Clinical case: *Platynosomum fastosum* Kossack, 1910 infection in a cat: First reported case in Trinidad and Tobago

S.A.S. MONTSERIN¹, K. MUÑOZ¹, R. SEEBARANSINGH¹, A. K. BASU*¹

¹School of Veterinary Medicine, Faculty of Medical Sciences, The University of the West Indies, St. Augustine, TRINIDAD AND TOBAGO

*Corresponding author: asokebasu@gmail.com

**SUMMARY**

This study is the first recorded case of *Platynosomum fastosum* infection in a cat, resident in Trinidad and Tobago. The history, clinical signs (strong emaciation, anorexia and chronic pain) coupled to marked dilated gallbladder evidenced by abdominal echography as well as haematological and biochemical findings indicating severe inflammation and hepatic / gallbladder injury have led to suspicion of cholangiohepatitis or cholecystitis. Histological findings have confirmed the diagnosis and parasites (adults and eggs) were identified in the bile at necropsy.

**Keywords** : cat, *Platynosomum fastosum*, liver, gallbladder, inflammation, Trinidad.

**Case report**

A ten-year old male domestic short haired cat from Chaguanas, Trinidad, was brought to the Veterinary Teaching Hospital (VTH), University of the West Indies, St. Augustine, with the complaint of anorexia for one week, recent weight loss and continuous crying. The cat was also known to roam a lot. The cat lived his entire life in Trinidad with no known exposure to any imported cat. On physical examination, mucosae were icteric. There was a slight mucus discharge from both eyes. The animal was emaciated (body condition score: 1.5/ 5) and approximately 8% dehydrated. All other parameters (heart rate, respiratory rate and temperature) were within usual values.

Abdominal echography revealed a severely distended gallbladder (38.7 mm). The walls were thickened (2.71 mm) and at one point folded inward. The hepatic parenchyma appeared coarse and hyperechoic. Fine needle aspirate of the gall bladder produced a pale yellow, blood tinged and cloudy fluid. These findings were highly suggestive of cholangiohepatitis or cholecystitis. Haematological analysis

**RESUME**

Cas Clinique : infection par *Platynosomum fastosum* Kossack, 1910 chez un chat: première observation à Trinidé et Tobago

Cette étude de cas présente la première description d’une infection par *Platynosomum fastosum* chez un chat de Trinidé et Tobago. Les commémoratifs recueillis et les signes cliniques (amaigrissement important, anorexie, douleur chronique) associés à la mise en évidence par échographie abdominale d’une forte dilatation de la vésicule biliaire et aux résultats hématologiques et biochimiques indiquant une inflammation sévère et des lésions du foie et du système biliaire ont conduit à une suspicion de cholangiohépatite ou de cholecystite. L’examen histopathologique a confirmé le diagnostic et de nombreux parasites (adultes et œufs) ont été identifiés dans la bile à l’autopsie.

**Mots-clés** : chat, *Platynosomum fastosum*, foie, vésicule biliaire, inflammation, Trinidé.
(Table I) showed a neutrophilia with significant left shift and toxic neutrophils, indicating severe inflammation coupled to lymphopenia suggesting stress. Throughout biochemistry (Table II) hyperproteinemia, hypernatremia, hyperphosphatemia, hypercholesterolemia and high urea and creatinine concentrations were evidenced and were associated with increased serum alanine transaminase (ALT), alkaline phosphatase (ALP), γ-glutamyl transferase (GGT) and creatine kinase (CK) activities.

The hospitalised cat was placed on intravenous fluids with multivitamins as well as being force fed. He was dewormed with ivermectin at a dose rate of 0.4 mg/kg subcutaneously once (as it is routinely used at this hospital since most cats are not up-to-date with their deworming and are also exposed to heartworm) and amoxicillin-clavulanic acid orally at 10 mg/kg twice daily since treatment with amoxicillin has been shown to be effective in cats with cholangitis/cholangiohepatitis [6]. The cat, however, subsequently died and necropsy was performed. Necropsy revealed an extremely emaciated cat (body condition score: 1/5) with a ruffled coat. There was mild icterus of the carcass. The liver parenchyma appeared normal but there was dilation of the intrahepatic ducts on the cut surface. The gall bladder was

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patient value</th>
<th>Usual values</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (10⁹/L)</td>
<td>33</td>
<td>5.5-19.5</td>
</tr>
<tr>
<td>Seg. (10⁹/L)</td>
<td>25.74</td>
<td>2.5-12.5</td>
</tr>
<tr>
<td>Bands (10⁹/L)</td>
<td>5.61</td>
<td>0.0-0.3</td>
</tr>
<tr>
<td>L (10⁹/L)</td>
<td>0.99</td>
<td>1.5-7.0</td>
</tr>
<tr>
<td>M (10⁹/L)</td>
<td>0.33</td>
<td>0.00-0.85</td>
</tr>
<tr>
<td>RBC (10¹²/L)</td>
<td>7.76</td>
<td>5-10</td>
</tr>
<tr>
<td>Toxic change</td>
<td>2+</td>
<td></td>
</tr>
<tr>
<td>Hb (g/L)</td>
<td>106</td>
<td>80-150</td>
</tr>
<tr>
<td>Ht (L/L)</td>
<td>0.388</td>
<td>0.24-0.45</td>
</tr>
<tr>
<td>MCV (fL)</td>
<td>50</td>
<td>39-55</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>13.7</td>
<td>13-17</td>
</tr>
<tr>
<td>MCHC (g/L)</td>
<td>273</td>
<td>300-360</td>
</tr>
<tr>
<td>Platelets (10⁹/L)</td>
<td>368</td>
<td>300-700</td>
</tr>
</tbody>
</table>

TP: Total protein; Alb: Albumin; A/G: Albumin / globulins; TBil: Total bilirubin; CK: creatine kinase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; GGT: γ-glutamyl transferase. University laboratory values.
grossly enlarged (figure 1) and the wall markedly thickened. The common bile duct was dilated and tortuous. By histology dilation of the hepatic biliary ducts with thickened walls was observed. The liver parenchyma showed areas of normal architecture with a central vein and portal tract. There were also areas of nodular regeneration that were separated from areas of normal architecture by dissecting fibrosis. A peribiliary inflammation involving mononuclear cells was seen (figure 2). There were also multiple biliary ducts in the portal tract indicating chronic inflammation and hyperplasia of the biliary ducts. The mononuclear infiltration was found to specifically contain lymphocytes and plasmocytes. In summary there was peribiliary portal fibrosis, chronic lymphocytic and plasmocytic cholecystitis and chronic inflammation of the liver with dissecting fibrosis coupled to nodular regeneration. The gall bladder contained yellow mucoid bile with myriad of ovoid, flattened dark brown helminth parasites measuring in average 4.1 mm x 1.7 mm. Microscopic examination of the parasite showed a smooth, thin, external cuticle. A sub terminal oral sucker along with a ventral sucker, both male and female reproductive organs and prominent vitelline glands were present. Numerous brown, operculated and oval eggs measuring in average 37.5 x 25.0 μm were also recorded in the bile. The parasites were identified as Platynosomum fastosum Kossack, 1910 (figure 3).

**Discussion**

The *P. fastosum* infection is usually asymptomatic and animals can harbour parasites without any important clinical abnormalities. The clinical signs range from none to obstruction of the biliary tract, with hepatic failure and death [15]. VIEIRA et al. [14] also indicate that the clinical signs of *P. fastosum* are very unspecific and even absent in many cases. When present, clinical signs may include: emaciation, anorexia, depression, vomiting, diarrhoea, and progressive jaundice. Clinical manifestation and severity of the disease is thought to be dependent on the severity of fluke infestation. With a heavy fluke burden in the biliary tree, significant irritation and inflammation occurs leading to fibrosis and possibly biliary obstruction. Cirrhosis occurs due to chronic inflammation and obstruction. According to RATNASABAPATHY and PRATHAP [11] cats clinically in good condition were found to have *Platynosomum* infection with lesions such as mottling and enlargement of liver.

Histological lesions of the bile ducts observed in the reported case are corroborated with other findings [11, 14]. Ultrasound showed a severely distended gall bladder with a thickened wall. Similar observations were reported by SALOMAO et al. [12]. In parallel, laboratory findings have also evoked liver and bile duct tree damage; indeed, severe inflammation was sustained by neutrophilia with left shift and toxic neutrophils whereas strong increases in serum ALT and ALP activities have indicated hepatocellular injury and stress and the marked increase in serum GGT activity was highly compatible with bile duct lesions observed in cholangiohepatitis or cholecystitis [3, 15]. It is also believed...
that the uraemia, phosphataemia and creatininemia resulted from a prerenal azotaemia as the cat was not drinking or eating for at least a week before presentation, these biochemical findings were similar to those found in another report [15]. The slight increase in serum protein could have been due to a hyperglobulinaemia which can be a common finding in these cases [5].

In spite of its occurrence in many other Caribbean islands, platynosomosis has not been reported earlier from Trinidad and Tobago. This study is the first recorded case of *P. fastosum* infection in a cat resident in Trinidad and Tobago. The heavy infection with *Platynosomum* in this resident cat reveals the presence of the parasite in Trinidad and Tobago. But the absence of any clinical sign until heavy worm burden leading to death might be the cause of unnoticed occurrences.

**Références**


