

A Case Report on Jaundice in Female Cat: Exploring Underlying Diseases, Diagnosis, and Treatment Approaches

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Abstract

Original Research Article

A 3-year-old female cat of a local breed was admitted to the hospital with the condition of anorexia, weight loss and yellowish colored urine. A physical examination revealed that the cat was icteric with normal temperature, respiration and heartbeat. Hematological and serum biochemical examinations showed elevated bilirubin, alkaline phosphatase, and alanine aminotransferase but normal hematological value suggestive of hepatic jaundice. Subsequent radiological and ultrasonographic findings also suggested it as a jaundice. The animal was treated silymarin, amoxicillin, vitamin C, and vitamin E, as well as a nutritionally balanced diet according to daily energy needs. Reevaluation of various blood parameters after 15 days of treatment revealed marked improvement.

Keywords: Cat, Jaundice, Hematology, Biochemistry.

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INTRODUCTION

Jaundice is a yellow discoloration of the skin, mucous membranes and sclera caused by an increased concentration of bilirubin in serum and tissues (Sherding, 2000). Jaundice develops when a feline's blood and bodily tissues have an excessive accumulation of the yellow pigment bilirubin (Bunch, 2005). Many tissues in the body will become discolored and many surfaces on the body, including the outermost layer of skin, will exhibit jaundice where icterus occurs for any length of time (Weir & Ward, 2022). Globally, illness is prevalent. Certain cat breeds are more prone to develop conditions that cause jaundice. The pathology of jaundice is well established. Breakdown of hemoglobin in senescent erythrocytes is the main source of bilirubin. A smaller amount of bilirubin is also produced by the liver's degradation of other proteins that contain hemoglobin, cytochromes, and other hemoglobin. Haeme from phagocytized senescent erythrocytes becomes separated by haeme oxygenase in the liver and spleen macrophages to produce biliverdin, a green pigment. Biliverdin reductase converts biliverdin to bilirubin. The yellow-

orange pigment known as unconjugated (indirectly reacting) bilirubin is released into the bloodstream and binds to albumin. Unconjugated plasma bilirubin is eliminated from the bloodstream by the liver (uptake), transformed by hepatocytes into conjugated (directly reacting) bilirubin, released into the biliary system, and ultimately eliminated into the gut together with bile (Kam *et al.*, 2025). Jaundice, however, could appear if the liver cannot manage to successfully get rid of the bilirubin (Kruzer, 2022). The most common reason for elevated bilirubin production is hemolysis, although it can also result from substantial internal bleeding or the breakdown of damaged red blood cells. Extravascular hemolysis produces bilirubin, as previously stated, in contrast to intravascular hemolysis, which happens while hemoglobin is released in the bloodstream and then combines with hemoglobin prior to being eliminated by hepatocytes, where hemoglobin degradation occurs. Hepatocellular dysfunction caused by reduced functional mass, insufficient perfusion, or anomalies in absorption or conjugation can also lead to hyperbilirubinemia and icterus (Gordon, 2011).

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Normal feline urine does not include bilirubin. Bilirubin in the urine is a sign of conjugated hyperbilirubinemia and liver disease in cats (Michaletos *et al.*, 2025). Severe bilirubinuria in concentrated urine can also cause green or orange-brown discoloration of the urine (Augusto, 2022). The most obvious signs of jaundice are yellowish-colored pinnae (ear flaps), sclera (the white portion of the eyes), and gingivae (gums). The gums, lips, and tongue, which are generally pink oral mucous membranes, become discolored and often turn yellow. The eyes appear yellowed due to the yellow color of the sclera, which is normally white, and the normally pink conjunctival membrane. The two hairless locations where the yellow color is most prominent are the inside of the ear pinnae and the bottom of the belly (Hollinger, 2021). According to the underlying cause, lethargy, vomiting, and loss of weight can also be signs of jaundice in cats (Wedderburn, 2022). The liver can heal completely with small amounts of damage. However, significant damage results in hepatocyte loss and bile duct proliferation. Fibrosis is characterized by significant hepatocyte loss or persistent damage. In cases of acute or chronic hepatitis, a conclusive diagnosis can be made

with the aid of a histological analysis of liver tissue (Antje *et al.*, 2022).

MATERIALS AND METHOD

Study area: The study was conducted in the Teaching and Training Pet Hospital and Research Centre (TTPHRC), a sister institute of Chattogram Veterinary and Animal Sciences University (CVASU), Dhaka, Bangladesh.

Case Presentation: A 2-year-old weighing 3.9 kg local breed female cat named Mithai was presented to TTPHRC with history of yellowish urine, anorexia, depression less water intake and marked dehydration. The cat had also the history of properly immunized and dewormed.

Clinical examination: Upon clinical evaluation, the cat's body temperature was measured at 102.2°F. 20/min was the RR. The heartbeat per minute was 125. Micturition and defecation were both normal. The only notable physical examination findings were hair loss and a yellowish mucous membrane. Additionally, there were no anomalies detected upon abdominal palpation.



Fig 1: Icteric Ear



Fig 2: Yellowish Gum

Laboratory examination: Blood was drawn from the cephalic vein for biochemical and hematological analysis. To locate the vein in the cat's arm before drawing blood, the animal was put in lateral recumbency. After that, anchor the vein with a tourniquet. An alcohol swab was used to sterilize the blood collection location. The entire blood collection process took three minutes. A total of 3 ml blood was collected and transferred 1.5 ml blood to EDTA tube for hematological analysis and 1.5 ml blood to red tube for biochemical analysis. Hematological analysis was carried out in automated cell counter (OV-560 RET, Balio, France) and biochemical analysis was carried out in semi-automated biochemical analyzer (Human 3000, GmbH, Germany).

Diagnostic test: Following diagnostic tests were performed to proper diagnosis and to exclude the diseases that having similar symptoms.

FIP Kit test: The process for diagnosing the FIP virus is quick and simple. We took advantage of TESTSEALAB's Rapid Test Kit. This test equipment was coated with an invisible T (test) and C (control) zone. The reagent will laterally flow across the test strip's surface when serum is poured into the sample well on that apparatus. If the test is positive, the T and C bands will both be visible. We took the kit out of the foil pouch for this test procedure. The cassette was placed horizontally on a surface, and a single droplet of serum was extracted using a discarded dropper sample, and three to four drops of the FIP assay buffer were immediately poured into the corresponding well. The cat was negative for FIP test.

X-ray Findings: In the x-ray, a bilateral picture was obtained. The results of the X-ray showed that the heart and lungs were sized normally and in the proper positions. The lung did not contain any accumulating blood or fluid. The liver is positioned correctly and is

normal size. The abdominal cavity did not contain any anomalies (Figure-3).



Fig 3: X-ray of patient

USG Findings: According to Larson (2016), ultrasonography is a helpful noninvasive imaging method for evaluating hepatic and biliary disorders. For scanning animal was positioned accordingly (Figure-4) The results of the ultrasonography showed that the heart

and lungs were both sized and positioned correctly. The liver is a somewhat echogenic, coarse-looking organ. The liver capsule was found to be moderately thickened (Figure-5).



Fig 4: Patient preparation for USG



Fig 5: USG image of Liver

RESULTS

Serum biochemical parameter: Serum biochemical examination revealed elevated SGPT (262.1 U/L),

Alkaline phosphatase (241.9 U/L) and Bilirubin (1.2 mg/dl). Other biochemical parameters are within normal limits (Table -1)

Table 1: Biochemical parameter results.

Parameters	Reference value	Test results
Total protein	5.2-8.2 g/dl	6.1
Albumin	2.5-3.9 g/dl	2.9
Globulin	2.3-5.3 g/dl	3.1
Bilirubin	0.1-0.4 mg/dl	5.9
Alanine aminotransferase (ALT/SGPT)	10-100 u/l	262.1
Alkaline phosphatase (ALP)	10-50 u/l	241.9
Serum Creatinine	0.6-1.6 mg/dl	1.2
Albumin Globulin ratio	0.6-1.2	0.93

Hematology parameter: Hematological examination revealed that RBC was 7.12 cu mm/ul, PCV was 44.4%, Hgb was 11.9 g/dL, MCV was 54.7, MCH was 14.7 pg,

MCHC was 26.9. In differential leukocytes count there were found no abnormalities (Table-2).

Table 2: Hematological findings

Test name	Reference value	Test results
Hemoglobin (HB%)	9-15 g/dl	11.9 g/dl
Total WBC count	5000-19000	14700
Neutrophils	40-70%	41%
Lymphocytes	10-45%	52%
Monocytes	2-8%	5%
Eosinophils	1-4%	2%
Basophils	0-1%	0%
RBC Count	6-10 m/ul	7.12
PCV	32-52	44.4%
MCV	39-55	54.7
MCH	13-18	14.7
MCHC	30-36	26.9
Platelet count	150000-500000	143000

Treatment: After the diagnosis the cat was treated with amoxicillin @ 10 mg/kg (Inj. Moxin 500, Opsonin Pharma Ltd.) silymarin @30 mg/kg (Cap. Silybin 140 mg, Square Pharma) vitamin B complex@1ml/ 10 kg (Inj. V-plex vet, Acme agrovet), vitamin E @30 unit/kg (Cap. E cap 200, Drug International Limited) and DNS 5 % fluid therapy @ 20 ml/kg. The treatment continued for

7 days. After 15 days, the patient's condition is further reevaluated.

Follow-up: The patient was reevaluated after 15 days of post treatment. The patient was fully recovered with no evidence of any clinical signs. Biochemical (Table-3) and hematological parameter (Table-4) are within normal range.

Table 3: Follow-up biochemical report (After 15 days)

Parameters	Reference value	Test results
Total protein	5.2-8.2 g/dl	8.0
Albumin	2.5-3.9 g/dl	3.3
Globulin	2.3-5.3 g/dl	4.2
Bilirubin	0.1-0.4 mg/dl	0.2
Alanine aminotransferase (ALT/SGPT)	10-100 u/l	42
Alkaline phosphatase (ALP)	10-50 u/l	57
Serum Creatinine	0.6-1.6 mg/dl	1.3
Albumin Globulin ratio	0.6-1.2	0.70

Table 4: Follow -up hematological report (After 15 days)

Test name	Reference value	Test results
Hemoglobin (HB%)	9-15 g/dl	16.4 g/dl
Total WBC count	5000-19000	18000
Neutrophils	40-70%	68%
Lymphocytes	10-45%	24%
Monocytes	2-8%	2%
Eosinophils	1-11%	6%
Basophils	0-1%	0%
RBC Count	6-10 m/ul	10
PCV	32-52	52.7%
MCV	39-55	51.8
MCH	13-18	16.5
MCHC	30-36	31.8
Platelet count	150000-500000	350000

DISCUSSION

Serum bilirubin concentrations higher than 2 mg/dl (35 μ mol/l), or five to ten times over normal, are often associated with jaundice. Jaundice is a highly specific sign of acute hemolysis or hepatobiliary disease because it correlates with hyperbilirubinemia, unlike

most other non-specific clinical signs of hepatobiliary disease (e.g., inappetence, lethargy, weight loss, vomiting, diarrhea, and dehydration). Jaundice, however, is not a very sensitive marker of liver illness because it is not present in more than 50% of cats with liver disease (Korman *et al.*, 2013). Concentrate

According to a biochemical analysis, the cat exhibits hyperbilirubinemia. Total bilirubin values above 2.5 to 3.0 mg/dL are indicative of clinical icterus (Susan & Moses, 2010). Extrahepatic cholestasis, inadequate absorption, prehepatic hemolysis, increased hemoprotein liberation, ruptured bile tract, bile duct occlusion, ruptured bile tract, etc. are among the factors that contribute to elevated bilirubin concentrations (Susan & Moses, 2010). According to other research, hyperbilirubinemia can cause both post- and pre-hepatic jaundice (Sherding, 2000). Hemolysis is the major anomaly that is associated with pre-hepatic jaundice. Increased bilirubin production is a result of increased erythrocyte breakdown (Salord Torres *et al.*, 2024). Blood phosphate and ATP values that are significantly below normal cause hemolysis (Valtolina & Favier, 2017). The hematological examination showed that the blood's RBC concentration was within the range of 7.12. The other statistics, such as platelet count, WBC count, and PCV, are similarly within the normal range. Also, the patient was FIP-negative. Thus, considering things from that angle, we concluded that hemolysis is not the reason for the jaundice. Pre-hepatic jaundice is thus not a serious issue in this case. In the serum biochemical

assay, alkaline aminotransferase (ALT) was discovered to be high. Elevations in ALT have been connected to hyperthyroidism, liver damage, and muscle damage (Susan & Mosses, 2010). The level of ALT rose with both inflammation and hepatocellular necrosis. Five days following the start of the action, it grew. (Otte & others, 2017) Additionally, alkaline phosphatase was over its typical reference range. Patients with intrahepatic cholestasis, cholangitis, or extrahepatic blockage have elevated serum levels of alkaline phosphatase (ALP), which is produced by the epithelial cells lining the bile canaliculi (Susan & Mosses, 2010). Similarly, another author asserted that elevated ALP concentrations in serum might be identified in cases of acute hepatitis and chronic cholangiohepatitis. Particularly low readings indicate cirrhotic liver disease (Neumann, 2004). The cat responded nicely to medication after receiving treatment (figure-6). After a week, the cat was clearly recovering, and after 14 days, it was completely recovered. After 15 days, a reexamination of the blood and serum for hematobiochemical parameters revealed a significant improvement, with bilirubin reaching the normal limit and a normal liver-specific enzyme.



Fig 6: Patient condition after 15 days

CONCLUSION

In cats, hepatic diseases are relatively frequent even though they might be challenging to diagnose because of their persistent, confusing, and non-specific clinical symptoms. In the present case, accurate diagnosis with combination therapy using antibiotics with supportive care was effective in eliminating the cause and early recovery of the animal.

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