***Review Article***

# Icterus in Cats

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## Abstract

Icterus in cats is characterized by the yellowing of the skin, eyes, and mucous membranes due to an increase in bilirubin levels in the blood. It is a serious symptom that necessitates a thorough evaluation to determine the underlying cause. It has been reported that icterus in cats does not have any age, breed, or gender predisposition. Also known as hyperbilirubinemia, icterus can result from various conditions such as liver damage, infections, bile duct obstructions, and hemolysis. Icterus is classified into three types based on its mechanism of formation: prehepatic, hepatic, and posthepatic icterus. Clinical signs, ultrasonography, radiography, and various laboratory tests are crucial for the diagnosis of icterus. Since icterus can develop due to various factors, accurate diagnosis forms the basis of treatment, and treatment procedures are developed separately for each cause.

**Keywords:** Cat, Icterus, pathophysiology

## Introduction

The term “icterus” is derived from the French word “Jaune,” and it refers to a significant clinical finding characterized by yellowish pigmentation of the skin, oral mucosa, and sclera due to elevated bilirubin levels. This condition is also commonly known as “jaundice.” Icterus is a frequently observed symptom in cats (Saraiva et al., 2019). Known as hyperbilirubinemia, icterus has been reported to occur as a result of various conditions such as liver damage, infections, bile duct obstructions, and hemolysis. It is understood that icterus is not associated with any breed or age predisposition (Abbas, Shamshad, Ashraf, & Javaid, 2016).

## Pathophysiology of Icterus

Bilirubin is an organic anion metabolized by the liver. It is produced as a result of the metabolism of various heme-containing proteins, including hemoglobin, myoglobin, cytochromes, catalase, and peroxidase. Approximately 80-85% of bilirubin synthesized from heme proteins originates from hemoglobin. In the reticuloendothelial system, macrophages present in organs such as the liver and spleen phagocytize aged or damaged erythrocytes. During this phagocytosis process, heme is released (Sherding, 2000). Heme oxygenases (HOs), encoded by the heme oxygenase 1 gene (HMOX1), catalyze a reaction in which heme is converted into carbon monoxide (CO), free iron (Fe), and biliverdin, a green pigment (Gozzelino, Jeney, & Soares, 2010).

Biliverdin is reduced to bilirubin IXa by biliverdin reductase (BVR), an enzyme found in various cellular components, including cell membranes. Unconjugated bilirubin binds to albumin molecules and is transported to hepatocytes via the cardiovascular system. This protein-bound unconjugated bilirubin cannot be excreted by the kidneys. Once transported to hepatocytes, unconjugated bilirubin is retained and carried by two proteins, Y (ligandin) and Z, which bind and transport bilirubin. Similar compounds, such as sulfobromophthalein and indocyanine green, also bind to these proteins, while bile acids do not. The binding of bilirubin to Y and Z proteins within hepatocytes limits its diffusion back into the plasma. Subsequently, bilirubin is transported to the endoplasmic reticulum, where it is conjugated with glucuronic acid, forming bilirubin glucuronide or bilirubin diglucuronide (conjugated bilirubin) via the enzyme uridine diphosphate-glucuronosyltransferase (Dandrieux, 2022; Joon et al., 2018; Turgut, 2000). This conjugation process renders bilirubin water-soluble (Bosma, 2003; Dandrieux, 2022).

Following conjugation, bilirubin is excreted into bile canaliculi and stored in the gallbladder until it is released into the duodenum. In the small intestine, conjugated bilirubin is first converted back to unconjugated bilirubin by bacteria and then further metabolized into tetrapyrrole compounds such as urobilinogen and stercobilinogen. Most urobilinogen (90%) is oxidized to urobilin and excreted in feces, while the remainder is absorbed, either reused in bilirubin synthesis via the enterohepatic circulation or excreted by the kidneys as urochrome. Stercobilinogen, a colorless compound, is oxidized to stercobilin, which gives feces its characteristic color (Dandrieux, 2022; Méndez-Sánchez et al., 2019; Sherding, 2000; Turgut, 2000).

The formation of urobilinogen is influenced by the amount of conjugated bilirubin entering the intestine, intestinal flora, and intestinal transit time. Therefore, measuring urobilinogen in urine samples is not considered a reliable diagnostic tool (Sherding, 2000).

Figure 1 illustrates the schematic mechanism of jaundice formation (Vandana Pushpendra, & Saket Singh, 2019).

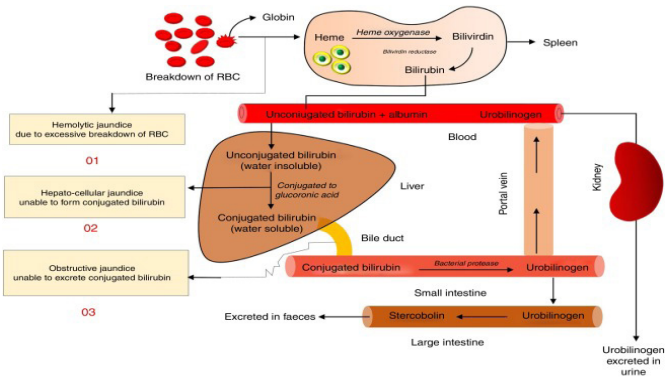


**Figure 1. Mechanism of icterus formation in cats.**

### Classification of Icterus

Jaundice, as a significant clinical finding, is classified into three types based on its mechanism of formation.

#### Prehepatic Icterus (Hemolytic/Hyperfunctional)

Prehepatic icterus occurs as a result of excessive erythrocyte destruction (Abbas et al., 2016). Cats have a high red blood cell (RBC) turnover rate, and RBC destruction can occur both in lesions and in circulation. The breakdown of RBCs leads to the release of various hemoglobin products, including bilirubin and biliverdin. Due to their limited glucuronidation capacity, cats are known to have a restricted ability to metabolize and recycle bilirubin and biliverdin. Glucuronidation is a process that converts bilirubin into a water-soluble form, facilitating its excretion. The limited glucuronidation capacity in cats results in the accumulation of bilirubin and biliverdin, contributing to the development of icterus (Niels, 2014).

Prehepatic icterus in cats can be caused by various factors. Infectious diseases are a relatively common cause of prehepatic hemolysis in cats. Identifying the specific cause of prehepatic icterus in cats is critical for accurate diagnosis and appropriate treatment.

#### Hepatic Icterus (Toxic/Retention)

Hepatic icterus develops due to the effects of toxic substances on liver epithelial cells, leading to hydropic degeneration, fatty infiltration, or necrosis. In such cases, liver cells either undergo excessive damage and fail to process bile, resulting in the accumulation of unconjugated bilirubin in the bloodstream, as seen in hemolytic icterus, or the swollen liver cells obstruct the bile canaliculi. Bile is excreted by the bile epithelial cells but cannot reach the gallbladder or intestines. Consequently, conjugated bilirubin accumulates in the liver and is reabsorbed into the bloodstream. In this type of icterus, both conjugated and unconjugated bilirubin levels increase in the blood (Şennazlı, 2016).

**Posthepatic Icterus (Obstruction/Reabsorption)**

Posthepatic icterus is characterized by the accumulation of bilirubin resulting from the obstruction of bile flow from the liver to the intestines. This type of icterus arises from a range of pathophysiological processes involving cholestasis mechanisms. Cholestasis can develop due to various factors, including bile duct obstruction, hepatobiliary diseases, or dysfunction of bile acid transporters (Zollner & Trauner, 2008)..

## Clinical Findings

In cases of icterus resulting from prehepatic icterus, urine color can vary from dark brown to yellow. Additionally, anemia, yellowish discoloration of the sclera and skin, and elevated bilirubin levels are commonly observed clinical findings (Bektaş et al., 2010).

Common diseases causing prehepatic icterus and the clinical signs associated with these conditions are summarized in the table below (Table 1) (Webb, 2016).

**Table 1. Causes and Clinical Signs of Prehepatic Icterus**

| Causes of Prehepatic Icterus | Anorexia | Lethargy | Fever | Lymphadenopathy | Vomiting and Diarrhea | Weight Loss | Anemia | Abdominal Pain | Other Clinical Signs |
|---|---|---|---|---|---|---|---|---|---|
| Mycoplasma Species | X | X | X | | | | | | Hypothermia, Physiological murmur, Increased heart rate/ respiratory distress secondary to anemia |
| Cytauxzoon felis | | X | X | X | | | | | Dehydration, Shock signs, Respiratory distress, Hypothermia |
| FIP | X | X | X | | | X | | | Effusive (wet) FIP: Ascites, pleural effusion; Non-effusive (dry) FIP: Ocular and neurological signs |
| Babesia Species | X | X | X | | | X | | | |
| FeLV | | | | | | | X | | Immunosuppression, Secondary infections |
| FIV | | | X | X | | X | | | Oral inflammation, Secondary infections, Lymphoma |
| IMHA | X | X | | | X | | | | Symptoms of IMHA, Pica |
| Erythrocyte Pyruvate Kinase Deficiency/ Osmotic Fragility | X | X | | | | X | | | Pica |
| Neonatal Isoerythrolysis | | X | | | | | | | Stopping nursing, Disseminated intravascular coagulation, Pigmenturia, Acute kidney injury, Death |
| Transfusion Reactions | | | X | | X | | | | Erythema or pruritus, Dyspnea or tachypnea, Tachycardia or bradycardia, Tremors, Convulsions, Shock, Cardiopulmonary arrest |
| Hypophosphatemia | X | X | | | | X | | | Acute hemolysis, Weakness, Tachypnea, Tachycardia |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Micro-angio-pathic Hemol-ytic Anemia | X | X | X | | | | X | | Hyperthermia, Ob-tundation, Cardio-pulmonary arrest |
| Drugs, Toxins, Poiso-ning, Oxi-dative Stress | | | | | | | | | Non-specific clinical findings depending on the underlying etiology, May include signs associated with he-molytic anemia and allergic reactions |

The clinical signs of hepatic icterus include yellow discoloration of the mucous membranes and sclera, along with symptoms such as abdominal pain, high fever, vomiting, eating disorders, gastrointestinal bleeding, diarrhea, anemia, edema, and loss of appetite. If left uncontrolled, hepatic icterus can lead to severe conditions such as kernicterus, coma, and even death (Mathew, 2008).

Common diseases causing hepatic icterus and the clinical signs associated with these conditions are summarized in the table below (Table 2) (Webb, 2016).

**Table 2. Causes and Clinical Signs of Hepatic Icterus**

| Cau-ses of Hepatic Icterus | Anorexia | Lethargy | Fever | Lymphadenopathy | Vomiting and Diarrhea | Weight Loss | Anemia | Abdominal Pain | Other Clinical Signs |
|---|---|---|---|---|---|---|---|---|---|
| Hepatic Lipido-sis | X | X | | | X | | | | Idiopathic or secondary hepatic lipidosis. Clinical signs vary based on the underlying etiology. |
| Cholan-gitis | X | X | X | | X | | | X | Bacterial, acute or chronic neutrophi-lic, lymphocytic. Dehydration, hepa-tomegaly. |
| FIP | | | | | | | | | Clinical signs of FIP. |
| FCV (Feline Calici-virus) | | | X | | | | | | Oral ulceration, upper respiratory tract signs, edema, ulcerative dermati-tis, conjunctivitis. |
| Fran-cisella tularen-sis | X | X | X | | | | | | Tachypnea, tachy-cardia. |
| Drugs, Toxins | | | | | | | | | Non-specific poi-soning symptoms, acute liver failure, death. |
| Amylo-idosis | | X | | | | | | | Genetic and other types of amylo-idosis. Sudden death, acute gastric bleeding. |
| Sepsis / SIRS | X | | X | | X | | | | Specific underlying etiology, clinical signs associated with collapse, bradycardia, and hypotension. |

The clinical signs of posthepatic icterus include lethargy, loss of appetite, weight loss, vomiting, dehydration, excessive urination, excessive water consumption, palpable masses in the abdominal region, abdominal pain, abdominal distension, diarrhea, and icterus (Harvey, Holt, Barr, Rizzo, & Tasker, 2007; Jensen & Chan, 2014; Jifcovici, Caraty, Vincken, & Bongartz, 2021; Linton et al., 2015). Additionally, symptoms such as brownish urine, pale-colored stools, generalized pruritus, high fever, biliary colic, and significant weight loss can occur. Figures 2, 3, 4, and 5 show various images of cats with icterus.

Common diseases causing posthepatic icterus and the clinical signs associated with these conditions are summarized in the table below (Table 3) (Webb, 2016).

**Table 3. Causes and Clinical Signs of Posthepatic Icterus**

| Causes of Post-hepatic Icterus | Anorexia | Lethargy | Fever | Lymphadenopathy | Vomiting and Diarrhea | Weight Loss | Anemia | Abdominal Pain | Other Clinical Signs |
|---|---|---|---|---|---|---|---|---|---|

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Chole-lithiasis | | | | | | | | | Clinical signs associated with extrahepatic bile duct obstruction (EHBO). |
| EHBO | | X | X | | X | X | X | X | Coagulopathy, hypotension, and shock. |
| Triadi-tis | X | X | X | | X | X | | X | Clinical signs may progress in cases of EHBO and hepato-megaly. |
| Liver Para-sites (Platy-noso-mum concin-num) | X | X | | | X | | X | | |

In cats, bilirubinuria often occurs alongside hyperbilirubinemia before the clinical symptoms of icterus become apparent. In most cats with hyperbilirubinemia (72%), liver disease is present. A significant proportion of these conditions are hepatic problems that develop secondarily to diseases affecting other body systems (Turgut, 2000).

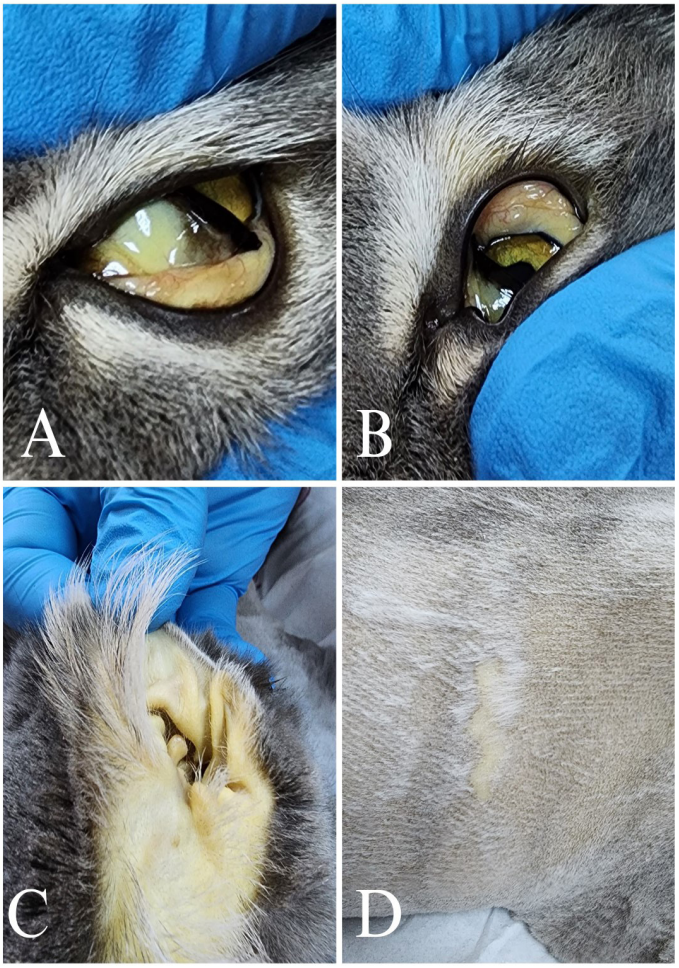


Figure 2. Clinical Signs of Icterus in Cats (Feline infectious peritonitis in a cat, sourced from the archives of Atatürk University, Faculty of Veterinary Medicine, Animal Hospital.) A and B: Noticeable yellow discoloration in the ocular mucosa. C: Yellow discoloration in and around the ears. D: Yellowing of the skin.

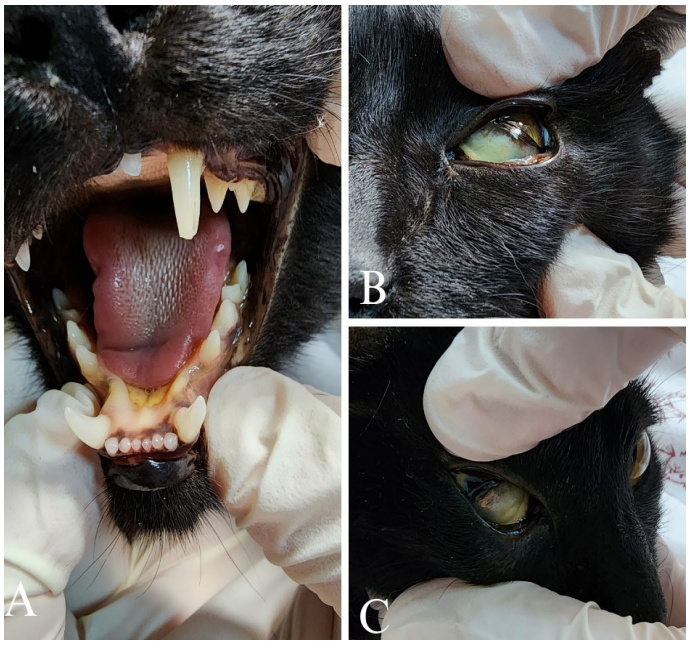


Figure 3. Clinical Signs of Icterus in Cats (A cat with hepatic lipidosis, sourced from the archives of Atatürk University, Faculty of Veterinary Medicine, Animal Hospital.) A: Oral mucosa of a cat with icterus. B and C: Ocular mucosa of a cat with icterus.


Figure 4. Clinical Signs of Icterus in Cats (Icterus resulting from Babesia infestation, sourced from the archives of Atatürk University, Faculty of Veterinary Medicine, Animal Hospital.) A and B: Noticeable yellow discoloration around the ears of a cat with icterus. C: Ocular mucosa of a cat with icterus. D: A cat with icterus.

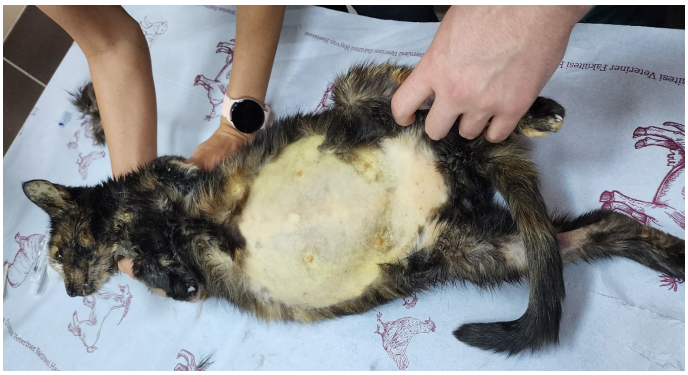

Figure 5. Clinical Signs of Icterus in Cats (A cat diagnosed with feline infectious peritonitis, sourced from the archives of Atatürk University, Faculty of Veterinary Medicine, Animal Hospital.)

In addition to clinical signs, the diagnosis and severity of icterus can also be determined using various laboratory parameters.

**Diagnostic Approach in Icteric Cats**

**Diagnostic Approach to Prehepatic Icterus**

In any case of prehepatic icterus, hemolytic anemia should first be investigated. Routine hematology analysis (+/- manual packed cell volume (PCV)) can quickly identify the presence of anemia, followed by blood smear examination to investigate hemolysis. Blood smears should be screened for polychromasia and anisocytosis to confirm regeneration; aggregate reticulocyte counts can be performed to further measure the degree of regeneration. However, it is important to note that in the early stages of the disease (the first 3–4 days), regeneration may be absent or minimal (Huang et al., 2021; Practice, 2020).

In icteric dogs and cats, if the erythrocyte count is low, with reticulocytosis and bilirubinemia, hemolytic diseases should be considered. In hemolytic anemias (regenerative anemias; hemorrhagic, autoimmune hemolytic anemia), reticulocytosis, hemoglobinemia, hemoglobinuria, erythrocytic autoagglutination, spherocytosis, positive Coombs' test results, splenomegaly, and/or hepatomegaly are commonly observed. Determining conjugated and unconjugated bilirubin concentrations is not significant in differentiating hemolytic icterus from hepatic icterus and may lead to misinterpretation. In hemolytic cases, unconjugated bilirubin may initially predominate. However, over time, conjugated bilirubin may replace unconjugated bilirubin. Even if unconjugated bilirubin predominates, the patient may still have hepatic disease. Clinicians should not be misled by elevated ALT activity, as severe acute hemolytic anemia can also cause high ALT levels due to acute hypoxia (Turgut, 2000).

In icterus caused by hemolysis, bilirubin concentration rarely exceeds 3–4 mg/dL in dogs and cats. Bilirubin concentrations above 3–4 mg/dL may indicate the presence of hepatic and/or posthepatic disease in addition to hemolysis (Turgut, 2000).

**Diagnostic Approach to Hepatic Icterus**

In the absence of hemolysis and bile duct obstruction, icterus should be suspected to have a primary hepatic origin. Liver disease does not always result in icterus, and when it does, it typically causes only mild to moderate increases in bilirubin levels rather than significant elevations. This is often accompanied by elevated liver enzyme levels. Elevated bilirubin levels due to liver disease confirm the presence of hepatic dysfunction. A detailed clinical history is essential to rule out the possibility of toxin or drug exposure, and additional serological testing may be beneficial to investigate infectious causes (e.g., FIP, toxoplasmosis) (Practice, 2020; Rothuizen, 2020).

**Diagnostic Approach to Posthepatic Icterus**

After ruling out the possibility of hemolysis, the likelihood of posthepatic icterus should be investigated. Posthepatic icterus is often associated with elevated hepatic enzyme levels, with ALP typically being higher than ALT. However, ultrasonography is always the most useful diagnostic tool. In normal patients, the intrahepatic bile ducts are not visible, while in normal cats, the common bile duct can be easily identified and should measure less than 4 mm in diameter. Bile duct obstruction leads to dilation of the bile ducts and the biliary tree, which will become evident during ultrasound examination. The gallbladder itself

may or may not appear enlarged. Comprehensive investigations for mass lesions as potential causes of biliary obstruction should be conducted, as these are often of pancreatic origin (e.g., pancreatitis, pancreatic carcinoma) or hepatic origin (e.g., primary neoplasia, hepatic metastases, liver cysts) (Griffin et al., 2021; Practice, 2020). Additionally, bilirubin concentrations can rise significantly in cases of posthepatic icterus (>20 mg/dL) (Turgut, 2000).

## Therapeutic Approach to Icterus

In cats, icterus arises from various causes, and treatment protocols are developed specifically for each underlying etiology. Accurate diagnosis is the cornerstone of effective treatment. Diagnosis established through a combination of anamnesis, systemic examination, laboratory findings, and imaging techniques is crucial for determining the prognosis and appropriate treatment of prehepatic, hepatic, and posthepatic icterus.

However, as a general treatment protocol, fluid therapy based on hydration status, pain management (e.g., buprenorphine, 0.01 mg/kg sublingually every 8 hours), and antiemetic therapy (e.g., maropitant, 1 mg/kg subcutaneously every 24 hours) can be addressed relatively effectively and significantly impact clinical outcomes. Ursodeoxycholic acid (5−15 mg/kg once daily) has been used in cases of bilirubin cholelithiasis, EHBO, and PK deficiency but should not replace antibiotics or prednisolone in lymphocytic or neutrophilic cholangitis. Supportive therapy may include S-adenosylmethionine (90 mg/cat once daily), silymarin (2−5 mg/kg once daily), and/or vitamin E (50 IU once daily) (Center, Randolph, Warner, Flanders, & Harvey, 2022; Webb, 2016).

## Treatment Options for Prehepatic Icterus

As previously mentioned, prehepatic icterus can arise from various causes. Identifying the underlying cause and implementing a specific treatment protocol is of utmost importance. The primary goal of treatment is to eliminate hemoplasma infection and restore normal RBC function.

The diagnostic and treatment options for prehepatic icterus are presented in the table below (Table 4) (Webb, 2016).

**Table 4. Diagnostic and Treatment Options for Prehepatic Icterus**

| Differential Diagnosis | Diagnosis Methods | Treatment Options |
|---|---|---|
| **Diagnostic and Treatment Options for Prehepatic Icterus** | | |
| Mycoplasma Species | CBC, Blood smear test, Serum biochemistry profile, Polymerase chain reaction (PCR) | Doxycycline, 5 mg/kg PO every 12 hours for 14 days<br>Pradofloxacin, 5 mg/kg PO every 24 hours for 14 days<br>Enrofloxacin, 5 mg/kg PO every 24 hours for 14 days |
| Cytauxzoon felis | CBC, Blood smear test, PCR | Atovaquone, 15 mg/kg PO every 8 hours<br>Azithromycin, 10 mg/kg PO every 24 hours |
| FIP / Babesia Species | CBC, Blood smear test, PCR | FIP: Supportive therapy, Polyprenyl immunostimulant, Pentoxifylline, 10 mg/kg PO every 12 hours<br>Prednisolone, 2–4 mg/kg PO every 24 hours<br>Babesiosis: Imidocarb dipropionate, 2.5 mg/kg IM<br>Doxycycline, 10 mg/kg/day PO for 21 days |
| FeLV / FIV | FeLV: p27 antigen test<br>FIV: Antibody test | Blood transfusion, Supportive therapy, Antiviral therapy<br>Medical therapy: Prednisolone, 2.2 mg/kg PO every 12 hours<br>Cyclosporine, 5 mg/kg PO every 24 hours<br>Chlorambucil, 2 mg/cat every 3 days<br>Mycophenolate mofetil, 10 mg/kg PO every 12 hours |
| Immune-Mediated Hemolytic Anemia (Primary) | Saline agglutination<br>Coombs test | Prednisolone, 2.2 mg/kg PO every 12 hours<br>Mycophenolate mofetil, 10 mg/kg PO every 12 hours |

| | | |
|---|---|---|
| Erythrocyte PK Deficiency / Increased Erythrocyte Osmotic Fragility | Genetic testing | Breeding management<br>Supportive therapy<br>Ursodeoxycholic acid, 5–15 mg/kg PO every 24 hours |
| Neonatal Isoerythrolysis | Appropriate blood typing before breeding | Supportive therapy, Cardiovascular support |
| Transfusion Reaction | Pre-testing of donors, Blood typing, Cross-matching | Supportive therapy, Cardiovascular support |
| Hypophosphatemia | Electrolyte testing | Supportive therapy, Treatment of the underlying disease |
| Microangiopathic Hemolytic Anemia | CBC with platelet count<br>Coagulation times | Intensive care, Cardiovascular support, Treatment of the underlying disease |
| Drugs, Toxins, Poisoning, Oxidative Stress | Various toxin assays<br>Blood smear test | Plasmapheresis<br>Elimination of exposure<br>Support for affected organs<br>Symptomatic treatment |

## Therapeutic Options for Hepatic Icterus

The treatment of hepatic icterus resulting from liver diseases in cats often requires careful selection and administration of medications. A comprehensive evaluation should be conducted to identify the underlying causes of icterus, and specific treatment methods tailored to liver diseases should be implemented. The diagnostic and therapeutic options for hepatic icterus are provided in Table 5 (Webb, 2016).

**Table 5. Diagnostic and Therapeutic Options for Hepatic Icterus**

| Differential Diagnosis | Diagnosis | Therapeutic Options |
|---|---|---|
| Hepatic Icterus Diagnostic and Treatment Options | | |
| Hepatic Lipidosis<br><br>(Idiopathic or Secondary) | CBC, Serum biochemistry profile, Urinalysis, FeLV/FIP testing, Bile acids, Feline pancreatic lipase (fPLI) blood test, Ultrasound-guided fine needle aspiration (FNA) of the liver | Vitamin K1, 1 mg SC every 12 hours<br>E-tube feeding |
| Sepsis / Systemic Inflammatory Response Syndrome | Various diagnostic methods | Supportive therapy<br>Symptomatic treatment |
| Cholangitis (Bacterial, Acute or Chronic Neutrophilic, Lymphocytic) | Ultrasound-guided or Laparoscopy-assisted cholecystocentesis and liver FNA Cytology Culture and antibiogram | Amoxicillin-clavulanic acid, 62.5 mg/cat every 12 hours, Enrofloxacin, 5 mg/kg every 24 hours, Metronidazole, 7.5 mg/kg every 12 hours, Prednisolone, 2 mg/kg every 24 hours, reduced to 0.5–1 mg/kg every 24 hours, Cyclosporine, 5 mg/kg PO every 24 hours, Ursodeoxycholic acid, 10–15 mg/kg every 24 hours |
| Drugs and Toxins | Various toxin tests | S-Adenosylmethionine, 90 mg/cat every 24 hours, Silymarin, 2–5 mg/kg every 24 hours, Vitamin E, 50 IU every 24 hours |
| Infectious Diseases (FIP, FCV) | Infectious disease testing | Supportive therapy<br>Symptomatic treatment |
| Amyloidosis (Genetic, Other) | Liver FNA Cytology | Supportive therapy |

## Therapeutic Options for Posthepatic Icterus

Posthepatic icterus in cats is commonly a result of bile duct obstruction caused by various potential factors. It is crucial to identify the underlying cause of this condition as the first step, followed by specific treatment aimed at resolving the primary issue. The diagnostic and therapeutic options for posthepatic icterus are detailed in the table below (Table 6) (Webb, 2016).

**Table 6. Diagnostic and Therapeutic Options for Posthepatic Icterus**

| Differential Diagnosis | Diagnosis | Treatment Options |
|---|---|---|
| **Diagnostic and Therapeutic Options for Posthepatic Icterus** | | |
| Cholelithiasis (Cholelithiasis) | Abdominal ultrasound | Ursodeoxycholic acid (5-15 mg/kg once daily), surgical intervention |

| | | |
|---|---|---|
| Extrahepatic Bile Duct Obstruction (EHBO) | Abdominal ultrasound, fine needle aspiration (FNA) of the affected tissue | Ursodeoxycholic acid (5-15 mg/kg once daily), systematic therapy, surgical or medical intervention |
| Triaditis | fPLI blood test, abdominal ultrasound, liver FNA, cholecystocentesis, cytology, culture and antibiogram, endoscopic biopsy of the small intestine | Supportive care: hydration, perfusion, acid-base balance; Buprenorphine (0.01 mg/kg sublingual every 8 hours), Maropitant (1 mg/kg SC once daily), antibiotics, E-tube feeding, single-dose anti-inflammatory glucocorticoids |
| Liver Parasites (Platynosomum concinnum) | Fecal examination, ultrasound-guided cholecystocentesis, bile cytology, abdominal ultrasound | Praziquantel (10-30 mg/kg once daily for 3 days), surgical intervention if EHBO is present |

## Conclusion

In conclusion, icterus in cats is often the result of various underlying pathologies, including hepatobiliary diseases, hematological disorders, toxic agents, and parasitic infestations. Clinical signs play a crucial role in the diagnosis of icterus in cats. Additionally, laboratory parameters are essential for identifying the cause of icterus, assessing the patient's condition, establishing a definitive diagnosis, and formulating an effective treatment plan. Treatment approaches may vary depending on the underlying cause of icterus. An individualized treatment plan should be developed for each patient.

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