Pathogenesis and outcome of extrahepatic biliary obstruction in cats

Extrahepatic biliary obstruction (EHBO) was confirmed at surgery or necropsy in 22 cats. Biliary or pancreatic adenocarcinoma was diagnosed by histopathology in six cats and one cat had an undiagnosed mass in the common bile duct. The remaining 15 cats had at least one of a complex of inflammatory diseases including pancreatitis, cholangiohepatitis, cholelithiasis and cholecystitis. The most common clinical signs were jaundice, anorexia, lethargy, weight loss and vomiting. Hyperbilirubinaemia was present in all cases. Distension of the common bile duct and gall bladder was the most commonly observed finding on abdominal ultrasound. Nineteen cats underwent exploratory laparotomy for biliary decompression and diversion. Mortality in cats with underlying neoplasia was 100 per cent and, in those with non-neoplastic lesions, was 40 per cent. Long-term complications, in those that survived, included recurrence of cholangiohepatitis, chronic weight loss and recurrence of obstruction. Based on these findings, the prognosis for EHBO in cats must be considered guarded.

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INTRODUCTION

Extrahepatic biliary obstruction (EHBO) is uncommon in cats and can be caused by any pathological process that obstructs the flow of bile from the liver and gallbladder to the duodenum. Pancreatitis and neoplasia are the most important causes of EHBO in dogs (Fahie and Martin 1995), but the most important causes in cats have not been well established. Causes of EHBO in cats may include pancreatitis, neoplasia, cholelithiasis, parasitic infection, diaphragmatic hernia and foreign body obstruction (Gibson 1952, Barsanti and others 1976, Feldman and others 1976, Naus and Jones 1978, Meijer and Kimman 1980, Wolf 1984, Martin and others 1986, Jenkins and others 1988, Lewis and others 1991, Boothe and others 1992, Cornell and others 1993, Fahie and Martin 1995, Leveille and others 1996, Pastor and others 1997).

The feline extrahepatic biliary tract is anatomically more similar to the human tract than the canine tract. It consists of the hepatic ducts, gallbladder, cystic duct and common bile duct. The common bile duct usually enters the duodenum at the major duodenal papilla along with the major pancreatic duct. In some cats, an accessory pancreatic duct enters the duodenum through the minor duodenal papilla, 2 cm distal to the major duodenal papilla (Boyden 1957, Crouch 1969). The result of this ductal fusion is a frequent concurrence of pancreatic and biliary disease.

Early surgical decompression of the biliary tract has been advocated to alleviate clinical signs (Bjorling 1991). However, indications for surgical intervention are poorly defined and there is considerable morbidity and mortality associated with operative procedures in cats with EHBO (Martin and others 1986). Decompression can be achieved either temporarily via percutaneous cholecystostomy catheter drainage (Lawrence and others 1992) or by definitive surgical procedures such as cholecystoduodenostomy, cholecystojejunostomy, cholecystotomy and choledochotomy (Martin and others 1986, Bjorling 1991, Fossum 1997).

There is a paucity of information in the veterinary literature regarding the pathogenesis of EHBO and the results of surgical treatment in cats. The aim of this study was to document the clinicopathological findings in cats with confirmed EHBO, and to report perioperative complications and long term follow-up in cases undergoing surgical decompression.

MATERIALS AND METHODS

The medical records of cats presented to the University of Pennsylvania Veterinary Hospital between May 1988 and August 2000 were reviewed. Cats were included in the study if they had clinical signs and laboratory data consistent with hepatobiliary disease, imaging findings consistent with biliary obstruction, and complete obstruc-

Table 1. Clinical signs in 22 cats with extrahepatic biliary obstruction

	n	Per cent
Icterus	22	100
Anorexia	21	95
Lethargy	18	82
Weight loss	18	82
Vomiting	12	55
Dehydration	10	45
Polyuria	4	18
Polydipsia	3	14
Palpable cranial abdominal mass	3	14
Painful abdomen	1	5
Distended abdomen	1	5
Diarrhoea	1	5
Dyspnoea	1	5

tion of the extrahepatic biliary tract confirmed at exploratory laparotomy or necropsy. For the purposes of this study, EHBO was defined as complete obstruction of the cystic or common bile ducts, as evidenced by an inability to express bile from the gallbladder into the duodenum or an inability to catheterise these structures from either a cholecystotomy or duodenotomy incision. Cats were excluded from the study if obstruction was partial, or suspected but not confirmed.

Information gathered from the medical records included signalment, history, progression, results of physical examination, evidence of intercurrent disease, results of laboratory tests, imaging findings, surgical pathology and histopathological examination of samples taken at surgery or necropsy.

Serum biochemical data from all cats and complete blood counts from 21 of 22 cats were available for analysis. Other diagnostic tests included serum thyroxine assay, urinalysis and serological tests for feline leukaemia virus (FeLV), feline immunodeficiency virus (FIV), feline coronavirus (feline infectious peritonitis, FIP) and *Toxoplasma gondii*. Coagulation profiles consisted of one-stage prothrombin time (OSPT), activated partial thromboplastin time (APTT) and plasma fibrin degradation products (FDPs) assay. The OSPT and APTT were expressed as percentage prolongation above the reference range, with a range of ±25 per cent considered normal.

The results of thoracic and abdominal radiography as well as abdominal ultrasonographic examinations were reviewed by one author (R.C.M.), who was blinded to the clinical, surgical and histopathological findings. Abdominal radiographs were examined for evidence of mass lesions, loss of serosal detail, gas accumulation or lithiasis in the right cranial quadrant, suggestive of biliary tract disease or associated pancreatitis (Smith and others 1998). Ultrasonographic examinations were evaluated for extrahepatic biliary tract distension or tortuosity, cholelithiasis and masses, as well as pancreatic and hepatic lesions (Leveille and others 1996, Smith and others 1998).

Exploratory laparotomy was performed via a ventral midline incision, followed by routine exploration of all abdominal organs. Presence of EHBO was confirmed by failed attempts at manual gallbladder expression or catheterisation through a duodenotomy or cholecystotomy incision. Cholecystoduodenostomy, cholecystojejunostomy, choledochoduodenostomy or cholecystectomy were carried out according to standard techniques (Martin 1993, Fossum 1997). In some cases, owners elected intraoperative euthanasia. Biopsy specimens from the liver, gallbladder, pancreas and duodenum were fixed in neutral-buffered 10 per cent formalin solution prior to routine processing for histological examination. Surgical procedures were performed directly by, or under the supervision of, board-certified surgeons.

Long-term follow-up was obtained either from the medical records or by questionnaire. Owners and/or their referring veterinarians were specifically questioned about postoperative complications, long-term morbidity and any recurrence of signs.

RESULTS

Twenty-two cats were confirmed to have EHBO either at surgery (n=19) or necropsy (n=3). Fifteen of the 22 (68 per cent) were domestic shorthaired cats; two (9 per cent) were Siamese; and there was one each of domestic long-haired, Burmese, Abyssinian, Himalayan and Maine coon breeds. Eleven of the 22 cats (50 per cent) were spayed females, nine (41 per cent) were castrated males and two (9 per cent) were intact males. The mean weight of the cats at presentation was 3·7 kg (range 1·9 to 6·3 kg) and the mean age at presentation was 8·8 years (range nine months to 18 years).

The most common presenting clinical signs (Table 1) were icterus (100 per cent), anorexia (95 per cent), lethargy (82 per cent), weight loss (82 per cent), vomiting (55 per cent) and dehydration (45 per cent). At the time of presentation, five cats were febrile (>39·5°C) with a mean temperature of 40°C (range 39·5 to 40·5°C) and two cats were hypothermic (<37·8°C). The mean time from onset of clinical signs to presentation was 20 days (range two days to five months). Ten cats had been treated with antibiotics (amoxycillin, ampicillin or metronidazole) prior to referral, five had received intravenous fluid therapy, and one had received prednisone. Three cats had previously confirmed hyperthyroidism (two were clinically hyperthyroid at presentation and one was euthyroid after thyroidectomy). Five other cats tested for thyroid hormone concentrations upon admission were within the reference range. All animals tested for FeLV (nine cats), FIP (five) and *T gondii* (three) were negative. One of six cats tested for FIV was positive.

Complete blood counts were available for 21 cats and were within the reference range in 10 of these animals. Nine cats had a leucocytosis (mean leucocyte count 28 $\times 10^9$/litre; reference range 5·5 to 19·5 $\times 10^9$/ litre), primarily due to a mature neutrophilia in all cases. Eight cats were anaemic (packed cell volume <0·30 litres/litre; range 0·15 to 0·29 litres/litre). Serum biochemical analysis revealed elevations of alkaline phosphatase (ALP) in 17 of 22 (77 per cent) cats; alanine transaminase (ALT) in 17 of 21 (81 per cent) cats; gamma-glutamyl transferase (GGT) in 11 of 11 (100 per cent) cats; and aspartate aminotransferase (AST) in 12 of 12 (100 per cent) cats. Plasma ammonia concentrations were elevated in eight of 10 (80 per cent) cases, and serum total bilirubin concentration was elevated in 22 of 22 (100 per cent) cats. Serum cholesterol was normal in 21 of 22 (95 per cent) cases. Only seven of 22 (32 per cent) cats had decreased serum albumin concentrations and the decrease was mild in all cases. Two of 22 (9 per cent) cats had moderate elevations of blood urea nitrogen and serum creatinine (Table 2).

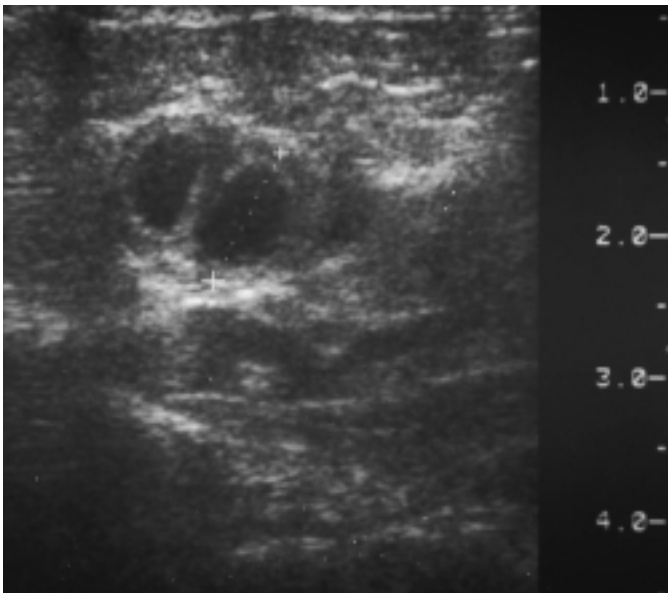

FIG 1. Ultrasonogram (case 15) showing dilation and tortuosity of the common bile duct. Note the thickening of the duct wall (centimetre markers to the right). This cat had cholangiohepatitis with bile sludge causing complete obstruction of the duct

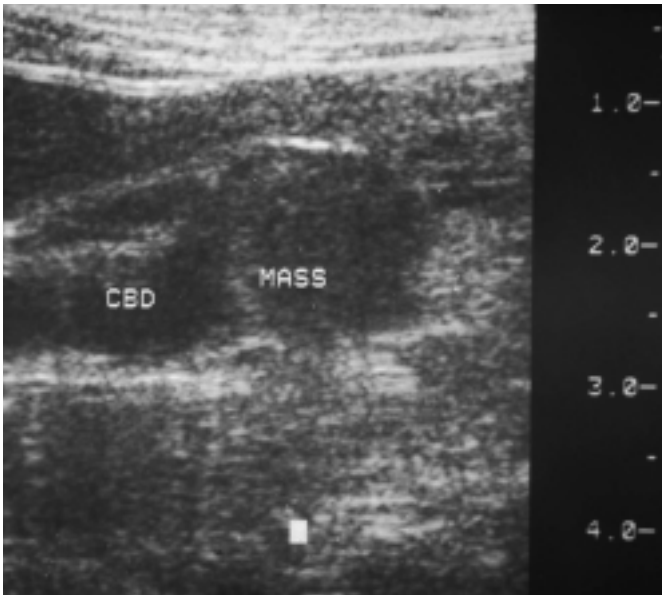

FIG 2. Ultrasonogram (case 21) showing a mass in the wall of the dilated common bile duct. A biliary adenocarcinoma was subsequently diagnosed on histopathological examination of a biopsy specimen

In eight of 18 (44 per cent) cats, the OSPT was prolonged (mean 37 per cent prolongation; range 27 to 69 per cent), and in 10 of 18 (56 per cent) cats the APTT was prolonged (mean 46 per cent prolongation; range 29 to 105 per cent). In four of 17 (24 per cent) cats the plasma FDP assay (reference range <5 µg/ml) was abnormal (>10<40 µg/ml in three cats and >40 µg/ml in one cat). All eight cats with prolonged OSPT had an APTT prolongation.

Urine was analysed in 12 cats. In six cases bacteria were noted on microscopic examination of urine sediments but none were cultured. In six cats, no bacteria were seen and, in two of these, urine culture revealed no bacterial growth. Bile pigments, haemoglobin and bile crystals were detected in the urine of 11, six and one of the cats, respectively.

Thoracic radiographs were performed in 14 cats. Results were normal in 11 (79 per cent) cases. One cat had a mild pleural effusion, one had sternal lymphadenopathy and one had pulmonary hyperinflation. Abdominal radiographs were available for review in 12 cats. Abdominal masses in the right cranial quadrant were suspected in four cases. Hepatomegaly was visible in five cases. Radiopaque choleliths were present in the gallbladder of three cats.

Results of abdominal ultrasound examination were available for review in 21 of 22 cats. Some organs were not imaged in all 21 cats. The most commonly observed abnormalities were in the common bile duct; distension and tortuosity were seen in 17 of 20 (85 per cent) and 15 of 19 (79 per cent) cats, respectively (Fig 1), and masses compressing the common bile duct were observed in four of 20 (20 per cent) cats, two of which were at the duodenal papilla. Choleliths within the common bile duct were observed in four of 20 (20 per cent) cats. Gallbladder distension was evident in 13 of 21 (62 per cent) cats, a thickened wall in 11 of 18 (61 per cent) cats, and calculi within the gallbladder were observed in three cats. Pancreatitis was suspected in six cases and appeared as irregular ill-defined areas of mixed echogenicity. Of these six cases, one was shown to have a biliary adenocarcinoma (Fig 2) (no pancreatic histology), one had a pancreatic carcinoma (and concurrent pancreatitis), two were confirmed histologically as having severe chronic active pancreatitis and two had gross evidence of pancreatitis at surgery. Focal peritoneal effusion was noted in four cats. Pancreatic masses were suspected in four cats (three were pancreatic adenocarcinoma and one was a biliary adenocarcinoma). The pancreas appeared ultrasonographically normal in six cats and was not observed in six others. In two cases, subsequently confirmed to have pancreatitis (one histologically and one on gross examination at surgery), no ultrasonographic changes were seen.

Nineteen cats underwent exploratory laparotomy. Biliary diversion was achieved in 14 animals by either cholecystoduodenostomy (10 cats) or cholecystojejunostomy (four cats). Cholecystojejunostomy was only performed if the duodenum or gallbladder could not be adequately mobilised to allow tension-free anastomosis. In one cat, a duodenotomy followed by catheterisation of the common bile duct allowed biliary decompression. A stent was then placed in the common bile duct. This cat was euthanased three days postoperatively due to persistent hypotension and, at necropsy, the stent was found to be in the duodenal lumen and the gallbladder was again non-expressible. Two cats had cholecystectomy (one with choledochotomy) performed after cholelith removal. One cat had a cholecystostomy tube placed. One cat was euthanased intraoperatively at the owner's request.

Intraoperative hypotension (systolic blood pressure <80 mmHg measured by Doppler ultrasonography) occurred in 13 of 19 (68 per cent) cats. In most cases, this occurred 60 to 90 minutes after the onset of surgical anaesthesia. Of these 13 cats, 11

Table 2. Summary of serum biochemistry findings in cats with extrahepatic biliary obstruction

	Reference range	Mean	Median	Range
Total bilirubin (n=22)	1·71-8·55 µmol/litre	203	162	30·8-513
Alkaline phosphatase (n=22)	23-107 U/litre	337	221	18-1152
Alanine aminotransferase (n=22)	20-107 U/litre	542	435	52-1951
Gamma-glutamyl transferase (n=11)	1-5 U/litre	48	47	8-102
Aspartate aminotransferase (n=12)	1-37 U/litre	273	263·5	48-635
Cholesterol (n=21)	2·7-9 mmol/litre	5·2	5·3	2·1-9
Ammonia (n=10)	6·3-20 µmol/litre	29	32·5	9·7-41·6
Albumin (n=22)	27-39 g/litre	30	30	22-40
Blood urea nitrogen (n=22)	2·6-4·9 mmol/litre	3·4	2·9	1·4-9·2
Creatinine (n=22)	44-177 µmol/litre	133	118	44-371

Table 3. Summary of case details, diagnosis and outcome in 22 cats with extrahepatic biliary obstruction

Case number	Breed	Age* (years)	Weight (kg)	Sex	Surgery	Diagnosis	Outcome
1	DSH	2	2·7	FN	Cholecystojejunostomy	CH, CL	Died 19 months postoperatively (unknown cause)
2	DSH	11·4	4·3	MN	Cholecystoduodenostomy	CH	Lost to follow-up
3	DLH	11·4	4·8	MN	Choledochotomy and cholecystectomy	CC, CL	Alive six years postoperatively
4	DSH	10·5	4·3	FN	Cholecystoduodenostomy	P, CH, HL	Lost to follow-up
5	DSH	8·5	4·4	M	Cholecystoduodenostomy. Revision choledochoduodenostomy at 20 months due to cholelithiasis	P, CH, CC, CL	Further bouts of cholangiohepatitis and cholelithiasis. Alive four years postoperatively
6	DSH	13	2·9	MN	Cholecystojejunostomy	P, CH	Euthanasia 15 months postoperatively (unknown cause)
7	DSH	8·5	5·2	MN	Cholecystojejunostomy	CL	Cardiopulmonary arrest immediately postoperatively
8	DSH	18	2·3	FN	NS	CH, CC, CL	Euthanasia on presentation
9	DSH	2	3·6	MN	Cholecystoduodenostomy and jejunostomy tube	P, HL	Lost to follow-up
10	DSH	15	3·5	MN	Cholecystoduodenostomy and jejunostomy tube	P, CH, CC, HL	Euthanasia seven days postoperatively (persistent hypotension and abscessed jejunostomy tube)
11	Burm	0·9	2·8	M	NS	CH, CC	Cardiopulmonary arrest on presentation
12	Hima	10	2·5	FN	Cholecystectomy	CH, CC, CL	Cardiopulmonary arrest immediately postoperatively
13	Siam	7·8	2·3	FN	Cholecystoduodenostomy and jejunostomy tube	P, CH, CC	Euthanasia one month postoperatively (unknown cause)
14	Siam	8·7	4·6	FN	Biliary stent placed and jejunostomy tube	P, CH, CC, IBD	Euthanasia three days postoperatively (persistent hypotension and abscessed jejunostomy tube)
15	Abys	4	2·5	FN	Cholecystoduodenostomy	CH (Fig 1)	Died three months postoperatively (unknown cause)
16	DSH	6·5	2·5	FN	Cholecystojejunostomy	PA	Cardiopulmonary arrest immediately postoperatively
17	DSH	14·6	4·7	FN	Cholecystoduodenostomy	PA	Cardiopulmonary arrest 48 hours postoperatively
18	DSH	7	4·1	FN	Exploratory laparotomy	PA, CH	Intraoperative euthanasia
19	Mcoo	13	4·4	MN	NS	PA, CH	Euthanasia on presentation
20	DSH	2·7	1·9	FN	Cholecystostomy and jejunostomy tube	BA	Cardiopulmonary arrest 72 hours postoperatively
21	DSH	9	6·3	MN	Cholecystoduodenostomy	BA (Fig 2)	Intraoperative euthanasia
22	DSH	10	4·6	MN	Cholecystoduodenostomy	UnDx mass	Cardiopulmonary arrest 48 hours postoperatively

* Age at first presentation
NS No surgery performed, diagnosis obtained at necropsy
DSH Domestic shorthair, DLH Domestic longhair, Burm Burmese, Hima Himalayan, Siam Siamese, Abys Abyssinian, Mcoo Maine coon, M Male, F Female, N Neutered, CH Cholangiohepatitis, CC Cholecystitis, CL Cholelithiasis, HL Hepatic lipidosis, P Pancreatitis, IBD Inflammatory bowel disease, PA Pancreatic adenocarcinoma, BA Biliary adenocarcinoma, UnDx Undiagnosed

received at least one vasopressor drug (phenylephrine [seven cats], epinephrine [four], ephedrine [two] or dopamine [one], at various dosages). Systolic blood pressure increased after vasopressor therapy in seven cats and failed to improve in four cats (two that received epinephrine and one each that received dopamine and phenylephrine). Eleven of 19 cats received whole blood transfusions and 10 cats had to be withdrawn from isoflurane anaesthesia and were maintained by total intravenous anaesthesia in an effort to maintain normotension. Persistent refractory postoperative hypotension resulted in euthanasia in two cats at the owners' request.

Four of eight liver biopsies, five of 10 bile cultures and the one cholelith sample submitted were positive on aerobic or anaerobic bacterial culture. *Escherichia coli* was cultured from the liver in all four cases. *Clostridium*, *Bacteroides*, *Enterococcus* and *Streptococcus* species were recovered from bile cultures. *E coli* and *Enterococcus* species were cultured from the cholelith submitted.

Histological interpretation of biopsy or necropsy specimens was available in 21 cases. In most, but not all, cats, the pancreas, gallbladder and liver were examined. Pancreatic adenocarcinoma was diagnosed in four cats. Two of these cats had evidence of metastases; one to the liver, the other to the lungs. Two cats had biliary adenocarcinoma. One cat had a mass in the common bile duct which did not undergo histological examination. The remaining 15 cats had at least one of the following: cholangiohepatitis, cholecystitis, pancreatitis, cholelithiasis, hepatic lipidosis and inflammatory bowel disease (Table 3). Seven of 15 (47 per cent) cats had pancreatitis (three observed at surgery and four confirmed on histology); 13 of 14 (93 per cent) and four of 14 (40 per cent) cats, that underwent liver biopsy, had cholangiohepatitis and hepatic lipidosis, respectively; six of 15 (40 per cent) cats had cholelithiasis; and eight of nine (89 per cent) cats that had gallbladder biopsies had cholecystitis (Table 3). Histological examination of the small intestine was performed in only one cat and revealed mild lymphocytic enteritis. Cholelith composition was analysed in one case and was shown to be 100 per cent calcium carbonate.

Seven cats suffered cardiopulmonary arrest and died; one shortly after presentation and the other six within 72 hours of surgery. Six cats were euthanased at the request of the owner; two at presentation, two at surgery due to a poor prognosis, and two due to persistent hypotension and jejunostomy tube site abscessation (at postoperative days 3 and 7, respectively). Three cats were lost to follow-up. Six cats survived to discharge with a mean follow-up time of 26 months (range one to 72 months). Three of these cats died of unknown or unrelated disease one, three and 15 months postoperatively, with no known complications. One cat presented six years prior to its EHBO episode for a common bile duct tear which was managed by cholecystoduodenostomy. At exploratory laparotomy this cat had a totally occluded stoma site and cholelithiasis. This cat died of unknown causes 19 months after revision cholecystojejunostomy and had chronic diarrhoea and weight loss throughout this period. Two other cats remained alive at the time of writing. One had had recurrent bouts of cholangiohepatitis and needed a revision choledochoduodenostomy 20 months after initial cholecystoduodenostomy for recurrent cholelithiasis. The other cat had had no complications in a follow-up period of six years. The overall mortality in cats with underlying neoplasia was 100 per cent within 72 hours of presentation. In cats without neoplasia, the overall mortality within one week of surgery was 40 per cent.

DISCUSSION

In this study, the aetiologies of feline EHBO appeared to fall into two groups: neoplasia of either biliary or pancreatic origin, and inflammatory diseases including pancreatitis, cholangiohepatitis, cholecystitis and cholelithiasis. The pathogenesis of these latter diseases, and their relationship to EHBO, is incompletely understood. An association has been established between cholangiohepatitis, pancreatitis and inflam-

matory bowel disease (Weiss and others 1996), and it has been suggested that cholangiohepatitis and pancreatitis may be extraintestinal manifestations of inflammatory bowel disease due to concurrent pancreatic and hepatobiliary reflux (Baez and others 1999). This theory seems conceivable because of the shared pancreatic and common bile duct anatomy prior to entry into the duodenum in cats (Thune and others 1990). Inflammatory changes are commonly found in both the pancreatic and common bile ducts of cats at necropsy (Jubb and others 1993) and histological evidence of inflammatory bowel disease was found in one cat in the present study that had a gastrointestinal tract biopsy.

The common bile duct passes adjacent to the pancreatic parenchyma before fusing with the pancreatic duct and entering the duodenum (Crouch 1969). Therefore, pancreatic swelling, oedema or fibrosis can cause obstruction of the common bile duct due to compression. This has been confirmed by cases in this study as well as by others (Martin and others 1986, Boothe and others 1992, Fahie and Martin 1995, Leveille and others 1996). The relationship between cholangiohepatitis and EHBO is less certain. Obstruction may be causal, coincidental or a consequence of cholangiohepatitis. Among the cases without neoplasia (15 cats), 93 per cent that had liver biopsies had cholangiohepatitis. At least one cat (case 2, Table 3) in this study that underwent necropsy examination had a primary cholangitis with obstruction caused by proliferating mucosa within the common bile duct, suggesting cholangitis to be the sole cause of EHBO in this patient. Another cat (case 8, Table 3) with a cholelith lodged in the common bile duct, exhibited severe epithelial hyperplasia with surrounding granulation tissue proximal to the site of obstruction and had evidence of severe suppurative and non-suppurative cholangiohepatitis in the liver. Distal to the obstruction, the common bile duct was normal with no evidence of inflammation, suggesting that, in this case, cholangiohepatitis may have been a consequence of obstruction. Two cats with pancreatic carcinomas also had cholangiohepatitis. The role of obstruction and biliary stasis in the pathogenesis of cholangiohepatitis in these cases is unknown but may be significant. The authors suggest that cholangiohepatitis may be a primary cause of EHBO but it may be more commonly a secondary change that occurs as a result of obstruction. This hypothesis is supported by experimental studies demonstrating the development of cholangiohepatitis after ligation of the common bile duct in cats (Stewart and Lieber 1935, Center and others 1986).

Cholelithiasis is uncommon in cats and can be asymptomatic (Center 1996). Choleliths can be a cause or result of EHBO. Experimental ligation of the common bile duct results in bile sludging and cholelith formation in the canine gallbladder within three days of ligation (Bernhoft and others 1983). In cats, although bile sludging does occur, prolonged (up to 42 days) experimental ligation of the common bile duct failed to induce cholelith formation (Stewart and Lieber 1935). Little is known of the lithogenicity of feline bile in health or disease, and cholelith composition has not been extensively investigated in cats. Feline cholelith composition has been reported primarily as cholesterol, calcium and bilirubin, although precise composition analyses have not been reported in most cases (Gibson 1952, O'Brien and Mitchum 1970, Naus and Jones 1978, Hirsch and Doige 1983, Wolf 1984, Heidner and Campbell 1985, Jorgenson and others 1987). The only cholelith analysed in this study was composed entirely of calcium carbonate. Bile stasis and bacterbilia have been incriminated in biliary lithogenesis (Bernhoft and others 1983, Kirpensteijn and others 1993), although a causal relationship has not been proven in the cat. In this study, six cats had choleliths detected at surgery or necropsy. Bile cultures were performed in four of these cases, of which three were positive.

Cholecystitis was found in eight cases in this study. This condition has been associated with EHBO and cholelithiasis in dogs (Church and Matthiesen 1988), and is thought to occur due to the proinflammatory effects of bile stasis after bile duct occlusion or irritant effects of choleliths present in the gallbladder (Center 1996). In this study, four of seven cases with cholecystitis had concurrent cholelithiasis. In seven of eight cases where liver and gallbladder were biopsied, concurrent cholecystitis and cholangiohepatitis were found. This suggests that cholecystitis may be part of the same underlying pathological process that induces cholangiohepatitis secondary to EHBO. The role of bacterbilia in the aetiology of cholecystitis is not known in cats. In one report, 81 per cent of dogs with necrotising cholecystitis had positive bile or gallbladder wall cultures (Church and Matthiesen 1988). Five of seven cats with cholecystitis in this study had bile cultures performed. All five (100 per cent) of these were positive for bacterial growth.

The indications for exploratory laparotomy in cats with a tentative diagnosis of EHBO are not well defined. In the absence of haemolytic or primary hepatic disease, it is generally accepted that an increasing serum bilirubin level over a seven- to 10-day period, combined with supportive radiographic or ultrasonographic evidence of obstruction, is an indication for exploratory surgery (Matthiesen 1989, Center 1996, Fossum 1997). Serum ALT, ALP, AST, GGT, amylase and lipase levels are less specific indicators of biliary obstruction (Center 1996). These findings are supported by the data in this study that show serum bilirubin levels elevated in 100 per cent of cases, while serum ALT and ALP levels were elevated in 77 per cent and 81 per cent of cats, respectively. It was also noted that the increases in ALT and ALP were smaller and less consistent over time, compared to reference ranges, than those for total bilirubin. Although 100 per cent of the cats had elevated AST and GGT in this study, the specificity of these enzymes for EHBO is unknown. Serum cholesterol assay did not appear to aid diagnosis of

EHBO as 95 per cent of the cats had normal levels; in contrast, hypercholesterolaemia is a common finding in dogs with EHBO (Center 1996). Biochemical abnormalities must never be interpreted in isolation. It is known that acute pancreatitis can cause a temporary, potentially reversible, bile duct occlusion in cats (Fossum 1997). This demonstrates the importance of obtaining radiographic or ultrasonographic evidence of an obstructive process that could potentially be surgically palliated by biliary decompression or diversion.

On ultrasound examination, 85 per cent of the cats had common bile duct distension and 62 per cent had gallbladder distension, when evaluated subjectively. In two cats, neither structure was distended. Evidence of gallbladder distension, and a dilated and tortuous common bile duct or hepatic ducts is suggestive of obstruction, although ultrasound examination cannot always confirm current obstruction without induction of gallbladder emptying with either a fatty meal or cholecystokinin (Smith and others 1998). Others have found the diameter of the common bile duct to be less than 4 mm in normal cats, and at least 5 mm in most cats with EHBO (Leveille and others 1996). In contrast, after prolonged distension of the common bile duct in dogs, loss of elasticity of the duct can result in permanent distension, although the gallbladder enlargement usually resolves (Raptopoulos and others 1985). Gallbladder wall thickening was subjectively observed in 61 per cent of cats in this study. Wall thickening is a non-specific sign of EHBO in cats (Hittmair and others 2001). Thus, ultrasonographic features of EHBO may not be specific for current obstruction and should not be considered in isolation as an indication for surgery.

Radiographic features of EHBO are rarely specific (Smith and others 1998). Radiodense calculi in the right cranial abdominal quadrant may be suggestive of biliary disease but can be present in asymptomatic unobstructed cats (Center 1996). Scintigraphy can give accurate information on gallbladder emptying and, where available, may be a valuable additional diagnostic test (Boothe and others 1992, Newell and others 1996).

Perioperative morbidity and mortality in cats with EHBO were high in this study. Nineteen cats underwent ventral midline laparotomy. Within 48 hours of surgery postoperative mortality was 57 per cent (excluding cats that were euthanased or without follow-up). The precipitating factors that led to cardiopulmonary arrest in these cases are not known. Six further cats were euthanased at the request of the owner due to poor prognosis (four cases) or poor recovery from surgery (two cases).

EHBO has been associated with numerous perioperative complications including hypotension (Alon and others 1982), decreased vasopressor response (Finberg and others 1981), decreased myocardial contractility (Green and others 1986), acute renal failure (Pitt and others 1981, Dixon and others 1983), coagulopathies including disseminated intravascular coagulation (Wardle 1975), gastrointestinal haemorrhage (Dixon and others 1984), delayed wound healing (Bayer and Ellis 1976) and high mortality (Pitt and others 1981, Dixon and others 1983). A mean postoperative mortality rate (from multiple studies) in people with EHBO of 13 per cent has been reported (Pain and others 1985). Principal risk factors are anaemia, hyperbilirubinaemia and malignancy (Pitt and others 1981, Dixon and others 1983). Unfortunately, small case numbers in this study precluded meaningful risk factor analysis. Intraoperative hypotension was very prevalent and necessitated discontinuation of inhalant anaesthetic gases, or vasopressor and blood product support, to maintain normotension in some cats. In some cases, response to hypertensive measures was poor. Two cats were euthanased postoperatively due to persistent and refractory hypotension and abscessation of their jejunostomy tube sites.

The physiological basis of these complications is poorly understood. It is hypothesised that the absence of bile salts in the intestinal tract leads to bacterial overgrowth and endotoxin absorption (Bailey 1976). Impaired clearance of endotoxins due to reduced reticuloendothelial function in the liver (Wen Ding and others 1994) leads to peripheral endotoxaemia (Bailey 1976). Endotoxin is a potent renal vasoconstrictor that is capable of causing acute tubular necrosis (Wardle 1970). Gastrointestinal bleeding may occur due to endotoxin-mediated gastric ischaemia and increased acid secretion (Dixon and others 1984). Decreased fibroplasia and angiogenesis, causing delayed healing in abdominal wounds of jaundiced patients has been shown, but is not consistently seen in clinical cases of EHBO (Bayer and Ellis 1976). Hypotension and decreased myocardial contractility has been demonstrated, although the mechanism remains unclear (Alon and others 1982, Green and others 1986). As many postoperative complications of EHBO appear to be endotoxin-related, therapeutic intervention to prevent endotoxaemia may be of benefit. Bile salt supplementation, administration of polymixin B, cimetidine, lactulose and bowel irrigation have all been shown to prevent endotoxaemia (Pain and others 1985) and may have been beneficial to cats in this study.

This retrospective study did have some limitations. Case management and surgical technique will inevitably vary from surgeon to surgeon as well as over time. It should be noted that the prevalence of cholangiohepatitis, cholecystitis and pancreatitis occurring with EHBO may have been overestimated in this study due to the likelihood that surgical biopsies were taken from grossly abnormal organs which are therefore more likely to have histopathological change. Small case numbers preclude the identification of specific risk factors for perioperative complications in feline EHBO surgery. Furthermore, the effect of chronicity of disease (mean time from onset of clinical signs to presentation was 20 days) on outcome is unknown.

Conclusions

EHBO is uncommon in cats. In this group of cats, it was most often associated with malignant neoplasia or a complex of inflammatory diseases including pancreatitis, cholangiohepatitis, cholelithiasis and cholecystitis. These cats had persistent hyperbilirubinaemia and ultrasonographic evidence of obstruction, and these parameters were used as indications for exploratory laparotomy and biliary decompression. Based on this clinical experience, the prognosis for cats undergoing laparotomy should be considered guarded; perioperative morbidity and mortality is high. The majority of cats in this study had a prolonged disease course, and it is possible that surgical intervention early in the course of disease may improve results. Long-term complications seen after biliary decompression include recurrent cholangiohepatitis, chronic weight loss and recurrence of obstruction.

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