahepatic biliary distension was not appreciated ultrasonographically 1 month later.

Two years following initial presentation (time of writing) the owners reported occasional vomiting (every 1–2 weeks); however, this was roughly the frequency of vomiting prior to initial presentation. Ultrasonographically, there was persistent absence of intrahepatic biliary distention.

Discussion

To our knowledge, this is the first suspected case of EHBDO as a result of migration and entrapment of the common bile duct by a foreign body in a cat. Other reported causes of EHBDO in cats include inflammatory disease, specifically cholecystitis, cholangiohepatitis, pancreatitis, cholelithiasis, hepatic lipidosis and inflammatory bowel disease.[2,5–9] Neoplastic causes of EHBDO are also reported, specifically carcinoma of the biliary tract, pancreatic carcinoma, unclassified biliary tract carcinoma, squamous cell carcinoma involving the duodenal papilla, and duodenal lymphoma.[5] Finally, a duodenal foreign body, biliary foreign body and *Platynosomum concinnum* are also reported causes of EHBDO in cats.[10–12] A teratoma arising from the common bile duct has rarely been documented in humans.[13] In humans, lesions have been described as a lobulated polypoid mass, which was not evident in this case; hence, we feel that this differential is unlikely.[13] Another suggestion is that this could represent a form of feline eosinophilic gastrointestinal sclerosing fibroplasia (FGESF) due to the foreign body leading to an overzealous fibrous tissue reaction causing biliary duct obstruction.[14,15] Cases of FGESF often have hyperglobulinaemia or hypereosinophilia, which was not documented at any stage in this case.[15] Histopathology would have been required to rule out this differential.

It is our opinion that the obstruction was due to a combination of physical constriction from the hair shafts and obstructive adhesions (induced by a foreign body reaction to the hair shafts). We cannot exclude concurrent extra and intraluminal inflammation such as cholangitis as a cause of biliary obstruction as cholecystocentesis or hepatic, pancreatic or gastrointestinal biopsies were not collected for culture and susceptibility and histopathological evaluation.

Prior to surgery, ultrasound failed to identify the hair constricting the bile duct. We speculate that this was because inflammation of the surrounding pancreatic and omental tissue made it difficult to delineate these structures.

Given the history of recent trauma resulting in a deep laceration, it is likely the abdominal cavity was penetrated and this was not appreciated during the initial surgical exploration. Foreign body migration distant from the original penetration site is well recognised in dogs and cats.[16–19] Unusual locations of migration are reported, for example in one case report, a grass awn migrated from the neck to the heart in cat leading to fatal endocarditis and myocarditis.[17] It is therefore not unreasonable to suggest that hair shafts could have migrated from the original laceration site to the common bile duct in this case. Another consideration is that hair was ingested and perforated the gastrointestinal tract; however, there was no evidence of gastrointestinal damage at exploratory laparotomy.

Six month survival in cats after biliary diversion is reportedly poor, ranging from 27–67%.[5,6,20] Only three cases had biliary diversion surgery performed in the study where 67% (2/3 cases) long-term survival was reported.[6] A poorer prognosis is seen with neoplastic lesions compared with inflammatory disease.[5]

Long-term complications include reflux of bile into the stomach (alkaline gastritis), cholangitis and stricture or leakage of the anastomosis.[5,21,22] Alkaline reactive gastritis is a clinical entity that has been linked to duodenogastric

reflux of bile constituents after cholecystectomy in humans.[23] In humans it is managed with proton pump inhibitors; hence, omeprazole was used indefinitely in this case.[23] Recurrent cholangitis has been reported in cats following cholecystoduodenostomy.[5] It is thought that this may be related to stricture at the anastomosis site, resulting in inadequate drainage of the biliary system.[7] Based on previous recommendations to prevent stricture formation, a large (2.5 cm) biliary–intestinal anastomosis was made at the time of surgery and mucosal surfaces were carefully approximated and sutured in the form of a stoma.[7] Additionally, because recurrent biliary tract infection secondary to enterobiliary reflux has been reported as a complication of biliary diversion surgery, tylosin was administered indefinitely in the hope of favouring the growth of a desirable microflora in the gastrointestinal tract.

Conclusions

To our knowledge, this is the first reported case of intra-abdominal migration of foreign matter (specifically hair shafts) contributing to EHBDO through physical constriction and a secondary foreign body reaction causing adhesions surrounding this. This should be included in the differential diagnosis of a cat with jaundice, particularly following abdominal trauma. In addition, lacerations over the abdominal cavity need to be explored carefully for evidence of penetration. If present, the abdomen should be explored and flushed thoroughly to prevent complications.

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References

1 Simpson K, Twedt DC, McDonough SP, et al. **Culture-independent detection of bacteria in feline pancreatitis**. *Proceedings of the ACVIM forum*; 2011 June 5–18; Denver, CO. Red Hook, NY: Curran Associates, Inc., 2011, pp. 507–509.

2 Gaillot HA, Penninck DG, Webster CR, et al. **Ultrasonographic features of extrahepatic biliary obstruction in 30 cats**. *Vet Radiol Ultrasound* 2007; 48: 439–447.

3 Bonczynski JJ, Ludwig LL, Barton LJ, et al. **Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats**. *Vet Surg* 2003; 32: 161–166.

4 Kilpinen S, Spillmann T, Syrja P, et al. **Effect of tylosin on dogs with suspected tylosin-responsive diarrhea: a placebo-controlled, randomized, double-blinded, prospective clinical trial**. *Acta Vet Scand* 2011; 53: 26.

5 Mayhew PD, Holt DE, McLear RC, et al. **Pathogenesis and outcome of extrahepatic biliary obstruction in cats**. *J Small Anim Pract* 2002; 43: 247–253.

6 Eich CS and Ludwig LL. **The surgical treatment of cholelithiasis in cats: a study of nine cases**. *J Am Anim Hosp Assoc* 2002; 38: 290–296.

7 Tangner CH, Turrel JM and Hobson HP. **Complications associated with proximal duodenal resection and cholecystoduodenostomy in two cats**. *Vet Surg* 1982; 11: 60–64.

8 Harvey AM, Holt PE, Barr FJ, et al. **Treatment and long-term follow-up of extrahepatic biliary obstruction with bilirubin cholelithiasis in a Somali cat with pyruvate kinase deficiency**. *J Feline Med Surg* 2007; 9: 424–431.

9 Bacon NJ and White RA. **Extrahepatic biliary tract surgery in the cat: a case series and review**. *J Small Anim Pract* 2003; 44: 231–235.

10 Brioschi V, Rousset N and Ladlow JF. **Imaging diagnosis – extrahepatic biliary tract obstruction secondary to a biliary foreign body in a cat**. *Vet Radiol Ultrasound* 2014; 55: 628–631.

11 Della Santa D, Schweighauser A, Forterre F, et al. **Imaging diagnosis – extrahepatic biliary tract obstruction secondary to a duodenal foreign body in a cat**. *Vet Radiol Ultrasound* 2007; 48: 448–450.

12 Jenkins CC, Lewis DD, Brock KA, et al. **Extrahepatic biliary obstruction associated with *Platynosomum concinnum* in a cat**. *Compend Contin Educ Pract Vet* 1988; 10: 628–632.

13 Demircan M, Uguralp S, Mutus M, et al. **Teratoma arising from anomalous common bile ducts: a case report**. *J Pediatr Surg* 2004; 39: e1–2.

14 Craig LE, Hardam EE, Hertzke DM, et al. **Feline gastrointestinal eosinophilic sclerosing fibroplasia**. *Vet Pathol* 2009; 46: 63–70.

15 Linton M, Nimmo JS, Norris JM, et al. **Feline gastrointestinal eosinophilic sclerosing fibroplasia: 13 cases and review of an emerging clinical entity**. *J Feline Med Surg* 2015; 17: 392–404.

16 Vansteenkiste DP, Lee KC and Lamb CR. **Computed tomographic findings in 44 dogs and 10 cats with grass seed foreign bodies**. *J Small Anim Pract* 2014; 55: 579–584.

17 Doyle CG, Allen JW and Ettinger SJ. **Foxtail-associated endocarditis in a cat**. *J Feline Med Surg* 2011; 13: 116–119.

18 Cherbinsky O, Westropp J, Tinga S, et al. **Ultrasonographic features of grass awns in the urinary bladder**. *Vet Radiol Ultrasound* 2010; 51: 462–465.

19 Brennan KE and Ihrke PJ. **Grass awn migration in dogs and cats: a retrospective study of 182 cases**. *J Am Vet Med Assoc* 1983; 182: 1201–1204.

20 Buote NJ, Mitchell SL, Penninck D, et al. **Cholecystoenterostomy for treatment of extrahepatic biliary tract obstruction in cats: 22 cases (1994–2003)**. *J Am Vet Med Assoc* 2006; 228: 1376–1382.

21 Srivengadesh G, Kate V and Ananthakrishnan N. **Evaluation of long-term results of choledochoduodenostomy for benign biliary obstruction**. *Trop Gastroenterol* 2003; 24: 205–207.

22 Matthiesen DT. **Complications associated with surgery of the extrahepatic biliary system**. *Prob Vet Med* 1989; 1: 295–315.

23 Santarelli L, Gabrielli M, Candelli M, et al. **Post-cholecystectomy alkaline reactive gastritis: a randomized trial comparing sucralfate versus rabeprazole or no treatment**. *Eur J Gastroenterol Hepatol* 2003; 15: 975–979.