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CASE SERIES
A series of six cases of sphincter of Oddi pathology in the cat (2008–2009)

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The sphincter of Oddi (SO) is located within the wall of the duodenum as the terminal part of the common bile duct. Six cats are reported with obstructive processes within their SO. Three of them may have had some form of sphincter dysfunction associated with the pre-existing complex known as 'inflammatory bowel disease' (IBD), two may have had the equivalent of the infant human condition known as 'bile plug syndrome' and the sixth had sphincter dysfunction associated with a tumour at the confluence of the common and right hepatic duct. In all six cases, the sphincter obstructions were surgically managed. The outcomes for 4/6 were favourable but 1/6 was euthanased intraoperatively, and 1/6 had a metastatic neoplasia and was euthanased 2 months postoperatively.

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The anatomy of the sphincter of Oddi (SO) in the cat was first described in detail by Boyden.[1] He reported in his study of the muscular arrangement in the cat's choledochoduodenal junction that in the proximal part of the intra-mural sphincter the pancreatic and bile ducts are separated by a muscular septum. Boyden[1] suggested that this anatomical arrangement may selectively protect the bile duct against duodenal reflux.

Mammalian species have one of two types of SO, 'pump' or 'resistor' sphincter.[2] A resistor sphincter is the type found in the cat, where choledochoduodenal flow occurs along a hydrostatic bilioduodenal pressure gradient.[2,3] In the cat, the SO is 12.36 ± 0.47 mm long and the external diameter of its common bile duct (CBD) is given as 5.63 ± 0.43 mm.[4]

The terminal ductus choledochus tapers into a nozzle that opens into the ampulla in which concentric retrograde saccules are located.[5] The lining mucosa of these saccules is flattened in profile.[6] This apparatus is closely associated with its terminal muscular sphincter.[5] This anatomical arrangement may serve to prohibit the reflux of duodenal contents into the bile duct.[5] Carlbuig et al[2] demonstrated that reflux of duodenal fluid into the bile duct failed to occur in the cat even with duodenal to bile duct pressure gradient as high as 90 cm H_2O.

The pancreatic duct merges with the bile duct within the duodenal wall, such that the ampulla is a common structure.[1] Thune et al[3] found in the cat, no functional or morphological evidence of a common sphincteric ampulla between the bile and pancreatic ducts with each duct having its own sphincter muscle ensheathing its terminal part.

In the cat, basal flow is higher in the pancreatic duct than in the bile duct, and the pressure within one duct is not affected by flow in the other.[3]

The term 'sphincterotomy' in this paper, is given to the procedure which longitudinally incises the ampulla.

Reports of SO pathology in the cat with inter-current small bowel pathology have been reported infrequently.[7,8] This paper reports six feline cases presented to the Animal Health Trust (AHT) during 2008–2009 where pathology was observed in the SO, and describes the procedures and outcomes of their treatments.

Case reports

Case 1

The cat was a 15-year-old female neutered domestic shorthair. The owner reported that the cat had been losing weight for some period of time, but eating as normal. The only abnormal findings on routine clinical examination were a bilateral goitre, a grade 2

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systolic cardiac murmur and small kidneys. On abdominal ultrasound examination, her liver was found to be enlarged with severe patchy nodular echogenicity; the gall bladder wall was thickened and the bile duct was tortuous but its diameter appeared to be near the smaller end of the normal range (4.7 mm). Both kidneys were smaller than the normal range, with a loss of corticomedullary definition. There was a moderate amount of free intra-abdominal fluid. This fluid was not sampled for either cytological or biochemical analysis. Abnormal systemic biochemical parameters are shown in Table 1.

The cat underwent exploratory coeliotomy to obtain multiple liver and gut biopsies. The relevant findings were: multiple biliary cysts under Glisson's capsule; a bilobed and turgid gall bladder (which could not be emptied with manual pressure); and a CBD >10 mm in diameter with obvious neovascularisation as it entered the duodenal wall. The free fluid observed by ultrasonography was not found. A transduodenal surgical approach to the major duodenal papilla (MDP) was made as previously described.[9] A 2–3 mm long dorso-medial orad sphincterotomy was performed and a small calculus was removed from the intra-mural portion of the SO. A trans-abdominal wall 1.5 mm outside diameter (OD) silicone Foley catheter (Veterinary Foley Catheter; Smiths Medical) was placed through a stab incision in the duodenal wall, about 5 mm distal to the distal limit of the duodenotomy incision, into the duodenal lumen. The catheter was advanced through the MDP into the CBD such that the tip was between the most distal hepatic duct and the duodenal wall. Its balloon was gently inflated with saline solution until it just occluded the lumen of the duct. The duodenotomy was closed in a routine manner. The stoma in the duodenal wall through which the catheter passed was closed with a purse string suture of 3/0 polyglytone (Caprosyn; Johnson and Johnson). The duodenum was pexied to the abdominal wall to minimise tension on the catheter and the abdominal wall was closed in a routine manner. The catheter was removed by traction, 24 h postoperatively.

Histological findings comprised biliary and oval cell hyperplasia in the liver with bridging fibrosis and moderate oedema and diffuse moderate to marked non-suppurative cholangiohepatitis. There was moderate lymphoplasmacytic enteritis in the ileum and duodenum, and marked lymphoplasmacytic enteritis with villous blunting and fusion in the jejunum.

The cat is currently on a management regime for its hyperthyroidism (carbimazole – Vidalta; Intervet) and inflammatory bowel disease (IBD) (diet and corticosteroids) and has been clinically well for the follow-up period of 8 months. Periodic ultrasonography of the abdominal cavity has failed to demonstrate the presence of free fluid. The aetiology of the initial free fluid remains unknown.

Case 2

A 10-year-old male neutered domestic longhair cat was referred to the AHT as an after hours emergency in a severely depressed state, with a history of having been missing for 48 h. On presentation he was very lethargic, reluctant to move, resting in lateral recumbency and with some pain on palpation of his epaxial lumbar muscles. He was in poor body condition (score 1/5).

Abdominal ultrasound demonstrated an empty gall bladder with thickening and hyperechogenicity of the mesentery, and a small amount of free intra-abdominal fluid. Some fluid was removed with ultrasound guidance and was examined by one of the attending clinicians. The free fluid was cytologically consistent with an infectious or biliary peritonitis. An aliquot was submitted for bacteriological culture. A detailed, routine haematology and biochemistry analysis of the cat's blood were not undertaken before the surgery. An emergency exploratory coeliotomy revealed approximately 200 ml of flocculated, yellow fluid within the abdominal cavity; the liver had a mottled surface with a red/grey colour; the CBD was dilated and approximately 7 mm in diameter, and all visible hepatic bile ducts were dilated; the gall bladder was small and essentially empty; a firm, linear mass was palpated within the intra-mural portion of the SO; all of the

Table 1. Pre-operative abnormal biochemical values from five cats.

Reference interval	ALT	AP	Cholesterol	CK	Glucose	K	P	Amylase	Albumin	Bilirubin	Bile acid
	0–80 IU/l	0–50 m/l	1.9–3.9 mmol/l	21–56 IU/l	3.5–7.5 mmol/l	4.0–5.0 mmol/l	1.1–1.6 mmol/l	900–3000 IU/l	25–35 g/l	0.0–5.0 μmol/l	0.0–15.0 μmol/l
Cat 1	NE	188	7.6	167	15.2	3.9	NE	NE	NE	NE	29
Cat 3	590	102	NE	NE	NE	5.3	1.9	622	NE	NE	NE
Cat 4	573	468	NE	682	NE	NE	NE	NE	24	54.2	54
Cat 5	NE	NE	NE	NE	NE	NE	0.9	448	19	60	NE
Cat 6	213	57	4.3	1095	7.7	NE	0.9	862	NE	NE	NE

*Cat 2 was not evaluated.ALT = alanine aminotransferase; AP = alkaline phosphatase; CK = creatinine kinase.K = potassium; P = phosphorus. NE = not examined.

abdominal contents were stained brownish-red in colour and the pancreas was friable and easily fractured.

Biopsies were taken from the liver, pancreas and mesenteric lymph node. The surgical approach to the MDP was as for case 1. A 0.3 mm OD polyurethane catheter (Milacath: Mila International) was passed into the CBD and when it was retrieved it contained an 8 mm long plug of inspissated bile. A 1.5 mm OD silicone Foley catheter was placed as for case 1. The common, cystic and hepatic bile ducts and the gall bladder were dilated with saline. No leakage was found. The previously palpated intra-mural mass was now no longer palpable. The duodenotomy was closed in a routine manner. Following a 500 ml volume lavage of body temperature normal saline solution of the abdominal cavity, the cat had a sudden decrease in its saturated oxygen level resulting in an acute cardiorespiratory crisis and in spite of a vigorous attempt at resuscitation was pronounced brain dead. He was euthanased.

On histological examination, the lymph node showed a moderate lymphoid and plasma cellular hyperplasia and an increase of polymorphnuclear granulocytes within the medullary sinuses. There was chronic-active mainly peripheral pancreatitis, mild to moderate lymphoplasmacytic infiltrates in a majority of portal fields with multifocal penetration of the limiting plates by single cells or small clusters and early bile duct hyperplasia.

Case 3

A 4-month-old entire male, Maine Coon cat presented for investigation of diarrhoea and vomiting over the past 7 days, and inappetence.

Abnormal findings on clinical examination were depression, mild dehydration, an unkempt hair coat, fluid filled bowel loops, enlarged abdominal lymph nodes and discomfort on palpation of his abdomen.

Abdominal ultrasonography demonstrated a diffusely hyperechoic liver; a grossly thickened gall bladder wall (approximately 2 mm thick); enlarged intra-abdominal lymph nodes and a small 1 mm diameter nodule at the cranial pole of the right kidney.

The only abnormal finding on haematology was a monocytosis (1.6×10^9/l), the abnormal biochemical parameters are shown in Table 1. He was negative on routine tests for feline leukaemia virus, and feline immunodeficiency virus.

An exploratory coeliotomy revealed viscous and bloody bile within the gall bladder; moderately dilated cystic and CBDs; an inflamed duodenum and jejunum and oedematous pancreas. Biopsies were collected from the liver, stomach, small bowel (duodenum, jejunum, and ileum) and pancreas. A sample of gall bladder bile was submitted for cytology, and for bacterial culture. A firm circular mass was palpable within the SO. A dorso-medial sphincterotomy was performed as described above and a biliary calculus was removed from within the SO. The duodenotomy and abdominal wall were routinely closed.

The calculus was not biochemically assayed.

Results of histopathology revealed moderate hepatocellular atrophy and multifocal arteriolar reduplication in the liver; moderate lymphoplasmacytic gastritis and moderate lymphoplasmacytic enteritis with multifocal moderate villous blunting in the small intestine. A sample of bile was sterile on bacterial culture.

The role played by the cholelith in the vomition and inappetence is unknown.

The cat was discharged as a suspected case of IBD with concomitant cholelithiasis. It has been managed with diet modification and corticosteroids and is currently free of clinical signs.

Case 4

This was a 12-year-old, neutered male, domestic shorthair cat which was referred with a history hyperthyroidism (effectively managed with carbimazole − Vidalta; Intervet). However, he had been vomiting a coloured liquid several times per day during the past 2 weeks. On clinical examination he was found to have a grade 2−3 systolic heart murmur and a moderate left goitre. There were no other abnormal clinical findings. His systolic blood pressure was normal at 126 mm Hg.

Abnormal haematological values were a leukocytosis of 21.0×10^9/l with a mature neutrophilia (18.9×10^9/l). Abnormal biochemical parameters are shown in Table 1.

On abdominal ultrasound there was a hyperechoic hepatic parenchyma which was interpreted as cholangitis with a suspected obstruction to the CBD with a focal dilation of 12.5 mm between the last observed right hepatic duct and the duodenal wall; dilated pancreatic ducts (5 mm) were followed into a hypoechoic pancreatic parenchyma; there was also a generalised intra-abdominal lymphadenopathy.

At exploratory coeliotomy, a 20 mm diameter mass (a) was located on the right side of the CBD at the junction with the most distal (right) hepatic duct. This mass was adherent to and appeared to be compressing the CBD. There was a similar sized mass (b) on the left side of the CBD which appeared to be a regional lymph node. There was free bile within visibly dilated pancreatic ducts. A duodenotomy and sphincterotomy were performed as described for case 1. The MDP was swollen, and it was not possible to pass a 1.3 mm OD silicone Foley catheter into the sphincter. The sphincterotomy was extended to permit the passage of the catheter. The catheter on passage continued to enter the pancreatic duct. It required manipulation of the sphincter to gain access to the CBD. The catheter passed freely down the intramural sphincter and the CBD to the level of the obstruction (a total distance of about 19 mm).

Mass (a) was removed from a friable but thickened CBD wall. The CBD at this level was found to be strictured. This stricture was boogied with use of the

balloon of the catheter, with restrictive bands of tissue being resected free from the wall as required. A 2–3 mm rupture of the wall occurred during this procedure. The associate hepatic duct was freed to its origin at the liver, ligated proximally and divided at that site. Its distal length was opened dorsally to the point where the duct joined the common duct. This modified hepatic duct was used as an advancement flap to seal the defect in the CBD, using 4/0 polydioxanone (PDS 11; Johnson and Johnson) suture material in a simple interrupted pattern. The CBD was then tested for leaks, and none were found. After the closure of the defect, the catheter passed freely though the entire length of the duct, along the cystic duct and into the gall bladder. The entire extra-hepatic ductile system was liberally flushed with saline solution, and the duodenotomy routinely closed. The liver surface appeared to be within normal limits, and no masses were palpated within the liver parenchyma. The right and left middle lobes of the liver were biopsied using the guillotine method. The pancreas was not biopsied.

The abdominal wall was closed in a routine manner.

Histopathology findings revealed an intra-ductile cholangiocellular carcinoma with intrahepatic metastasis. Postoperatively the cat was maintained on a low allergy diet, and progressed well following the surgery but was euthanased some 2 months later due to recurrence of the clinical signs. An autopsy was not performed.

Case 5

A 3-year-old neutered male domestic shorthair cat was presented with a history of having been missing for 5 days. On returning home he was underweight, depressed and anorexic.

On clinical examination he was found to be depressed and cachectic. His heart rate was >200 bpm, with a weak pulse, he was pyrexic (40.6 °C) and on abdominal palpation there was a mid-abdominal mass which was considered possibly suggestive of an intussusception.

The abnormal ultrasound findings included severe distension of the stomach and small intestines with fluid and no peristalsis and a shadowing luminal structure in the bowel with reactive mesentery. The abnormal haematology results were haemoglobin 7.1 g/l (8.0–15.0); red blood cells (RBC) 4.86×10^{12}; packed cell volume (PCV) 21.6; band neutrophils 1.6×10^{9}; and lymphocytes 0.5×10^{9}/l. Abnormal biochemical parameters are shown in Table 1.

In light of the ultrasound and laboratory findings, the cat was given antibiotics (amoxicillin and clavulanate potassium (Augmentin; GlaxoSmithKline) 20 mg/kg IV q6 h, metronidazole (metronidazole injection; Baxter) 10 mg/kg IV q12 h), and Hartmann's solution (Hartmann's solution; Animalcare) containing potassium chloride (Sterile Potassium 15%; Hamchan Pharmaceuticals) 20 mmol/l was administered intravenously at a rate of 1.5 × maintenance. Once stabilised, the cat underwent an exploratory coeliotomy.

The significant findings at surgery were a severe inflammatory response within the omentum and pancreas, approximately 50 ml of fetid brown (suspect chyme) free fluid within the abdominal cavity, over a 45 cm length of terminal ileum were the following cranial to caudal, a 40 mm long plication of two adjacent loops held together with soft fibrin, two small bowel wall ruptures incompletely sealed with omental plugs, and an avascular intussusception with tearing of associated omental vessels. Five free chymoliths were located within the jejunum. Compression of the enlarged gall bladder resulted in a bulging of the intra-mural portion of the SO. At the bulge there was clearly visible through the duodenal wall an 8 mm long plug of bile; the plug failed to move during the compression. A sample of gall bladder bile was collected for bacterial culture. A longitudinal duodenotomy as described in case 1 was performed and the inspissated bile was flushed from the sphincter until normal bile flow was established. The duodenotomy and ileal defects were closed in a routine manner. The intussuscepted length of bowel was resected and the bowel was routinely closed.[10] The abdominal cavity was lavaged with a large volume of sterile normal saline followed by a lavage of (V/V) 1/100 dilution of povidone iodine (Povidone–Iodine antiseptic solution 1% w/v; Vetasept) in normal saline solution (Sodium Chloride 0.9% w/v; Animalcare) followed by further lavage with normal saline solution. The abdominal wall was closed in a routine manner.

The bile was acellular and no bacterial were cultured from it.

The cat was hospitalised for the first 72 h after surgery during which time it continued to receive the antibiotics. Fluid was continued for the first 24 h. It was discharged with antibiotics for a further 4 weeks. The cat made an uneventful recovery and at last recheck (some 3 months postoperatively) was free of any client concerns.

Case 6

A 9-year-old neutered male domestic shorthair cat that had been acquired from a rescue centre 6 months presented to the AHT with a history of intermittent vomiting (twice daily for 3 weeks), anorexia (past 3 days), and episodic (every few days) facial twitching that lasted for a few seconds (past 5 days). On clinical examination he was found to be mildly dehydrated with some loss of muscle mass (body score was 2.5/5) and a palpably thickened duodenum. His pulse rate was 180, and when stressed rose to 240 bmp. The abnormal biochemical findings are shown in Table 1.

Abdominal ultrasound revealed; sand like material in the gall bladder, with a tortuous dilated cystic duct (up to 7 mm wide); the CBD was distended with three apparent calculi within it, the most distal being within the SO; the left pancreatic duct was distended.

An exploratory coeliotomy revealed a pale yellow liver, a turgid bilobed gall bladder that failed to compress on manual palpation, a grossly distended (8 mm diameter) and tortuous cystic duct and hepatic ducts that ranged in diameter from 1 to 2 mm. The SO was distorted into an oblique orad plane instead of one more parallel to the long axis of the duodenum (Fig 1a). The intra-mural sphincter was grossly dilated with the pancreatic duct being dilated (2 mm) and clearly distinguished from the bile duct (3–4 mm diameter) (Fig 1b). The right pancreatic duct contained bile for about its first 40 mm (Fig 1c), the right limb of the pancreas was atrophic and about 12–15 mm in diameter and the pancreas contained many apparently fibrotic nodules.

A duodenotomy was performed as described for case 1. The aV was raised about 5 mm into the lumen of the duodenum (Fig 1d). The lumen was extremely small (Fig 1d) with only a 25 gauge needle sized trickle of bile flowing from it on palpation of the CBD, with most of the bile flowing down the pancreatic duct. A 1.6 mm OD silicone Foley catheter was passed into the SO. It preferentially went down the pancreatic duct. The sphincter was opened for a further 2 mm. The bile duct component of the sphincter was held open by a pair of Halsted forceps and the catheter passed into the CBD. The catheter balloon was partially inflated. The common bile and cystic ducts, and the gall bladder were flushed with sterile saline solution. The catheter was then removed and replaced by one passing via a trans-abdominal and a duodenal wall stoma as described in case 1 (Fig 1e). The duodenum; mucosa of the SO; jejunum, ileum, and

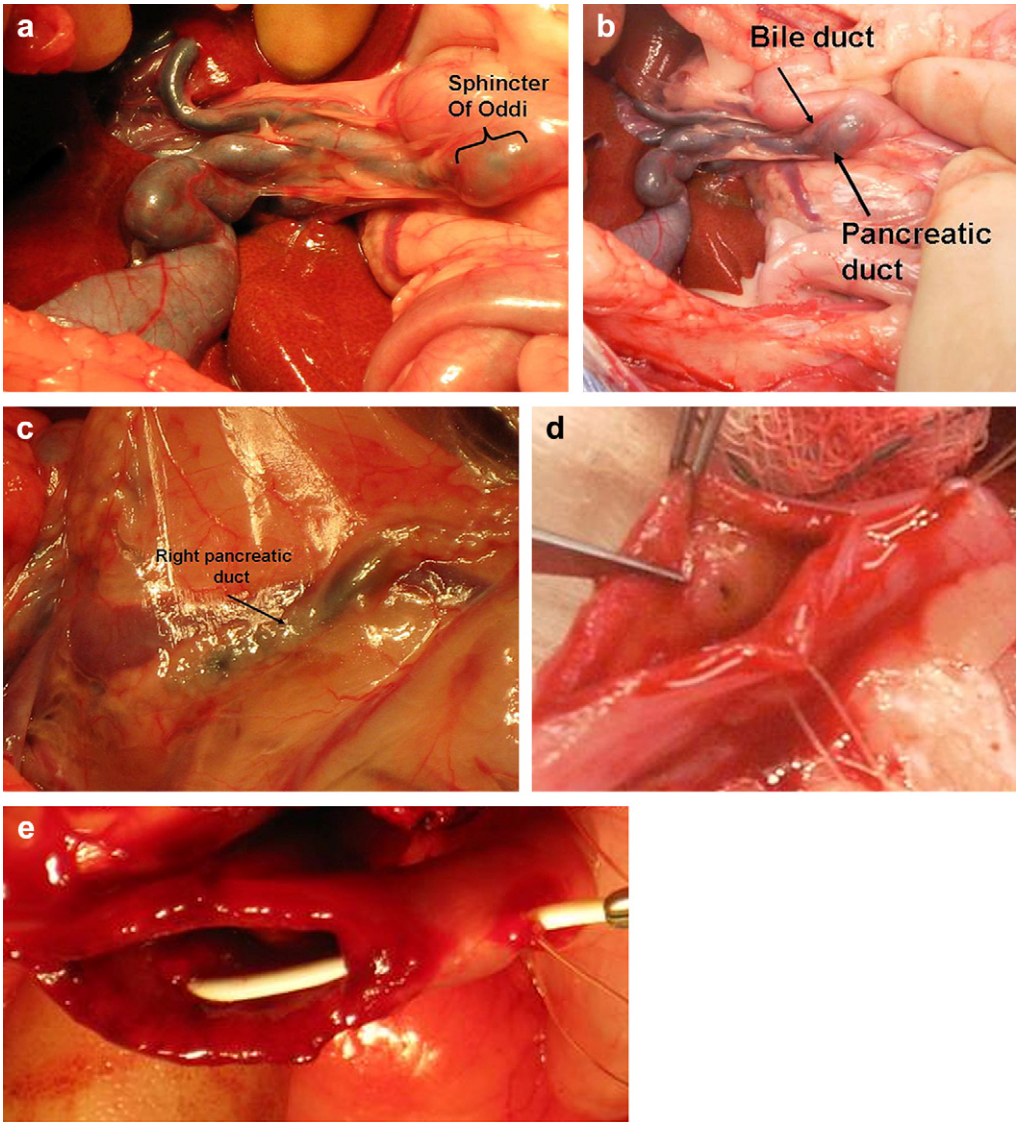

Fig 1. (a) SO. (b) Bile duct and pancreatic ducts within the sphincter. (c) Bile within the right pancreatic duct. (d) Intralumenal ampulla of Vater. (e) Duct of SO catheterised.

the left and right middle lobes of the liver were biopsied. Gall bladder bile was submitted for bacterial culture and cytology and the small bowel and abdominal wall were closed in a routine manner.

The bile was acellular and no bacterial were cultured from it.

Histopathology revealed chronic eosinophilic enteritis with blunting and fusion of villi; eosinophilic inflammatory changes in the mucosa of the SO; and mild to moderate lymphoplasmacytic infiltrates in a majority of portal fields with multifocal penetration of the limiting plates by single cells or small clusters.

The cat was discharged to the care of the owner on the third postoperative day. Once the histological findings were available the cat was prescribed a low allergy diet. The owner reports the cat has continued to do well at home 5 months after surgery.

Discussion

The presence of the calculus within the SO in the first case may have been a primary cause of the biliary outflow obstruction.

In the cat, there is a reciprocal relationship between the contraction of the gall bladder and the relaxation of the SO.[11] The resistance to flow at the choledochoduodenal junction is reduced when the hydrostatic pressure in the gall bladder and bile ducts increased.[12] The reflex relationship is under both excitatory and inhibitory neural regulation.[13]

Experimental obstruction to the CBD in the cat results in significant and potentially irreversible changes within the liver.[14] In the experimental rat, the bile duct diameter increased from 0.8 ± 0.2 mm in the control group to 3.2 ± 0.4 mm and 8.3 ± 2.1 mm at 3 and 5 days, respectively, following the ligation of the CBD. In addition, the thickness of the bile duct wall after processing for histology was 80 ± 10 μm in the control animals, and following ligation for 3 and 5 days was 160 ± 20 μm and 370 ± 30 μm, respectively. There was a loss of elastin within the wall and an increase in collagen[15] suggesting that a return to within normal limits for the duct was unlikely. Should similar histological studies be carried out in the cat extra-hepatic bile duct obstruction for 'several weeks'[14] may not be required before significant and potentially irreversible changes occur in its extra-hepatic bile ducts.

The pancreatic duct in its terminal part is wider in diameter than that of the terminal part of the bile duct.[5] If the common orifice is obstructed, it is suggested that the volume of the ampulla, including all of the saccules may be considerable. If it is filled with bile which cannot flow on into the duodenum when the ensheathing muscles contract, the elevation of pressure may result in regurgitation of bile into the pancreatic duct.[5] This may be the underlying pathophysiology in cases 4 and 6.

The interstitial cells of Cajal are thought to be the pacemakers of SO motility.[16] They have been located within the gall bladder the extra-hepatic bile ducts and in the SO in guinea pigs.[17] In humans, the concentration of these cells has found to be greater in the ampulla and the CBD prompting speculation that their presence at these sites may be implicated in conditions such as SO dysfunction.[18] It has been found that these cells are damaged in inflammatory conditions, and it has been speculated that inflammatory conditions of the SO may also damage these pacemaker cells.[16] How this pacemaker role may be affected by changes in both humoral and neural factors in feline IBD remains to be determined, but hypothetically, damage to these cells may result in SO dysfunction in this species.

Surgical intervention or local pharmacological activity may impact upon normal ampulla function.

Cholecystectomy in the cat damages peridochal nerves and impairs reflex regulation of the SO.[19]

Several substances have been studied that either result in oedema of the ampulla mucosa, or prevent muscular activity within the SO. Two of these are histamine and nitric oxide.

Mast cell degranulation with the release of histamine has been implicated in the presence of IBD.[20] The mucosa of the ampulla, when challenged with histamine, becomes oedematous occluding its meatus.[5] Whether the concentration of duodenal histamine in feline IBD is sufficient to result in the mucosa changes reported by Tansy[6] have yet to be determined, but in cases 1, 3 and 6, local inflammation may have been partially responsible for the clinical status of these cats.

Nitric oxide is released in high concentrations in IBD.[21] Nitric oxide is a potent inhibitor of muscular activity in the SO.[22] Intersphincteric nitric oxide has been found to reduce SO motility in the pig.[23]

Inspissated bile within the SO in cases 2 and 6 suggest that, in these cases, there may well have been some pathology within the ampulla prohibiting free flow of bile into the duodenal lumen.

In guinea pigs following partial ligation of their CBDs, calcium bilirubinate stones developed in all but one animal.[24] In case 3, it is unknown if the increased viscosity of the bile preceded the calculus formation or if the presence of the calculus decreased the flow of bile and thus encouraging the increased viscosity of the gall bladder bile.

In case 4, inspissation of bile within the SO may have occurred due to sluggish bile flow through the sphincter allowing the precipitation of solutes from the bile, or the inspissation may have been associated with increased resistance in the SO due to atypical neural stimulation due to the presence of the tumour affecting the peridochal nerves in the region of the tumour.

The gall bladder has been demonstrated to be a semi-permeable membrane.[25] Bile plug syndrome in infants is thought to be due to dehydration and

sepsis.[26] It is possible that dehydration and/or sepsis 'bile plug syndrome' may have contributed to the surgical findings in cases 2 and 5.

It is also possible, that dehydration may have contributed to the increase viscosity of the gall bladder bile in the third case.

Human bile instilled into the pancreatic duct of the rabbit fails to induce histological evidence of pancreatitis. However, when coliform bacteria infected bile was instilled into the pancreatic duct, but infected bile lead to pancreatitis.[27] Similar findings were reported in the cat where bile alone failed to induce pancreatitis and that bacterial infection was found to be necessary before pancreatitis was induced.[28] In addition, infected bile increased the basal pancreatic ductile pressure by 30%.[28] Infected bile caused acute oedematous pancreatitis at basal duct pressures and at high pressure additional focal necrosis was observed.[28] In spite of bile being visible within the pancreatic ducts in cases 4 and 6 there was no clinical evidence of acute pancreatitis in either of these cases. As histopathology of surgically acquired pancreatic biopsies at a single point in time cannot illuminate the true course of a disease process, had such biopsies been performed in these two cats it would not have been possible to state whether any observed changes within the pancreas preceded or were due to the presence of the bile within the pancreatic ducts.

A consistent protocol in the pre-operative management of these cats may have improved the understanding of the pre-existing conditions in more detail. Empirical biopsying of the pancreas and SO tissue where a sphincterotomy has been performed may also add more information to these types of cases.

In conclusion, investigation of SO pathology perhaps should be included more frequently in the assessment of cats with suspected IBD, and in those cats that are jaundiced from dehydration or sepsis. In those cats where the biliary outflow obstruction fails to clear with conservative management, sphincterotomy with or without stenting has a favourable outcome.

In cats with IBD, attention needs to be focused on the neurohumoral factors that may be at play in any sphincter dysfunction.

Whether these cats, like their human counterparts are in pain with this condition is another facet that requires further investigation.

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