ULTRASONOGRAPHIC FEATURES OF EXTRAHEPATIC BILIARY OBSTRUCTION IN 30 CATS

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The goals of our study were to review the ultrasonographic features of spontaneous extrahepatic biliary obstruction in cats and to determine whether these features can assist in differentiating tumor, inflammation, and choleliths as the cause of obstruction. Thirty cats with a presurgical ultrasound examination and confirmed extrahepatic biliary obstruction were studied. A common bile duct diameter over 5 mm was present in 97% of the cats with extrahepatic biliary obstruction. Gallbladder dilation was seen in <50% of the cats. Ultrasound identified all obstructive choleliths (calculus or plugs) in the common bile duct. However, neither common bile duct diameter nor appearance or any other ultrasonographic feature allowed differentiation between tumor and inflammation as the cause of obstruction. A short duration of clinical signs (10 days or less) seemed to be associated with obstructive cholelithiasis. *Veterinary Radiology & Ultrasound, Vol. 48, No. 5, 2007, pp 439–447.*

Key words: biliary obstruction, cat, extrahepatic, ultrasonography, ultrasound.

Introduction

EXTRAHEPATIC BILIARY OBSTRUCTION is uncommon in cats, and its underlying causes are often unknown. The most common causes of extrahepatic biliary obstruction in cats include tumors and inflammation of the common bile duct, pancreas or duodenum, and choledocholithiasis.[1–4] In studies on extrahepatic biliary obstruction in cats, high morbidity and mortality rates were associated with surgical biliary diversion.[5–9] Mortality was associated with the underlying cause, with tumors leading to the highest mortality rate,[6] and choleliths the lowest.[8]

A detailed description of ultrasonographic changes associated with extrahepatic biliary obstruction in cats is not available. In cats, a common bile duct diameter of 5 mm or more is considered to be dilated and supportive of extrahepatic biliary obstruction,[10] and a gallbladder wall thickness greater than 1 mm is considered to be an accurate sign of gallbladder disease.[11]

The goals of our study were (1) to describe the ultrasonographic features occurring with spontaneous extrahepatic biliary obstruction in cats and (2) to determine whether these features can assist differentiating tumor, inflammation, or a cholelith as the primary cause of obstruction.

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Materials and Methods

The medical records collected between 1994 and 2005 at Tufts University Cummings School of Veterinary Medicine (TUSVM, MA, USA) and Imagerie Médicale Vétérinaire (IMV15, Paris, France) were searched for cats with confirmed extrahepatic biliary obstruction. The inclusion criteria were presurgical abdominal ultrasonographic examination, and surgical or post-mortem confirmation of extrahepatic biliary obstruction.

Extrahepatic biliary obstruction was confirmed at surgery by the inability to express bile from the gallbladder or any dilated biliary duct into the duodenum, or by the inability to catheterize the common bile duct from a duodenostomy, cholecystotomy, or choledochotomy incision.

Signalment, clinical presentation and history, serum chemistry values, hematologic results, radiographic, ultrasonographic, surgical, histopathologic, and cytologic reports and outcome were reviewed.

Radiographs were assessed for liver size and contour, presence of gas or mineralized material within the biliary system, presence of a mass, or decreased serosal detail in the abdomen.

Ultrasound examinations were performed using a high-definition ultrasound system* equipped with a 5–8 MHz linear curved array transducer. All videotapes and static images were assessed for diagnostic quality and reviewed.

Ultrasonographic assessments for gallbladder included shape, size, luminal content, wall appearance, and thickness. In a sagittal plane, an oval to pear-shaped gallbladder was considered to be normal. The gallbladder size was

*ATL HDI 3000 and ATL HDI 5000, Philips Medical Systems, Bothell, WA, USA (TUSVM) and ATL HDI 3500, Société Polygone Paris, France (IMV 15).

evaluated subjectively as normal or enlarged. Subsequently, gallbladder volume was calculated using the extrapolated ellipse formula: volume = 0.52 × (height × length × width).[12–14] A value of 0 ml was used for the gallbladder volume of any cat without a visible gallbladder. Gallbladder content was described as anechoic or echoic, and the presence of shadowing was reported.[15,16]

The maximal internal diameter, shape and course, luminal content, wall echogenicity, and thickness of the cystic duct and common bile duct were recorded. Cystic duct diameter was assessed distal to the gallbladder neck. The proximal common bile duct was defined as the portion of the duct located between the cystic duct and the porta hepatis and the distal common bile duct between the porta hepatis and the duodenum (Fig. 1). Common bile duct diameter was measured at the porta hepatis and/or near the duodenal papilla. Maximal common bile duct diameter was used for statistical analysis. The regularity of the common bile duct wall thickness was evaluated. The presence of free peritoneal fluid and changes in echogenicity of the regional fat were noted.

The location, shape, size, and luminal content of visible extrahepatic ducts and intrahepatic ducts were assessed. Any extrahepatic duct or intrahepatic duct visible on ultrasound was considered to be dilated as these ducts are not visible in normal cats. When available, color flow Doppler confirmed the absence of a flow signal in these ducts.

An attempt was made to differentiate intrahepatic ducts from extrahepatic ducts. Intrahepatic ducts were recognized as small tubular or stellate structures located peripherally within the liver or as larger tubular structures converging on the porta hepatis close to and parallel to a portal vein (Fig. 1). Extrahepatic ducts were differentiated from intrahepatic ducts by their more central location, near the porta hepatis. Fusion of extrahepatic ducts with the common bile duct was used as a gold standard for extrahepatic duct identification (Fig. 1).

Liver was assessed for size, contour, echogenicity, and echotexture.

Duodenal wall thickness, duodenal papilla diameter, and maximal thickness of the pancreas were measured and compared with published normal values.[17,18]

Results are presented as the number of cats for categorical variables, and medians/ranges for continuous variables. Cats in our study were divided into three groups based on the primary cause of extrahepatic biliary obstruction: tumor, inflammation, and cholelithiasis. Because quantitative ultrasonographic data were not normally distributed, nonparametric one-way analysis of variance (Kruskal–Wallis) with exact P-values was used to compare these three groups with respect to ultrasonographic measurements. Spearman correlations between the duration of clinical signs and the degree of dilation of the biliary tree (common bile duct, extrahepatic ducts, and intrahepatic ducts) were estimated. The three groups were compared regarding the mortality rate vs. percentage of discharged animals, using Fisher's exact test. Values of $P < 0.05$ were considered to be statistically significant.

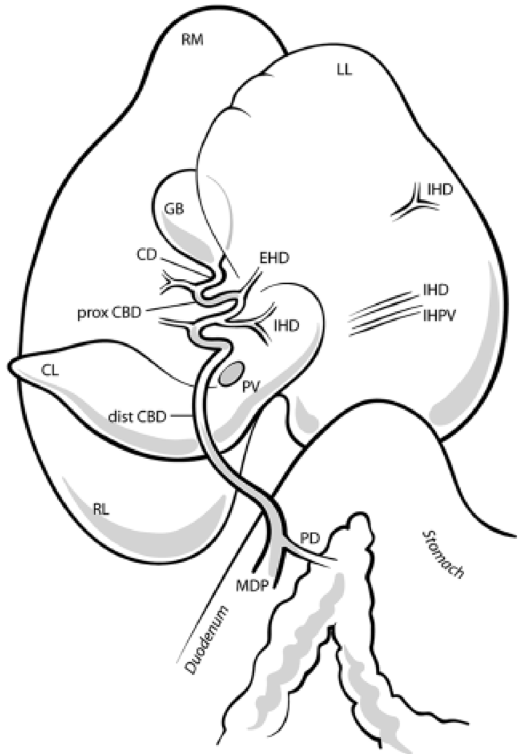

FIG. 1. Schematic illustration of the feline biliary tract. Visceral surface of the liver, proximal duodenum, and part of the pancreas are drawn. Inspired from references 40, 41, 42. GB, gallbladder; CD, cystic duct; prox CBD, proximal common bile duct; dist CBD, distal common bile duct; EHD, extrahepatic duct; IHD, intrahepatic duct; PD, main pancreatic duct; MDP, major duodenal papilla; P, pancreas; PV, portal vein; IHPV, intrahepatic portal vein; LL, left lateral liver lobe; RM, right medial liver lobe; RL, right lateral liver lobe; CL, caudate lobe of the liver.

Results

Thirty cats (24 domestic short hair, three domestic long hair, two Burmese, and one Maine Coon) fulfilled the inclusion criteria (24 from TUSVM and six from IMV15). Seventeen cats were neutered males and 13 were neutered females. Age ranged from 3 to 17 years, with a median of 10 years.

Cats were confirmed at surgery (29/30) or necropsy (1/30) to have extrahepatic biliary obstruction. The underlying cause was neoplasia in 12/30 cats, inflammation in 11/30 cats, and a cholelith in 7/30 cats. Tumor types were: carcinoma of the biliary tract (6/12), pancreatic carcinoma (3/12), unclassified pancreatic/biliary tract carcinoma (1/12), squamous cell carcinoma involving the duodenal papilla (1/12), and duodenal lymphoma (1/12). Underlying inflammatory diseases were chronic pancreatitis (3/11), pericholedochitis (3/11), duodenitis (2/11), choledochitis (2/11), and pancreatic abscess (1/11). Choleliths obstructing the common bile duct were confirmed as calculus made of calcium carbonate or palmitate in 5/7 cats, and amorphous plugs of bile salts and cholesterol in 2/7 cats.

The most common clinical signs were decreased appetite/anorexia (23/30), lethargy (18/30), and vomiting (16/30). Other clinical signs observed less frequently were weight loss (8/30), diarrhea (2/30), and polyuria/polydipsia (1/30). Icterus (22/30), painful cranial abdomen (4/30), hyperthermia (2/30), hepatomegaly (1/30), and cranial abdominal mass (1/30) were noted on physical examination.

The median time for the duration of clinical signs before presentation was 21 days in the tumor group (range 4–180 days), 22 days in the inflammation group (range 3–120 days), and 6 days in the cholelith group (range 1–10 days). The difference between the duration of clinical signs in the cholelith group and the two other groups was statistically significant ($P<0.02$).

During hospitalization, 11 cats died; six had a tumor, four had inflammation, and one had a cholelith. Euthanasia was requested by the owners in 7/11 cats. There was no significant difference in mortality rate among the three groups (Fisher's exact P-value = 0.35).

There was an increase in total bilirubin in 25/28 cats (median 11.1 mg/dl, range 0.1–91 mg/dl—reference values 0.1–0.3 mg/dl), alkaline phosphatase in 25/30 cats (median 384 U/l, range 33–1064 U/l—reference values 10–72 U/l), alanine aminotransferase in 27/30 cats (median 559 U/l, range 29–1848 U/l—reference values 29–145 U/l), gamma-glutamyltransferase in 17/23 cats (median 13 U/l, range 0–78 U/l—reference values 0–5 U/l), and cholesterol in 11/23 cats (median 257 mg/dl, range 99–474 mg/dl—reference values 77–258 mg/dl).

Hematologic results were available in 25/30 cats. Increased segmented neutrophils were noted in 11/24 cats (median $9790 \times 10^3/\mu l$, range $949 \times 10^3/\mu l$–$37{,}037 \times 10^3/\mu l$—reference values $2100 \times 10^3/\mu l$–$10{,}000 \times 10^3/\mu l$). A mild increase in monocytes was noted in 2/23 cats ($1875 \times 10^3/\mu l$ and $1992 \times 10^3/\mu l$—reference values 0–$1640 \times 10^3/\mu l$). Anemia was observed in 9/25 (median hemoglobin concentration of 11.1 g/dl, range 5.4–15 g/dl—reference values 10–15 g/dl and median hematocrit of 32.4%, range 14–44%, reference values 31–46%). Lymphopenia was noted in 14/25 cats (median $850 \times 10^3/\mu l$, range 182–$5320 \times 10^3/\mu l$—reference values $1080 \times 10^3/\mu l$–$6020 \times 10^3/\mu l$).

Abdominal or whole-body radiographs were available in 16/30 cats. Radiographic findings included suspected cranial abdominal mass (6/16), mineralized opacities located in the biliary tree or the gallbladder (7/16), and hepatomegaly (4/16).

All ultrasonographic images were of good diagnostic quality. However, some tapes did not include all the required scan planes for retrospective measurement of quantitative parameters. Consequently, some parameters were assessed in <30 cats.

Ultrasonographically, the gallbladder was seen in 29/30 cats. At surgery in the cat without gallbladder visualization, a bile duct carcinoma was found obstructing the cystic duct and preventing gallbladder and common bile duct filling.

Subjectively, the gallbladder was considered dilated in 13/30 cats. Subsequently, gallbladder length, height, and width were measured in 27 cats (Table 1). The median gallbladder volume of the tumor group and of the cholelith group were the largest and the smallest, respectively, but no statistically significant differences were noted among groups. In 12/13 cats with subjective impression of gallbladder dilation, calculated gallbladder volume was over 10 ml. In 15/17 cats with subjective impression of normal gallbladder size, calculated gallbladder volume was <8 ml.

The gallbladder wall was seen and its thickness was measured in 22/29 cats. The median thickness did not vary significantly between groups (Table 1). Histopathologic examination of the gallbladder wall was performed in 9/30 cats. The gallbladder wall was thick (>1 mm) in all nine cats and histopathologic changes included inflammation (7/9), mucosal hyperplasia (2/9), intramural hemorrhage (1/9), and mural fibrosis (1/9).

The echogenicity of the gallbladder wall was evaluated in 29 cats and was classified as hyperechoic to the surrounding liver parenchyma (12/29), isoechoic (13/29), hypoechoic (3/29), and double-rimmed (1/29). No correlation was found between echogenicity of the gallbladder wall and the final diagnosis.

TABLE 1. Ultrasonographic Quantitative Parameters of the Gallbladder in 30 Cats and by Cause of Extrahepatic Biliary Obstruction

	Gallbladder Volume			Gallbladder Wall Thickness		
	n	Median* (ml)	Range (ml)	*n*	Median** (mm)	Range (mm)
Population ($N=30$)	27	7.2	0–21.9	22	1.7	0.7–6.5
Tumor ($N=12$)	11	10	0–21.9	7	1.8	0.8–2.6
Inflammation ($N=11$)	10	7.2	0.2–21	9	2.3	0.8–6.5
Choleliths ($N=7$)	6	5.5	3.2–14.8	6	1.5	0.7–2

*Not significantly different ($P=0.61$) between tumor, inflammation, and choleliths.**Not significantly different ($P=0.29$) between tumor, inflammation, and choleliths.

The gallbladder content was evaluated in 29 cats. Content was anechoic (4/29), echoic and amorphous (18/29), echoic and organized (3/29), and hyperechoic with shadowing (4/29). One of the cats with an organized echoic nonmobile content had a mucocoele and the four cats with hyperechoic content and associated shadowing had mineralized choleliths.

The cystic duct was visible in 24/30 cats. The shape and course were curvilinear and short (12/24), tortuous (8/24), and pouch-shaped (4/24). The echogenicity of the cystic duct wall was classified as isoechoic to the surrounding liver parenchyma (17/24), hypoechoic (3/24), and hyperechoic (4/24). The thickness of the cystic duct wall was measured in 8/24 cats and the median thickness was 1.1 mm (range 0.9–2.8 mm). The content of the cystic duct lumen was anechoic in 10/24 cats, echoic and amorphous in 12/24 cats, and hyperechoic with distal shadowing in 2/24 cats. The median cystic duct diameter did not differ significantly among the three groups (Table 2). The content of the cystic duct lumen was anechoic in 10/24 cats, echoic and amorphous in 12/24 cats, and hyperechoic with distal shadowing in 2/24 cats.

TABLE 2. Ultrasonographic Quantitative Parameter of the Cystic Duct in 30 Cats and by Cause of Extrahepatic Biliary Obstruction

	Cystic Duct Diameter		
	n	Median* (ml)	Range (ml)
Population ($N=30$)	24	7	3–13
Tumor ($N=12$)	9	5.2	3.3–11.6
Inflammation ($N=11$)	8	8.2	3–13
Choleliths ($N=7$)	7	6.7	4–10.6

*Not significantly different ($P=0.60$) between tumor, inflammation, and choleliths.

The common bile duct was identified in 29/30 cats. One cat had no visible common bile duct and was the cat with a nonvisible gallbladder. The shape of the proximal common bile duct was recorded in 21/29 cats and appeared tortuous in all cats. The course of the distal common bile duct was assessed in 13/29 cats and was straight or slightly undulating in all cats. The median common bile duct diameter did not differ significantly among the three groups (Table 3). The diameter of the common bile duct was relatively uniform in 24/29 cats. A focal dilation (diverticulum) was seen in the remaining 5/29 cats ranging from 16 to 63 mm. The luminal content of the common bile duct was classified as anechoic (7/29), mildly echoic and amorphous (13/29), echoic and organized (2/29), and hyperechoic with shadowing (6/29). Hyperechoic contents with distal shadowing were diagnosed as calculus in 5/6 cats and echoic intraluminal masses were found to be amorphous plugs in 2/3 cats. The common bile duct lumen was focally and markedly reduced in 10 cats by either an intraluminal mass (7/10) or by a focal thickening of the wall (3/10) (Fig. 2). The analysis of the intraluminal masses revealed calculi (5/7) or amorphous bile plugs (2/7). The common bile duct wall thickness was measured in 20/29 cats. The median thickness did not vary significantly between groups (Table 3). An uneven and asymmetrical common bile duct wall thickness was observed in 4/20 cats that proved to be a bile duct carcinoma in all four cats.

In 11/30 cats, a nodule/mass was identified in the region of the distal common bile duct, the papilla, or the proximal duodenum (Fig. 3). The masses were homogenous and hypoechoic (7/11), isoechoic (1/11), inhomogeneous (1/11), or cavitated (2/11). None of these patterns was reliably associated with a specific cause (inflammation or tumor). The nodules/masses ranged from 5 to 52 mm in diameter. They were diagnosed as a tumor in 5/11 cats, and inflammation in 6/11 cats.

A dilation of either extrahepatic ducts or intrahepatic ducts was seen in 27/30 cats. A dilation of extrahepatic ducts was seen in 18/27 cats (Fig. 4). The luminal content of dilated extrahepatic ducts was anechoic in 13/18 cats, moderately echoic and amorphous in 2/18 cats, and hyperechoic with shadowing in 3/18 cats. No significant difference in the median maximum diameter of extrahepatic ducts was noted among the three groups (Table 4).

A dilation of intrahepatic ducts was seen in 23/27 cats. Intrahepatic ducts appeared as isolated peripheral tubules in the liver of 5/23 cats and as branching tubules connected to dilated extrahepatic ducts in 18/23 cats (Fig. 4). In 2/23 cats, intrahepatic ducts appeared as straight anechoic

TABLE 3. Ultrasonographic Quantitative Parameters of the Common Bile Duct in 30 Cats and by Cause of Extrahepatic Biliary Obstruction

	CBD Diameter			CBD Wall Thickness		
	n	Median* (ml)	Range (ml)	n	Median** (mm)	Range (mm)
Population ($N=30$)	29	8.9	5.2–63	20	1.7	0.8–16
Tumor ($N=12$)	11	12	5.8–63	7	2.4	0.8–16
Inflammation ($N=11$)	11	7	6–25	8	1.8	1.2–3
Choleliths ($N=7$)	7	7.9	5.2–14	5	1.4	1.3–2.5

*Not significantly different ($P=0.48$) between tumor, inflammation, and choleliths.**Not significantly different ($P=0.72$) between tumor, inflammation, and choleliths.CBD, common bile duct.

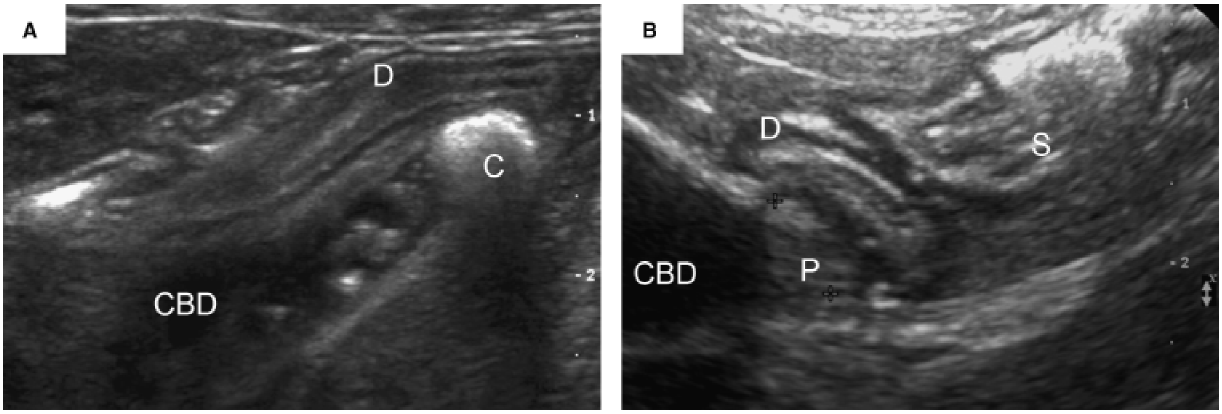

FIG. 2. Sonogram of the distal common bile duct near the duodenum in two cats. The distal common bile ducts were dilated (7 mm in cat A and 14 mm in cat B). (A) The intraluminal structure (C) was hyperechoic and produced a strong distal shadowing; it was a calcium carbonate calculus. In B, the intraluminal mass (P) was moderately echoic with no associated shadow; it was a bilirubin and bile salts plug. CBD, common bile duct; D, duodenum; S, stomach.

tubules parallel to intrahepatic portal veins. The luminal content of intrahepatic duct was anechoic (12/23), echoic and amorphous (3/23), hyperechoic with shadowing (5/23), and unevenly compartimentalized (3/23). The median maximum diameter of intrahepatic duct did not significantly vary among the three groups (Table 4).

Concomitant dilation of both intrahepatic duct and extrahepatic ducts was noted in 14/27 cats. Intrahepatic ducts

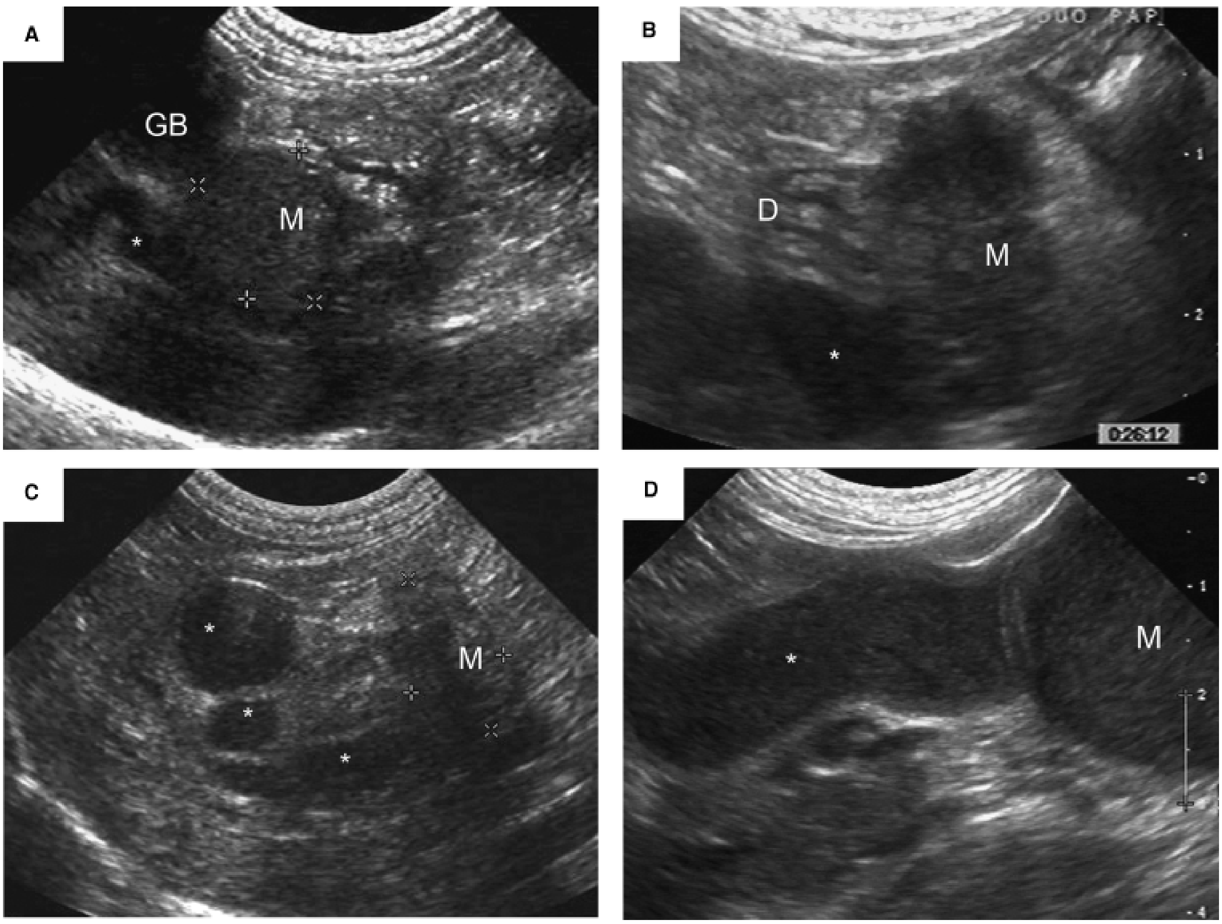

FIG. 3. Sonogram of the region located between the gallbladder and duodenal papilla in four cats. Hypoechoic masses (M) located close to a dilated common bile duct (stars) and close to the gallbladder (GB). (A–C) The masses are irregular and had a coarse echotexture. (D) The cavitated lesion had a thick wall. The final diagnosis was common bile duct carcinoma (A), pancreatic carcinoma (B), chronic pancreatitis (C), and pancreatic abscess (D).

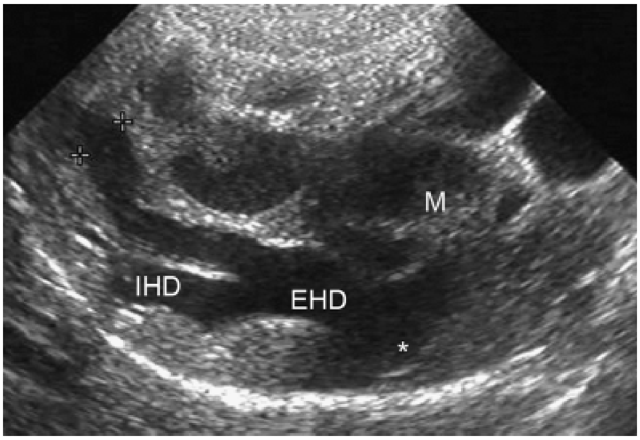

FIG. 4. Transverse sonogram of the liver in a cat with a common bile duct carcinoma. The common bile duct, identified in real time, has been hightlighted with a star. It received a large extrahepatic duct (EHD-7.8 mm). Two intrahepatic ducts (IHD) were identified as they joined the visible extrahepatic duct (one duct has been measured using calipers: 5 mm).

were dilated without dilation of extrahepatic ducts in 9/27 cats and extrahepatic ducts were dilated without dilation of intrahepatic ducts in 4/27 cats.

The liver was evaluated in all 30 cats. Hepatic ultrasonographic changes were present in 15/30 cats and included hepatomegaly (13/30), diffusely hyperechoic liver parenchyma (11/30), single nodule (2/30), or cystic changes within the parenchyma (2/30). In the remaining 15 cats, the liver appeared normal. Nine out of these 15 cats had a histopathologic diagnosis of hepatitis (5/9), cholangiohepatitis (2/9), cholangiocarcinoma (1/9), and adenocarcinoma (1/9).

The duodenum wall was diffusely thickened in 2/30 cats (5.5 and 6 mm in thickness). The layering was altered in one cat, and lost in the second cat. The histopathologic diagnosis was inflammation and fibroplasia of the muscular tunic in the first cat and duodenal lymphoma in the second.

The duodenal papilla diameter was measured ultrasonographically in 11/30 cats. In 4/11 cats, there was thickening of the papilla (5.6–11 mm). Histopathologic examination in these cats revealed duodenitis (3/4) and pericholedochitis (1/4).

The pancreas was seen in 19/30 cats and considered to be thickened in 12/19 cats. Seven out of these 12 cats were in the inflammatory group, 2/12 in the neoplasia group, and 3/12 in the cholelith group. Ultrasonographic changes were observed in the pancreatic parenchyma of five cats. They included mild diffuse hypoechogenicity (3/5) and diffuse coarse pattern (2/5). In six cats, a mass near the proximal duodenum was suspected to originate from the pancreas based on its anatomic location. The pancreatic origin was confirmed in 5/6 cats.

No significant difference was noted for any of the quantitative ultrasonographic parameters (gallbladder volume, gallbladder wall thickness, cystic duct diameter, common bile duct diameter, common bile duct wall thickness, extrahepatic ducts diameter, and intrahepatic duct diameter) between the three groups of cause of extrahepatic biliary obstruction. The only statistically significant difference between the three groups was the duration of the clinical signs ($P = 0.0184$) (Table 5).

There was a significant correlation between the degree of dilation of the common bile duct, extrahepatic ducts, and intrahepatic duct with the duration of clinical signs, with Spearman' correlation coefficients of 0.48 ($P = 0.01$), 0.56 ($P = 0.002$), and 0.48 ($P = 0.01$), respectively.

Discussion

In 97% of the cats in our study, the common bile duct diameter was larger than 5 mm. In accordance with other data, our results support common bile duct diameter being a useful indicator of extrahepatic biliary obstruction in cats.[10,21] The only cat with a common bile duct diameter below 5 mm had a cystic duct carcinoma that prevented normal bile flow into the common bile duct and gallbladder.

Rates lower than 97% of common bile duct distension in cats with extrahepatic biliary obstruction have been reported,[6,9] and this discrepancy cannot be easily explained.

In our study, the degree of dilatation of the common bile duct did not aid in differentiating the cause of obstruction. Based on our results, the degree of common bile duct dilation mostly seems to be influenced by the duration of biliary obstruction as there was an association between the

TABLE 4. Ultrasonographic Maximum Diameter of Extrahepatic and Intrahepatic Ducts in 30 Cats and by Cause of Extrahepatic Biliary Obstruction

	EHD Diameter			IHD Diameter		
	n	Median* (ml)	Range (ml)	*n*	Median** (mm)	Range (mm)
Population ($N = 30$)	18	4.4	2.5–15	23	2.5	1.2–8
Tumor ($N = 12$)	9	5.6	3–15	11	2.5	1.7–8
Inflammation ($N = 11$)	6	4	2.5–7	8	2.6	1.4–5
Choleliths ($N = 7$)	3	4.3	3.8–4.5	4	2.5	1.2–4.9

*Not significantly different ($P = 0.32$) between tumor, inflammation, and choleliths.**Not significantly different ($P = 0.69$) between tumor, inflammation, and choleliths. EHD, extrahepatic ducts; IHD, intrahepatic ducts.

TABLE 5. Duration of Clinical Signs in 30 Cats and by Cause of Extrahepatic Biliary Obstruction

		Duration time	
	n	Median* (Days)	Range (Days)
Population ($N=30$)	28	12	1–180
Tumor ($N=12$)	11	21	4–180
Inflammation ($N=11$)	10	22	3–120
Choleliths ($N=7$)	7	6	1–10

*Significantly different ($P<0.02$) between tumor, inflammation, and choleliths.

degree of common bile duct dilation and the duration of clinical signs, although the strength of the correlation was moderate.

In our study, a calculated gallbladder volume equal or greater than 10 ml agreed with the subjective impression of gallbladder dilation. However, gallbladder dilation was not a reliable sign of extrahepatic biliary obstruction as it was seen in only 43% of the cats with extrahepatic biliary obstruction. This is in accordance with two previous studies where only 38%[9] or 62%[6] of the cats with extrahepatic biliary obstruction had gallbladder distention. In experimental bile duct obstruction in cats, distension of the gallbladder and biliary tree was seen in all cats.[19,21] The low percentage of cats in our study with spontaneous extrahepatic biliary obstruction that had gallbladder dilation might be explained by several factors including the location, extension, duration, and type of the underlying cause as well as compliance of the gallbladder and elasticity of the surrounding liver parenchyma. In our study, the absence of visualization of the gallbladder in one cat was due to a peculiar location of a mass that was occluding the cystic duct and preventing gallbladder filling. Only 20% of cats with choleliths had a distension of the gallbladder. This result contrasts with a report of 100% gallbladder distension due to cholelithiasis in nine cats.[8] In all cats with obstructive choleliths and normal gallbladder volume, histopathologic examination, when performed, revealed moderate to severe hepatic inflammatory changes. One can speculate that underlying liver changes could have affected biliary tree compliance.

Dilation of intrahepatic and/or extrahepatic ducts along with the common bile duct may improve the detection of biliary obstruction. Extrahepatic and intrahepatic ducts are usually not visible ultrasonographically in normal cats.[10,20] In our study, 90% of the cats had dilated intrahepatic and/or extrahepatic ducts, indicating that this sign is an important ultrasonographic feature of extrahepatic biliary obstruction in cats.

Dilation of intrahepatic ducts without dilation of extrahepatic ducts was seen in 30% of the cats in our study. In the cat with no visible gallbladder and common bile duct, intrahepatic duct dilation was the only ultrasonographic feature suggestive of obstruction. A similar pattern of hepatic duct dilation has been reported previously in two cats with parasitic extrahepatic biliary obstruction.[24,30] In dogs and humans, intrahepatic duct dilation has been considered to be an early sign of biliary obstruction.[23,25] Dilation of extrahepatic ducts without dilation of intrahepatic ducts was seen in 13% of the cats in our study. This uncommon pattern of hepatic ducts dilation is believed to represent an early transitional phase in the obstructive sequence.[22,23]

Factors that determine the preferential dilation of extrahepatic ducts vs. intrahepatic ducts have not yet been elucidated. The site, severity, and chronicity of obstruction as well as the compliance of the biliary tree and the elasticity of the liver are potential factors contributing to the variation in dilation of the different segments of the biliary tree.[31,32]

Dilation of intrahepatic and/or extrahepatic ducts is not a pathognomonic sign of extrahepatic biliary obstruction as it has been associated with nonobstructive hepatobiliary diseases.[26–28] For this reason, intrahepatic and/or extrahepatic duct dilation should always be interpreted in the light of clinical and biologic changes as well as other ultrasonographic findings that might be suggestive of extrahepatic biliary obstruction.

In our study, the diameter of the intrahepatic and extrahepatic ducts was not different between tumors, inflammatory diseases, and choleliths and was not helpful in differentiating the cause of obstruction.

Obstructive choledocholithiasis was reliably diagnosed in our study, and it was possible to distinguish accurately between mineralized and nonmineralized choleliths.

Variable ultrasound detection rates of obstructive cholelithiasis ranging from 20%[6] to 88% have been reported.[8] This range may be due to technical difficulties in assessing the distal portion of the common bile duct as bowel gas can prevent accurate assessment.[29] In our study, all obstructive choleliths were located in the distal common bile duct close to or into the duodenal papilla. They had a fixed location and seemed to occlude the common bile duct lumen entirely as outlined by the drastic and focal difference in diameter proximal and distal to the obstruction site.

In most of the cats with no obstructive cholelith, it was not possible to differentiate inflammatory disease from tumor accurately, even in the presence of a mass associated with a distended common bile duct. An extraluminal mass close to the distal common bile duct was seen in 37% of the cats. This is in agreement with other studies.[6,9] However, in those studies, it was not indicate whether ultrasound was useful in determining the origin of the mass and in differentiating tumor from inflammatory disease. In our study, we correctly identified a pancreatic origin of the mass in 55% of the cats. In 18% of these masses, the nature of the lesion was accurately identified by the ultrasound examination. In the first cat, the mass had several features consistent with a pancreatic abscess, that was surgically confirmed. In the second cat, a large mass was noted invading the distal common bile duct and was suspected to be an aggressive tumor. A pancreatic or biliary carcinoma was confirmed histologically. However, in the remaining 82% of the masses, ultrasonographic features were not helpful in differentiating tumor from inflammatory diseases as the size, shape, echogenicity, and location of the masses were similar in the two groups.

The common bile duct wall was seen in 67% of the cats. Its thickness was not different between the tumor, inflammation, and cholelith groups. Irregular wall thickening was noted in 13% of the cats. All these cats had a confirmed carcinoma of the common bile duct. This observation should be assessed further to characterize its predictive value more completely for diagnosing common bile duct carcinoma.

Changes in the ultrasonographic appearance of the liver were noted in only 50% of the cats. In 30% of the cats, the liver appeared normal despite histopathologic changes of hepatitis/cholangiohepatitis or carcinoma. This supports the limited sensibility of ultrasound in detecting infiltrative liver diseases.

Ultrasonographic changes in the pancreas were noted in 67% of the cats with pancreatic tumor and in 67% of the cats with chronic pancreatitis. However, none of the ultrasonographic features allowed a thorough discrimination between pancreatic tumor and pancreatitis because similar changes were noted in both groups.

Ultrasonographic features of biliary tract diseases should be interpreted along with the duration of clinical signs. We found a shorter duration of clinical signs in the cholelith group than in the inflammation or tumor groups. This is consistent with previous reports on extraheptaic biliary obstruction caused by cholelithiasis and tumors or inflammatory diseases.[6,7,33–36] Based on this observation, we speculated that a cholelith is more prone than inflammation and neoplasia to induce an acute/subacute complete obstruction of the biliary tree as it occludes the biliary lumen. A longer duration of clinical signs with inflammatory and neoplastic diseases might be secondary to a slowly progressive obliteration of the biliary lumen.

Clinical signs, blood chemistry data, and hematologic results were in accordance with previous reports on extrahepatic biliary obstruction in cats and were similar among the three groups.[6,8,9,37–39] These findings led to the suspicion of hepatobiliary disease but did not help to confirm extrahepatic biliary obstruction or the cause of obstruction.

The causes of extrahepatic biliary obstruction in this study were arbitrarily classified into three groups for comparison. Separating cholelith from inflammatory disease and even from neoplasia might be questionnable as a cholelith might be a cause but also a consequence of extrahepatic biliary obstruction.[6] Whatever the exact physiopathologic relationship among choleliths, inflammation, tumor, and obstruction, the lowest morbidity and mortality have been found after surgical treatment of extrahepatic biliary obstruction when cholelithiasis was responsible for the obstruction and the highest rate when the etiology was neoplastic.

Finally, this study has several limitations inherent to its retrospective design. The long time frame and the gathering of data collected at two institutions increased the number of uncontrollable variables associated with the equipment quality and various operators. However, the design allowed us to study a larger population of cats with extrahepatic biliary obstruction.

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