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Ultrasonographic and clinicopathologic features of segmental dilatations of the common bile duct in four cats

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Abstract

Case series summary This case series documents ultrasonographic and clinicopathologic features of four cats with marked segmental dilatations of the common bile duct (CBD). All cats had additional ultrasonographic changes to the hepatobiliary system, including hepatomegaly, tubular to saccular intra/extrahepatic biliary duct dilatation and biliary debris accumulation. Based on all available data the presence of extrahepatic biliary duct obstruction (EHBDO) was ruled out in 3/4 cases and was equivocal in one case. One cat underwent re-routing surgery to address the CBD dilatation after multiple recurrent infections, one cat was euthanized and had a post-mortem examination and two cats were medically managed with antibiotics, liver protectants, gastroprotectants and cholerectics.

Relevance and novel information The ultrasonographic features of the CBD in this population of cats were supportive of choledochal cysts (CCs). The maximal diameter of the CBD dilatations exceeded 5 mm in all cases, a sign that has been previously reported to be consistent with EHBDO. In our study, dilatations were segmental rather than diffuse. Given the high morbidity and mortality associated with hepatobiliary surgery in cats, segmental dilatation of the CBD should not prompt emergency surgery. Some cats may respond to medical management. Careful planning for cyst resection was beneficial in one cat. Evaluation of CC morphology (eg, size, location, concurrent intrahepatic anomalies) may assist in selecting cats that could benefit from surgical intervention.

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Introduction

Ultrasonography is a baseline imaging modality for evaluating the feline hepatobiliary system. The normal ultrasonographic appearance of the feline biliary tree has been previously described.[1–4] The gall bladder (GB), cystic duct and common bile duct (CBD) can be reliably identified in clinically normal feline patients.[1,2] The remaining components of the biliary tree (extrahepatic ducts and intrahepatic structures, including interlobular ducts, interlobar ducts and larger hepatic bile ducts) are not ultrasonographically visible unless there is pathological dilatation.[1,2,4] In the cat, a CBD diameter of up to 4 mm is considered normal.[2,4,5]

An abnormal increase in the diameter of the CBD in cats with hepatobiliary disease is largely dependent on the presence of an obstructive lesion.[5,6] Extrahepatic biliary duct obstruction (EHBDO), which may be secondary to numerous etiologies (pancreatitis, duodenitis, neoplasia, cholelithiasis, mucus plugs, fibrosis, foreign bodies, liver flukes), is the most commonly reported cause of CBD dilatation in the cat.[4–13] A previous study of cats with confirmed EHBDO demonstrated that a CBD diameter of

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5 mm or greater was highly supportive of obstruction.[4] A later investigation of the ultrasonographic features of EHBDO in cats described the obstructed CBD as undulating to tortuous in appearance with diffuse dilatation of uniform diameter in the majority of cats.[5] The literature describing ultrasonographic changes to the CBD in non-obstructive processes reports less severe CBD dilatation. In a population of cats with surgical or necropsy-confirmed non-obstructive hyperbilirubinemia, maximal CBD diameter did not differ significantly from normal cats,[4] and in cats with cholangitis or liver fluke infestation CBD dilatation to or beyond 5 mm was not reported in conjunction with a patent biliary tract.[9,14,15]

Segmental dilatations of the CBD, also referred to as choledochal cysts (CCs), have been described in human patients and may vary in morphology; the classification of CCs depends on anatomical features such as the shape (cystic, fusiform, diverticular), extent (focal, diffuse) and location (extrahepatic, intrahepatic or both) of the dilatation(s).[16–20] In this context (and throughout this report) the term 'cyst' denotes a morphological appearance of a rounded, fluid-filled structure with a thin wall as opposed to a true histological cyst (eg, non-communicating, epithelium-lined, fluid-filled sac). Only two case reports of cats with segmental CBD dilatation consistent with CCs have been previously published.[21,22] In both cases, cats presented with non-specific clinical signs and icterus on physical examination. Cystic structures adjacent to the liver and concurrent biliary dilatation were identified via ultrasonography, but an etiology for these cysts could not be determined. Exploratory laparotomies were performed owing to concern for EHBDO; surgery confirmed the presence of segmental CBD dilatations suggestive of CCs without evidence of an obstructive component.[21,22]

In our experience, cases of segmental CBD dilatation (consistent with CCs) have been encountered in cats where maximal CBD diameter has exceeded the published 5 mm diameter cut-off for obstructive disease; however, the presence of EHBDO was not clinically suspected. Although emergency surgical intervention is unnecessary in these situations, such cases can still present a therapeutic challenge in determining appropriate management strategies, especially in electing medical vs surgical options. The presence of concurrent extrahepatic and/or intrahepatic bile duct dilatation in addition to a CC may be a complicating factor. Therefore, the goal of this project was to describe the ultrasonographic features, clinicopathological changes, and outcomes of four cats with segmental CBD dilatations suggestive of CCs without definitive evidence of EHBDO.

Case series description

Case 1

A 6.5-year-old, female spayed, indoor-only, domestic shorthair (DSH) cat was presented for a history of chronically elevated serum liver enzyme activity. Over the previous 2 years, the cat had multiple episodes of infectious cholangitis, which had cultured positive for *Enterococcus species*, *Escherichia coli* and *Corynebacterium species*, and had responded to prior medical management with antibiotics. The cat was also diagnosed with lymphoplasmacytic and eosinophilic enteritis by endoscopic biopsy 3 years prior and was being treated with corticosteroids and chlorambucil. The cat was currently asymptomatic.

The cat was normal on physical examination and in good body condition (body condition score [BCS] 6/9, body weight 5.6 kg). Routine serum biochemical examination was unremarkable. In the 12 months prior the cat had variably elevated liver enzyme activities, including alanine aminotransferase (ALT; range 203–4238 U/l; reference interval [RI] 25–145 U/l) and aspartate aminotransferase (AST; range 84–1104 U/l; RI 5–42 U/l). Seven months prior the cat had mild serum hyperbilirubinemia (0.7 mg/dl; RI 0.1–0.3 mg/dl), which occurred in conjunction with a diagnosis of septic cholangitis confirmed by an *E coli*-positive bile culture.

The liver was enlarged, diffusely hyperechoic and coarse in echotexture. The GB was subjectively normal in size with mild wall thickening (1.5 mm; normal <1 mm).[23] There was a single, large (~1.7 cm diameter × 3.0 cm length), smoothly margined, fusiform, segmental dilatation of the proximal to mid-aspect of the CBD (Figure 1a). The distal CBD leading up to the duodenal papilla (DP) was normal in appearance and diameter (4 mm). The DP was slightly thickened (5.5 mm, normal <4 mm in transverse plane).[24] There was concurrent diffuse, mild, tubular dilatation of intrahepatic (up to 1.9 mm diameter) and extrahepatic bile ducts (up to 5.7 mm diameter). Diffusely throughout the biliary tree, and within the CBD segmental dilatation, there was moderate echogenic sediment accumulation and multifocal intrahepatic mineralization. All findings were similar to an initial ultrasound examination performed 2 years prior. Ultrasound-guided bile aspiration (at the level of the CC) and percutaneous hepatic tissue core biopsies were performed.

The bile culture was positive for *E coli* and *Enterococcus* species. Hepatic biopsies revealed mildly increased amounts of peribiliary connective tissue and mild lymphocytic infiltrates associated with portal areas. Several scattered portal areas contained distinct, follicle-like clusters of lymphocytes. In rare portal areas, small bile duct profiles were obscured by small-to-moderate numbers of neutrophils that entered the biliary epithelium. Small numbers of eosinophils were infrequently admixed with portal inflammation. Diffusely, hepatocytes were mildly expanded by diaphanous vacuolation (glycogen), likely secondary to oral corticosteroid administration. Findings were consistent with mild, chronic biliary injury. Although rarely captured, the presence of neutrophils entering bile ducts supported a bacterial etiology.

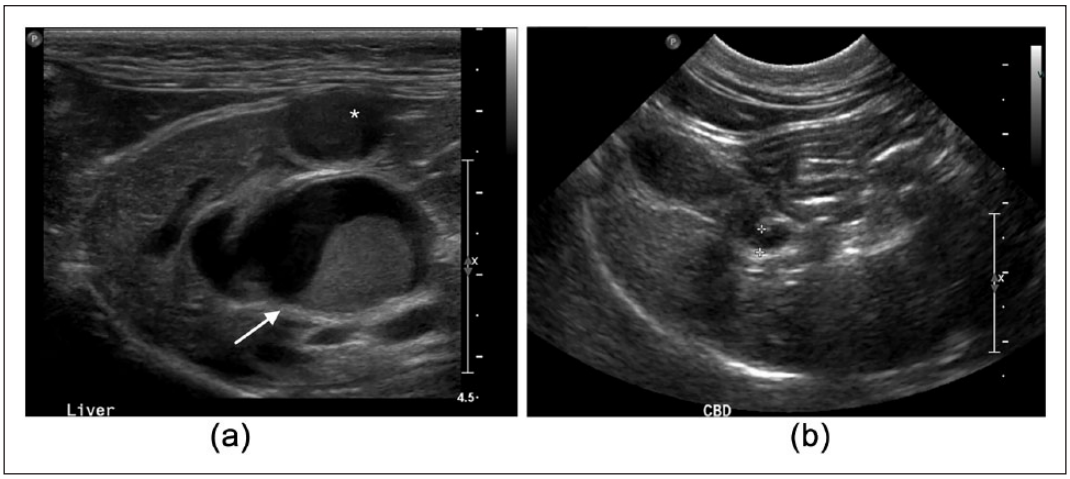

Figure 1 (a) Sagittal ultrasonographic image of the right liver of case 1. The largest biliary structure represents the segmentally dilated common bile duct (CBD; arrow) with mild wall thickening. There is a collection of organized, dependent echogenic debris within the CBD dilatation. The tubular structure noted ventral to the CBD represents a dilated extrahepatic duct containing echogenic bile (asterisk). (b) Sagittal image of the gall bladder and CBD at follow-up ultrasound examination 27 months postoperatively. The CBD focal dilatation is absent and the distal CBD measures 3.4 mm in diameter (calipers).

Although this cat did not have hyperbilirubinemia at the time of ultrasound and primary mechanical EHBDO was not suspected, the marked focal CBD dilatation was thought to be a predisposing factor for recurrent bacterial infections. Ultimately, the cat underwent a choledochoduodenostomy. The cat recovered from surgery without complication. Numerous follow-up ultrasound examinations were obtained at 10 days, 18 days, 23 months and 27 months postoperatively. There was no recurrence of CBD dilatation (Figure 1b) and there was resolution of the previous intrahepatic duct dilatation. Over the follow-up examinations there was persistent hepatomegaly with coarse hepatic echotexture and unchanged thickening of the walls of the GB and intra/extrahepatic biliary ducts. Despite ultrasonographic evidence of normal diameter of the bile ducts, the cat continued to have recurrent episodes of cholangitis (often associated with vomiting), albeit reduced in frequency, for the next 3 years. The cat remained free of clinical signs for 14 months prior to its death secondary to an unrelated chylothorax.

Case 2

A 5-year-old male castrated, indoor-only DSH cat was presented for evaluation of lethargy, reduced appetite, weight loss and abdominal distension. On physical examination the cat was bright and alert but in poor body condition (BCS 3/9, body weight 4.8 kg). Additional abnormalities included mild icterus of the pinnae and mucus membranes, a distended, non-painful abdomen with suspected cranial organomegaly and a grade II/VI parasternal heart murmur.

Blood work revealed a mild normocytic, normochromic, non-regenerative anemia (hematocrit [HCT] 29%; RI 30–50%), mild serum hyperbilirubinemia (total bilirubin 1.7 mg/dl; RI 0.1–0.3 mg/dl) and elevated serum liver enzyme activities (ALT [1110 U/l; RI 25–145 U/l], AST [291 U/l; RI 5–42 U/l], alkaline phosphatase [ALP; 312 U/l; RI 10–79 U/l) and gamma-glutamyl transpeptidase [GGT; 13 U/l; RI 0–5 U/l]).

On abdominal ultrasound the liver was enlarged and distorted by numerous coalescing anechoic structures, presumed to represent intrahepatic biliary ducts in the absence of color flow Doppler signal. There was marked, diffuse tubular-to-saccular dilatation of the intrahepatic ducts (up to 1.1 cm diameter) causing almost complete effacement of the hepatic parenchyma. The GB could not be reliably identified from the abnormal surrounding intrahepatic ducts. There was marked, irregularly fusiform dilatation of the proximal CBD (Figure 2a) with a concurrent, focal, rounded, cystic dilatation of the distal aspect of the CBD measuring 5.2 cm in diameter. The DP was within normal limits. The extrahepatic bile ducts were diffusely tortuous and markedly dilated (up to 2.8 cm diameter). The dilated biliary structures occupied the majority of the abdomen displacing adjacent organs (eg, spleen, small intestines). Diffusely throughout the entire biliary tree, there was marked echogenic sediment accumulation. There was a small volume of anechoic peritoneal effusion.

In the light of the non-specific clinical signs, the mild hyperbilirubinemia – despite marked biliary dilatation – and the lack of ultrasonographic identification of an obstructive lesion, primary mechanical EHBDO was not suspected. Despite the older age of the cat, the widespread intra- and extrahepatic changes of this magnitude were suspected to represent a developmental anomaly. Diagnostic and therapeutic bile sampling (for cytology/culture and relief of abdominal discomfort) and abdominal CT were offered for further evaluation. Surgical intervention was not considered owing to the

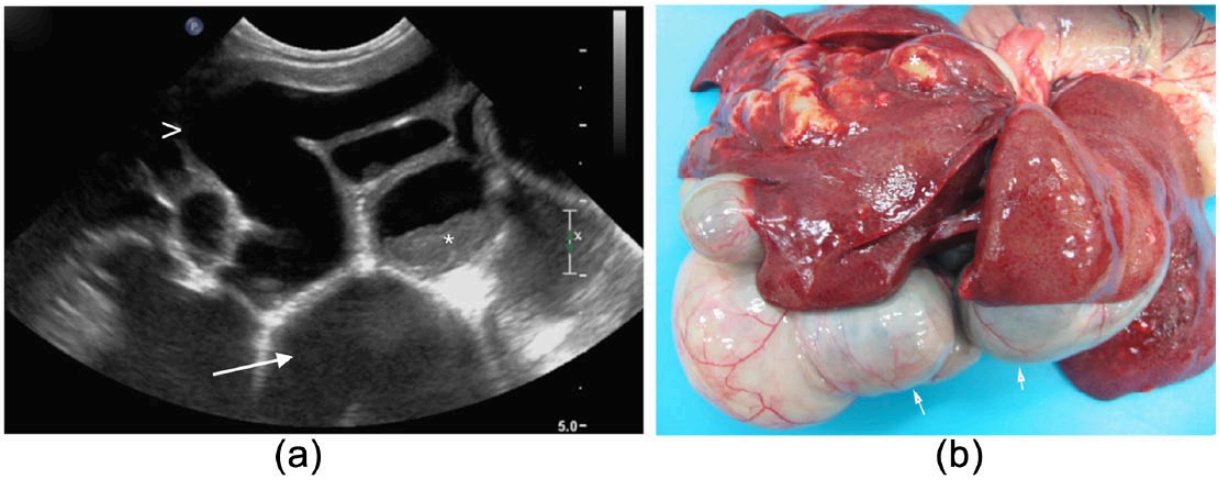

Figure 2 (a) Sonogram of the right liver of case 2. Multiple dilated bile ducts are visible, including extrahepatic (arrowhead) and intrahepatic ducts (asterisk). There is variable echogenic-dependent debris in the visible intrahepatic bile ducts. The structure in the far field is the proximal aspect of the common bile duct dilatation (arrow). (b) Post-mortem image of the liver of case 2. The extrahepatic bile ducts (arrows) are severely, irregularly dilated and the hepatic capsular surface is multifocally deformed by irregularly dilated intrahepatic bile ducts (asterisk).

extent of the biliary changes. The owner elected humane euthanasia with necropsy.

On post-mortem examination, the abdomen was filled by numerous, coalescing, cystic dilatations of the external biliary tree up to approximately 6.0 cm in diameter (Figure 2b). The liver contained numerous, irregularly dilated intrahepatic bile ducts. It was not possible to express bile through the DP by applying manual pressure to the GB, but the severe saccular dilatation of the extrahepatic biliary tree likely affected the reliability of this test as an indicator of ante-mortem obstruction. A catheter was passed anterograde through the CBD into the proximal duodenum.

Histologically, large- and medium-sized intrahepatic bile ducts varied from irregularly dilated and filled with mucinous material to collapsed with irregularly folded walls. These bile ducts were surrounded by abundant, concentric, fibrous tissue with mild biliary hyperplasia and minimal lymphocytic inflammation. In the smallest levels of the biliary tree, portal areas exhibited milder biliary hyperplasia and fibrosis with infrequent, mild bridging fibrosis. In total, the gross, histologic, and clinical features were thought to favor a primary biliary developmental abnormality.

Case 3

An 8-year-old female spayed, indoor-only DSH cat was presented for evaluation of chronic vomiting with a frequency of 1–2 times per week that had not responded to diet change or antibiotics. Abnormalities on physical examination included poor body condition (BCS 3/9, body weight 2.7 kg), mild palpable renal asymmetry and a grade II/VI heart murmur.

Blood work for this cat was performed 11 days prior to the reported ultrasound. Biochemical changes included a mild anemia (HCT 26%; RI 30–50%) and elevated creatinine (2.5 mg/dl; RI 0.9–2.1 mg/dl). Serum liver enzyme activities and total serum bilirubin were within normal limits.

On abdominal ultrasound the liver was enlarged with a coarse echotexture. The GB had a bilobed morphology characterized by a heart-shaped appearance of the GB fundus with an incomplete luminal septum. The GB was subjectively normal in volume with wall thickening (2 mm). There was a single, large (approximately 2.4 cm diameter $\times$ 3.7 cm length), fusiform, focal dilatation of the distal aspect of the CBD (Figure 3). The most distal CBD adjacent to the DP was tubular in appearance, but slightly dilated (up to 4.5 mm diameter). The DP was hyperechoic and slightly thickened (5.3 mm). There was concurrent diffuse, moderate, tubular dilatation of intrahepatic bile ducts (up to 3.5 mm diameter). Extrahepatic bile ducts were diffusely, moderately dilated and saccular in appearance (up to 5.4 mm diameter). Diffusely throughout the biliary tree there was thickening of the biliary walls (up to 2 mm), a moderate amount of echogenic debris and numerous small shadowing biliary calculi. There was also diffuse small intestinal wall thickening (over 3 mm) with altered wall layering characterized by muscularis thickening and a hyperechoic line within the mucosal layer parallel to the submucosa suggestive of mucosal fibrosis.

Given the mild clinical signs and normal serum liver enzyme activity and bilirubin, the biliary changes were suspected to be secondary to chronic inflammation or infection and surgical intervention was not recommended

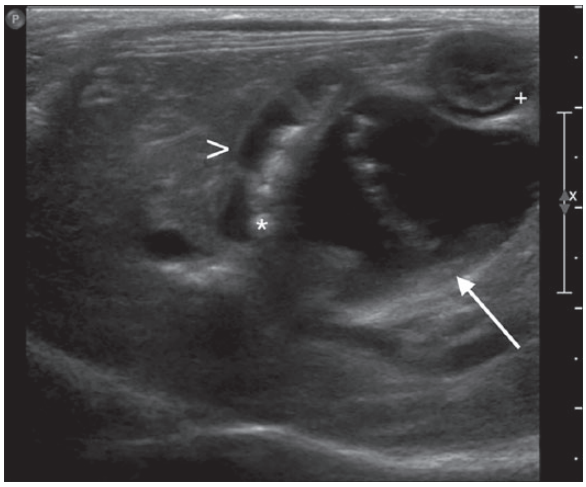

Figure 3 Transverse sonogram of the segmental common bile duct (CBD) dilatation in case 3 (arrow). There are dependent, shadowing calculi (asterisk) within a saccular, dilated extrahepatic bile duct adjacent to the CBD (arrowhead). A small intestinal segment can be seen in the near-field; there is evidence of wall thickening (up to 4.5 mm) with thickening of the muscularis layer (cross).

at this time. Liver biopsy and bile culture were recommended but declined by the owner. Empirical medical management for cholangitis was initiated with antibiotics (amoxicillin–trihydrate/clavulanate potassium, 24 mg/kg PO q12h), antacids (famotidine, 2 mg/kg PO q24h) and choleretics (ursodeoxycholic acid, 18 mg/kg PO q24h) for a 3 month period. The cat returned for follow-up examination at the end of the antibiotic course, at which time it was clinically normal. Recheck ultrasound showed unchanged biliary findings compared with the prior study with persistent focal CBD dilatation, biliary wall thickening and multifocal biliary calculi. A biochemical panel revealed normal liver enzymes and total bilirubin.

Twenty-one months later the biliary system findings were again unchanged. Serum liver enzymes and total serum bilirubin remained normal. The cat remained free of any clinical signs of biliary disease.

Case 4

An 11-year-old male castrated, indoor-only DSH cat was presented for evaluation of 1 week of lethargy with previous chronic history of vomiting, weight loss despite normal appetite and intermittent diarrhea. The owner also reported intermittent sneezing and increased upper respiratory noise. Abnormalities on physical examination included poor body condition (BCS 1/9, body weight 2.5 kg), unkempt haircoat, pale, tacky mucus membranes, cranial organomegaly, suspected pain on abdominal palpation, moderate serous nasal discharge and referred upper airway sounds on thoracic auscultation.

Blood work revealed a mild anemia (HCT 28%; RI 30–50%), elevated liver enzyme activities (ALT [195 U/l; RI 25–145 U/l], AST [106 U/l; RI 5–42 U/l], ALP [91 U/l; RI 10–79 U/l] and GGT [23 U/l; RI 0-5 U/l]) and low creatinine (0.4 mg/dl; RI 0.9–2.1 mg/dl). Total bilirubin was within normal limits. A complete blood count showed leukocytosis (white blood cells 19.7 K/µl; RI 4.5–15.7 K/µl) with a mature neutrophilia (segmented cells 17.8 K/µl; RI 2.1–10.0 K/µl).

On abdominal ultrasound the liver was enlarged and hyperechoic. The GB was not reliably identified. There was a large (approximately 3.2 cm diameter × 5.0 cm length), fusiform, focal dilatation of the proximal aspect of the CBD (Figures 4a,b). The most distal 2–3 cm of the CBD was tubular in shape and mildly dilated (5.7 mm diameter). The DP was within normal limits. There was

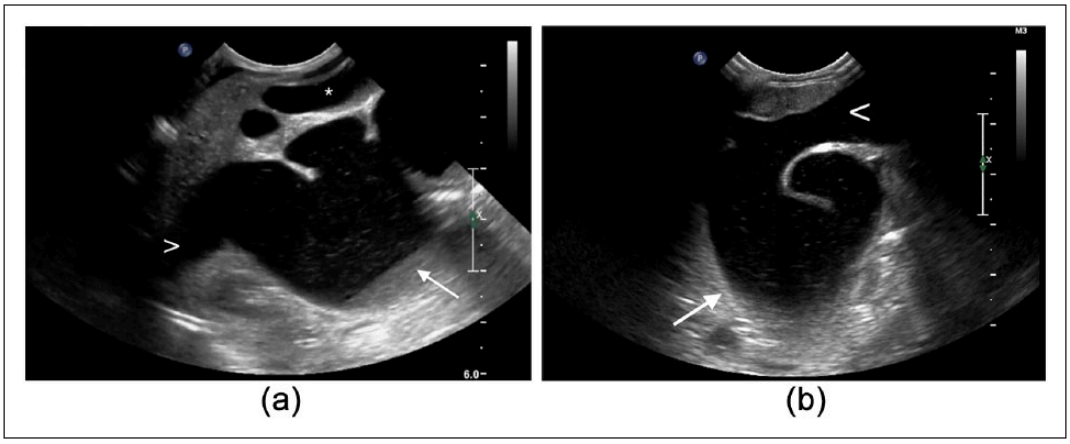

Figure 4 (a) Sagittal and (b) transverse sonograms of the right liver of case 4. There is a fusiform focal dilatation of the proximal aspect of the common bile duct (arrow). Dilated extrahepatic ducts can be seen coalescing with the focal dilatation of the common bile duct (CBD; arrowhead). In the near field there is a dilated intrahepatic bile duct (asterisk). Echogenic, dependent sediment is present in the CBD and the intrahepatic duct.

concurrent diffuse, marked, tubular-to-saccular dilatation of intrahepatic bile ducts (up to 8 mm diameter). Extrahepatic bile ducts were diffusely, markedly dilated and saccular in appearance (up to 1 cm diameter). Diffusely throughout the biliary tree there was a small amount of echogenic sediment. A small volume of anechoic peritoneal effusion was noted.

An EHBDO was ruled out owing to normal total bilirubin. Cholecystocentesis and bile culture were recommended but declined. Given the concurrent diffuse, saccular intrahepatic dilatation, and the absence of EHBDO, medical management was favored. The cat was medically managed for presumptive cholangitis/cholangiohepatitis; it was treated with antibiotics (amoxicillin–trihydrate/clavulanate potassium, 15 mg/kg PO q12h; metronidazole 10 mg/kg PO q12), liver protectants (*s*-adenosylmethionine, 35 mg/kg PO q24h), antacids (omeprazole, 2 mg/kg PO q24h) and antiemetics (maropitant citrate, 1 mg/kg PO q12h). The owner reported improved appetite but persistent diarrhea 1 week following initial examination. Additional follow-up information was not available for this cat; however, the referring veterinarian records indicated that the cat was euthanized within 1 month.

Discussion

In this case series we identified four cats that presented with histories of chronic vomiting, reduced appetite and lethargy. All had hepatomegaly, biliary debris, intra-/extrahepatic bile duct dilatation and marked segmental dilatations of the CBD suspected to represent CCs. Despite the severe biliary duct dilatations observed on ultrasound, only one cat in this series had mild hyperbilirubinemia. In that cat (case 2), the presence of obstruction was equivocal on necropsy owing to the inability to express the GB during gross examination, despite the ability to pass a catheter through the lumen. However, the extreme distortion of the extrahepatic biliary tree in this cat likely interfered with manual biliary expression. Additionally, the relatively mild biochemical changes in the face of extensive and severe biliary dilatation in this cat were not supportive of complete obstruction.

CCs are defined as segmental cystic dilatations of extrahepatic and/or intrahepatic biliary ducts. The etiology of CCs is strongly debated; however, it is likely multifactorial in nature. Congenital and acquired etiologies have been described in the human literature.[16,17,25] Congenital CCs may be related to malformations of the intrahepatic bile ducts resulting from anomalous remodeling of the biliary primordium called the 'ductal plate'; these conditions can be collectively referred to as ductal plate malformations and have been reported in cats.[18,25,26] Although the choledochus is not derived from the ductal plate the presence of concurrent intrahepatic anomalies falls within the accepted six-type classification scheme of CCs, as type IV.

A common hypothesis for the development of acquired CCs in humans is the presence of an abnormal pancreaticobiliary junction (APBJ), which is reported in 57–96% of patients with CCs.[17,25] In humans, APBJ is described as confluence of the pancreatic duct and CBD into a common channel prior to insertion into the duodenal wall; this configuration permits distal mixing of bile and pancreatic enzymes and is thought to lead to mechanical weakening of the CBD wall with secondary pathologic dilatation.[17] Overall, the presence of CCs, with or without concurrent APBJ, often leads to biliary stasis, predisposing the patient to chronic inflammation (choledochitis, cholangitis, pancreatitis), cholelith formation, infection and possibly increased risk for biliary malignancy.[8,16–18,21,25,27]

CCs are rarely reported in the veterinary literature with only two prior case reports in cats, to our knowledge.[21,22] In our case series, the CCs were round to fusiform in shape and varied in size with the largest CC measuring over 5 cm in diameter. The CBD was affected anywhere along its length. In the two previously published cases one cat had a round, 1.5 cm diameter cyst of the distal CBD,[22] and the other cat had a fusiform 10 cm diameter cyst of the proximal CBD.[21]

In humans, CCs are sub-categorized based on morphology with the most recent classification scheme divided into six types.[17,19,20,25] The differentiation of CC type is based on shape (cystic, fusiform/saccular or diverticular), location (extrahepatic, intrahepatic or both) and extent of the CC (segmental or diffuse).[17] Surgical resection of the CC is the treatment of choice in humans for cysts affecting extrahepatic bile ducts (eg, types I–III) to address secondary inflammation and stasis and reduce the risk for regional malignancy.[16,28] Treatment for cysts involving both extra- and intrahepatic structures (type IV) is considered challenging because despite resection of the extrahepatic cyst(s), patients continue to have serious complications from the unaddressed intrahepatic component such as recurrent cholangitis, hepatic abscessation and even sepsis. More invasive surgical interventions such as partial hepatectomy or liver transplantation have been performed in humans with type IV CCs, when necessary.[28] The two previously reported CCs in cats were morphologically suspected to be type I[21] and type I, II or III; [22] both cats underwent combined surgical and medical management. Of the cats presented in this series two cats were suspected to have type I CCs (cases 1 and 3) and two were suspected to have type IV CCs (cases 2 and 4).

Owing to the inherently high morbidity and mortality of biliary surgery in the cat, the decision to pursue and choose the appropriate surgical intervention in cases of CCs should be considered with caution.[6,10] In our cats,

there was no need for emergency surgical intervention, allowing time for assessment of response to medical management. This is especially pertinent for this population because the cats with CCs in our series and past reports often had comorbidities. In the two cases that had liver histopathology performed (cases 1 and 2) both had evidence of inflammation, although this was mild in case 2. One cat (case 1) had multiple positive bile cultures compatible with bacterial cholangitis/cholangiohepatitis. This is similar to the two prior case reports where both cats had chronic cholangitis, one had evidence of chronic pancreatitis and one cultured positive for *E coli*. Underlying intestinal disease was confirmed in case 1 via endoscopic biopsies and was suspected in case 3 based on ultrasonographic findings. Despite the presence of a CC, cat 3 did not demonstrate any biochemical changes in support of hepatobiliary disease. The clinical signs in this cat were mild and non-specific (chronic intermittent vomiting), and therefore it is difficult to evaluate the clinical impact of the CC, especially in the light of intestinal changes seen on ultrasound. Surgery was not considered indicated in this cat, but re-check blood work and ultrasound assessment were recommended over time.

One cat in our series (cat 1) underwent surgery on the assumption that it could address the segmentally abnormal bile flow suspected to predispose to repeated infections. The CC in this cat was large, fusiform and located within the proximal to mid aspect of the CBD, consistent with a type I CC. The morphology of the CC was comparable with the one previously reported by Best et al.[21] In that study, the cat had a combined partial cyst resection, omentalization and cholecystojejunostomy with postoperative medical management. Cat 1 had a choledochoduodenostomy that was successful in improving clinical signs associated with recurrent cholangitis and led to resolution of the extra-/intrahepatic biliary dilatation. In the case reported by Grand et al,[22] the cat also underwent surgical resection; however, owing to differences in morphology duodenotomy, biliary stenting, cyst resection, CBD reconstruction, and cholecystostomy tube placement were elected. On follow-up examination resolution of biliary ultrasonographic changes was also observed postoperatively.[22]

Two of the cats in our study were not considered good surgical candidates based on biliary morphology. The multifocal, saccular dilatation of the intrahepatic ducts seen in cats 2 and 4 was distinct from the remaining cats in this series, and the case reported by Best et al,[21] where dilatation was tubular; therefore, these cats were suspected to have type IV CC. The GB was not clearly identified in either cat as a result of severe intrahepatic changes. In these cats, it was speculated that resection of the extrahepatic component of the CC would be unlikely to lead to resolution of hepatobiliary disease. Chronic, life-long, medical management with regular biochemical assessment may therefore be recommended. In addition, given the severity of morphological changes to the CBD and adjacent extrahepatic ducts, the degree of surgical biliary resection and/or diversion is difficult to predict and, to our knowledge, there is no published information on this topic in veterinary medicine. Although we have retrospectively linked the intra- and extrahepatic findings in these cats as type IV CC, the possibility that the intrahepatic component was actually secondary to severe, chronic inflammatory disease and biliary remodeling/fibrosis, or due to a concurrent ductal plate malformation cannot be ruled out.[8,29–31]

We speculate that surgical management for CCs could be considered when the dilatations are focal in nature, when intrahepatic duct involvement is absent or mild/tubular, or when cats have continued clinical signs and biochemical abnormalities despite appropriate medical management. When surgery is indicated, a more detailed anatomical characterization of the biliary tree for surgical planning purposes may be beneficial. Although ultrasonography is a widely available imaging modality for assessing the biliary tract in cats, there are inherent limitations in evaluating markedly abnormal biliary anatomy. In cases with equivocal ultrasound findings additional imaging techniques may include: percutaneous transhepatic cholangiography (PTHC), endoscopic retrograde cholangiopancreatography (ERCP), CT and magnetic resonance cholangiopancreatography (MRCP). The latter is considered the gold standard for anatomic evaluation in humans.[17,27] Abdominal MRI studies are currently not commonplace in veterinary medicine; however, a recent article reported the use of MRCP for evaluating cats with pancreatitis and cholangitis – the CBD was identified in all 10 cats.[32]

Definitive exclusion of EHBDO requiring emergency surgery can be challenging, especially in situations where underlying hepatobililary, pancreatic, or intestinal disease may be causing cholestasis and intermittent or partial obstruction. In this case series, thickened bile ducts and biliary debris were present and may have contributed to intermittent or partial obstruction in some cats. Performing serial biochemical panels and ultrasonographic examinations over time is recommended to monitor hepatobiliary disease and rule out an obstructive component. One additional benefit to the contrast procedures mentioned above, such as PTHC and ERCP, is the potential for concurrent evaluation of anatomy and biliary patency. ERCP has been described in the cat;[33] however, this study reported only a 50% success rate in cannulating the CBD. We are not aware of any reports of the use of PTHC in cats in the veterinary literature. Scintigraphic studies have also been used in human and veterinary medicine to further evaluate biliary patency in cases of questionable obstruction.[34]

Future studies are indicated to further define the incidence of CCs in cats, as well as the relationship between the presence of CCs and concomitant hepatobiliary disease. Evaluation of long-term outcome data for cats with CCs that received medical vs surgical management, especially in the light of differences in cyst morphology, is also needed to make appropriate treatment recommendations.

Conclusions

Segmental dilatations of the CBD may be consistent with CCs. Cats with marked ultrasonographic biliary distension may present with mild, non-specific clinical signs and have minimal biochemical changes; however, some cats with CCs can demonstrate serious sequelae such as biliary stasis, debris/cholelith accumulation and repeated infection. The presence of comorbid conditions (cholangitis, cholangiohepatitis, pancreatitis, inflammatory bowel disease) can complicate management and may contribute to intermittent or partial obstruction. Although emergency surgical intervention is not indicated when EHBO is unlikely, partial or complete CC resection and/or biliary re-routing has led to clinical improvement in some cases.[21,22] Long-term medical management for concurrent disease (cholangitis, biliary stasis, inflammatory bowel disease) is often required. The detailed morphology of the biliary system may assist in selecting cats that would benefit from surgical intervention.

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