

A Case Report of Feline Cholangiohepatitis: Clinical Presentation and Diagnostic Work-Up

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Abstract. An 8-year-old male Maine Coon cat weighing 4.1 kgs presented with jaundice and constipation for over one week. The chief complaint included lethargy, anorexia, and reduced water intake. A veterinarian had previously administered glycerin and bisacodyl enemas, but complete evacuation of the stool was not achieved. Hematological and serum biochemical analyses revealed non-regenerative anemia, lymphocytopenia, neutrophilia, and eosinopenia. Liver function tests showed elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total bilirubin, bile acids, and amylase levels. Abdominal ultrasonography demonstrated a markedly enlarged gallbladder with a thickened wall and a slightly hyperechoic intraluminal mass suggestive of sludge. Furthermore, dilatation of the hepatic artery and portal vein was observed, consistent with hepatic inflammation. Based on the clinical presentation, hematological and biochemical profiles, and ultrasonographic findings, cholangiohepatitis was diagnosed with a guarded to poor prognosis (*dubia ad infausta*). Intravenous fluid therapy, a hepatic-support diet, and hepatoprotective medication were all part of the therapeutic strategy to promote liver function. Close observation of liver enzyme levels and the patient's reaction to treatment will be crucial in addition to these measures. This case report presents the diagnostic work-up to feline cholangiohepatitis identified through the clinical manifestation of jaundice.

1 Introduction

Cholangiohepatitis is an inflammatory condition of the liver parenchyma that extends into the bile ducts (Rondeau, 2009). This condition occurs more commonly in cats than in dogs (Ettinger et al., 2017). Affected animals often do not show clinical symptoms until a large part of their liver is damaged. Clinical manifestations of this disease include jaundice,

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decreased appetite, fever, vomiting, diarrhea or constipation, and abdominal pain upon abdominal palpation.

Diagnosis confirmation of cholangiohepatitis cannot rely solely on clinical symptoms. Further diagnostic investigation through hematology, serum biochemistry, ultrasonography, and fine-needle aspiration is necessary (Ettinger et al., 2017). Cats diagnosed early generally have a favorable prognosis. However, late-stage diagnosis is associated with a poor prognosis (Ettinger et al., 2017; Rondeau, 2009). This clinical case report aims to provide a diagnostic work-up for feline cholangiohepatitis.

The geographic distribution and prevalence of cholangiohepatitis in cats differ by region. In northeastern Brazil, 42.6% of cats infected with the trematode *Platynosomum fastosum* were diagnosed with cholangiohepatitis (Braga et al., 2016); conversely, in the metropolitan region of Porto Alegre, the prevalence among necropsied cats was only 1.7% (Argenta et al., 2018). Cholangiohepatitis was verified in 7.3% of all feline cases treated at veterinary facilities in Lithuania (Zamokas et al., 2008). These differences show how geography, environment, and diagnosis can all affect how common a disease is.

However, there is still not enough information and epidemiological data about cholangiohepatitis in Southeast Asia, especially Indonesia. Because Indonesia has a lot of cats and more people are going to the vet, it's important to know how common this disease is, what its symptoms are, and what puts people at risk. This information is very important for improving early detection, guiding clinical management, and shaping public health strategies to stop serious liver disease in the local cat population. This shows how important it is to do research in certain areas to fill in the gaps in what we already know.

2 Case Description

History

An 8-year-old male Maine coon cat weighing 4.1 kg was seen at the Veterinary Clinic at Universitas Gadjah Mada in Indonesia. The cat had jaundice. This cat lives indoors all the time, gets all of its vaccinations, and is dewormed on a regular basis. The owner said that the cat had been constipated for more than a week and was also lethargic, not eating, and drinking less water. A veterinarian had previously given glycerin and bisacodyl enemas, but they did not work to get all of the stool out.

Clinical Examination

Clinical examination revealed a poor body condition score (score 2/9) and a normal rectal temperature (38.7°C). The respiratory rate was 40 breaths per minute, and the pulse rate at 168 beats/minute. The submandibular and retropharyngeal lymph nodes were not enlarged. Decreased skin turgor (dehydrated). The conjunctiva and gingiva were pale yellow (figure 1), with a capillary refill time of more than 2 seconds. The hair coat was clean with normal shedding, and no ectoparasite infestation was observed. The nasal cavity was dry, the breathing pattern was thoracoabdominal, and pulmonary auscultation revealed bronchial sounds. Cardiac auscultation revealed clear differentiation between systole and diastole. The oral examination revealed a clean mouth without lesions and slow intestinal peristalsis, and the anus appeared clean. Abdominal palpation revealed a hard mass suspected to be feces. The conspicuous symptom was generalized jaundice of the skin and sclera. Based on this

examination, hospitalization was indicated. During hospitalization, the cat vomited partially digested food.

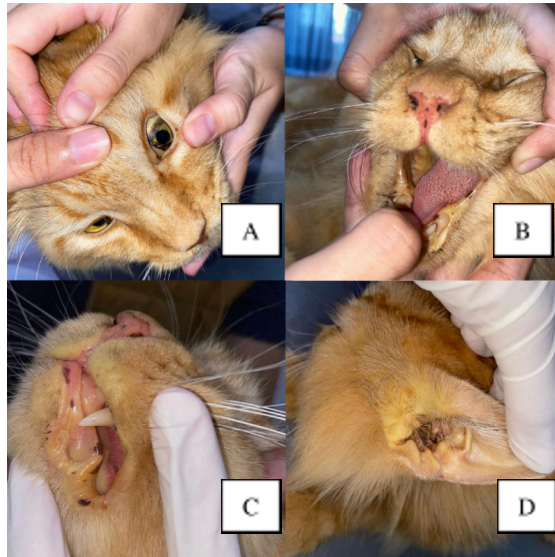


Fig. 1. Jaundice was presented in the conjunctiva and sclera (A), oral cavity (B), gingiva (C), and ears (D).

Hematology and Biochemistry

Complete blood count revealed microcytic-normochromic anemia, neutrophilia, lymphocytopenia, and eosinopenia. Although the total white blood cell count remained within the normal range, neutrophils were increased in both absolute ($83,1\%$) and relative ($14,8 \times 10^3/uL$).

Serum biochemistry revealed elevated alanine aminotransferase/ALT (283 IU/L), aspartate aminotransferase/AST (162 IU/L), alkaline phosphatase/ALP (564 IU/L), gamma-glutamyl transferase/GGT (23 IU/L), total bilirubin (22.5 mg/dL), bile acids (>120 IU/L), and amylase (1715 IU/L). The increases in ALP and GGT were more pronounced than those in AST and ALT, suggesting hepatobiliary involvement, particularly affecting the gallbladder. Severe hyperbilirubinemia explained the clinical manifestation of jaundice. Further biochemical parameters were within normal limits.

Table 1. Hematology and Biochemistry result

Parameter	Unit	Result	Reference Range	Flag
Hematology				
RBC	$10^6/uL$	6,73	6,00-10,00	
HGB	g/Dl	8,9	9,5-15,0	Low
HCT	gr/dL	6,2	6,0-7,5	
MCV	fl	37,5	41,0-54,0	Low
MCH	pg	13,3	13,5-17,5	Low
MCHC	g/Dl	35,4	31,0-36,0	
RDW-CV	%	18,4	16,0-23,0	
WBC	$10^3/uL$	17,83	5,50-19,50	
Neutrophil	A %	83,1	35,0-75,0	High

Basophil	R	10 ³ /uL	14,80	2,50-12,50	High
	A	%	0	0,0-1,0	
Eosinophil	R	10 ³ /uL	0	0,00-0,10	
	A	%	0,8	2,0-12,0	Low
Limfosit	R	10 ³ /uL	0,15	0,00-1,50	
	A	%	13,9	20,0-55,0	Low
Monosit	R	10 ³ /uL	2,48	1,50-7,00	
	A	%	2,2	1,0-4,0	
Platelet	R	10 ³ /uL	0,40	0,00-0,85	
		10 ³ /UI	262	150-600	
MPV		Fl	11,8	9,9-16,3	
PDW			13,9	12,0-17,5	
PCT		%	0,309	0,090-0,700	
LIVER FUNCTION					
GPT/ALT		IU/L	283	28,0-76,0	High
GOT/AST		IU/L	162	5,0-55,0	High
Alkaline phosphatase		IU/L	564	0,0-62	High
Gamma GT		IU/L	23	1,0-7,0	High
Bilirubin total		mg/dL	22,5	0,0-0,4	High
Globulin		gr/dL	3,7	3,4-5,2	
Albumin		gr/dL	2,6	2,4-4,1	
A/G			0,69	0,6-1,5	
Glucose		mg/dL	81,58	70-150	
Cholesterol		mg/dL	138,13	70,0-150,0	
Triglyceride		mg/dL	52,2	20-90	
Bile acid total		IU/L	>120	Random: <15,0 Puasa: 0,0-5,0	High
KIDNEY FUNCTION					
BUN		mg/dL	16,20	15,0-34,0	
Creatinine		mg/dL	1,1	0,8-2,3	
BUN/Creat		ribu	59	27-182	
OTHER					
Amylase		IU/L	1715,00	500-1500	High
Creatinine kinase		IU/L	182,00	64-440	
ELECTROLIT					
Calcium		mg/dL	8,18	7,5-10,8	
phosphate anorganic		mmol/L	1,87	0,97-2,26	

Ultrasonography Findings

Abdominal ultrasonography demonstrated a markedly enlarged gallbladder with a thickened wall and a slightly hyperechoic intraluminal mass suggestive of biliary sludge. Biliary sludge occurs because of cholestasis, leading to the deposition of components such as cholesterol crystals, bile salts, calcium bilirubinate, mucin, and debris cells (Griffin, 2019). Additionally, dilatation of the hepatic artery and portal vein was observed, consistent with hepatic inflammation.

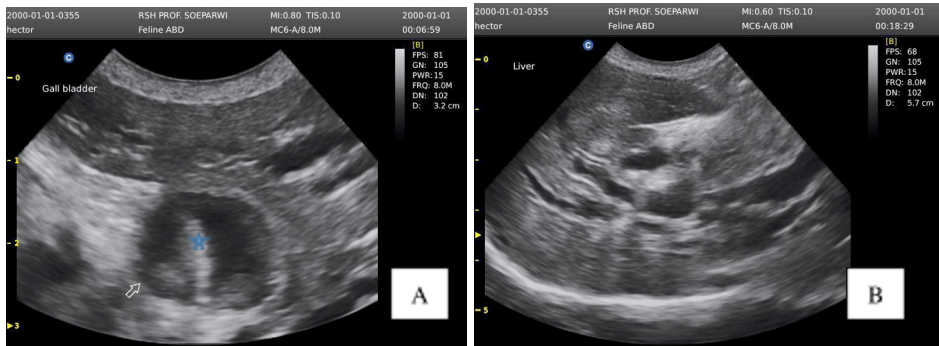
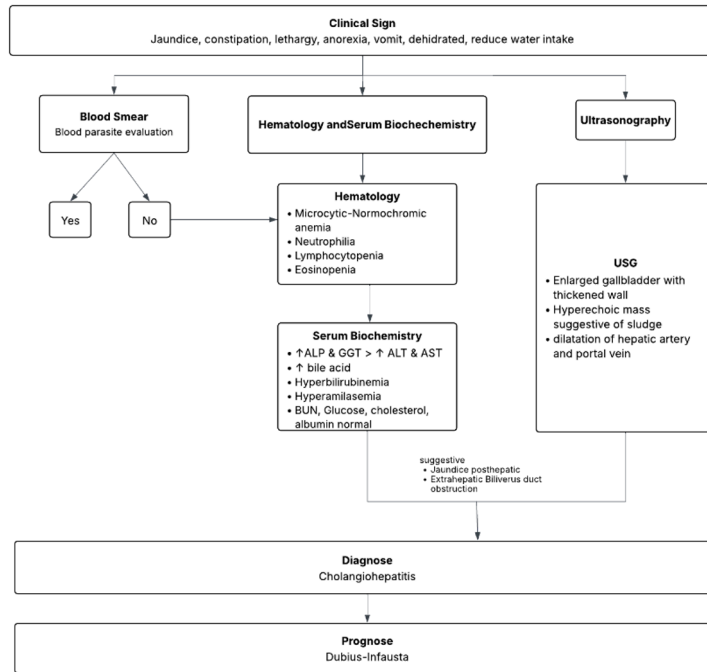


Figure 2. Abdominal ultrasonography findings on gallbladder (A) show an enlarged gallbladder (arrows) with a thickened wall and a hyperechoic mass suggestive of sludge (star). Liver ultrasonography (B) showed dilatation of the hepatic artery and portal vein.

3 Discussion

The diagnostic process starts with anamnesis and a physical exam, which showed jaundice, constipation, loss of appetite, and tiredness. After that, supporting tests were done, such as blood chemistry and haematology tests and an ultrasound. Haematology results indicated microcytic-normochromic anaemia, neutrophilia, lymphocytopenia, and eosinopenia. Because of the neutrophilia result and the erythropoietin response from the liver and kidneys, inflammation needs to be looked into. Blood chemistry tests were done to check the function of the liver and kidneys. The blood chemistry tests showed that the BUN, glucose, cholesterol, and albumin levels were all normal, which ruled out kidney damage. An increase in ALP and GGT levels that is greater than the increase in AST and ALT levels is a sign of liver problems that have spread to the gallbladder. This is backed up by higher bile acid levels and higher blood amylase levels. Ultrasonography continued to confirm problems with the liver and gallbladder. The ultrasound showed that the gallbladder was bigger than normal and had a mass that was hyperechoic, which is thought to be biliary sludge. The edges of the gallbladder wall were also thick and hyperechoic. This series of examinations led to a diagnosis of cholangiohepatitis with a dubious-to-poor prognosis.

Diagram 1. Diagnostic Work-Up : Comprehensive Overview (Ettinger, XX)



Cholangiohepatitis in this case suspected to originate from bacterial migration from the digestive tract (bacterial ascending). Several bacteria that may cause this condition include *Escherichia coli*, *Clostridium perfringens*, *Actinomyces spp*, *Fusobacterium spp*, *Bacteroides spp*, *Staphylococcus spp*, and *α-Hemolytic Streptococcus spp*. Bacteria migrate from the digestive tract to the bile ducts, spread to the gallbladder, and progressively ascend to the liver. The bacteria cause an infection of the bile duct epithelium and infect hepatocytes, marked by the infiltration of neutrophil inflammatory cells (Edwards, 2004). This inflammation results in clinical symptoms such as anorexia, decreased activity, and jaundice. Debris from inflammatory cells accumulates in the gallbladder, obstructing bile flow. The prolonged obstruction of bile flow causes stasis in the gallbladder, leading to the deposition of cholesterol crystals, bile salts, calcium bilirubinate, mucin, and cellular debris, forming a material known as biliary sludge (Griffin, 2019). The presence of biliary sludge causes gallbladder distension, which can stimulate the vagus nerve and induce vomiting of food. Additionally, gallbladder sludge hinders bile acid secretion into the intestines, where bile acids aid in fat emulsification and digestion. Undigested fat can affect intestinal motility, resulting in constipation or steatorrhea in cats, along with nausea and vomiting. Clinical symptoms of constipation may also occur due to anorexia-induced dehydration, increasing water absorption in the colon and resulting in dry, hard feces (Ettinger et al., 2017). Clinical signs in some cases of cholangiohepatitis in cats include vomiting, anorexia, lethargy, diarrhea, polyuria, polydipsia, some experiencing abdominal distension, dysuria, neurological abnormalities, and icterus. The liver has a good regenerative capacity, so animals with cholangiohepatitis may not show visible clinical signs until a large portion of the liver is damaged (Rondeau, 2009). In this case, the observed clinical symptoms were jaundice, anorexia, decreased activity (lethargy), vomiting, and constipation.

4 Conclusion

An 8-year-old male Maine Coon cat presented with generalized jaundice, lethargy, anorexia, and constipation. Laboratory findings showed a cholestatic hepatobiliary pattern, and abdominal ultrasonography revealed gallbladder enlargement with wall thickening and biliary sludge, supporting a diagnosis of cholangiohepatitis with a guarded-to-poor prognosis. Appropriate management and follow-up should focus on clinical monitoring and serial evaluation of liver-related parameters.

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The authors declare no competing interest.

This clinical case report provides the clinical presentation and the diagnostic work-up on cholangiohepatitis in cat emphasizing the need for further investigation.

RFL and DKW examined and treated the cat. RFL collected data, literature, and prepared manuscripts. DSD and AN designing studied. All authors have read and approved the final manuscripts.

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