

Severe hepatic lipidosis in a dog: a case report

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Abstract

Hepatic lipidosis (fatty liver) is a rare condition in dogs, primarily caused by improper feeding and care practices. It involves the accumulation of triacylglycerols in hepatocytes due to various toxic factors and dietary carbohydrate deficiencies. This case report describes the clinical, hematological, and pathological findings of an abandoned 8-year-old dog suffering from hepatic lipidosis that was referred to an animal shelter. The dog was unconscious, immobile, depressed, and severely obese. Pronounced abdominal distension and icteric mucous membranes were immediately noticeable during the initial examination. A blood sample was collected, and the case was referred to a veterinary hospital for diagnostic imaging. Unfortunately, before the referral could be completed, the dog suddenly died. Immediately following the death, a postmortem necropsy was performed to determine the cause. Necropsy revealed an extremely enlarged liver with a yellowish discoloration and abnormal kidneys with nodular lesions. Based on these findings, fatty liver was strongly suspected. Complete blood count and biochemical parameters, including triglycerides, cholesterol, aspartate transferase, alanine transaminase, and alkaline phosphatase, were subsequently evaluated. Complete blood count results indicated leukocytosis, thrombocytopenia, and an increase in red cell distribution width. Biochemical analyses revealed hypertriglyceridemia and elevated levels of alanine transaminase, aspartate transferase, and alkaline phosphatase. Histopathological examination confirmed severe fatty degeneration in the liver, alongside marked degenerative and necrotic changes in the kidneys. The findings of the present study contribute to the limited data on canine hepatic lipidosis and underscore the necessity of raising awareness about its clinical presentation, diagnostic markers, and histopathological characteristics.

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Introduction

Liver damage arises from factors, like hepatitis B/C, aflatoxin B1, obesity, diabetes, and diet.¹ Hepatic lipidosis, caused by toxic influences and carbohydrate deficiency, involves triacylglycerols accumulation due to gluconeogenesis and glucocorticoid-induced fat mobilization.² This disrupts phospholipid synthesis, fatty acid oxidation, and high-density lipoproteins production, exacerbating fat buildup.³ Lipid metabolism disruptions are central to lipidosis.⁴ Hepatic lipidosis, marked by triglyceride accumulation in 80.00 - 90.00% of hepatocytes, leads to oxidative stress, generating reactive oxygen and nitrogen

species, initiating cirrhosis, lipid peroxidation, and inflammation.^{5,6} These processes escalate to chronic hepatitis, cirrhosis, or hepatocellular carcinoma *via* cytokine release, gene mutations, and apoptosis.⁷

Liver disease manifests through various clinical signs depending on the liver function affected, as the liver plays a central role in metabolism, detoxification, and storage. Dysfunction in these processes can impact other body systems, leading to symptoms, such as depression, loss of appetite, vomiting, weight loss, diarrhea, polyuria, polydipsia, abdominal distension, lethargy, icterus, and ascites.⁸ Neurological issues, often resulting from hypoglycemia and hepatic encephalopathy, can be presented

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as seizures, disorientation, head pressing, blindness, or behavioral changes. However, many of these signs are non-specific and not exclusive to liver disease.⁹ Hepatic lipidosis is one of the most common liver diseases in cats,¹⁰ whereas it is relatively rare in dogs.¹¹

Various strategies have been proposed for the prevention of hepatic lipidosis, such as weight management and obesity prevention, early intervention for anorexia, and nutritional prophylaxis (use of high-protein, moderate-fat diets for at-risk cases).^{12,13} Additionally, recommended treatments for this condition include aggressive nutritional support (tube feeding), anti-emetics, fluid therapy, vitamins K and B and electrolyte supplementation, and L-carnitine. Cats often show a worsening of symptoms during the initial days of treatment compared to their condition at presentation. Clinicians should not become discouraged too early.^{12,14}

This study reports clinical signs, as well as hematological and histopathological changes in a dog suffering from a severe form of hepatic lipidosis.

Case Description

A nearly 8-year-old intact male mixed-breed dog weighing 9.20 kg, abandoned by its owner, was found and referred to the Urmia Animal Shelter, Urmia, Iran. The dog was presented in an unconscious state, unable to move, depressed, and severely obese. Marked abdominal distension was immediately apparent during the initial examination (Fig. 1). Clinical evaluation revealed a heart rate of 105 beats *per min*, which was within normal limits. Mild tachypnea (43 breaths *per min*) was observed, and the rectal temperature measured 39.60 °C. The veterinarian of the animal shelter collected blood samples, and intra-venous lactated Ringer's solution (Iran Injectable and Pharmaceutical Products Co., Tehran, Iran) was administered.



Fig. 1. Appearance of the referred dog in **A)** dorsal and **B)** lateral views. The dog exhibited marked obesity and was non-ambulatory.

The patient was scheduled for referral to a veterinary hospital for advanced diagnostic imaging. However, prior to departure from the shelter, the dog experienced sudden death. A postmortem necropsy through ventral midline incision was immediately performed to investigate the cause of death. Significant yellow fat accumulation severely

hindered access to the abdominal cavity. Upon entering the cavity, extensive liver tissue damage caused by severe fat accumulation was evident. Due to the observed abnormalities in the liver and kidneys, tissue samples were collected from both organs (Fig. 2). These samples were preserved in 10.00% buffered formalin solution and sent to a histopathological laboratory for further evaluation.

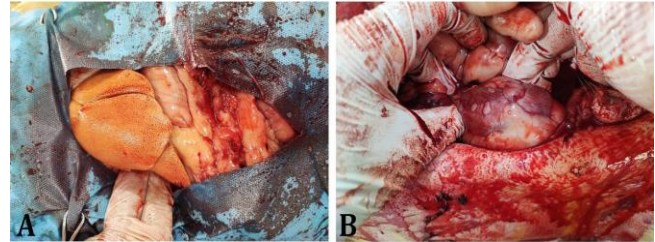


Fig. 2. Gross necropsy findings in a dog. **A)** The liver was markedly enlarged and icteric, occupying a substantial portion of the abdominal cavity, and **B)** The kidney appeared abnormally large, with prominent nodular lesions on its surface.

A complete blood cell count was conducted using an automated hematology analyzer (Celltac α MEK-6510; Nihon Khoden, Tokyo, Japan), and biochemical parameters, including triglycerides, cholesterol, aspartate transferase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP) were measured using a chemistry auto analyzer (BS-480; Mindray, Nanchang, China). The complete blood cell counts revealed leukocytosis ($49.90 \times 10^3 \mu\text{L}^{-1}$), thrombocytopenia ($101 \times 10^3 \mu\text{L}^{-1}$), and an increase in red cell distribution width (14.90%). Biochemical analysis indicated hypertriglyceridemia (455 mg dL^{-1}) and elevated levels of ALT, AST, and ALP (Table 1).

Table 1. Complete blood cell count and biochemical values of triglycerides, cholesterol, AST, ALT, and ALP of the dog.

Parameters	Values
White blood cells ($\times 10^3 \mu\text{L}^{-1}$)	49.90
Red blood cell ($\times 10^6 \mu\text{L}^{-1}$)	5.61
Hemoglobin (g dL^{-1})	12.30
Hematocrit (%)	37.00
Mean corpuscular volume (fL)	66.00
Mean corpuscular hemoglobin (pg)	21.90
Mean corpuscular hemoglobin concentration (g dL^{-1})	33.20
Platelet ($\times 10^3 \mu\text{L}^{-1}$)	101
Red cell distribution width (%)	14.90
Triglycerides (mg dL^{-1})	455
Cholesterol (mg dL^{-1})	87.00
Aspartate transferase (U L^{-1})	291
Alanine transaminase (U L^{-1})	337
Alkaline phosphatase (IU L^{-1})	2,663

Figure 3 depicts blood smears from the patient. Intense leukocytosis with neutrophilia, accompanied by the presence of band cells and metamyelocytes, indicated a moderate regenerative left shift. Also, activated monocytes with a moderate regenerative left shift suggested a degenerative infectious disease. Leuko-erythroblastic changes were evidenced by the presence of nucleated red

blood cells, reticulocytes, band cells, and metamyelocytes. This simultaneous erythroid, myeloid, and neutrophilic left shift reflected bone marrow stimulation, being typically associated with acute anemia, severe infections, or malignancies, indicating a poor prognosis. Furthermore, anisocytosis was evident in the blood smear, along with the presence of target cells and elliptocytes.

Histopathological analysis revealed severe fatty degeneration in the liver, along with significant degenerative and necrotic changes, glomerulonephritis, hyperemia, and hemorrhage in the kidneys. Figures 4 and 5 illustrate the histopathological examination of tissue samples obtained from the patient.

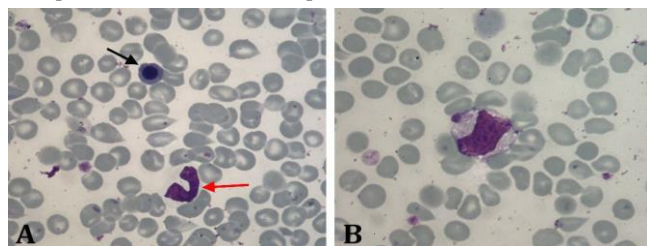


Fig. 3. Hematological assessment of the referred dog. **A)** Black arrow indicates a nucleated red blood cell and red arrow shows a band neutrophil, **B)** A monocyte is obvious in the center of the slide. In the whole slide anisocytosis can be observed (Giemsa staining, 400 \times).

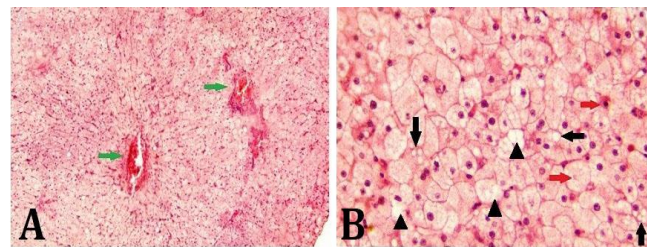


Fig. 4. Photomicrographs of the liver tissue. **A)** Normal parenchyma of liver was replaced with fatty vacuoles and severe fatty degeneration is seen in the liver. Central veins (green arrows) and sinusoidal spaces are congested (Hematoxylin and Eosin staining, 100 \times), **B)** Fatty vacuoles (black arrows) are seen. Red arrows show hepatocytes that have undergone necrosis due to the fatty degenerative changes. Hepatocyte nuclei are pyknotic or lysed. Arrowheads indicate hepatocellular ballooning lesions (Hematoxylin and Eosin staining, 400 \times).

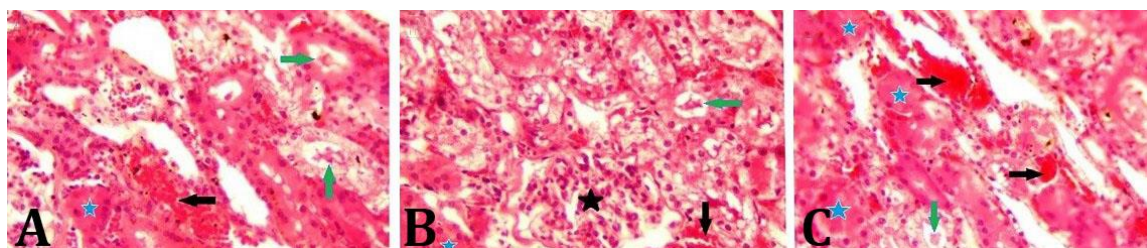


Fig. 5. Photomicrographs of the kidney tissue. Severe degenerative and necrotic changes are evident in the kidney tissue. Lesions of glomeruli include glomerular capillaries swelling being attached to the Bowman's capsule (black asterisk) and severe tubular lesions indicating glomerulonephritis. Hyperemia and hemorrhage are seen in the interstitial tissue in the kidney parenchyma (black arrows). Also, severe degenerative lesions in tubules, such as swelling of proximal tubules (blue asterisks), hydropic degeneration, and tubular necrosis are seen. The casts (green arrows) are seen in many tubules (Hematoxylin and Eosin staining; 400 \times).

Discussion

Hepatic lipidosis is rarely reported in dogs. It is primarily caused by improper feeding and care practices. Triacylglycerols accumulate in hepatocytes due to various toxic factors and carbohydrate deficiencies in the diet. One key mechanism of lipidosis, often associated with hyperlipidemia, involves increased triacylglycerol buildup coupled with a reduced rate of clearance. Fatty liver dystrophy in dogs is characterized by non-specific clinical symptoms, requiring laboratory and diagnostic imaging methods for accurate detection.⁴

Zemlyanski has studied biochemical parameters in 39 dogs with hepatic lipidosis, revealing significant increases in triglycerides, cholesterol, AST, and ALT levels compared to the healthy controls.⁴ These findings highlight marked disruptions in lipid metabolism and liver function associated with the condition. In our case, the cholesterol level was lower than that reported by Zemlyanski. However, the levels of triglycerides, AST, and ALT were respectively 10.00, 4.10, and 4.25 times higher than those reported in Zemlyanski's study. These findings may indicate a more severe form of hepatic lipidosis in the present case. Additionally, in line with Zemlyanski's study, anemia, leukocytosis, and the presence of band neutrophils were observed in our case.

Hunt *et al.*, conducted a study on the evaluation of hepatic steatosis in dogs with congenital portosystemic shunts and found that all dogs exhibited serum biochemical indicators of liver dysfunction, characterized by elevated levels of liver enzymes, such as ALT and ALP, along with decreased concentrations of key hepatic metabolites, including serum urea nitrogen, albumin, and cholesterol. In the mentioned study, triglyceride levels in all studied dogs were found to be within the normal range.¹⁵ Consistent with the findings of Hunt *et al.*, in the present case, liver enzymes were higher than the normal range, and the cholesterol level was lower than the normal range; however, in contrast to the results of the mentioned study, the triglyceride level in the present study was significantly elevated.¹⁵

Armstrong and Blanchard, have reported that cats affected by hepatic lipidosis often show non-regenerative anemia and mild leukocytosis. The most notable laboratory abnormality in these cases was a significant elevation in serum ALP, while increases in ALT and AST were generally less pronounced. Additionally, the study highlighted hypertriglyceridemia as a common finding, occasionally accompanied by hypercholesterolemia.¹⁶ Valtolina and Favier, have found that cats with hepatic lipidosis typically exhibit elevated serum ALP and ALT levels, along with hematological evidence of non-regenerative anemia and mild leukocytosis. Their study also noted that thrombocytopenia is uncommon in cats with this condition.¹⁷ The findings of the present study largely align with those described in these two reports, but some notable differences were observed. In the current case, leukocytosis was more severe, and the levels of ALP, ALT, and AST showed more significant alterations than expected. Hypertriglyceridemia was markedly more severe, and contrary to the findings of the aforementioned studies, thrombocytopenia was observed in this case.

Armstrong and Blanchard, investigated histopathological changes following hepatic lipidosis in cats and found widespread mixed micro-vesicular and macro-vesicular lipidosis.¹⁶ Also, they observed that steatosis was predominantly macrovesicular, characterized by lipid-filled vacuoles exceeding the size of hepatocyte nuclei, often displacing them. Intra-hepatic cholestasis was frequently observed, identified by the presence of bile as fine linear deposits between hepatocytes. Another study conducted by Webb, on hepatic lipidosis in cats reported diffuse lobular alterations with over 50.00% of hepatocytes containing cytoplasmic lipid-filled vacuoles.¹⁴ Furthermore, hepatic lipidosis leads to intra-hepatic cholestasis, presenting structural changes being distinct from those associated with extra-hepatic cholestasis caused by bile duct obstruction. Arunorat *et al.*, have evaluated liver damage following hepatic lipidosis in dogs and indicated hepatocyte fatty degeneration at periportal areas alongside with infiltration of mixed inflammatory cells.¹⁸ In agreement with previous studies, in this report, normal parenchyma of liver was replaced with fatty vacuoles, and severe fatty degeneration was observed in the liver. Additionally, central veins and sinusoidal spaces were congested. In the present case, histopathological assessment of kidney revealed severe degenerative and necrotic changes and glomerulonephritis.

Fluid and electrolyte therapy, enteral feeding, food regimen alterations, anti-emetic therapy, cobalamin therapy, treatment of coagulation disorders, and treatment of hepatic encephalopathy are available treatment options in cats and dogs suffering from hepatic lipidosis.¹⁶ The prognosis of hepatic lipidosis in cats is significantly influenced by the presence of concurrent conditions. Cats with idiopathic hepatic lipidosis generally have better

survival rates (50.00%) compared to those with conditions, like acute pancreatitis (20.00%). Rapid and aggressive nutritional intervention improves survival outcomes. Notably, recurrence of hepatic lipidosis in cats that recover appears to be rare.¹⁴ Data regarding dogs with hepatic lipidosis are limited. However, in the present case, severe form of the disease, very late referral, and renal involvement led to death before performing any treatments.

In this case, the concurrent presence of hepatic lipidosis and renal lesions suggests a potential metabolic link between the liver and kidneys. Hepatic lipidosis, marked by excessive lipid accumulation in hepatocytes, may reflect systemic lipid dysregulation, which has been implicated in kidney injury through mechanisms, such as glomerulosclerosis and tubulointerstitial fibrosis.¹⁹ Additionally, nephrotic syndrome and other renal disorders can cause significant alterations in lipid metabolism, potentially contributing to hepatic dysfunction.²⁰ This bidirectional relationship indicates that lipid metabolic disturbances could simultaneously or sequentially affect both organs. While a direct causal relationship remains to be confirmed, our findings support the need for further investigation into the shared metabolic and pathological pathways influencing liver and kidney health in such cases.

Due to limited access to magnetic resonance imaging, computed tomography scans, and advanced biochemical tests for some veterinarians, as well as the challenges in diagnosing this rare disease in dogs, Vali *et al.*, conducted a study on the diagnosis of hepatic lipidosis in dogs using ultrasonography.²¹ By analyzing the liver's mean gray levels, they observed a significant difference between healthy livers and those affected by hepatic lipidosis. It is hoped that the implementation of such novel diagnostic methods will help prevent delays in treatment of affected dogs.²¹

This case report highlights the severe and rapidly progressing nature of hepatic lipidosis in dogs, emphasizing the importance of early diagnosis and intervention. The advanced stage of the disease, compounded by concurrent renal involvement, underscores the critical need for prompt and comprehensive medical attention to improve outcomes. The findings contribute to the limited data on canine hepatic lipidosis and underscore the necessity of raising awareness about its clinical presentation, diagnostic markers, and histopathological characteristics. Future studies are essential to further explore effective preventive and therapeutic strategies for managing this condition in dogs.

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Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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