



ISSN (E): 2277- 7695

ISSN (P): 2349-8242

NAAS Rating: 5.03

TPI 2021; 10(1): 24-26

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Received: 19-11-2020

Accepted: 21-12-2020

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## Biochemical evaluation of canine hepatic disorders

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### Abstract

A total of 88 dogs were diagnosed with hepatic disorders based on clinico, hemato-biochemical and diagnostic imaging. Out of which, 32 dogs were diagnosed with diffuse parenchymal disorders with ascites, 32 dogs with diffuse parenchymal disorders without ascites, and 24 with focal parenchymal disorders. Elevated activity of ALT, AST, ALP, GGT, total bilirubin, direct bilirubin and globulin with decreased levels of total protein, albumin, glucose and serum electrolytes (sodium, potassium and chloride) were common biochemical findings recorded in all hepatic disorders affected dogs.

**Keywords:** hepatic disorders, dogs, serum biochemistry

### Introduction

Laboratory investigation of hepatic disorders in dogs is frequently necessary to rule hepatic disease in or out, to assess the functional impact on the liver, and to decide whether hepatic disease is the primary problem or a complication of something else. The selection and interpretation of laboratory tests to resolve these problems is based on an understanding of relevant functional anatomy and pathophysiology. The hepatocellular pattern of disease is characterized by increases in leakage enzymes such as AST, GLDH, and ALT and the cholestatic pattern by increases in induced enzymes (ALP and GGT).. For this reason, the primary biochemical data base for ruling hepatobiliary disease in or out always should involve some screening tests of hepatic function, such as albumin, protein, bilirubin, glucose etc. As the liver is physiologically and anatomically diverse, a battery of tests that adequately identifies hepatic disease or its underlying cause must be undertaken (Kumar *et al.* 2013) [3].

### Materials and Methods

Dogs presented with the clinical signs of anorexia, ascites, jaundice, pale mucous membranes, vomiting, lethargy, polyuria and polydipsia and other manifestations suggestive of hepatic disorders were selected for the present study.

Blood was collected from the peripheral (cephalic/saphenous) veins of dogs suffering with hepatic disorders in dogs using into sterile clean vaccutainers containing clot activator for serum separation and was centrifuged at 3000rpm for 15 minutes and the serum collected was transferred into eppendorf tubes and stored at -20o C for estimation of biochemical parameters. Serum samples were analyzed using star21 plus semi-automatic biochemical analyzer and commercially available diagnostic kits supplied by M/S Rapid diagnostics Pvt Ltd, New Delhi. Further, blood and serum was collected from apparently healthy dogs to obtain normal values.

### Results and Discussion

The mean levels of alanine amino transferase, aspartate amino transferase, alkaline phosphatase, gamma glutamyl transferase, total bilirubin, direct bilirubin, total protein, albumin, globulins, glucose, sodium, potassium and chloride in healthy control dogs were 32.80±1.60 U/L, 43.05±1.22 U/L, 59.62 ±2.08 U/L, 3.04±0.15 U/L, 0.45±0.03 mg/dl, 0.19±0.02 mg/dl, 6.22± 0.03 g/dl, 2.81±0.07 g/dl, 3.41±0.07 g/dl, 108.24±1.87 mg/dl, 142.16±0.38 mEq/L, 4.05±0.35mEq/L and 103.12±0.35 mEq/L, respectively.

The mean alanine amino transferase (ALT) activity on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, and focal parenchymal disorders were 203.19±19.16, 197.58± 34.72 and 109.76±6.79 respectively. Increased levels were significant ( $P<0.01$ ) in diffuse parenchymal disorders with ascites and ( $P<0.05$ ) in diffuse parenchymal disorders without ascites and focal parenchymal disorders as compared with healthy control.

ALT and AST are indicative of altered hepatocellular membrane permeability, hepatocellular necrosis and inflammation with degree proportional to number of injured hepatocytes (Kramer and Hoffman, 1997) [2].

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The mean aspartate amino transferase (AST) activity on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites and focal parenchymal disorders were  $184.84 \pm 22.32$ ,  $154.18 \pm 19.61$  and  $141.10 \pm 10.31$  respectively. Serum concentrations of ALT and AST are the most commonly measured markers of hepatocellular leakage in dogs (Lidburg and Steiner, 2013)<sup>[4]</sup>.

The mean alkaline phosphatase (ALP) activity on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders were  $284.82 \pm 18.39$ ,  $251.98 \pm 20.73$  and  $85.72 \pm 2.84$  respectively. A significant ( $P < 0.01$ ) increase in this parameter was noticed in diffuse parenchymal disorders with ascites and a significant increase ( $P < 0.05$ ) among the dogs affected with diffuse parenchymal disorders without ascites and focal parenchymal disorders as compared with healthy control. The mean gamma glutamyl transferase (GGT) activity on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders were  $6.64 \pm 0.51$ ,  $6.70 \pm 0.57$  and  $4.74 \pm 0.18$  respectively. These values were significantly ( $P < 0.05$ ) elevated in diffuse parenchymal disorders with ascites, without ascites and focal parenchymal disorders as compared with healthy control. Elevation in the serum ALP levels along with GGT was seen with the administration of drugs like corticosteroids. (Meyer, 2013)<sup>[13]</sup> And was the most common among cholestatic disorders.

The mean total bilirubin and direct bilirubin levels were none significantly elevated in diffuse parenchymal disorders with ascites, without ascites and focal parenchymal disorders. Total bilirubin concentrations indicate the hepatic ability for uptake, conjugation and excretion of the bilirubin (Sevelius and Jonsson, 1995)<sup>[8]</sup>.

The mean total protein levels on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders were  $4.77 \pm 0.10$ ,  $5.45 \pm 0.09$ ,  $5.32 \pm 0.12$  respectively. These values were significantly decreased in diffuse parenchymal disorders with ascites ( $P < 0.01$ ) and ( $P < 0.05$ ) in diffuse parenchymal disorders without ascites,

focal parenchymal disorders as compared with healthy control. The mean albumin levels on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders and biliary tract disorders were  $2.01 \pm 0.03$ ,  $2.34 \pm 0.09$ ,  $2.68 \pm 0.09$  and  $2.35 \pm 0.07$  g/dl, respectively. A significantly ( $P < 0.01$ ) lower levels were noticed in diffuse parenchymal disorders with ascites and a significant decrease ( $P < 0.05$ ) in diffuse parenchymal disorders without ascites and biliary tract disorders. The reduction was non-significant observed in focal parenchymal disorders as compared with healthy control. The mean globulin levels on day zero in dogs with diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders were  $3.43 \pm 0.01$ ,  $3.44 \pm 0.09$ ,  $3.65 \pm 0.14$  respectively. A significantly ( $P < 0.01$ ) elevated levels were recorded among focal parenchymal disorders and a significant ( $P < 0.05$ ) increase in diffuse parenchymal disorders with ascites and without ascites was seen as compared with healthy control. These findings of the present investigation corroborated well with the findings of James and Pillai (2011)<sup>[1]</sup>, who observed hypoproteinemia in dogs affected with hepatic cirrhosis. Most of the plasma proteins were synthesized and catabolised by the liver and form a vulnerable indicator of compromised hepatic function.

The mean glucose values were significantly ( $P < 0.01$ ) decreased in all the groups of hepatobiliary disorders when compared with healthy control. Hypoglycemia was a very specific sign of marked reduction in liver function and in cases of end stage chronic liver failure (Webster, 2010)<sup>[9]</sup>.

There was a non-significant decrease in sodium, potassium and chloride levels among diffuse parenchymal disorders with ascites, without ascites, focal parenchymal disorders and biliary tract disorders when compared with healthy control (Table 1). These findings are in agreement with Ram Prabhu *et al.* (2002)<sup>[6]</sup>, who stated that chronic vomiting will typically cause decreased serum electrolytes in dogs owing to their loss in the vomitus.

**Table 1:** Mean biochemical findings in healthy and hepatic disorders in dogs.

S. No	Parameter	Healthy control	Diffuse parenchymal disorders with ascites	Diffuse parenchymal disorders without ascites	Focal parenchymal disorders
1.	ALT(U/L)	$32.80 \pm 1.60$	$203.19 \pm 19.16^{**}$	$197.58 \pm 34.72^*$	$109.76 \pm 6.79^*$
2.	AST(U/L)	$43.05 \pm 1.22$	$184.84 \pm 22.32^{**}$	$154.18 \pm 19.61^{**}$	$141.10 \pm 10.31^{**}$
3.	ALP(U/L)	$59.62 \pm 2.08$	$284.82 \pm 18.39^{**}$	$251.98 \pm 20.73^*$	$85.72 \pm 2.84^*$
4.	GGT(U/L)	$3.04 \pm 0.15$	$6.64 \pm 0.51^*$	$6.70 \pm 0.57^*$	$4.74 \pm 0.18^*$
5.	TB(m g/dl)	$0.45 \pm 0.03$	$1.17 \pm 0.04$	$1.07 \pm 0.05$	$0.93 \pm 0.06$
6.	DB(mg/dl)	$0.19 \pm 0.02$	$0.75 \pm 0.05$	$0.67 \pm 0.03$	$0.38 \pm 0.03$
7.	Total Protein (g/dl)	$6.22 \pm 0.03$	$4.77 \pm 0.10^{**}$	$5.45 \pm 0.09^*$	$5.32 \pm 0.12^*$
8.	Albumin(g/dl)	$2.81 \pm 0.07$	$2.01 \pm 0.03^{**}$	$2.34 \pm 0.09^*$	$2.68 \pm 0.09$
9.	Globulins(g/dl)	$3.41 \pm 0.07$	$3.43 \pm 0.01^*$	$3.44 \pm 0.09^*$	$3.65 \pm 0.14^{**}$
10.	Glucose (mg/dl)	$108.24 \pm 1.87$	$90.86 \pm 1.39^{**}$	$100.76 \pm 0.21^{**}$	$104.80 \pm 1.20^{**}$
11.	Creatinine (mg/dl)	$1.08 \pm 0.07$	$1.21 \pm 3.03$	$1.16 \pm 0.03$	$0.95 \pm 0.05$
12.	Sodium (mEq/L)	$142.16 \pm 0.38$	$134.77 \pm 1.23$	$138.13 \pm 1.37$	$137.91 \pm 1.18$
13.	Potassium (mEq/L)	$4.05 \pm 0.35$	$3.56 \pm 0.08$	$3.60 \pm 0.08$	$3.69 \pm 0.09$
14.	Chloride (mEq/L)	$103.12 \pm 0.35$	$97.87 \pm 0.85$	$96.13 \pm 1.23$	$100.63 \pm 3.01$

\* Significant at ( $P < 0.05$ ), \*\* Significant at ( $P < 0.01$ )

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