A Case Study on Diagnosis and Management of Chronic Hepatitis in A Shelter Bitch

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A Case Study on Diagnosis and Management of Chronic Hepatitis in A Shelter Bitch

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List of Abbreviations

Abbreviation	Full Form
CH	Chronic Hepatitis
CBC	Complete Blood Count
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
ALP	Alkaline Phosphatase
GGT	Gamma Glutamyl Transferase
RBC	Red Blood Cell
WBC	White Blood Cell
BID	Twice a Day
SID	Once a Day
NSAIDs	Non-Steroidal Anti-Inflammatory Drugs
PU/PD	Polyuria/Polydipsia
CVASU	Chattogram Veterinary and Animal Sciences University
PO	By Mouth (Per Os)
SC	Subcutaneous
IV	Intravenous
Injection	Inj.
Syrup	Syp.
Tablet	Tab.

Abstract

Chronic hepatitis (CH) is a progressive inflammatory liver disease that poses significant diagnostic and therapeutic challenges in veterinary practice. This case study documents the diagnosis and management of CH in a 7-year-old shelter bitch presenting with lethargy, jaundice, anorexia. Clinical examination, laboratory findings, and imaging revealed hallmark features of CH, including hepatomegaly, ascites, increased hepatic echogenicity, anemia, hypoalbuminemia, and elevated liver enzymes. The radiographic and ultrasound examinations revealed advanced liver disease. Management provided comprehensive support through the administration of intravenous fluids, antibiotics, liver-protecting medications such as silymarin, vitamin supplementation, and hematinic. This case study underscores the importance of non-invasive diagnostic modalities in resource-limited settings for the diagnosis and treatment of canine chronic hepatitis, and emphasizes the need for standardized therapeutic approaches. Chronic hepatitis in dogs requires further investigation through large-scale, collaborative studies in future research to develop more effective diagnostic and therapeutic strategies.

Keywords: *Hepatitis, chronic, dog, hepatomegaly.*

Chapter 1: Introduction

Chronic hepatitis is the most common canine liver disease when secondary reactive hepatopathies are ruled out. Chronic hepatitis is a persistent inflammatory condition of the liver that is characterized by hepatocellular damage, inflammation, and progressive fibrosis, often leading to cirrhosis if left untreated. In most cases, an underlying cause is never determined. (Cohn and Cote, 2019)

Chronic hepatitis (CH) has been widely recognized in dogs across various breeds and in both general and specialized veterinary settings for decades (Andersson and Sevelius, 1991; Dill Macky, 1995; Fuentealba et al., 1997; Sevelius, 1995; Strombeck and Gribble, 1978).CH is often perceived as a challenging disease for its tendency to progress to cirrhosis, leading to a generally poor prognosis. Chronic hepatitis is a significant contributor to illness and death in dogs, with studies reporting a postmortem prevalence of 12% in cases from first-opinion veterinary practices (Watson et al., 2010). Furthermore, it is the most frequently diagnosed form of hepatitis in dogs referred to university veterinary hospitals, comprising 66% of hepatitis cases, of which 64% were identified as idiopathic (Poldervaart et al., 2009).

Chronic hepatitis may result from drug therapy (eg, phenobarbital, NSAIDs, or other drugs), infectious diseases (such as leptospirosis), toxin-induced liver injury, and other forms of acute hepatitis. Diets containing high levels of copper can result in copper-associated hepatitis in dogs that are genetically susceptible. Idiopathic chronic hepatitis (ICH) in dogs has been associated with familial predisposition, particularly in breeds such as the Bedlington Terrier, Cocker Spaniel (both American and English), Dalmatian, Doberman Pinscher, Skye Terrier, West Highland White Terrier, and potentially the Labrador Retriever (Tilley et al., 2021). The following conditions have been associated with the onset of chronic hepatitis: leptospirosis, leishmaniasis, canine adenovirus type 1, and canine acidophil cell hepatitis (suspected but not as well-documented). These diseases may be contagious, with leptospirosis and leishmaniasis being zoonotic (Cohn and Cote, 2019).

Idiopathic chronic hepatitis appears more common in bitches, with the average age of onset between 5 and 6 years, although dogs of any age or breed can be affected. Typical clinical signs include anorexia, depression, weakness, polyuria/polydipsia, ascites, jaundice, weight loss, and vomiting (Birchard and Sherding, 2006).

Chronic hepatitis can be suspected in any dog with historical, physical, biochemical, or imaging evidence of hepatobiliary disease. Diagnosis is confirmed via histologic assessment of a liver biopsy. Unexplained persistent abnormal liver enzyme activities should initiate investigation for diagnosis of possible chronic hepatitis (Cohn and Cote, 2019).

The objective of this study are-

- To evaluate the clinical, radiographic and ultrasonographic features associated with chronic hepatitis.
- To evaluate the therapeutic approach in treating dogs with Idiopathic Chronic Hepatitis.

Again, by integrating laboratory test results with the imaging and clinical observations, this study seeks to establish a diagnostic framework for field veterinarians, in absence of advanced tools like liver biopsy and histopathologic examination, bile acid testing, hepatic metal quantitative analysis etc., emphasizing practical approach in resource limited settings.

Chapter 2: Case presentation

2.1 Case Description

A 7-year-old female dog named “Vuri” was presented at the Teaching and Training Pet Hospital and Research Centre, Dhaka with a history of lethargy, loss of appetite, weight loss, yellow-colored urine and a striking yellow discoloration of the mucous membranes, strongly indicative of jaundice. The owner reported that the dog’s vaccinations and deworming schedules were overdue, raising concerns about potential infectious or systemic causes.

2.2 Diagnostic Protocol

2.2.1 Clinical Examination

The dog appeared visibly weak with a body temperature of 102°F and signs of dehydration, suggesting a more severe underlying issue. The poor condition of the animal, combined with jaundiced gums and eyes, pointed to significant liver involvement. Abdominal palpation hinted at discomfort and possible hepatic enlargement, underscoring the urgency for diagnostic imaging and laboratory tests to confirm the suspected hepatic dysfunction and guide in treatment protocol.

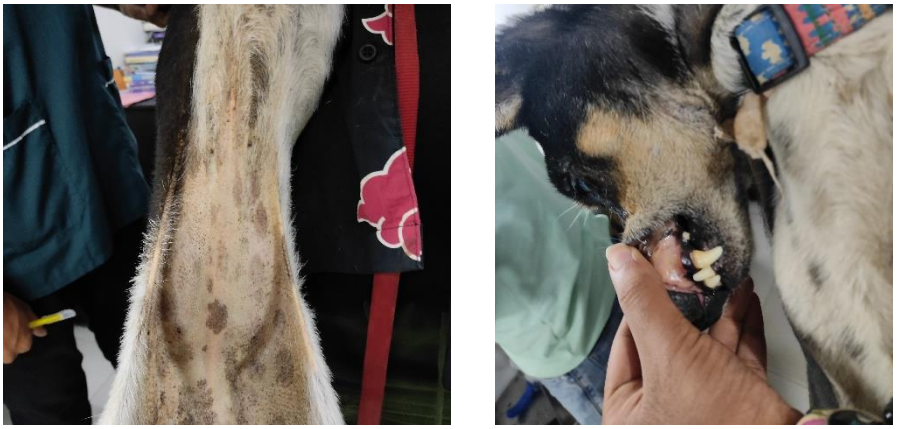

Figure 1. Yellowish discoloration of ventral abdomen and gum

2.2.2 Radiographic Examination

- The thoracic cavity shows no clear signs of abnormalities.
- The diaphragm appears intact and normal in shape. (Figure 2.a)
- The gastric axis (the line from the fundus to the pylorus) seems slightly shifted caudally (Figure 2.b). Caudal displacement supports the possibility of hepatomegaly.

- The caudoventral border of the liver extended slightly beyond the costal arch with a rounded edge indicates hepatomegaly.
- Accumulation of fluid in abdominal cavity with decreased serosal details, indicates ascites.

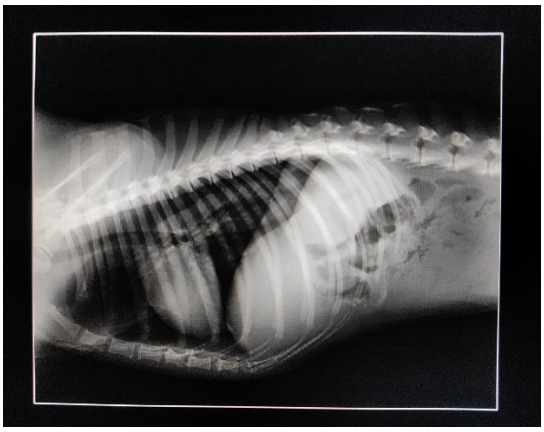

Figure 2.a Radiographic examination of thorax

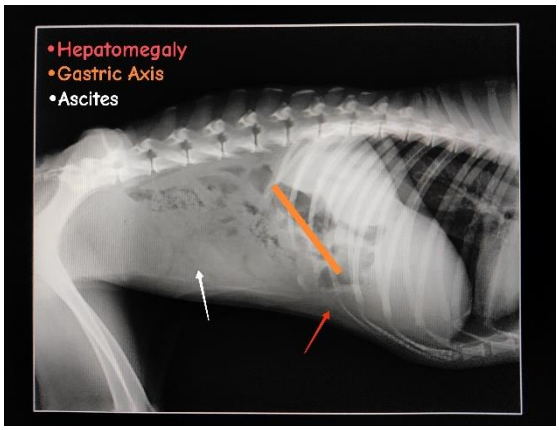

Figure 2.b Radiographic examination of abdomen

2.2.3 Ultrasonographic Examination:

- Increased echogenicity of liver possibly due to fibrotic or fatty changes in the liver, which is characteristic of chronic liver disease. Fibrosis disrupts normal parenchymal architecture, altering echogenicity due to increased collagen and scar tissue formation.
- Fluid in the abdomen or ascites. Free abdominal fluid can develop due to portal hypertension (Cynthia, 2020) resulting from increased venous pressure associated with chronic liver damage.
- Thickened intestinal wall may be due secondary or chronic inflammation.

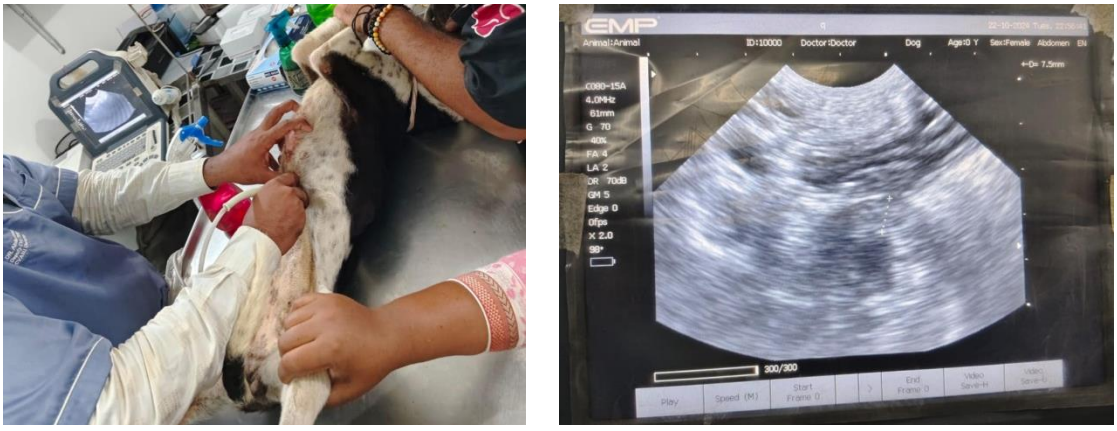

Figure 3. Ultrasonographic examination

2.2.4 Hematological Assessment

Table no.1: Hematological test result:

Test Name	Result	Reference Value
Hemoglobin (Hb%)	**9.6 g/dl**	12 - 18 g/dl
RBC Count	**4.79x10^6/cumm**	5.5 - 8x10^6/cumm
HCT/PCV	**32.4%**	37 - 55%
MCV	71.8 fL	60 - 77 fL
MCH	20.9 Pg	19.5 - 26 Pg
MCHC	29.1 g/dl	32 - 36 g/dl
RDW_CV	16.8%	11 – 19%
RDW_SD	40.4%	35.2-45.3%
Total WBC Count	16,940/cumm	6,000 - 17,000 /cumm
Neutrophils	76%	60 - 77%
Lymphocytes	09%	12 - 30%
Monocytes	09%	03 - 10%
Eosinophils	06%	02 - 10%
Basophils	00%	00 - 01%
Platelet Count	**112,000 /cumm**	164,000 - 510,000 /cumm
PCT	0.095%	0.09 - 0.50%

Reference source: (Tilley et al., 2021, p. 1441)

A complete blood count (CBC) (Table no.1) revealed low hemoglobin (9.6g/dL), decreased RBC count (4.79x10^6/cumm), reduced hematocrit (32.4%), lower platelet count (112,000/cumm) etc. which are common scenario in chronic liver disease.

2.2.5 Biochemical Analysis

Table no.2: Biochemical test result:

Parameters	Test Results	Reference Value
Calcium	**7.2**	7.5-11.3 mg/dl
Phosphorus		2.1-6.3 mg/dl
Magnesium		1.8-2.4 mEq/L
Potassium		4-5.6 mEq/L
Sodium		141-156 mEq/L
Chloride		105-115 mEq/L
Glucose	30	60-125 mg/dl
Total Protein	7.6	5.1-7.8 g/dl
Albumin	2.54	2.6-4.3 g/dl
Globulin		2.3-4.5 g/dl
Albumin Globulin Ratio		0.75-1.9
Bilirubin	**5.9**	0-0.4 mg/dl
Alanine Aminotransferase (ALT/SGPT)	**96**	5-60 u/l
Aspartate Aminotransferase (AST/SGOT)	126	5-55 u/l
Alkaline Phosphatase (ALP)	**341**	10-150 u/l
Gamma Glutamyl Transferase (GGT)		0-10 u/l
Creatine Kinase		10-200 u/l
Cholesterol		112-328 mg/dl
Blood Urea Nitrogen (BUN)	21	7-27 mg/dl
Serum Creatinine	1.3	0.4-1.8 mg/dl
Amylase		500-1500 u/l

Reference source: (Tilley et al., 2021, p. 1441)

Biochemical test result (Table no.2) reflects several abnormalities. The test result shows, Hypocalcemia (7.2 mg/dL), hypoglycemia (30 mg/dL), elevated bilirubin level(5.9mg/dL),

hypoalbuminemia (2.54 mg/dL), Increased ALP (341u/l) and ALT (96u/l) etc. These parameters are strong indicators and suggesting hepatitis in the bitch.

2.3 Treatment

Considering history, clinical signs, imaging and laboratory findings the bitch was diagnosed with Chronic Hepatitis. Supportive therapy was given to the dog as CH in dog is mostly idiopathic. Broad-spectrum antibiotic Amoxicillin @10mg/kg (Inj. Moxin 500mg, Opsonin Pharma Ltd.) was given subcutaneously BID for 7 days to check secondary bacterial infection. Fluid therapy using isotonic solution e.g. sodium chloride 0.9% (Inj. NS 500 ml, OSL Pharrma Ltd.) 200 ml intravenously SID for 7 days for correcting dehydration and electrolyte imbalance, promoting toxin excretion. Cyanocobalamin or vitamin B_{12} @25mcg/kg (Inj. Cynomin 1mg, Jayson Pharmaceuticals Ltd.) was administered subcutaneously three days interval to facilitate metabolism and correct anemia. Betacarotene, Vitamin C, Vitamin E (Tab. EC Plus, Orion Pharma Ltd.) was given @1 tablet/day for 7 days to scavenge free radicals and prevent oxidative injury. Hematinic mixture (Syp. Hemobest Pet, TTk Healthcare Ltd.) was administered @2ml PO, BID for 15 days to correct anemia. Silymarin (Liq. Milk Thistle, Wild and Organic) @20-50 mg/kg, PO, BID for 1 month as hepato-protective agent.

Chapter 3: Discussion

Chronic hepatitis is insidious, as clinical signs are not always readily evident until a well-established pathologic process has developed. Lethargy, weight loss, anorexia, vomiting, jaundice, ascites etc. are consistent clinical signs. PU/PD, diarrhea, depression, weakness has also been reported (Cohn and Cote, 2019). Clinical signs (lethargy, loss of appetite, weight loss, jaundice) found in the bitch also supports the saying of Cohn and Cote (2019) as mentioned earlier. Idiopathic chronic hepatitis appears more common in bitches, with the average age of onset between 5 and 6 years, although dogs of any age or breed can be affected (Birchard and Sherding, 2006).

Further clinical and laboratory examination revealed and supports the presence of Chronic Hepatitis in the bitch. The radiographic examination shows hepatomegaly (Figure 2.b) as the caudo-ventral border extends beyond the costal arch. Again, caudal displacement of gastric axis also supports the enlargement of liver (Cynthia, 2020) and decreased serosal details due to ascites (Figure 2.b) which is found in advanced staged hepatopathy (Cynthia, 2020) caused by more sodium and water retention as mentioned by Webster et al. (2019).

Increased echogenicity found in ultrasonography (Figure 3.) is also indicator of chronic inflammation and fibrosis, cirrhosis of the part of the liver which aligns with the findings of Wu et al. (2020). Ascites was found that may result from portal hypertension and hypoalbuminemia as found in the bitch which is similar to the findings mentioned by Webster et al. (2019).

A complete blood count (CBC) (Table no.1) revealed low hemoglobin (9.6g/dL), decreased RBC count ($4.79x10^6$/cumm) and reduced hematocrit (32.4%) indicate mild anemia (Webster et al., 2019), which is common in chronic liver disease. This is possibly due to reduced erythropoiesis (from nutritional deficiency or chronic inflammation), or hemolysis. The white blood cell count seems to be within the normal range. Yet a predominance of neutrophil (76%), which indicates neutrophilia, a response to inflammatory or infectious process. The platelet count (112,000/cumm) is markedly reduced, indicating thrombocytopenia, a common scenario in chronic liver disease which is consistent with findings of Webster et al. (2019).

Biochemical test result (Table no.2) reflects several abnormalities. Hypocalcemia (7.2 mg/dL) may result from impaired hepatic production of vitamin D metabolites which is essential for the

absorption of Ca^{+} from the intestine. Severe hypoglycemia (30 mg/dL) is seen which is a hallmark of advanced liver disease e.g. CH (Yang et al., 2023) Again hypoalbuminemia (2.54 mg/dL) may be due to less hepatic synthesis as hepatic parenchyma is damaged or cirrhosis of the liver (Conner, 2016). Elevated bilirubin level(5.9mg/dL) reflects compromised bilirubin conjugation and clearance, consistent with cholestasis or hepatobiliary disease (Rothuizen and Van Den Brom, 1987). Increased ALP (341u/l) and ALT (96u/l) is also associated with chronic hepatis. As CH progresses and hepatic parenchyma decreases, ALP and GGT activities increase compared to ALT (Webster et al., 2019; Fuentealba et al., 1997).

After diagnosing the bitch with chronic hepatitis, a therapeutic management was initiated immediately. Treatment for chronic hepatitis is mostly empirical and symptomatic, as for idiopathic chronic hepatitis or advance stage liver disease treatment aims to slower fibrosis rather than targeting root cause, as effective reversal of cirrhosis is not possible in dogs. Only a few specific therapies available, such as for copper chelators (D-penicillamine etc.) in case of copper accumulation in Bedlington terrier as mentioned by Rolfe and Twedt (1995). Here, a broad-spectrum antibiotic Amoxicillin @10mg/kg body weight was given twice a day to modify gut microflora, control secondary bacterial infection. Fluid therapy was administered to correct dehydration, electrolyte imbalance. Cyanocobalamin was administered to facilitate metabolism and correct anemia as it helps in RBC maturation. Vitamin E were used as an anti-oxidant to prevent oxidative damage that is found in hepatopathies mentioned by Cohn and Cote (2019). Hematinic mixture was administered to correct anemia, improve weakness. Silymarin is used as hepato-protective agent for its antioxidant, anti-inflammatory, anti-fibrotic, choleretic effect. Though it may inhibit certain cytochrome-p450 enzymes and p-glycoprotein (Webster et al., 2019). Thus caution is needed while using high dose of the disease.

There is a scarcity of clinical trials evaluating common treatments like copper chelators, corticosteroids, colchicine, and ursodeoxycholic acid (UDA). The majority of suggestions are either from anecdotal evidence or extrapolated from human medicine.

It’s evident that the knowledge of the etiology and treatment of CH is still at its infancy as compared to CH in humans. Many works should be done to describe the etiologies and determine effective treatments for canine disease. Previous studies in dogs provided information on infectious and genetic causes of CH. But the etiologies of CH are yet unknown due to lack of large scale,

multi-center studies. In many studies liver biopsy was given priority to define the stage and condition of the hepatopathy. The WSAVA's recent efforts to standardize histopathological descriptions of canine CH should serve as the foundation for larger, multi-center studies of the disease's etiology and management, ideally in individual breeds, which should advance our knowledge in the future.

Conclusion

This study reveals the complexity of diagnosing and managing chronic hepatitis in dogs, particularly when advanced diagnostic facilities are not available. By harmonizing clinical symptoms, laboratory data, and imaging results, a reliable diagnosis was obtained. Therapeutic efforts were aimed at slowing disease progression, alleviating symptoms, and improve the condition of the bitch. Despite limited knowledge about its causes and optimal treatment, chronic hepatitis in dogs remains a significant contributor to morbidity and mortality in the canine population. This study highlights the importance of practical approaches for diagnosis and management, especially in settings with limited resources, and emphasizes the need for more research for better understanding of the disease in dogs.

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Biography

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