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Diagnostic and Therapeutic Studies in Ascitis in Dogs

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Abstract: The present study was conducted on the dogs presented in the Small Animal Medicine OPD of Referral Veterinary Hospital of the Faculty of Veterinary Science and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology-Jammu, between June 2010 and August 2011. Anaemia, weight loss, diarrhoea, jaundice and ascites with hepatic encephalopathy were the chief clinical signs observed. Haemogram revealed anaemia, thrombocytopenia and increased clotting time. Biochemical status showed severe hypoprotenemia and hypoalbuminemia with nonsignificant increase in globulin.

Key words: Jaundice, ascites, thrombocytopenia, anechoic fluid, status

INTRODUCTION

The most wide spread form of interspecies bonding occurs between humans and dogs. It is a wonderful companion animal and is loved for its obedience, loyality and devotion. Ascites is accumalation of edematous transudate in the peritoneal cavity. It is a common problem in dogs and may occur due to increased portal vein pressure or due to decreased production of albumin as observed in present study and due to excessive intake of sodium chloride and sodium retention by the kidney which greatly enhance its occurrence. Also, it has been attributed to conditions like chronic hepatic failure, congestive heart failure, malnutrition and parasitism (Randhawa *et al*., 1988).

MATERIALS AND METHODS

The present study was conducted on the dogs presented in the Small Animal Medicine OPD of Referral Veterinary Hospital of the Faculty of Veterinary Science and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology-Jammu, between June 2010 and August 2011. Six apparently healthy dogs with no clinical condition in the age group of 3-5 years irrespective of sex and breed were chosen to act as control for the present study. Normal clinical parameters, haematology and blood biochemical profiles were obtained from this group. After properly restraining the animal, blood samples were collected taking all the aseptic precautions and avoiding haemolysis. About 1 mL of blood was collected in vacutainer containing disodium salt of Ethylenediamine-Tetra Acetic acid (EDTA, 1 mg mL^{-1}) 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 min 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 Leukocytic Count (TLC), total thrombocytic count and Differential Leukocytic Count (DLC) was done as per the methods described by Jain (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, uro-genital 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

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liberally over the abdomen to provide essential acoustic coupling of the transducer to the patient. The animals 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.

RESULTS AND DISCUSSION

Out of 49 dogs with hepatic disorders 16 dogs aged between 3-19 years were found to be suffering from ascites as noticed on clinical examination and confirmed by biochemical examination and ultrasonography, three dogs had acute hepatitis, 11 were having chronic hepatitis and two were having cholestasis/cholangio hepatitis. Also, abdominal palpation revealed fluid filled abdominal cavity which was confirmed by performing USG of abdomen revealing fluid in the abdomen. Anechoic fluid was also seen in peritoneal cavity. Some animals had a tucked up abdomen as felt on palpation.

Haematobiochemical studies (pre-treatment and post treatment) in dogs suffering from ascites are given in Table 1 and 2, Fig. 1a, b, respectively. Haematological examination revealed non-significantly decreased Hb (9.89±0.38 g dL^{-1}), PCV (31.70±1.29%), TEC (4.97±1.10×10^6 μL^{-1}) and platelet count (3.32±0.39×10^5 μL^{-1}). TLC (11.48±0.98×10^3 μL^{-1}) remained almost unaffected. Clotting time (3.01±0.15 min) increased significantly. Differential leucocyte count revealed neutrophils, lymphocytes, monocytes, eosinophils and basophils as 68.00±1.37, 25.16±1.24, 4.35±0.20, 2.35±0.14 and 0.14±0.05%, respectively. Plasma biochemical profile revealed significantly decreased total protein (4.49±0.12 g dL^{-1}), albumin (1.90±0.10 g dL^{-1}), globulin (2.57±0.05 g dL^{-1}), A/G (0.74±0.04) ratio, blood glucose (62±3.13 mg dL^{-1}) and cholesterol (120±9.40 mg dL^{-1}). Mean values of ALT (269.50±16.30 IU L^{-1}), AST (225±11.60 IU L^{-1}), ALP (278.03±14.70 IU L^{-1}) GGT (9.56±0.72 IU L^{-1}), BUN (29.60±2.74 mg dL^{-1}) and bilirubin (1.95±0.12 mg dL^{-1}) increased significantly. Creatinine (1.52±0.10 mg dL^{-1}) increased non-significantly as compared to control group. The results of present investigation indicated that the dogs were suffering from ascites due to liver damage.

Post treatment haematological examination revealed significant increase in Hb (11.38±0.32 g dL^{-1}), PCV (34.20±1.38%), TEC (5.73±0.15×10^6 μL^{-1}) and platelet (3.85±0.13×10^5 μL^{-1}) count when compared to pre-treatment and non significant decrease as compared to control group. TLC (12.28±0.42×10^3 μL^{-1}) increased

Table 1: Haematological studies (pre-treatment and post treatment) in ascites

Parameters	Control (n = 6)	0 day (n = 11)	15 day (n = 6)
Hb (g/dL)	11.95±0.490^a	9.89±0.380^b	11.38±0.32^a
PCV (%)	34.70±2.010^a	31.70±1.290^a	34.20±1.38^a
TEC (×10^6/μL)	5.92±0.280^a	4.97±0.530^a	5.73±0.15^a
TLC (×10^3/μL)	11.53±0.780^a	11.48±0.980^a	12.28±0.42^a
Platelets (×10^5/μL)	3.62±0.190^a	3.32±0.390^b	3.85±0.13^a
Clotting time (min)	2.06±0.520^a	3.01±0.150^b	2.33±0.14^a
DLC (%)			
Neutrophils	65.00±1.710^a	68.00±1.370^a	67.21±1.20^a
Lymphocytes	25.66±1.540^a	25.16±1.240^a	24.76±1.04^a
Monocytes	5.58±0.310^a	4.35±0.200^a	5.38±0.24^a
Eosinophils	3.25±0.170^a	2.35±0.140^a	2.39±0.15^a
Basophils	0.16±0.110^a	0.14±0.050^a	0.26±0.09^a

Table 2: Biochemical studies (pre-treatment and post treatment) in ascites

Parameters	Control (n = 6)	0 day (n = 11)	15 day (n = 6)
ALT (IU/L)	47.25±2.08^a	269.5±16.30^b	66.06±10.28^a
AST (IU/L)	30.22±1.98^a	225±11.60^b	48.80±7.900^a
ALP (IU/L)	64.22±6.60^a	278.80±14.70^b	65.40±3.200^a
GGT (IU/L)	8.80±0.52^a	9.56±0.72^a	6.69±0.510^a
Glucose (mg/dL)	90.66±2.10^a	62±3.13^b	81.6±2.4000^c
Cholesterol (mg/dL)	214.22±2.87^a	120.80±9.40^b	179.76±6.700^c
Total protein (g/dL)	6.30±0.05^a	4.49±0.12^b	5.63±0.110^b
Albumin (g/dL)	2.97±0.04^a	1.92±0.10^b	2.47±0.100^c
Globulin (g/dL)	3.35±0.03^a	2.57±0.05^b	3.16±0.040^a
A/G ratio	0.88±0.01^a	0.74±0.04^b	0.78±0.030^b
Bilirubin (mg/dL)	1.04±0.12^a	1.95±0.39^a	0.91±0.120^a
BUN (mg/dL)	20.26±1.84^a	29.60±2.74^b	23.28±3.070^a
Creatinine (mg/dL)	1.35±0.14^a	1.52±0.10^a	1.19±0.080^a

Values within a column having superscript with at least one common letter do not differ significantly at 5% level ($p<0.05$) from each other

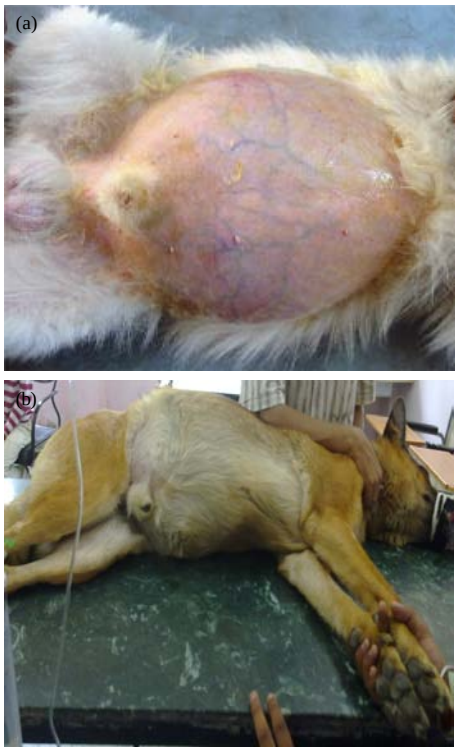

Fig. 1: a, b) Abdomen filled with ascitic fluid

non-significantly as compared to control and pre-treatment. Clotting time (2.33±0.14 min) decreased significantly as compared to pre treatment and non-significantly increased as compared to control. Differential leucocyte count revealed neutrophils, lymphocytes, monocytes, eosinophils and basophils as 67.21±1.20, 24.76±1.04, 5.38±0.24, 2.39±0.15 and 0.26±0.09%, respectively. Biochemical profile post treatment revealed significant decrease in ALT (66.06±10.28 IU L^{-1}), AST (48.80±7.90 IU L^{-1}), ALP (65.40±3.20 IU L^{-1}), GGT (6.69±0.51 IU L^{-1}), BUN (23.28±3.07 mg dL^{-1}) and bilirubin (0.91±0.12 mg dL^{-1}), respectively. Plasma glucose (81.60±2.40 mg dL^{-1}) and plasma cholesterol (179.76±6.70 mg dL^{-1}) increased significantly as compared to pre-treatment and also showed a significant decrease as compared to control. Total protein (5.63±0.11 g dL^{-1}) increased non-significantly as compared to pre treatment and was significantly lower than control. Albumin (2.47±0.10 g dL^{-1}) showed significant increase and decrease as compared to pre-treatment and control, respectively. Globulin (3.16±0.04 g dL^{-1}) increased significantly as compared to pre treatment and non-significantly decreased as compared to control. However, cholesterol post treatment was significantly lower than control. Creatinine (1.19±0.08 mg dL^{-1}) decreased non-significantly as compared to pre-treatment and control. A/G (0.78±0.03) ratio increased non-significantly compared to pre-treatment and was significantly lower than control. Ultrasonography of the abdomen revealed that almost all the afected dogs suffered from enlargement of liver and in one case there was presence of tumour in the liver. 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.

In the present study, ascites was observed in 16/49 (32.65%) 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. These findings were in accordance with those of Skardova (1991). Also, factors like excessive sodium chloride intake greatly and sodium retention by kidney greatly enhances development of ascitis (Kaneko *et al*., 2008).

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. 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 isoenzymes are present in kidneys, intestine, placenta and bone but elevation in its level in >1 year old dog is usually indicative of hepatic origin unless bone disease coexists, since isoenzymes 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). 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. The findings of the present study were comparable to that of Sevelius (1995). But hypoalbuminemia may also occur without impairement 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 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 Varshney 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 by 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. Haemolysis may produce substrates in the form of proteins that would require deamination and consequently lead to hyperammonaemia. Similar observations were made by Chohan *et al*. (2009). This might be due to the renal abnormalities and the urinary retention due to obstruction.

CONCLUSION

Haemogram revealed anemia thrombocytopenia and increased clotting time with neutrophilic leucocytosis. The indicators of ascitis were decreased total protein, decreased A/G ratio, hypercholesterolemia and hypoglycemia. Ultrasonography with biochemical observations was observed to be a significant diagnostic tool in ascitis.

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