Archives of Current Research International

Volume 25, Issue 7, Page 307-316, 2025; Article no.ACRI.139306
ISSN: 2454-7077

Serum Biochemical Alterations in Dogs Affected by Hepatic Dysfunction in Jaipur, Rajasthan, India

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: https://doi.org/10.9734/acri/2025/v25i71335

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://pr.sdiarticle5.com/review-history/139306

Original Research Article

Received: 28/04/2025
Accepted: 30/06/2025
Published: 07/07/2025

ABSTRACT

Hepatic dysfunction in dogs manifests through a variety of clinical and subclinical signs, many of which are reflected in changes in serum biochemical parameters. The liver plays a pivotal role in metabolism, detoxification, protein synthesis, and regulation of various biochemical processes. Alterations in serum markers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), bilirubin, bile acids, urea, cholesterol, and serum proteins serve as essential indicators for diagnosing and monitoring liver diseases. The present study was aimed to assess biochemical changes in dogs with hepatic

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Cite as: *Pathan, Nabeel Khan, Nazeer Mohammed, Rashmi Singh, Pradeep Kumar, Dharm Singh Meena, and Harshita Rathore. 2025. "Serum Biochemical Alterations in Dogs Affected by Hepatic Dysfunction in Jaipur, Rajasthan, India". Archives of Current Research International 25 (7):307-16. https://doi.org/10.9734/acri/2025/v25i71335.*

dysfunction. During the period from August 2024 to January 2025, a total of 200 dogs with suspected hepatic dysfunction on the basis of clinical signs were screened in this study from Jaipur, Rajasthan, India. Serum Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP), Gamma-Glutamyl Transferase (GGT), total bilirubin, blood glucose and total protein were measured on an automated biochemical analyzer. Among 200 suspected dogs, twelve (12) were diagnosed with hepatic dysfunction which was compared with ten (10) healthy control dogs. Relevant biochemical changes found in these dogs were increased activities of ALT, AST, ALP, GGT and total bilirubin, with reduction in the levels of total protein and glucose. These observations indicate that biochemical profiling is a useful method for diagnosing and monitoring hepatic dysfunction in dogs.

Keywords: Canine liver disease; serum biochemistry; ALT; liver dysfunction; veterinary diagnostics dogs; hepatic dysfunction; biochemical parameters; diagnosing.

1. INTRODUCTION

Hepatic dysfunction in dogs represents a significant challenge in veterinary medicine due to the organ's complex and multifaceted role in maintaining systemic homeostasis. The liver is central to a wide range of physiological functions including metabolism of carbohydrates, lipids, and proteins; storage of vitamins and minerals; detoxification of drugs and endogenous toxins; synthesis of plasma proteins; and regulation of immune responses (Assawarachan *et al.,* 2021; Chapman & Hostutler, 2013; Vijayakumar *et al.,* 2008). Because of its strategic position between the gastrointestinal tract and systemic circulation, the liver is exposed to a multitude of substances, making it particularly vulnerable to various forms of injury, both acute and chronic.

Liver diseases in dogs can be classified as congenital or acquired and may manifest as hepatocellular injury, cholestasis, or vascular abnormalities such as portosystemic shunts. Common causes include infectious agents (e.g., leptospirosis, canine adenovirus), toxins (e.g., aflatoxins, certain drugs), neoplasia, autoimmune conditions, and metabolic or genetic disorders (Chaturvedi *et al.,* 2013; Das & Lodh, 2024; Watson, 2004). Clinical signs are often non-specific in the early stages and may include lethargy, anorexia, vomiting, diarrhea, jaundice, or ascites. Hence, early and accurate diagnosis is critical for effective treatment and prognosis.

Serum biochemistry serves as a fundamental diagnostic tool for the evaluation of liver function and integrity. Alterations in liver-specific enzymes, such as alanine aminotransferase (ALT), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT), along with non-specific indicators like bilirubin, urea, cholesterol, and serum proteins, can provide essential clues regarding the presence, type, and extent of hepatic damage (Dixit *et al.,* 2010; Elhiblu *et al.,* 2015; Harvey *et al.,* 2022). For example, increased ALT and AST levels are often associated with hepatocellular injury, while elevated ALP and GGT are indicative of cholestasis or bile duct obstruction. Furthermore, alterations in protein synthesis and metabolic markers such as hypoalbuminemia and hypocholesterolemia can signify chronic or advanced liver disease.

However, interpreting serum biochemical data is not always straightforward. Many markers are not entirely liver-specific and may be influenced by extrahepatic factors such as age, concurrent diseases, medications, or physiological states like growth and pregnancy. Additionally, the presence of subclinical hepatic disease may go unnoticed without targeted testing. These challenges underscore the importance of understanding the diagnostic value, limitations, and interrelationships of various serum biochemical parameters.

Recent advances in veterinary diagnostics have led to the identification of novel biomarkers and enhanced the accuracy of liver function assessment. Markers such as serum bile acids, ammonia, symmetric dimethylarginine (SDMA), and even specific microRNAs are being investigated for their potential to detect hepatic dysfunction earlier and with greater specificity. Despite these innovations, traditional serum biochemistry remains the first-line diagnostic modality due to its availability, cost-effectiveness, and established clinical relevance (Watson, 2017).

Biochemical profiling is a valuable diagnostic tool for hepatic diseases as serum ALT, AST, ALP, GGT, bilirubin, glucose, total protein and albumin provides important insights into hepatocellular injury, cholestasis and synthetic dysfunction.

However, certain liver diseases are linked to mild enzyme changes, in the absence of accompanying clinical evidence, making the diagnosis even more difficult (Singh *et al.,* 2019). Therefore, careful biochemical examination is still necessary for early diagnosis and monitoring of hepatic dysfunction in dogs.This investigation aims to assess the serum biochemical parameters that show impairments in liver function.

2. REVIEW OF LITERATURE

2.1 Liver Enzymes as Indicators of Hepatic Injury

Liver enzymes are integral to the evaluation of hepatic injury in dogs. These enzymes are broadly classified into two categories: those that indicate hepatocellular injury (e.g., ALT and AST) and those that reflect cholestasis or biliary tract abnormalities (e.g., ALP and GGT). Alterations in the serum activity of these enzymes serve as early and often non-specific markers of liver dysfunction, preceding the development of overt clinical signs or significant histopathologic changes. However, interpreting these enzymes requires a nuanced understanding of their origin, half-life, specificity, and the pathological context in which they are elevated (Westgren *et al.,* 2014, Lakshmi *et al.,* 2023; Webster, 2010; Hirose, *et al.,* 2014).

2.1.1 Alanine aminotransferase (ALT)

ALT is one of the most commonly measured liver enzymes in canine serum biochemistry. It is primarily found in the cytoplasm of hepatocytes and is released into the bloodstream in response to hepatocellular membrane damage. ALT is considered relatively liver-specific in dogs, and elevations usually point to primary liver disease, particularly hepatocellular injury or necrosis (Verma *et al.,* 2024; Thornburg, 2000; Tennant, 1997; Solter *et al.,* 1994).

Elevated ALT levels may be seen in conditions such as chronic hepatitis, toxin exposure (e.g., xylitol, aflatoxin), drug-induced hepatotoxicity (e.g., phenobarbital), hepatic neoplasia, and infectious diseases like leptospirosis or infectious canine hepatitis. ALT can also rise following transient hepatic hypoxia or trauma. However, the magnitude of ALT elevation does not necessarily correlate with the severity or reversibility of hepatic injury. Moreover, ALT has a relatively short half-life (approximately 2–3 days in dogs), making it a useful marker for monitoring the progression or resolution of hepatic inflammation (Sultana *et al.,* 2022; Tantary *et al.,* 2014; Saravanan *et al.,* 2014).

2.1.2 Aspartate aminotransferase (AST)

AST is a mitochondrial enzyme present not only in hepatocytes but also in cardiac and skeletal muscle, red blood cells, and kidneys. As such, AST is less liver-specific than ALT and should always be interpreted alongside other markers. In hepatic disease, AST is typically elevated in more severe or advanced hepatocellular injury, particularly when mitochondrial damage occurs (Rothuizen, 2009; Pandya *et al.,* 2022; Oikonomidis & Milne, 2023; McCord & Webb, 2011).

An isolated increase in AST without concurrent ALT elevation may suggest non-hepatic causes such as muscle injury, strenuous exercise, or intramuscular injections. In contrast, a disproportionate elevation of AST relative to ALT in liver disease may indicate irreversible hepatic injury or necrosis. Evaluation of serum creatine kinase (CK) can help differentiate hepatic from muscular sources of elevated AST.

2.1.3 Alkaline phosphatase (ALP)

ALP is a group of isoenzymes produced by various tissues including liver, bone, intestine, and placenta. In dogs, the liver and bone isoenzymes predominate, but a third isoenzyme, the corticosteroid-induced isoenzyme (C-ALP), is also clinically significant. This form is unique to dogs and is induced by endogenous or exogenous corticosteroids (Lidbury & Suchodolski, 2016; Lawrence & Steiner, 2017; Lathamani & Nalinikumari, 2015).

Liver-associated ALP is predominantly located in the biliary epithelium and canalicular membranes, making it a marker for cholestasis or biliary obstruction. Elevation in ALP is common in hepatobiliary conditions such as gallbladder mucocele, extrahepatic bile duct obstruction, or vacuolar hepatopathy. However, elevated ALP is not always pathological—it can rise in young, growing dogs due to bone isoenzyme activity or in animals on corticosteroid therapy due to C-ALP induction (Lakshmi *et al.,* 2018; Kumar *et al.,* 2012; Kozat & Sepehrizadeh, 2017).

Given its relatively long half-life (approximately 3 days) and its responsiveness to non-hepatic stimuli, ALP should be interpreted in conjunction with other liver enzymes and imaging findings. A

significant elevation of ALP without a corresponding increase in GGT may suggest steroid hepatopathy or idiopathic vacuolar changes rather than cholestatic disease.

2.1.4 Gamma-glutamyl transferase (GGT)

GGT is a membrane-bound enzyme primarily located in the biliary epithelium. Its serum activity increases in response to biliary stasis or ductal cell proliferation. Unlike ALP, GGT is less influenced by corticosteroids or age in dogs, making it a more specific indicator of cholestatic liver disease. GGT is particularly useful in differentiating causes of increased ALP. For example, concurrent elevation in both ALP and GGT strongly suggests cholestatic or obstructive liver disease. In contrast, a high ALP with normal GGT often points to non-cholestatic causes such as vacuolar hepatopathy or corticosteroid influence. Marked elevations in GGT can also occur in hepatic neoplasia, gallbladder disease (e.g., gallbladder mucocele), and chronic hepatitis with ductular reaction. In cases of hepatic lipidosis, GGT may be less elevated compared to ALP, which can aid in distinguishing this condition from others (Lakshmi & Padmaja, 2021; Koenig, 2009; Johnson *et al.,* 2004; Jaffey, 2022; Kearns, 2009).

2.2 Bilirubin and Bile Acids in Hepatic Function Evaluation

2.2.1 Total and conjugated bilirubin

Hyperbilirubinemia in dogs may result from pre-hepatic (hemolysis), hepatic (hepatocellular damage), or post-hepatic (biliary obstruction) causes. Measurement of total and direct (conjugated) bilirubin helps localize the pathology (Bexfield & Watson, 2006; Brovida & Rothuizen, 2010; Chapman & Hostutler, 2013; Chaturvedi *et al.,* 2013).

2.2.2 Serum bile acids

Bile acid testing is a sensitive indicator of hepatic function, especially in detecting portosystemic shunts and hepatic insufficiency. Increased pre- and postprandial bile acid levels suggest decreased hepatic clearance or enterohepatic circulation dysfunction (Das & Lodh, 2024; Dixit, *et al.,* 2010; Kanemoto *et al.,* 2011).

2.3 Protein and Albumin Alterations

The liver synthesizes most plasma proteins, including albumin and clotting factors. Hypoalbuminemia is a hallmark of chronic liver disease due to reduced hepatic synthesis. Globulin levels may be elevated due to reactive hepatitis or inflammation. The albumin-to-globulin ratio can help differentiate hepatic insufficiency from other causes of hypoalbuminemia such as protein-losing enteropathies or nephropathies (Elhiblu *et al.,* 2015; Favier, 2009; Harvey *et al.,* 2022; Hassan *et al.,* 2022).

3. MATERIALS AND METHODS

A total of 200 dogs suspected of hepatic dysfunction exhibited clinical signs such as inappetence or anorexia, vomiting, diarrhea or constipation, icterus, ascites, pyrexia, lethargy, melena, polyuria, polydipsia, weight gain, weight loss, or various combinations thereof were screened during the period of August 2024 to January 2025 at the Veterinary Clinical Complex (VCC) of PGIVER and Government Veterinary Polyclinic Hospital, Panchbatti, Jaipur, Rajasthan. Additionally, 10 clinically healthy dogs were included as the control group for comparative evaluation. In all animals, 7 ml of venous blood was collected aseptically from the cephalic or saphenous vein out of which 5 ml was transferred into plain vials, allowed to clot and centrifuged at 2500 rpm for 30 minutes to obtain serum. Biochemical parameters, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total bilirubin, total protein and glucose were estimated using commercially available diagnostic kits (CPC Diagnostics) on an automated blood biochemistry analyzer. ALT, AST, ALP, and GGT activities were determined by IFCC methodology. Total bilirubin was measured by the Sulfanilic Diazotization (SDA) method, glucose by the GOD-PAP method and total protein by the direct biuret method. Statistical analysis was performed using standard methods (Snedecor & Cochran, 2004).

4. RESULTS AND DISCUSSION

Among the 200 dogs, 12 were diagnosed with hepatic dysfunction based on clinical signs and biochemical evaluation. The biochemical parameters assessed in these dogs revealed significant deviations from those of healthy controls indicating varying degrees of hepatocellular injury, cholestasis, metabolic disturbances and impaired hepatic synthetic function in the dogs. These alterations in

parameters as compared to healthy controls are listed in Table 1 and Fig. 1.

The mean ± SE value of alanine aminotransferase (ALT) in the hepatic dysfunction group (162.16 ± 5.61 IU/L) was significantly ($p<0.01$) higher in comparison to healthy controls (41.67 ± 2.24 IU/L). ALT is a cytosolic enzyme predominantly found in hepatocytes and its elevated levels of activity suggest the hepatocellular damage due to irreversible necrosis or reversible injury resulting in cytoplasmic leakage or blebbing. Common contributing factors includes inflammation, hypoxia, toxins, drugs and neoplasia, the level of serum elevation may be roughly proportional to the severity of the disease and the amount of liver mass affected. An underlying abnormality may be present even if there is no elevated enzyme activity due to a quantitative decrease in hepatocytes (for example, advanced cirrhosis or portosystemic shunting with atrophy) (Lawrence and Steiner, 2017; Chapman& Hostutler, 2013). These findings align with those of Tantary *et al.,* (2014) and Pandya *et al.,* (2022), who observed similar elevations in ALT levels in dogs with hepatic disorders. Similarly, aspartate aminotransferase (AST), a leakage enzyme presents in both cytoplasm and mitochondria showed a significant ($p<0.01$) increase in affected dogs (148.87 ± 7.14 IU/L) as compared to healthy controls (38.36 ± 2.90 IU/L). Although AST is found in a variety of tissues, the liver, skeletal muscles, and heart muscles exhibit the highest levels of activity. Furthermore, since erythrocytes contain a significant amount of AST, hemolysis, whether in vitro or in vivo, is anticipated to raise serum AST activity. However, there is some debate regarding the half-life of AST in dogs, it is most likely between 12 and 22 hours. According to reports, the half-life of AST in cats is 1.5 hours. During hepatocellular injury, serum AST activity rises and typically coincides with an increase in serum ALT activity. However, because AST has a shorter half-life than ALT, the increase is usually milder and may normalize earlier (Oikonomidis & Milne, 2023). Elevated AST levels are a hallmark of both acute and chronic hepatocellular injury and necrosis (Webster, 2010). These results are consistent with findings of Lakshmi & Padmaja, (2021) and Verma *et al.,* (2024), highlighting AST as a useful biomarker in hepatobiliary disorders.

In addition to hepatocellular injury, cholestasis indicators were also significantly altered. Alkaline phosphatase (ALP) activity was markedly elevated ($p<0.01$) in affected dogs (301.28 ± 29.27 IU/L) in comparison to healthy controls (76.89 ± 5.46 IU/L). For the majority of mammalian species, including dogs, elevated serum alkaline phosphatase (ALP) activity is regarded as a sensitive indicator of cholestasis. An elevated level of ALP is an indicative of both acute and chronic liver diseases, with significantly higher elevations indicating cholestasis. The most marked increases are generally seen in cases of cholangitis, biliary cirrhosis, or obstruction of the extrahepatic bile ducts, glucocorticoid therapy can also result in high activity of serum ALP (Tennant and Center, 2008; Solter*et al.,* 1994). Similar findings were also recorded by Elhiblu *et al.,* (2015) and Das & Lodh (2024). Gamma-glutamyl transferase (GGT) a sensitive marker of cholestasis and hepatic oxidative stresswas also significantly increased ($p<0.01$) in affected dogs (17.72 ± 2.74 IU/L) as compared to healthy controls (5.19 ± 0.40 IU/L). Dogs may have comparatively lower hepatic GGT than some other domestic animals, which could explain their low serum GGT activity (0–10 IU/L). Although measuring a dog's serum GGT can help distinguish between different alkaline phosphatase sources (Shull & Hornbuckle,1979). Its elevation may be linked to endocrine disorders, neoplasia, benign nodular hyperplasia, drug-induced hepatic changes or idiopathic breed predispositions (Alvarez & Whittemore, 2009). These results are corroborated with findings of Bhadesiya *et al.,* (2015) and Assawarachan *et al.,* (2021).

There was significant elevation ($p<0.05$) of total bilirubin concentration was observed in dogs with hepatic dysfunction (1.20 ± 0.38 mg/dL) as compared to healthy controls (0.20 ± 0.04 mg/dL). There are three possible causes of hyperbilirubinemia: pre- hepatic and post-hepatic. Increased bilirubin production, usually brought on by hemolysis or acute internal hemorrhage is the cause of pre-hepatic causes. The absence of severe anaemia, internal bleeding, and alterations in the hepatic enzymology ruled out these causes. Hepatic reasons of hyperbilirubinemiainclude impaired excretion of bilirubin due to hepatocellular damage or biliary obstruction, indicating decreased hepatic excretory function or impaired bile flow (Vijayakumar *et al.,* 2008; Harvey *et al.,* 2022). The finding of increased total bilirubin was also reported by Saravanan *et al.,* (2014) and Verma *et al.,* (2024).

Table 1. Mean ± SE values of biochemical parameters in healthy control and dogs affected with hepatic dysfunction

S. No	parameter	Healthy Control group (n=10)	Dogs affected with hepatic dysfunction (n=12)
1	ALT (IU/L)(**)	41.67±2.24	162.16±5.61
2	AST (IU/L) (**)	38.36±2.90	148.87±7.14
3	ALP(IU/L)(**)	76.89±5.46	301.28±29.27
4	GGT(IU/L) (**)	5.19±0.40	17.72±2.74
5	Total Protein (g/dl) (**)	6.26±0.18	4.64±0.38
6	Total bilirubin (mg/dl) (*)	0.20±0.04	1.20±0.38
7	Glucose (mg/dl) (**)	95.11±3.39	64.16±6.48

** The variations in mean value were significant (p<0.05) when compared with the mean value of healthy control group.*

*** The variations in mean value were highly significant (p<0.01) when compared with the mean value of healthy control group.*

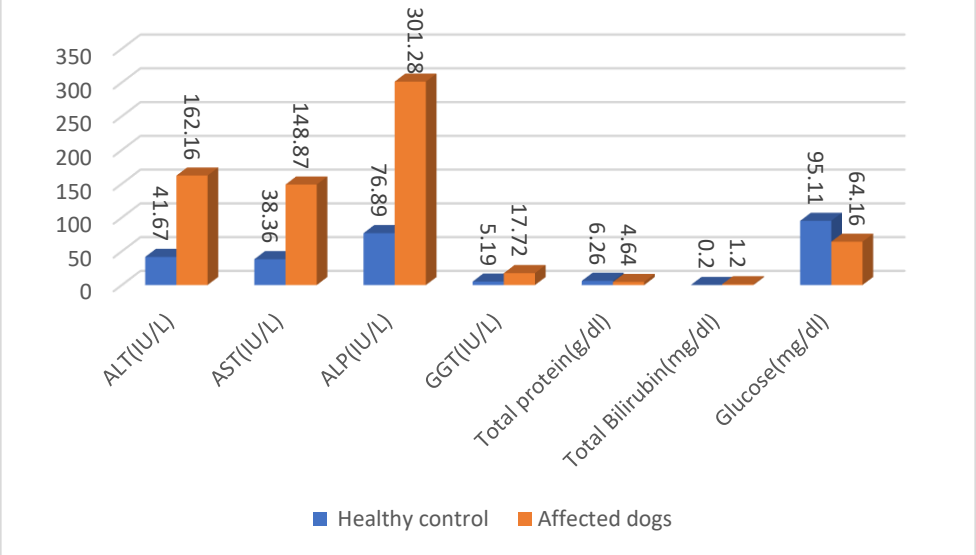

Fig. 1. Mean values of biochemical parameters in healthy control and hepatic dysfunction affected canines

In current study metabolic disturbances were evident as blood glucose levels were significantly decreased ($p<0.01$) in affected dogs (64.16 ± 6.48 mg/dL) in comparison to controls (95.11 ± 3.39 mg/dL).Three sources provide the body with glucose: are intestinal absorption from the breakdown of carbohydrates, the breakdown of glycogen (the form of glucose that is stored) through glycogenolysis, which occurs primarily in the liver but also in the muscle; and the synthesis of glucose (gluconeogenesis), which is primarily carried out by the liver from non-carbohydrate sources such as lactate, pyruvate, amino acids and glycerol, but also significantly by the kidneys (Koenig, 2009). Hypoglycemia in hepatic disease is attributed to loss of hepatic functional mass due to conditions like portosystemic shunts or acute and chronic liver failure. The liver diseases also prevent the breakdown of insulin, and this further exacerbates the hypoglycaemia (Schoeman, 2012). These observations agree with findings by Lathamani & Nalinikumari, (2015) and Hassan *et al.,* (2022).

Finally, total protein concentration was significantly reduced ($p<0.01$) in diseased dogs (4.64 ± 0.38 g/dL) compared to controls (6.26 ± 0.18 g/dL). The liver detoxifies ammonia, the main byproduct of protein metabolism, and produces a variety of plasma proteins. Disturbances in hepatic protein metabolism can lead to decreased production of protein, as a clinical consequence of liver diseases that cause portal hypertension. But even in cases of severe liver dysfunction, the liver's ability to produce

proteins is only slightly impacted because of its high degree of plasticity (Brovida & Rothuizen, 2010; Rothuizen, 2009). Hypoproteinaemia is commonly seen in chronic hepatic conditions such as cirrhosis or portosystemic shunting due to diminished hepatic synthetic capacity because of compromised hepatic synthetic function. Albumin, serum protein made exclusively in the liver, act as a sensitive marker of chronic liver disease (Tennant, 1997). Similar results have been reported by Chaturvedi *et al.,* (2013) and Lakshmi *et al.,* (2018).

5. CONCLUSION

The study demonstrates that liver dysfunction in the dogs results in marked biochemical alterations. These are elevated levels of ALT, AST, ALP, GGT and total bilirubin, while glucose and total protein levels are decreased. These alterations reflect the damage of liver cells, cholestasis and impaired liver functions. It is suggested that biochemical profiling is an effective approach for the early diagnosis, monitoring, and treatment of liver diseases in dogs.

6. STUDY LIMITATIONS

1. The small cohort size (12 vs. 10) limits the statistical power of the study and increases the likelihood of both false-positive and false-negative results.
2. Ultrasound and biopsy were not utilized in this study. Although diagnostic imaging and histopathological methods are commonly employed to assess hepatic conditions, this study focused solely on hepatic dysfunction without incorporating such tools.
3. The study was conducted within a limited geographic area and involved a small sample size. Therefore, the findings may not be generalizable. A larger, more geographically diverse sample could yield more reliable and representative results.

ETHICAL APPROVAL

Animal Ethic committee approval has been collected and preserved by the author(s)

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

ACKNOWLEDGMENT

The author is highly thankful to Major Advisor, Dean and Head, Department of Veterinary Medicine, postgraduate institute of veterinary education and research (PGIVER), Jaipur for their valuable guidance, motivation and support rendered during this study. The authors would also like to thank the faculty and staff of the Veterinary Clinical Complex for their cooperation and help.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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