



# Common bile duct obstruction caused by a helminth in a cat in the UK: ultrasonographic findings, histopathology and outcome

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## Abstract

**Case summary** An 11-year-old neutered female domestic shorthair cat presented to our hospital with a 5-day history of vomiting, lethargy, anorexia and hyperbilirubinaemia, despite intravenous fluid therapy, gastroprotectants and antibiotic treatment. An abdominal ultrasound revealed a markedly distended common bile duct (diameter 6.2 mm). The cystic duct and intrahepatic bile ducts were also dilated. A linear structure formed by two parallel hyperechoic lines was identified in the common bile duct and could be traced to the duodenal papilla. The cat underwent laparotomy for surgical decompression of the biliary tree. A tubular, brown-coloured structure was retrieved from the common bile duct. Histological examination was consistent with a degenerate helminth. The cat recovered uneventfully from the surgery and its demeanour and appetite improved rapidly over the following days. Liver and gallbladder wall histopathology was consistent with bacterial cholangitis and cholecystitis. *Escherichia coli* was cultured from both bile and liver parenchyma.

**Relevance and novel information** To our knowledge, this is the first reported case of extrahepatic biliary duct obstruction caused by a helminth in a cat in the UK. We hypothesised that the obstruction had been caused by the aberrant migration of an intestinal nematode that became lodged in the duodenal papilla. Ultrasound allowed prompt diagnosis and guided the treatment decision.

**Keywords:** Common bile duct; cholestasis; liver; helminth; ultrasound

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## Introduction

Several conditions can cause extrahepatic biliary duct obstruction (EHBDO) in cats. These include biliary or pancreatic neoplasia, cholangiohepatitis, pancreatitis, choleliths, choledochitis, duodenitis<sup>1–4</sup> and liver fluke.<sup>5</sup> In the literature, there are also isolated case reports of EHBDO caused by diaphragmatic herniation,<sup>6</sup> a grass awn lodged in the common bile duct,<sup>7</sup> duodenal foreign body<sup>8</sup> and involuntary transcavitary implantation of hair.<sup>9</sup>

Abdominal ultrasound is often performed in cats with suspicion of EHBDO. The diameter of the common bile duct in healthy cats is generally smaller than 4 mm.<sup>10</sup> Dilatation of the common bile duct above 5 mm in

diameter is highly suggestive of EHBDO,<sup>10</sup> although this finding is not present in all cats.<sup>1</sup> Other common ultrasonographic findings include tortuous common bile duct and dilatation of the intrahepatic biliary ducts. Dilatation of the gallbladder is present in <50% of cases.<sup>1</sup> The severity of the changes is correlated with the duration of the

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**Table 1** Progression of total bilirubin, gamma glutamyl transferase (GGT) and alanine aminotransferase (ALT)

Day	1	2	3	4	6	8	27
Total bilirubin ( $\mu\text{mol/l}$ ) (RI 0–15)	85	173	314	418	343	52.6	8.9
ALT (U/l) (RI 12–130)	902	ND	ND	ND	ND	203	101
GGT (U/l) (RI 0–4)	10	ND	ND	ND	ND	4	1

Surgery was performed on day 5

RI = reference interval; ND = not determined

obstruction but not its cause.<sup>1</sup> The identification of choleliths, foreign material or masses via ultrasound can aid in reaching a definitive diagnosis.<sup>1,2,6–8</sup>

Cats with EHBDO usually require surgery to decompress the biliary tree. Surgical options include cholecystectomy, cholecystoduodenostomy or cholecystojejunostomy.<sup>2</sup> Choledocal stenting and temporary decompression via percutaneous biliary drainage has also been used in selected cases.<sup>4,11</sup> The incidence of perioperative complications and mortality is high.<sup>2,3</sup> Unfortunately, there are currently no defined criteria to differentiate cases that will benefit from surgical treatment from those that can be managed medically.<sup>2</sup>

This report describes a case of an EHBDO caused by a helminth in a cat in the UK. We describe imaging and histopathological findings together with case management and outcome.

### Case description

An 11-year-old neutered female domestic shorthair cat was presented to our hospital with a 5-day history of vomiting and progressive lethargy and anorexia. The cat had been rehomed from a rescue centre when it was 2 years old. It had never travelled abroad and it received worming treatment (milbemycin oxime and paziquantel) every 3 months. Blood tests performed at the referring practice revealed a moderate hyperbilirubinaemia (day 1:  $85\mu\text{mol/l}$  [reference interval (RI) 28–51  $\mu\text{mol/l}$ ]), moderately elevated alanine aminotransferase (ALT), mildly elevated gamma glutamyl transferase (Table 1), mild hyperproteinaemia with mild hyperglobulinaemia and leukocytosis with a mature neutrophilia. The cat had received ranitidine and potentiated amoxicillin over 3 days, with no obvious clinical improvement. Abdominal ultrasound performed by the referring veterinarian revealed a distended gallbladder and bile duct with no obvious hepatic changes. Over the following 48h, the cat remained lethargic and anorexic, and the hyperbilirubinaemia worsened ( $173\mu\text{mol/l}$  on day 2 and  $314\mu\text{mol/l}$  on day 3). An oesophageal feeding tube was placed on day 3 and assisted feeding was initiated.

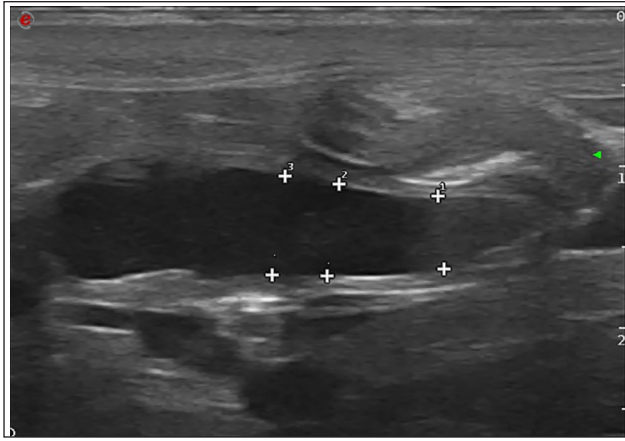
When the cat presented to our hospital (day 4), it was quiet but alert and responsive. It was markedly jaundiced. Abdominal palpation revealed moderate discomfort in the cranial abdomen and tachycardia was detected on



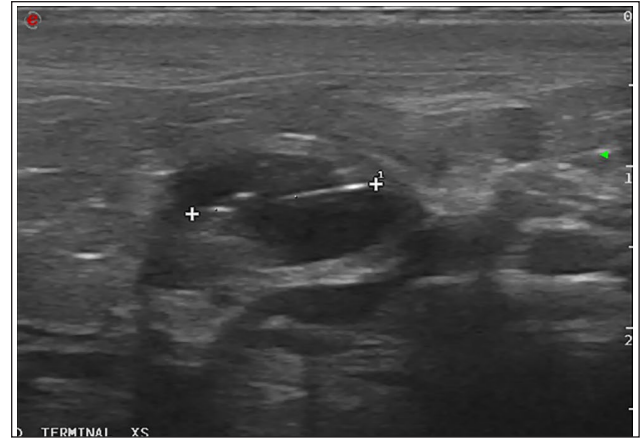
**Figure 1** Ultrasonographic image of the distended gallbladder with anechoic content. The gallbladder wall appears thickened and irregular

auscultation. The hyperbilirubinaemia had increased further (day 4:  $418\mu\text{mol/l}$ ). Hypokalaemia was also detected ( $2.8\text{mmol/l}$  [RI 3.5–5.5  $\text{mmol/l}$ ]). Abdominal ultrasound confirmed a moderately distended gallbladder, with a mildly irregular thickened wall (2.6mm) and clear anechoic content (Figure 1). Dilatation of the intrahepatic biliary tree and cystic duct was also present. The common bile duct was dilated and tortuous, and its maximum diameter was 6.2mm (Figure 2). A distinct, linear structure was identified within the lumen (Figure 3). It was formed by two parallel, hyperechoic lines, approximately 11 mm in length and 1.5 mm in diameter (Figure 4), and it extended through the distal common bile duct and terminated at the level of the duodenal papilla. There were no significant changes in the wall of the duct or in the surrounding tissues. The appearance of the liver, apart from the ductal dilatation, was normal. No other causes of biliary obstruction could be identified. No changes were identified in any other abdominal organ.

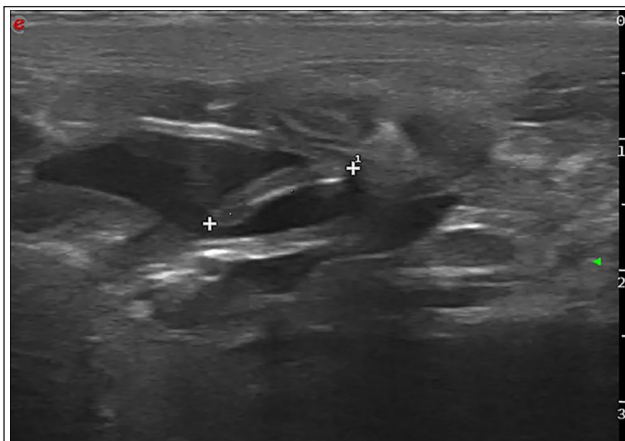
Surgical exploration with a view to biliary decompression was elected as the ultrasound findings were consistent with EHBDO and the cat was not responding to the medical treatment. Laparotomy confirmed dilatation of all components of the biliary tree. The gallbladder appeared grossly abnormal, with a thickened, irregular wall and cholecystectomy was performed. The common



**Figure 2** Ultrasonographic image of the common bile duct in long axis. The duct is dilated with a maximum diameter of 6.2 mm



**Figure 4** Transverse ultrasound image of the distended common bile duct and intraluminal linear structure



**Figure 3** Dilated common bile duct in long axis. In the lumen a linear structure delimited by two parallel hyperechoic lines is visible in this ultrasound image



**Figure 5** Tubular structure retrieved from the common bile duct

bile duct was flushed using sterile saline via a 4 Fr catheter inserted in the duodenal papilla. A large quantity of clear, thick, mucous material was removed from the duct. Together with this material, also a tubular, brown-coloured structure was removed (Figure 5). Biopsies of the liver parenchyma and gallbladder wall were collected for histopathology. Bile and a liver parenchyma biopsy were submitted for culture. Histopathological examination of the structure retrieved from the common bile duct was also requested.

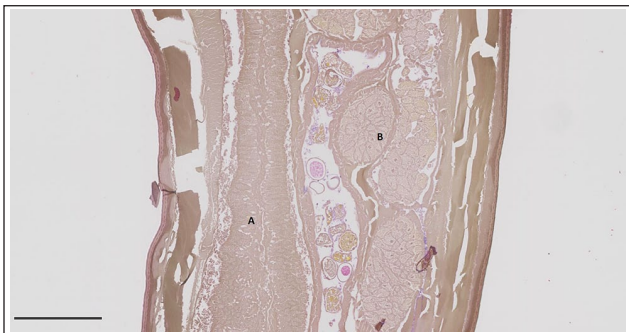
Postoperative support included intravenous fluid therapy with potassium chloride supplementation, maropitant (1 mg/kg IV q24h) and omeprazole (1 mg/kg IV q12h). Potentiated amoxicillin (20 mg/kg IV q8h) was continued pending the bile and liver tissue culture results. Analgesia was provided with a ketamine constant rate infusion and methadone as required based on regular pain score assessment using the Glasgow Feline

Composite Measure Pain Scale. Assisted feeding was restarted as soon as the cat recovered from the general anaesthesia. Fenbendazole (20 mg/kg PO q24h for 5 days) was added to the treatment plan given the suspicion that the structure retrieved from the common bile duct was a roundworm. Over the following days, the cat became progressively brighter and more active. Analgesia was tapered and discontinued on day 5. The cat tolerated the oesophageal tube feeding well and it started eating voluntarily on day 8. Its serum bilirubin decreased rapidly postoperatively (343  $\mu\text{mol/l}$  on day 6 and 53  $\mu\text{mol/l}$  on day 8). The cat was discharged from the hospital 5 days postoperatively (day 9).

The bile and liver parenchyma cultures revealed a profuse growth of non-haemolytic *Escherichia coli*, sensitive to potentiated amoxicillin (see Table 2 for the full list of the antibiotics tested). Antibiotic therapy was therefore continued. The liver histopathology was consistent with marked neutrophilic cholangitis with biliary duct distension and

**Table 2** Antibiotic sensitivity of the non-haemolytic *Escherichia coli* isolated from the cat's liver and bile

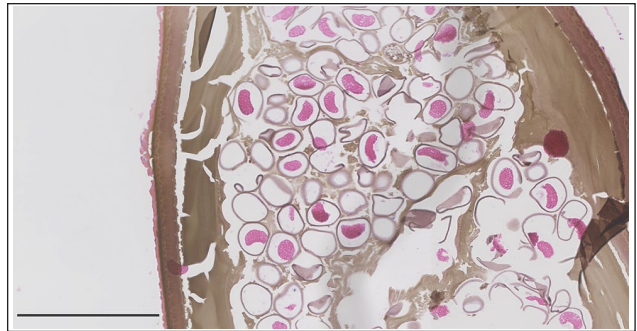
Ampicillin	Sensitive
Potentiated amoxicillin	Sensitive
Cephalexin	Sensitive
Cefovecin	Sensitive
Tetracycline	Sensitive
Potentiated sulfonamide	Sensitive
Enrofloxacin	Sensitive
Marbofloxacin	Sensitive



**Figure 6** Histopathology image of the degenerated helminth retrieved surgically from the common bile duct. (a) Digestive tract; (b) reproductive tract (haematoxylin and eosin,  $\times 100$ , scale bar=250  $\mu\text{m}$ )

periductular fibrosis. Moderate-to-marked, ulcerative, neutrophilic-to-lymphocytic and plasmacytic cholecystitis was identified on histological examination of the gallbladder wall. Histology of the tubular structure retrieved from the common bile duct revealed segments from a degenerated helminth (Figures 6 and 7). The helminth had an eosinophilic tegument with variably present spines. The specimen was very fragmented. No body cavity could be identified. Within the body of the helminth, fragments of a muscular pharynx were present. Paired caeca, which are typical of trematodes, could not be identified. It was not possible to determine if this finding was secondary to the degree of degeneration of the specimen rather than true absence. Scattered ova and vitellaria were also noted. The eggs had yellow-brown shells and eosinophilic, globular material was present in some of them, which suggested the eggs were embryonated. Their diameter was approximately 60–70  $\mu\text{m}$ . In some regions there was an infiltrate of degenerate neutrophils from the host admixed with slightly mucinous debris. Identification of the phylum was not possible due to the poor preservation of the specimen. PCR for *Toxocara cati* was performed on the specimen and yielded a negative result. Faecal analysis was performed on a sample taken before administration of fenbendazole, and no ova or larvae were detected.

Twelve months after the initial presentation, the cat is still well, active and eating with good appetite. The owner reported no recurrence of the clinical signs. Both



**Figure 7** Histopathology image of the degenerated helminth retrieved from the common bile duct. Several eggs are visible. Some of the eggs contain eosinophilic globular material, which suggests these eggs were embryonated (haematoxylin and eosin,  $\times 150$ , scale bar=250  $\mu\text{m}$ )

total bilirubin and ALT decreased to within the reference intervals 3 weeks after the cat was discharged (Table 1). Antibiotic therapy with potentiated amoxicillin was continued for a total of 6 weeks.

## Discussion

This case report describes EHBDO caused by a helminth. Liver disease and biliary obstruction caused by parasites has been described in cats affected by liver fluke.<sup>5</sup> This condition is caused by trematodes of the families Dicrocoeliidae and Opisthorchiidae,<sup>12,13</sup> and it is one of the three recognised forms of inflammatory liver disease in cats alongside neutrophilic and lymphocytic cholangitis.<sup>12</sup> Liver fluke has been reported in several countries worldwide but never in the UK. There are some differences between our findings and the ones typically recognised in cats affected by liver fluke.

The macroscopic appearance of the parasite retrieved from the common bile duct was not consistent with trematodes that affect the feline liver, which are usually lanceolated in shape and smaller in size (2.9–8 mm long and 0.9–2.5 mm wide).<sup>13</sup> The tubular shape of the helminth in this case was more consistent with a nematode. Furthermore, adult flukes on ultrasound appear as oval hypoechoic structures with a hyperechoic centre.<sup>14</sup> In our case, the parasite was identified in the common bile duct as a linear structure composed of two parallel hyperechoic lines. This finding resembles closely the appearance of intestinal roundworms.<sup>15</sup> When adult flukes are not identified, the ultrasonographic changes present in cats with liver fluke are similar to those present in cats affected by other causes of liver disease,<sup>16</sup> and they therefore cannot be used to reach a definitive diagnosis. The alterations identified by ultrasound in our case (thickened gallbladder wall, distension of intra- and extrahepatic bile ducts) had been previously reported in cats with EHBDO and they are not specific.<sup>1</sup> Histopathology of the helminth did not allow a

definitive identification of the parasite phylum. The lack of body cavity is typical of trematodes;<sup>17</sup> however, this could have been an artefact due to the poor preservation of the specimen rather than a true finding. The paired caeca, another feature that characterises trematodes, were not present.<sup>13</sup>

Taking all the findings into consideration, we therefore hypothesise that the EHBDO in this case had been caused by aberrant migration of an intestinal nematode. The cat was regularly receiving a product containing milbemycin oxime and praziquantel. Although the efficacy of this product is very high, some parasites can still survive and reach adulthood<sup>18</sup> in treated cats. It is also possible that ingestion of the embryonated eggs could have occurred after the last administration of the product. We attempted to identify the helminth further by performing faecal analysis, which did not identify any ova or larvae. In cats with liver fluke, eggs can sometimes be identified on bile cytology and this technique seems to have higher sensitivity compared with faecal analysis.<sup>19</sup> Unfortunately, bile cytology was not performed in this case. Additionally, PCR has also been used in some studies as a sensitive technique for the diagnosis of parasitosis in cats.<sup>20,21</sup> *T. cati* PCR was negative in this case, leaving the question of the identity of the helminth unanswered. However, the size of the eggs present in our specimen was similar to the size of the eggs of *T. cati* (62.3–72.7 µm).<sup>22</sup>

In this case, surgical decompression of the biliary tree was performed. Criteria to identify cats with EHBDO that would benefit from surgery rather than medical treatment alone are not clearly defined.<sup>2</sup> In our case, we decided to proceed with surgery owing to the ultrasonographic findings and the lack of response to the medical treatment. The helminth was already dead when it was retrieved from the common bile duct of our cat; therefore, we consider it unlikely that parasiticide treatment would have resolved the obstruction without the need for surgical intervention. The cat was routinely treated with milbemycin oxime and praziquantel. It is therefore impossible to establish if the death of the parasite had been caused by this treatment or it had found the environment of the common bile duct unfavourable for its survival.

## Conclusions

We report the case of an EHBDO caused by a helminth in a cat in the UK. Aberrant parasite migration should be included in the list of possible causes of EHBDO in cats. Ultrasound identified the parasite lodged in the common biliary duct, which allowed us to proceed promptly with surgical decompression of the biliary tree, confirming the utility of this imaging modality in the management of EHBDO in cats.

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**Ethical approval** This work involved the use of non-experimental animals only (including owned or unowned animals and data from prospective or retrospective studies). Established internationally recognised high standards ('best practice') of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not necessarily required.

**Informed consent** Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work for the procedure(s) undertaken. For any animals individually identifiable within this publication, informed consent for their use in the publication (verbal or written) was obtained from the people involved.

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