A Case Study on Repeated Jaundice in a Local Breed Female

Cat



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List of Abberviation

| Abbreviation | Elaboration | |
|--------------|---|--|
| % | Percentage | |
| etc. | Et cetera | |
| et. al | And his associate | |
| RBC | Red blood cell | |
| HR | Heart Rate | |
| RR | Respiration Rate | |
| RI | Reference Interval | |
| EDTA | Ethylendiamine Tetraaceticacid | |
| PCV | Packed Cell Volume | |
| AST | Aspartate Transaminase | |
| ALT | Alanine Aminotransferase | |
| ATP | Adenosine Triphosphate | |
| BUN | Blood Urea Nitrogen | |
| TTPHRC | Teaching & Training Pet Hospital & | |
| | Research Centre | |
| | | |
| CVASU | Chattogram Veterinary and Animal Sciences | |
| | University | |

Abstract

A 2-year-7-month-old female local breed cat was diagnosed with jaundice about 3 times this year. The cat was icteric with severe hyperbilirubinemia. Hematological and serum biochemical analysis revealed that the cat had severe hyperbilirubinemia associated with hypophosphatemia and FIV virus. It also had moderate elevation of alanine aminotransferase, alkaline phosphatase, total protein, blood urea nitrogen and creatinine. Our study revealed that hemolysis occurs due to hypophosphatemia and FIV virus infection and this condition leads to repeated jaundice. After being treated with medicinal drugs such as cynomin, cataphos, amphexin and given a nutritionally balanced diet accordingly to daily energy the cat recovered from this condition.

Keywords: Jaundice Bilirubinin, Hypophosphatemia, Medicine, FIV

Chapter 1: Introduction

Icterus is also known as jaundice, the term used to describe an excessive buildup of yellow pigment in the blood and tissues (Weir & Ward, 2022). The word "icterus" is derived from the Greek word "icteric" which refers to the disease that causes skin to turn yellow. The French term "jaune," which implies yellow, in which the word "jaundice" first appeared (Wedderburn, 2022). Jaundice is an indication of another illness (Kruzer, 2022). When bilirubin, a yellow pigment, builds up excessively in a cat's blood and body tissues, jaundice begins to form (Hollinger, 2021). When icterus is present for any period of time, many bodily tissues will become discolored, and most body surfaces, including the skin, will show jaundice (Weir & Ward, 2022). The disease is seen worldwide. Jaundice causing illnesses are more likely to develop in some cat breeds. Siamese and Oriental Shorthair cats, Abyssinians, and Somali cats are a few examples. Because a cat's skin is mostly covered by fur, jaundice is most noticeable in the gingivae (gums), sclera (white area of the eyes), and pinnae (ear flaps) is yellowish in color.

In cats which have black pigmented gums or skin, biliary cirrhosis may be difficult to spot. The normally pink oral mucous membranes (gums, lips, and tongue) exhibit discoloration and frequently take on a yellow hue. The conjunctival membrane, which is typically pink, and the sclera, which is typically white, both appear yellow, giving the eyes the appearance of being yellowed (Hollinger, 2021). The cat's normally white skin seems yellow. Because so much of the skin is covered in fur, it can often be difficult to notice this. The underside of the belly and the interior of the ear pinnae are two areas without hair where the yellow hue is most noticeable. Lethargy, vomiting, and weight loss can also be indicators of jaundice in cats, depending on the underlying reason (Wedderburn, 2022). Jaundice can have prehepatic, hepatic, or post-hepatic origins, but they are all brought on by hyper bilirubinemia (Zawie & Garvey, 1984). An abnormally high blood level of bilirubin, a substance found in red blood cells, is known as hyper bilirubinemia. It is caused by red blood cell degeneration, gallbladder illness, or liver disease. A brown to yellow pigment called bilirubin is produced by the bile. When a cat's old red blood cells naturally start to die, bilirubin is normally excreted in the cat's feces. However, if the liver is unable to properly eliminate the bilirubin, jaundice may develop (Kruzer, 2022).

Red blood cells (RBCs) are responsible for the majority of bilirubin production, either through natural quiescence (removal of old RBCs) or aberrant RBC destruction (hemolysis). The free heme group of the RBC is converted to biliverdin in the first phase, which takes place inside healthy macrophages. The iron is let go and might be kept. The biliverdin is transformed into unconjugated bilirubin and then released into the plasma, where it interacts with albumin to travel to the liver (Augusto, 2022). The hepatocyte separates the bilirubin from the albumin and prepares it for excretion into the biliary system to produce conjugated bilirubin. Along with bile salts, conjugated bilirubin is discharged into the colon where it is converted to urobilinogen by bacterial and intestinal enzymes (Augusto, 2022). About 10% of urobilinogen is either reabsorbed or broken down into stercobilin and urobilin, respectively (both of which are excreted in the feces). The majority of the reabsorbed urobilinogen enters the liver's hepatic circulation to be reabsorbed, while the remain bypasses the liver and is expelled in the urine. Jaundice becomes visible when bilirubin concentration exceeds 35 mol/L (Augusto, 2022). Increased bilirubin synthesis (prehepatic), reduced hepatic conjugation, or decreased excretion can all lead to hyper bilirubinemia (post hepatic).

Hemolysis is the most frequent cause of excessive bilirubin production, but it can also happen when there is significant internal bleeding or when damaged red blood cells are broken down. Contrary to intravascular hemolysis, which occurs when hemoglobin is free in the bloodstream and subsequently complexes with hemoglobin before being removed by hepatocytes where hemoglobin breakdown takes place, extravascular hemolysis results in the generation of bilirubin as previously mentioned. Hyperbilirubinemia and icterus can also result from hepatocellular dysfunction brought on by diminished functional mass, inadequate perfusion, or abnormalities in uptake or conjugation (Gordon, 2011). This happens when the liver is affected by inflammatory, infiltrative, and necrotic disorders. As a result of reduced bile flow outside of the liver, bilirubin excretion is decreased. Bile flow may be impacted by inflammatory, viral, malignant, neoplastic, and obstructive conditions that affect the cystic duct, gallbladder, common bile duct, or duodenum (Gordn, 2011). Production and excretion of bile, detoxification of numerous substances, as well as synthesis of plasma protein are the metabolic function of liver (McClaran et al., 2013). In hepatitis destruction of liver parenchyma occur due to apoptosis or necrosis and inflammatory reaction.

Liver can fully regenerate in small grade of destruction. On the other hand severe destruction causes loss of hepatocytes and proliferation of bile ducts. Fibrosis occur in chronic destruction or a severe loss of hepatocyte. With the help of histopathological examination of liver tissue definitive diagnosis can be done incase of acute or chronic hepatitis (Antje et al., 2022). The objective of this study is describe below:

•To asses the hematological and biochemical parameter in jaundiced cat.

• To diagnose the repeated jaundiced case in cat.

Chapter 2: Materials and Method

2.1 Study area: Teaching & Training Pet Hospital and Research Centre(TTPHRC) Bangladesh's first-ever veterinary clinic, opened its doors in October 2018 in the Purbachal neighborhood of Dhaka. The hospital is regulated by Chattogram Veterinary and Animal Sciences University oversees the hospital (CVASU). The hospital was built to give practical training to students and proper clinical care to pet animal. It contains a well-equipped operating room, as well as lab and x-ray equipment. It offers both pets and birds a variety of services, including care, vaccination, deworming, health checks, surgery, and more. Animals from zoos and wild environments are also brought here for medical care and checkups. Thus, it is a reliable source of proper clinical services to pet animal.

2.2 Data and information collection: Following initial registration and a historytaking process, a clinical examination was conducted on each case. Each case's clinico epidemiological findings were entered into the structured record keeping form. Address, time, total population, housing system, species, breed, age, sex, body weight, BCS, immunization, deworming, history of prior illnesses and treatments, length of illness, defecation, micturition, and vomiting were all included in the data, along with client demographic data (age, sex, education and job). Pulse, respiration, and rectal temperature were measured, and skin fold test, mucous membrane examination, and examination of various body organs were carried out using the techniques of palpation, percussion, and auscultation. Data on diagnoses and medication prescriptions were also kept on the structured record keeping sheet. created using the trade names of the medications and the primary.

2.3 Case Presentation: A 2 year 7 months old weighing 3.2 kg local breed female cat was referred to TTPHRC for evaluation of Repeated Jaundice about 3rd time in this year.

2.4 Case history: The owner reported that the cat first became sick in late January with the Symptoms of yellow gum, yellow earflap, yellow nose, weakness, appetite, lethargy and unusual behavior. The owner consults with a vet and Clinical examination revealed that temperature was 100.5°F, Heart Rate(HR) 128/min, Respiration Rate(RR) 18/min. With the treatment of following drug Ceftron (Cefriaxone), Silybin (Silymarin), Syrup Icturn (Chicory, Borage, differ & other natural ingredients), Melvet (Meloxicum), Aminovit Plus (Dextrose, Vitamin, Amino Acid) she became recovered.

In June the cat becomes sick again with the symptom of difficulty breathing, weakness, and off-feed. The cat recovered with the treatment of Moxaclav (Amoxicillin+ Clavulanic acid). On September 24, The cat became sick again with the sign of yellow gum, yellow mucus membrane, yellow ear flap, weakness. Clinical examination revealed that temperature was 104.6°F, HR 128/min, RR 18/min. Biochemical examination revealed that serum bilirubin was 1.32mg/dL (Reference Interval 0-0.2 mg/dL), Alanine Aminotransferase 36.44 U/dL (RI 0-80 U/dL), Aspartate Aminotransferase 87.93 U/dL (RI 0-80), Alkaline Phosphatase 5.49 U/L (RI 5- 180), Blood Urea Nitrogen 36.44 mg/dL (RI 5-30 mg/dL), Creatinine was 1.1 mg/dL (RI . 5 - 1.5 mg/dL), which is higher than the normal range. The cat recovered with the treatment of syrup Moxaclav (Amoxicillin + Clavulanic Acid) and Capsul Silybin (Silymarin), Tab Nefrotec (Pasanabheda, Shilapushpa).

On November, the cat became sick again with signs of weakness, off-feed, yellow gum, and yellow mucus membrane and referred to TTPHRC.

2.5 Clinical examination: On clinical examination cat was having body temperature 101.6°F. The RR was (20/min). The pulse rate was (125/min). Definition and micturition were normal. Physical examination was unremarkable except for yellowish mucus membrane and hair fall. Lung sound was taken. There is no grunting sound in the lungs. HR was (125/min). The lymph node was palpated no enlargement was found in them. There were also no abnormalities found in palpation of the abdomen.



[Fig 1: jaundiced cat]

2.5 Laboratory examination: For hematological and biochemical examination blood was collected from the cephalic vein. Before blood collection the cat was placed in lateral recumbency and palpate the cat's arm to find the vein. Then use a tourniquet to anchor the vein. Blood collection area was disinfected with alcohol swab. Blood collection was complete within 3 minutes. 3 ml of blood was collected. Anticoagulated blood was needed for hematological examination. 1.5 ml blood sample were collected with ethylenediamine tetracetic acid (EDTA) containing vial for Complete Blood Count. TTPHRC diagnostic lab did not have the facility of hematological test. The blood sample was taken into another lab for hematological test. For this the blood sample was taken into transport box. In transport box a layer of perforated sponge was placed at bottom and blood sample was sealed in Zip lock bag over the perforated sponge. Cover the sample with the second layer of perforated sponge and close the lid .Then the blood sample carried to the Vet Lab, Baridhara, Dhaka. Hematological test were performed in that lab. Another 1.5 ml blood was taken in red color vacutainer which do not contain EDTA which is used for serum biochemical test. A serum biochemical test was performed in the diagnostic lab of TTPHRC, Purbachol, Dhaka.

2.6 Diagnostic test: Diagnostic tests play a significant role to diagnose the disease. Following diagnostic tests were done to diagnose the disease.

2.6.1 FIV kit test: It is an easy and rapid procedure for FIV virus diagnosis. We used Rapid test kit from TESTSEALAB. This test device was coated by an invisible T (test) zone and C (control) zone. When serum is applied into the sample well on that device the reagent will laterally flow on the surface of the test strip. Both T and C band will show if the test is positive. In this test procedure, removed the kit from the foil pouch. The cassette was placed horizontally on surface. Took one drop of serum in disposable dropper from the serum

sample and immediately dripped 3-4 drop of FIV assay buffer into corresponding well. After 6 minutes both T and C band was appeared and diagnosed that the cat is positive for FIV virus.

2.6.2 X-ray findings: Bilateral view was taken in the x-ray. X-ray findings revealed that the lung and heart were right in their position and normal in size. There was no blood or fluid accumulated in the lung. The liver is normal in size and in the right position. There were no abnormalities found in the abdominal cavity.



[Fig 2 : X-ray of patient's liver]

2.6.3 USG finding: For the assessment of hepatic and biliary disorder, ultrasonography is the useful noninvasive imaging (Larson 2016). Ultrasonography finding revealed that the liver, lung, and heart were normal in size and right in position. The liver appear coarse, moderately echogenic organ. It was located between the diaphragm cranially and the stomach and right kidney caudally No fat was accumulated surrounding the liver.

Chapter 3: Results

3.1 Hematological Parameter :

Hematological examination revealed that RBC was 4g/L, PCV was 24.8%, Hgb was 7.6 g/dL, MCV was 35.6, MCH was 10.8 pg, MCHC was 24, Neutrophil was 60%, Leucocyte was 29%, Macrophage was 03%, Eosinophil was 08%.

| Parameters | Presenting | ReferenceValues |
|---------------------------------|------------|-----------------|
| | value | |
| Redbloodcells(RBC) | 4 | 5-10 |
| (g/L) | | |
| Packedcellvolume(PCV)(%) | 24.8 | 30-45 |
| Hemoglobin(Hgb) | 8.1 | 8-15 |
| (g/dL) | | |
| Meancorpuscularvolume (MCV) | 35.6 | 39-55 |
| Meancorpuscularhemoglobin (MCH) | 10.8 | 13-17 |
| (pg) | | |
| Meancorpuscularhemoglobin | 24 | 30-36 |
| Concentration (MCHC) | | |
| Whitebloodcells(WBC) | 25 | 5.5-19.5 |
| Neutrophils(%) | 60 | 22-82 |

| Lymphocytes | 29 | 9-56 |
|-------------|----|------|
| (%) | | |
| Monocytes | 3 | 1-4 |
| (%) | | |
| Basophils | 0 | 0-1 |
| (%) | | |
| Eosinophils | 08 | 2-12 |
| (%) | | |

3.2 Serum Biochemical Parameter:

Serum biochemical examination revealed that Phosphorus level was 1mg/dl, Glucose level was 71.7 mg/dl, Total protein was 9.7 g/dl, Albumin level was 2.9g/dl, Bilirubin value was 1.1 mg/dl, ALT was 241U/l, AST was 89 u/l, ALP was 63u/l, BUN value was 74 mg/dl and Creatinine was 4.4 mg/dl.

| Parameters | Presenting | Reference |
|----------------------------------|------------|-----------|
| | Values | Values |
| Phosphorus (mg/dl) | 1.0 | |
| Glucose (mg/dl) | 71.7 | 50-170 |
| Total protein (g/dl) | 9.7 | 5.2-8.8 |
| Albumin(g/dL) | 1.1 | .1–.04 |
| Bilirubin (U/l) | 241 | 2.4-3.7 |
| Alanine Aminotransferase (U/L) | 89 | |
| Aspartate Aminotransferase (u/l) | 63 | |
| Alkaline Phosphatase(μ/L) | 45 | 9.2-40 |
| Blood Urea Nitrogen (mg/dL) | 74 | 15-30 |
| Serum Creatinine (mg/dl(| 4.4 | |

3.3 Treatment : After came to TTPHRC, vet treated the cat with following drug including Cynomin (Cyanocobalamin) administered subcutaneously, Cataphos

(Butaphosphan) applied subcutaneously, Amphexin (Ampexin) applied subcutaneously, Syrup Liv (Chicory, caper bush) administered orally. This treatment was given for 7 days.

Chapter 4: Discussion

The largest internal organ of feline body is liver which makes up about 3-4% of it's Overall weight (Otte et al., 2017). In prehepatic jaundice excess bilirubin is produced which is beyond the liver's capacity to conjugate the bilirubin and excrete it .

Hematological examination revealed that RBC concentration in blood was 4 g/L which is slighty lower than the normal reference interval. RBC concentration in blood can be decreased due to intra or extravascular hemolysis. Intravascular hemolysis results in hemoglobinemia and hemoglobinuria which were not found in our study. Our study revealed that decreased RBC level in blood associated with jaundice. In a similar way other authors claimed that extra or intra both type of hemolysis can cause icterus (Susan & Moses, 2010). PVC level was found 24.8% which is lower than normal level which might occur due to loss of blood (hemorrhage), breakdown of erythrocyte in circulation (hemolysis) Or lack of production of erythrocyte by bone marrow (hypoplasia or anaplasia). Other study showed that PCV estimation always significant in an icteric patient. If PCV value is moderately or markedly decreased the icterus has prehepatic origin (Kumar et al. 2020). Which is similar with our study.

Bilirubin is metabolized in the liver and converted into conjugated bilirubin which removes from the body through feces and urine. Serum Biochemical examination revealed the cat has hyperbilirubinemia. Clinical icterus occurs when total bilirubin levels are above 2.5 to 3.0 mg/dL (Susan & Moses, 2010) The causes of an increase in bilirubin concentrations include extrahepatic cholestasis, poor absorption, intracellular transport, glucuronide conjugation, prehepatic hemolysis, increased hemoprotein liberation, raptured bile tract, bile duct occlusion, impaired hepatobiliary bilirubin processing, raptured biliary tract etc. (Susan & Moses, 2010). Albumin is extracellular transport protein for bilirubin in most animals including human. Other study showed that hyperbilirubinemia is seen in prehepatic hepatic and posthepatic jaundice (Sherding, 2000). The cat had decreased phosphorus in her blood. Serum biochemical analysis represents that the cat was suffering from hypophosphatemia. It can be caused by a transcellular transfer of phosphate into cell, increased renal excretion, decreased intestinal absorbtion or combination of these mechanism. Chronic metabolic acidosis may also contribute to increased urinary losses of phosphate. hypophosphatemia was followed by the development of hemolytic anemia. It was likely the origin of hemolytic anemia because of the temporal link between hemolysis and hypophosphatemia. Although mechanism of hemolysis leads to hypophosphatemia is uncertain. Depletion of erythrocyte adenosine triphosphate (ATP), which is essential for maintaining cell membrane integrity, may lead to hemolysis. Phosphate is required for the phosphorylation of glucose to start glycolysis, which in turn depends on the intracellular ATP concentration of erythrocytes (Adams et al., 1993).

Hemolysis occurs when intracellular phosphate and ATP concentrations are severely below a crucial The pentose monophosphate shunt's first stage, the phosphorylation of glucose to glucose-6-phosphate, may be impaired by hypophosphatemia (Adams et al. 1993) Other study reported that, In cattle, sheep and goat hypophosphatemia causes post parturiant hemoglobinuria and hemolysis. This condition arise after 2-6 week after parturition. Incase of diabetes mallitus, hepatic lipidosis and refeeding syndrome hypophosphatemia with secondary hemolysis is seen in dog and cat. Any bilirubin that is successful in becoming conjugated will be eliminated normally. However, it is the unconjugated bilirubin that persists in the bloodstream and is responsible for jaundice. In this cat hypophosphatemia probably caused hemolysis. Because of this hemolysis, unconjugated bilirubin was produced more and this unconjugated bilirubin cannot be eliminated through the urine and before bilirubin levels in the plasma increases which leads to Jaundice. This may explained that hypophosphatemia associated with hemolysis in our study. The diagnostic test revealed that the FIV virus was positive. Prevalence of FIV vitus is 1 to 28% worldwide. In case of FIV virus it destroy the immune system when persist in the body (Uhl et al., 2002). Hemolysis could be occur by FIV (Kruzer, 2022). This virus could be cause hemolysis and hemolysis lead to prehepatic jaundice (Gordn , 2011).

Alkaline Aminotransferase (ALT) was found elevated in serum biochemical analysis. Increases in ALT have been linked to liver and muscle damage, as well as hyperthyroidism (Susan & Mosses, 2010). ALT increased in hepatocellular necrosis, inflammation and hepato toxicosis. It was increased after 5 day of onset of action. (Otte et al., 2017) Alkaline phosphatase was also higher than its normal reference interval. Alkaline phosphatase (ALP) is produced by the epithelial cells that line the bile canaliculi, and patients who have intrahepatic cholestasis, cholangitis, or extrahepatic obstruction have higher serum levels of this enzyme (Susan & Mosses, 2010). In similar way other author claimed that incase of acute hepatitis and chronic cholangiohepatitis high ALP concentration in serum can be detected . Low values are especially indicative of cirrhotic liver disease (Neumann, 2004). Blood urea nitrogen (BUN) level was found greater than normal. Other authors claimed that

renal disease and obstruction of the flow of urine might cause elevated level of BUN (Morais, 2017). Other study showed that amount of BUN in plasma serve a reflection of an animal's protein metabolism (Chikhou et al., 1993). Other study reported that, BUN was associated with dehydration which is similar with our study. In our study the animal was dehydrated which caused the elevation level of BUN. Creatinine is removed from the body through urine by the kidney in a healthy body. Serum biochemical analysis revealed that creatinine was also elevated than the normal value. High creatinine level indicate kidney disease. Study reported that creatinine was associated and did not eat properly which might be the cause of elevated BUN and creatinine level.

After being treated with medicine the cat responded well. The cat was showing visible recovery after 5 days fully recovered after 14 days. Reexamination of blood and serum after seven days for hematobiochemical parameter showed marked improvement with normal liver specific enzyme as well as bilirubin reached in normal limit.

Limitations of this study

This study has several limitations. The first patient did not come to TTPHRC the first time when jaundice occur. If the owner came in the first stage of the disease then diagnostic tests and hematological and biochemical tests can be done in this time and diagnosis can be easy. The second one is there was no facility to do the liver biopsy test.

Conclusion

Jaundice is the commonest presentation of patients with liver and biliary disease. In this cat the main cause of this repeated jaundice is probably hypophosphatemia and FIV virus. Hemolysis occur due to hypophosphatemia and FIV virus infection. This hemolysis leads to excess production of bilirubin and finally jaundice occur. After using medicine and proper nutritional supplement phosphorus consistency in blood back to normal range and the cat recovered.

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