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RESEARCH ARTICLE

Diagnostic Studies in Dogs with Hepatic Disorders

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ABSTRACT

This communication describes the different diagnostic methods employed in diagnosis of hepatic disorders in canine. The current clinical study was conducted on a total of forty nine (n=49) dogs were suffering from various hepatic disorders. The diagnosis of disorders were made into acute hepatitis (n=14), chronic hepatitis (n=23) and Cholestasis/cholangiohepatitis (n=12). Ultrasonographic examination was carried out using 3-6.5 MHz transducer for various organs viz, hepatobiliary, urogenital and spleen. The ailing dogs with hepatomegaly were subjected to plain radiography by following the standard procedures. The mean values of haemoglobin, packed cell volume, total erythrocyte count and total platelet count were recorded to be 8.15±0.30g/dl, 25.33±1.06%, 3.81±0.14x106/µl, and 2.47±0.12x105/µl, respectively. A non-significant increase in the TLC (14.35±0.53x103/µl) was observed. Mean ALT, AST, ALP, Total bilirubin and BUN were 670.50±68.66 IU/L, 364.19±52.21 IU/L, 585.12±46.91 IU/L, 2.51±0.50 mg/dl and 25.69±1.90 mg/dl, respectively. Mean GGT and creatinine values were 16.38±5.53 IU/L and 1.51±0.11 mg/dl, respectively. Mean values of total protein, albumin, globulin, A/G ratio, plasma glucose and plasma cholesterol were 5.32±0.12 g/dl, 2.29±0.07g/dl, 3.01±0.07g/dl, 0.76±0.02, 66.28±2.03 mg/dl and 135.67±6.27mg/dl, respectively. Six (12.25%) dogs showed reduced parenchymal echogenicity with enhanced visualization of portal vessels suggestive of acute hepatitis. Dogs with hyperechoic bright liver and normal size were diagnosed as chronic hepatitis, which was observed in nine (18.36%) dogs. Bright and small liver with irregular margins was diagnosed as hepatic cirrhosis and was observed in four (8.16%) dogs. Cholestasis/cholangiohepatitis was diagnosed in 10 (20.40%) dogs having increased echogenicity of the wall with thickening of 2.9 to 3.7 mm. In three (6.12%) dogs which have been referred as the ultrasonographic murphy sign characteristic of acute cholecystitis.

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INTRODUCTION

Liver is the most important vital organ and the largest parenchymal gland of the body with vast reserves of function (70-80%), an almost embryonic capacity to regenerate and perform adequately despite often extensive pathological damage to its integrity. It provides a myriad of biochemical, synthetic, excretory and regulatory functions important to intermediary body metabolism (Center, 1998). Hepatic disorders are one of the top five causes of non accidental deaths in dogs. It is associated with varied and often vague clinical symptoms and thus frequently presents a diagnostic challenge to veterinary clinicians. However hepatic disorder is often treatable and has a predictable prognosis when a definitive diagnosis is made. Dogs affected by this condition are mostly young or middle aged adults of either sex with symptoms of hepatitis of varying severity. The symptoms included lethargy, depression, weight loss, vomiting, and jaundice. Idiopathic chronic hepatitis, however, appears to be highest in female dogs with signs of anorexia, depression, weakness, polyuria/polydipsia, ascites, jaundice, weight

loss, and vomiting (Nelson and Couto, 1998). As the liver has great functional reserve capacity, detection of the hepatic functional impairment by conventional means is only possible once a significant hepatic dysfunction (≥55%) is present (Hall and German, 2005). Decreased values of hemoglobin (Hb), packed cell volume (PCV), total erythrocytic count (TEC), total protein and glucose with increased leucocytes, total bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatise (ALP) with usually abnormal clotting time (increased), reduced protein synthesis and reduced vitamin K absorption. Furthermore, systemic diseases and various drugs can cause misleading increases in serum activities (secondary or reactive hepatopathies), and it can be a clinical dilemma to decide whether liver enzyme elevations are significant, and whether they represent primary or secondary liver disease (Nelson and Couto, 1998). Despite availability of a range of diagnostic tests of both hepatic damage and dysfunction, there is rarely a single test that adequately identifies hepatic disease or its underlying cause. The diagnosis of hepatic disorders due to non-specific symptoms and signs is difficult and as such require a number of diagnostic tests to reach a definite diagnosis (Rothuizen and Van, 1998). Different types of tests are performed to help in diagnose the disease process. The most commonly used diagnostic tests include clinical signs and history of disease, laboratory examination (icterus index, Van Den Berg test, examination of bile pigments in urine), hematobiochemical, radiographic and ultrasonographic examination. Ultrasonographic evaluation is an integral part in the assessment of liver disease in dogs (Angtuaco *et al.,* 2002). Indications for hepatic ultrasound usually include elevated liver enzymes and presence of free abdominal effusion. It is particularly useful in differentiating focal from diffuse disease, cystic from solid masses and obstructive from non-obstructive icterus (Konde and Pugh, 1996).

MATERIALS AND METHODS

The current study was conducted on the dogs in the year 2010 - 2011. Six apparently healthy dogs with no clinical condition brought for routine clinical examination in the age group of 3-5 years irrespective of sex and breed were chosen to act as control for the study. Normal clinical and physiological parameters, haematology and blood biochemical profiles were obtained from this group. A total of 49 dogs suffering from various hepatic disorders were examined during the current study on the basis of history, clinical symptoms, serobiochemical observations, ultrasonographic findings, duration and the progression of the disease. Clinical symptoms observed during the study were constipation, vomition, complete anorexia, weight loss, jaundice, ascites, anaemia and hepatic encephalopathy. The diagnosis of disease were made into acute hepatitis (n=14), chronic hepatitis (n=23) and Cholestasis/cholangiohepatitis (n=12). After properly restraining of the animals, blood samples were collected taking all the aseptic precautions and avoiding haemolysis. About 1 ml of blood was collected in a vacutainer containing disodium salt of ethylenediamine-tetra acetic acid (EDTA, 1mg/ml) for haematology, and about 4 ml blood was collected in heparinized vacutainer for biochemical estimation and an additional 2 ml of blood was collected in vacutainer containing sodium fluoride as anti coagulant for the estimation of blood glucose. Blood collected in heparin was immediately centrifuged at 3000 rpm for 12 minutes and plasma separated was stored at -20°C till further use. Whole blood was used for hematological study. Estimation of hemoglobin (Hb), packed cell volume (PCV), total erythrocytic count (TEC), total leukocyte count (TLC), total thrombocytic count and differential leukocytic count (DLC) was done as per the methods described by Jain et al. (1986). The biochemical estimations viz, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatise (ALP), gama glutamyl transferase (GGT), plasma glucose, plasma cholesterol, total protein, plasma albumin and globulin, A:G ratio, blood urea nitrogen (BUN), creatinine and bilirubin. Biochemical parameters were carried out spectrophotometrically using standard protocols in various reagent kits.

Ultrasonographic (USG) examination was carried out using SONOSITE-600M machine as per the procedures described by Nyland et al. (2002) using 3-6.5 MHz transducer for various abdominal organs viz. hepatobiliary, urogenital and spleen. The sick anorectic dogs were not subjected to fasting. The cranio-ventral abdominal hairs were shaved from costal arch cranially to the inguinal region caudally and laterally along the body wall. Acoustic coupling gel was applied liberally over the abdomen to provide essential acoustic coupling of the transducer to the patient. The dogs were gently restrained in dorsal recumbency by holding the forelimbs and hind limbs. The transducer was placed immediately behind the xiphisternum on the midline and angled cranio-dorsally to image a transverse section of liver. Transducer head was then rotated through 90° to image a longitudinal section of liver on midline. The liver was also imaged in both planes by moving the transducer laterally along the caudal aspect of the costal arch to the left and right of midline if stomach gas does not interfere with imaging. The ailing dogs with hepatomegaly were subjected to plain radiography by following the standard procedures. Animals were restrained in dorsal recumbency and left/right lateral recumbency for ventro-dorsal view and lateral view of the abdomen respectively.

Statistical analysis

The data recorded, wherever applicable, was statistically analyzed using chi square test and simple one way analysis of variance as per Snedocor and Cochran (1967).

RESULTS

Clinical signs observed in hepatic disorders in dogs are presented in Table 1. On clinical examination, only eight (16.32%) dogs were active and alert as regards of behaviour was concerned, while 34 (69.39%) were moderately dull and depressed, five (10.20%) were severely dull and depressed and two (4.08%) were in a comatose condition. Diarrhoea was observed in 31 (63.27%) dogs, out of which nine (18.37%) dogs had haemorrhagic diarrhoea, 22 (44.89%) dogs had non-haemorrhagic diarrhoea. Constipation was observed in 4

(8.16%) dogs and 18 (36.73%) dogs had normal defecation status. Vomiting was seen in 16 (32.65%) dogs out of which five (10.20%) dogs had blood tinged vomitus. Complete anorexia with weight loss was a predominant sign in 22 (44.89%), 17 (34.70%) dogs suffered from inappetence and 10 (20.40%) dogs had a normal appetite. Water intake was normal in most of the animals with a few animals showing signs of polydipsia (13/49) and polyurea (9/49). Jaundice was seen in 16 dogs (32.65%), out of which two dogs had acute hepatitis, six had chronic hepatitis and 8 had cholestasis/ cholangihepatitis. Ascites was observed in 11 dogs (22.45%), in which one had acute hepatitis, eight were having chronic hepatitis and 2 were having cholestasis/ cholangihepatitis. Five (10.20%) dogs had mild dehydration (+), 27 (55.10%) were moderately dehydrated (++) and 11 (22.45%) were severely dehydrated (+++) and dehydration was undetectable in rest of 6 dogs (12.25%). Conjunctiva was pinkish in seven (14.28%), congested in three (6.12%), pale in 17 (34.69%) severely pale in six (12.24%, and icteric in 16 (32.65%) dogs. Twenty nine cases (59.18%) were having mild to severe anaemia. Hepatic encephalopathy was observed in two (4.08%) dogs. Painful abdominal palpation was observed in 19 (38.78%) dogs.

The mean values of haemoglobin, packed cell volume, total erythrocytic count and total platelet count were recorded to be 8.15± 0.30 g/dl, 25.33±1.06%, 3.81±0.14 x 10^6/µl, and 2.47±0.12 x 10^5/µl, respectively, and these values decreased significantly from healthy control. Clotting time (2.90±0.09 min) increased significantly as compared to healthy control (2.06±0.52 min). A non-significant increase in the TLC (14.35±0.53 x 10^3/µl) was observed. Differential leukocyte count revealed neutrophils, lymphocytes, monocytes, eosinophiles and basophiles percentage as 68.20±1.17, 22.70±0.86, 5.24±0.22, 3.64±0.17 and 0.20±0.06 per cent, respectively. Mild increase in neutrophil count was observed in the affected animals (Table 2). Mean ALT, AST, ALP, Total bilirubin, and BUN were 670.50±68.66 IU/L, 364.19±52.21 IU/L, 585.12±46.91 IU/L, 2.51±0.50 mg/dl and 25.69±1.90 mg/dl, respectively. All these values were significantly increased as compared to control group. Mean GGT and creatinine values were 16.38±5.53 IU/L and 1.51±0.11 mg/dl, respectively and the values increased non-significantly as compared to control group. Mean values of total protein, albumin, globulin, A/G ratio, plasma glucose and plasma cholesterol were 5.32±0.12 g/dl, 2.29±0.07 g/dl, 3.01±0.07 g/dl, 0.76±0.02, 66.28±2.03 mg/dl and 135.67±6.27 mg/dl, respectively. A significant decrease was observed in all the values as compared to healthy control (Table 3).

In the present study USG was performed in 26 dogs in which twelve dogs showed multifocal hypoechoic to mixed echogenic liver parenchyma. Diffuse hyper echoic bright liver was observed in nine dogs and bright thickened gall bladder wall was recognized in seven dogs (Figure 1). Peritoneal fluid accumulation was detected by the presence of anechoic areas separating the various intra-abdominal structures in 11 dogs suggesting ascites. The presence of large amount of ascitic fluid in some dogs greatly enhanced the visualization of various abdominal organs. Six (12.25%) dogs showed reduced parenchymal

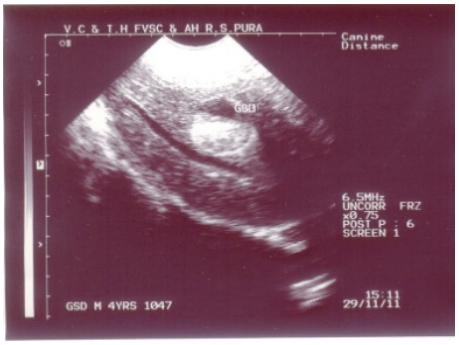

Figure 1: Ultrasonograph (2D) in transverse scan showing diffuse hyperechoic liver lobe and bright thickened gall bladder (GB) wall in a 4-year-old German shepherd male dog.

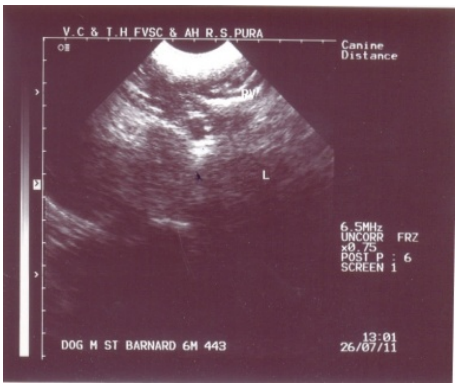

Figure 2: Ultrasonograph (2D) in transverse scan showing reduced liver (L) parenchymal echogenicity with enhanced visualization of portal vessels (PV) in a 6-month-old Saint Barnard male dog.

echogenicity with enhanced visualization of portal vessels suggestive of acute hepatitis (Figure 2). Dogs with hyperechoic bright liver and normal size were diagnosed as chronic hepatitis, which was observed in 9 (18.36%) dogs. Bright and small liver with irregular margins was diagnosed as hepatic cirrhosis and was observed in 4 (8.16%) dogs. Diffuse hyperechoic liver parenchymas with less distinct portal vessels associated with peritoneal fluid accumulation (ascites) were noticed in 8 dogs and diagnosed as chronic hepatitis. Cholestasis/ cholangio-hepatitis were diagnosed in 10 (20.40%) dogs having increased echogenicity of the wall with thickening of 2.9 to 3.7 mm. Two (4.08%) dogs showed biliary duct obstruction as evidenced by dilatation of bile duct. Pain was detected in the gallbladder region during scanning process with transducer in three (6.12%) dogs which has been referred as the sonographic Murphy sign characteristic of acute cholecystitis. Abdominal radiography of 11 dogs with hepatobiliary disorder and two dogs with urinary disorders did not provide any useful information. Generalized enlargement was associated with rounding of the caudoventral edge, particularly that of the left lateral lobe on the lateral view.

DISCUSSION

The clinical signs recorded in hepatic disorders in the present study were highly variable in nature. The

Table 1: Clinical signs observed in hepatic disorders

S. No	Clinical sign	Frequency
1	Diarrhoea	31 (63.27), haemorrhagic 9 (18.37) and non haemorrhagic 22 (44.89)
2	Constipation	4 (8.16)
3	Vomition	16 (32.65), Haemorrhagic 5 (10.20) and non haemorrhagic 11 (22.45)
4	Complete anorexia	22 (44.89), Inappetiance 17 (34.70)
5	Polydipsia	13 (26.53)
6	Polyurea	9 (18.36)
7	Jaundice	16 (32.65)
8	Ascites	11 (22.45)
9	Anaemia	29 (59.18)
10	Hepatic encephalopathy	2 (4.08)
11	Painful abdominal palpation	19 (38.78)
12	General depression	41 (83.67), active and alert 8 (16.32), dull and depressed 34 (69.39), severely dull and depressed 5 (10.40), comatose 2 (4.08)
13	Dehydration	43 (89.58). Nil 6 (12.24), mild + 5 (10.20,) moderate ++27 (55.10) severe +++11 (22.45
14	Conjunctival mucous membrane	pinkish 7 (14.28), congested 3 (6.12), pale 17 (34.70), severely pale 6 (12.24) and icteric 16 (32.65)

Figures in parenthesis indicate percentage

Table 2: Haematological studies in hepatic disorders

S. No	Parameters	Control (n=6)	0-day (n=49)	Reference Range
1	Hb (g/dl)	11.95±0.49	8.15±0.30*	3.30-12.30
2	P.C.V (%)	34.70±2.01	25.33±1.06*	8.90-40.07
3	T.E.C (x 10^6/µl)	5.92±0.28	3.81±0.14*	1.30-5.70
4	T.L.C (x 10^3/µl)	11.53±0.78	14.35±0.53	6.80-26.50
5	Platelets(x 10^5/µl)	3.62±0.19	2.47±0.12*	0.87-4.10
6	Clotting time (min)	2.06±0.52	2.90±0.09*	1.98-4.20
7	DLC (%)			
	Neutrophils	65.00±1.71	68.22±1.17	52-84.30
	Lymphocytes	25.66±1.54	22.70±0.86	13-31
	Monocytes	5.58±0.31	5.24±0.22	3.50-5.90
	Eosinophils	3.25±0.17	3.64±0.17	0.43-4.20
	Basophils	0.16±0.11	0.20±0.06	0.09-0.35

*significant at 5% (P<0.05)

Table 3: Biochemical studies in hepatic disorders

S. No	Parameters	Control (n=6)	0-day (n=49)	Reference Range
1	A.L.T (IU/L)	47.25±2.08	670.50±68.60**	62-2744
2	A.S.T (IU/L)	30.22±1.98	364.19±52.21*	42-2508
3	A.L.P (IU/L)	64.22±6.60	585.12±46.91**	58-2744
4	G.G.T (IU/L)	8.80±0.52	16.38±5.53	3.67-257
5	Glucose (mg/dl)	90.66±2.10	66.28±2.03*	38.70-109
6	Cholesterol (mg/dl)	214.22±2.87	135.67±6.27*	56-215
7	Total protein (g/dl)	6.30±0.05	5.32±0.12*	3.20-6.98
8	Albumin (g/dl)	2.97±0.04	2.29±0.07*	0.90-3.46
9	Globulin (g/dl)	3.35±0.03	3.01±0.07*	3.20-4.30
10	A/G ratio	0.88±0.01	0.76±0.02*	0.43-1.22
11	Bilirubin (mg/dl)	1.04±0.12	2.51±0.50*	0.28-21.50
12	B.U.N (mg/dl)	20.26±1.84	25.69±1.90*	13-63.72
13	Creatinine (mg/dl)	1.35±0.14	1.51±0.11	0.40-4.80

*Significant at 5% (P<0.05); ** Significant at 1% (P<0.01)

predominant signs recorded were inappetiance/anorexia, depression and dullness, vomition, weight loss and diarrhoea, constipation, ascites, jaundice and anaemia. The present findings were in agreement with the findings of Varshney and Hoque (2002). Vomiting could be exacerbated by eating and drinking. Besides gastric retention due to gastric hypomotility further contributes to vomition and this vomitus may contain gastric juices, food or bilious reflux. Diarrhoea could be due to extensive involvement of whole gastro intestinal tract due to inflammatory processes because of release of certain mediators of inflammation such as cachetin which further results in fever, nausea, anorexia and weight loss (Boomkens et al., 2004). Anaemia as observed in the present study may be because of haemorrhage or failure of erythropoiesis and haemolysis in icteric patients. Anaemia associated with hepatic disease is associated with chronic inflammatory reactions (defective iron utilization) and is usually moderate, nonregenerative, normocytic, and normochromic. Hepatic encephalopathy (HE) was observed in two dogs which could be due to a significant loss of liver function (60-70%) in acute or chronic liver disease. A number of nitrogenous waste products such as ammonia, aromatic amino acids, and methionine, gamma-aminobutyric acid (GABA) and fatty acids, which are either consumed in the diet or synthesized by the gastrointestinal flora. In animals with a significant loss of liver function, these toxins pass the blood-brain barrier

and cause nervous signs (Watson, 1998). Icterus or jaundice was reported in 16 cases and may be caused by the accumulation of bilirubin in the tissues. Bilirubin is a product of red blood cell destruction and is the major pigment in bile. During liver failure, the hepatocytes are unable to process bilirubin leading to the yellow discoloration. In the present study ascites was observed in 11/49 dogs which clinically resulted in abnormal collection of fluid in peritoneal cavity. Ascitis was a major clinical sign in chronic hepatitis which may be caused by cirrhosis of liver and is characterised by increased portal vein pressure. It may also be due to the decreased production of albumin as observed in the present study. Serum albumin is produced exclusively in the liver and is a major determinant of plasma and tissue oncotic pressure. Hypoalbuminemia associated with chronic liver disease is a major factor contributing to development of ascitis. Also factors like excessive sodium chloride intake greatly and sodium retention by kidney greatly enhances development of ascitis (Kaneko *et al.,* 2008).

There was significant fall in Hb, PCV, and TEC in hepatic disorders in dogs which was in accordance to findings of Chohan *et al.* (2009). Decrease in Hb is attributed to increased degradation of erythrocytes due to increased transit time through spleen because of reduced portal blood flow and or increased fragility of erythrocytes due to high levels of bile acids, besides impaired bone marrow responses, decreased erythrocyte survival time, decreased nutrient uptake due to inappetance or anorexia and reduced availability of micronutrients from liver (Bush, 2002). Neutrophilia was observed in all three conditions with significant neutrophilic leucocytosis in acute hepatitis. Mean platelet count was also significantly decreased. Several mechanisms have been suggested for thrombocytopenia in patients with hepatic disorder which include increased platelet sequestration in the spleen as a result of congestive splenomegaly, reduced production of thrombopoietin by the liver, increased platelet breakdown due to antibodies (Prins *et al.,* 2010) and increased consumption resulting from low grade disseminated intravascular coagulopathy. Clotting time was significantly increased, which could be due to improper synthesis of proteins by the liver required for clotting mechanisms. Webster (2005) opined that liver is the production site for all coagulation factors. Reduced hepatic synthesis results in a clinically significant hypocoagulable state. The activities of ALT, AST and ALP were significantly elevated. GGT was elevated non-significantly. Elevations of plasma transaminases such as ALT and AST were indicative of altered hepatocellular membrane permeability, hepatocellular necrosis and inflammation with degree proportional to number of injured hepatocytes (Kramer and Hoffman, 1997). ALP is a membrane bound enzyme found on hepatocyte cannalicular membrane and luminal surface of biliary epithelial cells and its iso-enzymes are present in kidneys, intestine, placenta and bone, but elevation in its level in more than one year old dog is usually indicative of hepatic origin unless bone disease coexists, since iso-enzymes from other organs are having extremely short half lives. Marked increase in activities of ALP and GGT has been reported in conditions causing cholestasis, cholangiohepatitis, biliary cirrhosis, biliary obstruction and cholecystitis of cholelithiasis (Bandyopadhyay, 2003). A significant increase in total bilirubin was observed in the present study. Hyperbilirubinemia is due to disturbance of the balance between rate of production of bilirubin and metabolism and excretion of bilirubin. In the present study the increase might be as a result of diminished excretion due to extensive hepatocyte damage or biliary obstruction or a combination thereof (Vijayakumar *et al.,* 2008). Also a significant decrease in total protein, albumin, globulin level and A/G ratio was observed. Liver being the main site of synthesis and degradation of most of the proteins, any hepatic disorder (chronic hepatitis and cirrhosis) are responsible for decrease in albumin concentration. Total plasma protein might also have decreased due to marked decline in the diet intake, malabsorption and ongoing protein losing enteropathies like gastroenteritis, gastrointestinal ulcerations and chronic gastritis. Inflammation also results in increased bowel permeability leading to fluid, electrolyte, protein, and cell loss. The findings of the present study were comparable to that of Sevelius (1995). But hypoalbuminemia may also occur without impairment in hepatic albumin synthesis due to either leakage of albumin from hepatic lymph or increase in volume of distribution as in cases of ascites. Significant decrease in cholesterol level was observed in all the three groups of patients which may be attributed to decrease in synthesis or absorption from the gut or excessive conversion of cholesterol into bile acids (Hall, 1985). There was a significant decrease in the plasma glucose levels which corroborated with the findings of Varshaney and Hoque (2002) in dogs with hepatic dysfunctions. Hypoglycaemia in the affected dogs might be due to inappetance/anorexia complemented by malabsorption from intestine. Hypoglycemia in patients with hepatic disorders results from decreased glycogenolysis and gluconeogenesis combined with hyper-insulinemia due to decreased hepatic metabolism. Blood urea nitrogen increased significantly which corroborates with the findings of Chohan *et al.* (2009). Ammonia loading may occur in dogs as a result of haemolysis, blood transfusions and gastrointestinal haemorrhage. This could lead to non-renal related elevations in serum urea concentrations via hyperureagenesis and could cause an increase in the UC ratio. Haemolysis may produce substrates in the form of proteins that would require deamination and consequently lead to hyperammonaemia. Since gastric ulceration has been reported as a major cause of an increased UC ratio in dogs (Prause and Grauer, 1998), it may play a role in the elevated UC ratio encountered in dogs with hepatic disorders. Nine dogs in this study showed typical clinical signs of gastric ulceration. Creatinine levels were non-significantly increased. Similar observations were made by Chohan *et al.* (2009). This might be due to the renal abnormalities and the urinary retention due to obstruction.

Ultrasonographic appearance showed multifocal hypoechoic to mixed echogenic liver parenchyma with diffuse hyperechoic bright liver and bright thickened gall bladder wall. Liver echogenicity was usually normal in cases of acute hepatitis, but it may be decreased with enhancement of periportal echoes. Focal hypoechoic

lesions suggestive of necrosis were noticed in six dogs which is in agreement with Lamb (1990). In present study diffuse hyperechoic liver parenchymas with less distinct portal vessels associated with peritoneal fluid accumulation (ascites) were noticed in eight dogs diagnosed as chronic hepatitis. Four dogs were diagnosed with hepatic cirrhosis as evidenced by hyperechoic 'bright' small liver with irregular margins which was in accordance with the findings of Cartee (1981). Irregular margins and hyperechogenicity was due to fibrosis which occurs in chronic hepatitis. Pain was detected in the gallbladder region during scanning of 2 dogs which has been referred as the ultrasonographic"Murphy sign" characteristic of acute cholecystitis. Abdominal radiography of 11 dogs with hepatobiliary disorder and 2 dogs with urinary disorders did not provide any useful information except for diagnosing hepatomegaly. In conclusions, ultrasonographic evaluation is a more reliable method in the diagnosis of liver disease in dogs.

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REFERENCES

Angtuaco TL, SK Lal and OGD Banaad, 2002. Current liver biopsy practices for suspected parenchymal liver diseases in the United States: the evolving role of radiologists. Am J Gastroenterol, 97: 1468-1471.

Bandyopadhyay S, 2003. Clinico-therapeutic aspects of cholecystic disorders in dogs. MVSc Thesis, Indian Veterinary Research Institute Izatnagar, India.

Boomkens SY, LC Penning and HF Egberink, 2004. Hepatitis with special reference to dogs. A review on the pathogenesis and infectious etiologies, including unpublished results of recent own studies. Vet Quarter, 26: 107-114.

Bush BM, 2002. Interpretation of laboratory results for small animal clinics. Blackwell Sciences Co, Lowa, USA. pp: 317-319.

Cartee RE, 1981. Diagnostic real time ultrasonography of the liver of the dog and cat. J Am Anim Hosp Assoc, 17: 731-737.

Center SA, 1998. Nutritional support for dogs and cats with hepatobiliary disease. J Nutri, 128: 2733-2746.

Chohan AS, BK Bansal, PS Dhaliwal and R Ranjan, 2009. Hematobiochemical changes in dogs suffering from hepatobiliary diseases. Ind J Vet Med, 29: 73-76.

Hall EJ and AJ German, 2005. Laboratory evaluation of hepatic disease. In: BSAVA Manual of Canine and Feline Clinical Pathology, Villiers E, Blackwood L (2nd ed), pp: 184-206.

Hall RL, 1985. Laboratory evaluation of liver disease. Vet Clin N Am: Small Anim Prac, 15: 3-19.

Jacobson LS and GE Swan, 1995. Supportive treatment of canine babesiosis. J S Afri Vet Assoc, 66: 95-105.

Jain NC, 1986. Schalm's Veterinary Haematology. Lea and Febiger, Philadelphia, USA.

Kaneko JJ, JW Harvey and ML Bruss, 2008. Clinical Biochemistry of Domestic Animals.6th ed Academic Press, New York.

Konde LJ and CR Pugh, 1996. Radiology and sonography of the digestive system. In: Handbook of small animal gastroenterology, Tams TR (1st ed), WB Saunders, Philadelphia, Pa, USA, pp: 75-135.

Kramer JW and WE Hoffman, 1997. Clinical enzymology. In: Clinical Biochemistry of Domestic Animals. Kaneko J, J Harvey and M Bruss, (eds) Academic Press London, p: 330.

Lamb CR, 1990. Abdominal ultrasonography in small animals: Examination of liver, spleen and pancreas. J Small Anim Pract, 31: 6-15.

Nelson RW and CG Couto, 1998. Diagnostic tests for the hepatobiliary system. In: Small animal internal medicine, Mosby Maryland Heights, USA, (2nd ed), pp: 487-539.

Nyland TG, JS Mattoon, EJ Herrgesell and ER Wisner, 2002. Liver, spleen. In: Small animal diagnostic ultrasound. Nyland TG, Mattoon JS (ed), WB Saunders, Philadelphia, pp: 93-143.

Prause LC and GF Grauer, 1998. Association of gastrointestinal hemorrhage with increased blood urea nitrogen and BUN/Creatinine ratio in dogs: a literature review and retrospective study. J Vet Clin Pathol, 27: 107–111.

Prins M, CJMM Schellens, LMW Van, J Routhuizen and E Teske, 2010. Coagulation disorders in dogs with hepatic disorders. Vet J, 185: 163-168.

Rothuizen J and DITS Van, 1998. Hepatitis in dogs: a review. Vet Clinics N Am: Small Anim Pract, 6: 9.

Sevelius E, 1995. Diagnosis and prognosis of chronic hepatitis and cirrhosis in dogs. J Small Anim Prac, 6: 521-528.

Snedecor GW and WG Cochran, 1967. Statistical method. 6th ed. Oxford and IBH Publishing Company, New Delhi.

Varshney JP and M Hoque, 2002. Clinico-pathological and ultrasonographic observations in canine hepatopathies. Ind J Anim Sci, 72: 423-427.

Vijayakumar G, V Pandayan, GA Balasubramanian and M Subramanian, 2008. Haematobiochemical changes in dogs with hepatic disorders. Ind Vet J, 85: 341-342.

Watson TDG, 1998. Recognition and management of portosystemic shunts in dogs. Ped Breeder For, 1: 9-15.

Webster CRL, 2005. History, clinical signs and physical findings in hepatobiliary disease. In: Textbook of Veterinary Internal Medicine, diseases of dog and cat. Ettinger SJ and Feldman EC (6th ed), Elsevier Saunders, USA. pp: 1422-1434.