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CASE REPORT
Suspected choledochal cyst in a domestic shorthair cat

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A 9-year-old female neutered domestic shorthair cat was presented with a history of polyphagia, weight loss and inappropriate urination. Clinical examination revealed jaundice and a mid-cranial abdominal mass. Further investigations revealed a large extra-hepatic cyst originating from the biliary tract (choledochal cyst). Concurrent chronic, active neutrophilic cholangitis and chronic lymphoplasmacytic pancreatitis were also noted. Surgical drainage, subtotal resection and omentalisation of the cyst, along with supportive medical management, were successful in relieving the clinical signs.

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A 9-year-old female neutered domestic shorthair cat was presented to the referring vet with a history of polyphagia, weight loss and urinating in the house. Her demeanour had also changed, becoming nervous and clingy over the previous few days. On clinical examination she was bright and alert but underweight (body weight 2.9 kg, body condition score 1.5/5). Vital parameters were within normal limits but the mucous membranes were jaundiced. In addition, a large, firm, non-painful mass was palpable in the mid-cranial abdomen. Routine haematology was unremarkable but serum biochemistry revealed significant elevations in alanine aminotransferase [609 U/l, reference interval (RI) 0–120], alkaline phosphatase (326 U/l, RI 0–170) and bilirubin (118 μmol/l, RI 0–12). Conscious radiographs revealed reduced contrast throughout the abdomen consistent with lack of abdominal fat but possibly also suspicious for mild ascites. In addition, displacement of the gastric axis dorsally and of the intestines caudally was suggestive of a mid-cranial abdominal space-occupying lesion (Fig 1).

The cat was referred for further investigations at which time repeat haematology revealed significant leukocytosis (36.11×10^9/l, RI 5.50–19.50) as a result of marked neutrophilia (30.07×10^9/l, RI 2.50–12.50), moderate monocytosis (1.90×10^9/l, RI 0.15–1.70) and mild basophilia (0.13×10^9/l, RI 0.00–0.10). Serum biochemistry confirmed persistence of the previous findings together with hyperglobulinaemia (56 g/l, RI 35–40) and hypoalbuminaemia (18 g/l, RI 25–39). Clotting times (prothrombin and activated partial thromboplastin time) were within normal limits and urinalysis was unremarkable. Abdominal ultrasonography revealed a large cystic structure (~10 cm diameter) just caudal to the liver. This consisted of a hyperechoic wall of variable thickness containing hypoechoic material. In addition, the gall bladder and associated biliary tree were distended and had a markedly thickened hyperechoic wall. The abdominal organs were otherwise unremarkable but a small pocket of free abdominal fluid was noted. No obvious communication between the gall bladder and cyst could be detected but ultrasound guided needle aspiration of both structures yielded a similar colourless, slightly cloudy fluid. Fluid analysis revealed a highly proteinaceous fluid which yielded a scant mixed bacterial growth. Cellularity was low and no inflammatory or neoplastic cells were noted.

Therapy with intravenous amoxicillin–clavulanate (Augmentin; GlaxoSmithKline) (60 mg q8h) and metronidazole (Macroflex; Marcopharma) (30 mg q12h) was started prior to exploratory midline coeliotomy. Following induction with alfaxalone (Alfaxan; Vetoquinol) (6 mg), anaesthesia was maintained with isoflurane (Isocare; Animalcare) and intravenous fluid therapy with compound sodium lactate (Hartmann's Solution; Animalcare) (18 ml/h) was supplied throughout. A large fluid filled structure was confirmed within the mid-cranial abdomen. This was juxtaposed to the caudal edge of the liver and attached to the common bile duct. The gall bladder was enlarged and the hepatic and cystic ducts were tortuous and distended. The liver parenchyma was diffusely firm

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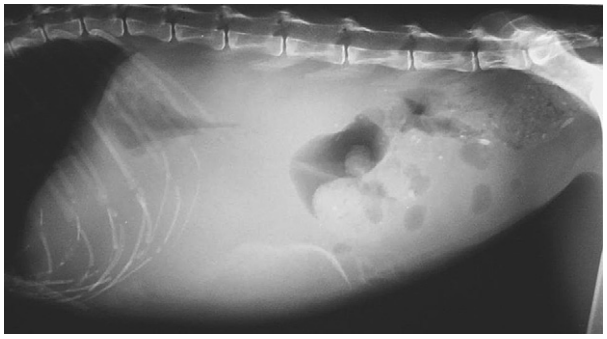

Fig 1. Right lateral abdominal radiograph showing reduced contrast throughout the abdomen which is obscuring the normal abdominal organs. This is likely to be a consequence of poor body condition with a lack of abdominal fat, possibly in association with mild ascites. Displacement of the gastric axis dorsally and of the intestines caudally is apparent, suggestive of a mid-cranial abdominal mass.

but friable and the pancreas was oedematous. An incision was made in the cyst and suction was used to empty it of its contents (Fig 2). This confirmed the presence of a colourless, slightly cloudy fluid as previously. However, a small amount of more concentrated, bile-like fluid was also present within the dependant portion of the cyst. The wall of the cyst was thick (~3 mm) with the consistency of normal feline small intestine, and was contiguous with the wall of the common bile duct. Examination within the cyst revealed potential communications with the duodenum distally and with the biliary tree proximally. However, both stomas were small, atresic and could not be catheterised. As the cyst was arising from the common bile duct (Fig 3), en mass resection was not considered a viable option and a subtotal resection of the cyst wall was, therefore, performed. After closing the stomas at either end with polypropylene (Prolene; Ethicon) purse-string sutures, the structure was omentalised; the original cystostomy incision was partially closed, and the remaining cavity was packed with an omental pedicle. Interrupted stay-sutures were placed at the stoma to keep the pedicle in place.

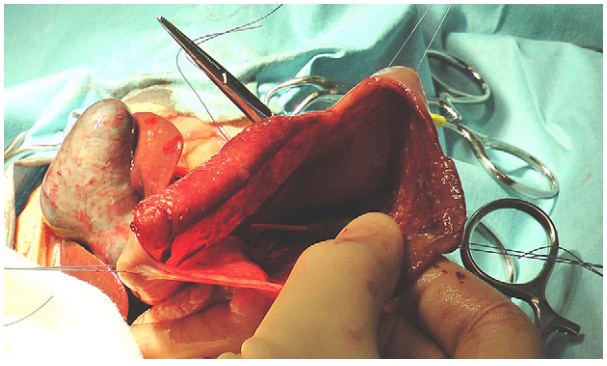

Fig 2. Intra-operative photograph showing opened choledochal cyst, aspirated of content. The thickened gall bladder is also visible to the left of the cyst.

Inspissated bile was removed from the gall bladder before the biliary tract was diverted via standard cholecystojejunostomy.[1] Biopsies of the pancreas, liver and cyst wall were taken for histopathology. Ascetic fluid was not noted as a finding at surgery, presumably because the volume was so small. The abdomen was lavaged with sterile saline and closed in a routine manner.

The cat's anaesthetic recovery was unremarkable and postoperatively she remained bright with a good appetite. However, repeat serum biochemistry revealed significant deterioration of the hypoalbuminaemia (13 g/l, RI 25–39) with a concurrent drop in total calcium (1.70 mmol/l, RI 2.00–3.00). Interestingly, ionised calcium levels were also depleted (0.97 mmol/l, RI 1.12–1.40). Intravenous colloid therapy with hydroxyethyl starch (Voluven; Fresenius Kabi) (1.5 ml/h) was instigated to maintain oncotic pressure and sublingual buprenorphine (Buprecare; Animalcare) (30 µg q8h) was administered for analgesia. Serum total and ionised calcium levels were monitored. Antibiotic therapy was continued intravenously for 3 days and oral therapies were then instigated; amoxicillin–clavulanate (Nisamox; Fort Dodge Animal Health) (50 mg q12h) and metronidazole (Metronidazole; Pharmavit) (33.3 mg q12h). Supportive therapies for suspected concurrent liver disease were also started; ursodeoxycholic acid (Destolit; Norgine) (37.5 mg q24h), S-adenosylmethionine (50 mg q24h)/vitamin E (10 mg q24h)/vitamin K (0.05 mg q24h) (Hepatosyl; Ceva Animal Health) and a high quality protein diet (Sensitivity Control; Royal Canin). Over the ensuing days the albumin levels gradually improved, serum calcium levels (total and ionised) normalised and the bilirubinaemia slowly resolved. Despite persistence of a suboptimal body condition score the cat remains bright and alert with an improved demeanour. The polyphagia has improved and the inappropriate urination has resolved.

Histopathology of the cyst wall revealed mature fibrous connective tissue with scattered clusters of well-differentiated glandular structures consistent with bile ducts/ductules. In addition, one of the sections was lined on one side by small, multi-loculated cystic structures covered by a single layer of well-differentiated cuboidal to attenuated epithelial cells. Hepatic and pancreatic sections revealed chronic, active neutrophilic cholangitis with concurrent chronic lymphoplasmacytic pancreatitis, respectively.

Extra-hepatic solitary cysts of the biliary system are rarely found in cats. Most cystic disease is multifocal and linked to polycystic disease, usually affecting the kidneys or liver.[2] In contrast, biliary cystadenomas are often solitary but these are found within the liver parenchyma.[3,4] Although uncommon, extra-hepatic cystic disease of the biliary tract is described in humans. These cystic dilations of the bile duct are referred to as choledochal cysts and usually contain bilious fluid, rich in pancreatic enzymes.[5] The cysts are divided into five types depending on their

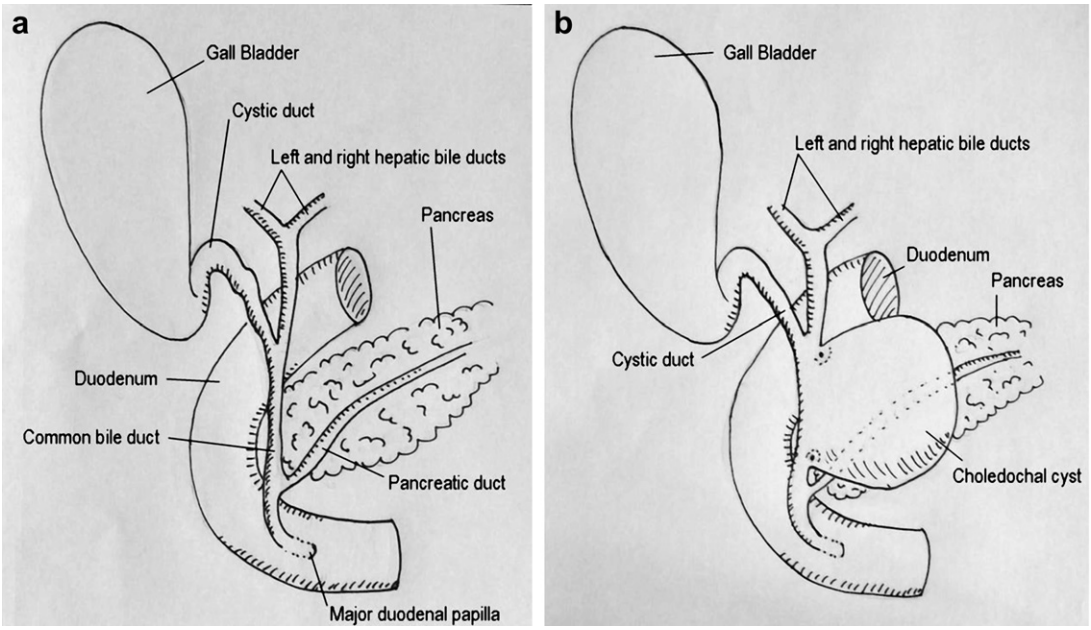

Fig 3. Diagrammatic representation of the normal feline biliary anatomy (a) compared with that of the abnormal choledochal cyst (b).

anatomical location,[5] type 1 being the most common and consistent with the findings in this case (ie, a cystic or fusiform dilation of the common bile duct) (Fig 3). Type 5 lesions, secular dilations of the intrahepatic bile ducts, also known as Caroli's disease have been described in a litter of Golden Retriever puppies that also had renal cysts.[5]

In humans, most cases are believed to be congenital as they predominantly occur in infants and children. However, approximately 20% are diagnosed in adults.[5] Consequently, several theories for late-onset cyst development have been postulated, including a weakness in the wall of the common bile duct (with or without obstruction distally) and reflux of pancreatic enzymes into the common bile duct due to an anomaly of the pancreaticobiliary junction (the common bile duct joins the pancreatic duct more proximally than normal giving it similar anatomy to the cat).[4,5] This reflux of pancreatic enzymes is thought to weaken the wall of the common bile duct causing a cyst to form. As far as we are aware, a type 1 choledochal cyst has not been previously described in a cat. It is possible that the cyst was congenital in origin and that gradual distension eventually culminated in late-onset clinical signs due to biliary obstruction and ascending bacterial infection from the intestine, leading to the associated neutrophilic cholangitis and pancreatitis. Alternatively, late-onset cyst development associated with underlying primary intestinal, pancreatic or biliary pathology cannot be ruled out. Bacterial culture of the bile and cystic fluid would have been useful in order to determine the involvement of infection in the pathogenesis and would also have facilitated sensitivity testing to enable targeted antibiotic use and screening for antibiotic resistance. It should also be noted that underlying intestinal disease cannot be excluded as a cause for this cat's polyphagia and persistent weight loss. In fact, although the cat is now doing well clinically with normal haematology and serum biochemistry results, weight gain has been suboptimal. An exclusion diet of home cooked chicken and rice appears to have been of some benefit with regards to this but instigation of prednisolone therapy is currently being considered for treatment of suspected inflammatory bowel disease. Intestinal biopsies at the time of exploratory coeliotomy would, therefore, have been extremely helpful to determine the presence of underlying primary intestinal disease, particularly given the possibility of triaditis with primary enteropathy resulting in pancreatic reflux, pancreatitis and choleangitis.

As in this cat, humans often present with signs of biliary obstruction (primarily jaundice) and a palpable mass in the anterior abdomen.[6] The profound weight loss seen in this case was likely to have been a consequence of increased caloric requirement (inflammatory process) and decreased protein production (liver disease). Low albumin levels may have resulted from a combination of reduced production (acute phase shift and hepatic dysfunction), increased loss (due to sequestration within the cystic fluid) and dilutional effects (post fluid therapy). Although total calcium levels are frequently reduced in association with hypoalbuminaemia, low ionised calcium levels would not usually be expected and it is possible that this finding was related to the concurrent pancreatitis. Polyphagia can be seen with feline inflammatory liver disease but this is usually associated with lymphocytic rather than neutrophilic cholangitis.[7] It is possible that the increased appetite could have resulted from an

underlying enteropathy or may simply have been a normal response to persistent weight loss. Mild polyuria can be associated with hepatic disease but this is rarely so clinically significant and is generally associated with a reduced urine specific gravity. Inappropriate urination may have, at least in part, been a behavioural change brought on by the stress of illness. However, as urinalysis was unremarkable, it is assumed that the main reason for inappropriate urination in this cat was increased pressure on the bladder subsequent to a large abdominal space-occupying lesion.

Malignant transformation of cyst tissue resulting in carcinoma formation has been described and complete cyst resection is, therefore, the treatment of choice in human patients.[6] In the cat described here a less invasive surgical option, avoiding the need for disruption of the pancreatic duct, was chosen to minimise peri-surgical morbidity. So far the clinical outcome appears to be favourable. However, as the underlying cause is unknown, the long-term prospects for the cat remain uncertain and monitoring for recurrent biliary obstruction and ongoing hepatic and pancreatic disease is ongoing.

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