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Haematological and Biochemical Observations in Canine Ascites Associated with Hepatopathy

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A B S T R A C T

Dogs of multiple breeds within the age group of 3-6 years, presented to VCC, Rajendranagar with clinical signs of distended abdomen, general weakness and inappetence were studied and grouped into two groups. The diseased animals (Group II), comprising of 8 affected animals and control (Group I), comprising of 6 apparently healthy animals. These animals were subjected to thorough clinical and physical examination, haemato-biochemical, serum biochemistry, ultrasonographical & radiographical examinations. The haematological tests revealed anaemia in the affected dogs, while the serum biochemistry revealed an abnormal increase in the liver enzymes with significantly decreased values of albumin, globulin, total proteins, and albumin/globulinratio. Radiography documented ground glass appearance of the abdomen, hepatomegaly and other hepatopathies in both the groups. Anechoic abdominal free fluid was noticed and samples were collected by ultrasound-guided abdominocentesis for analysis. The resultant diagnosis made was ascites associated with chronic hepatitis, hepatomegaly, and other hepatopathies in dogs.

Introduction

Ascites in canines is a very common condition with 68% occurrence and mainly observed as accumulation of clear serous fluid (exudate/transudate) in the peritoneal cavity (Kumar, 2002), characterized by abdominal distension. Ascites is a polyetiological progressive pathological condition that has been attributed to chronic hepatic failure, congestive heart failure, nephritic syndrome, malnutrition, ancylostomiasis and protein losing enteropathy in canine (Turkar *et al.,* 2009). It results in abdominal swelling, dyspnoea, lethargy, anorexia, vomiting, weakness, discomfort. Common clinical signs of ascites in dogs are inappetence, halitosis, vomiting, respiratory distress, lethargy, persistent distended abdomen, pedal oedema, melena with pale to icteric mucus membranes and in a few cases, distended jugular vein, cough, cyanotic tongue, seizures and syncope (Hernaez and Hamilton, 2016). On radiographic examination, ground-glass appearance of the abdomen with diffused fluid opacity is observed. Ultrasonography reveals mixed echogenicity of visceral organs, such as liver (hyper-echoic) floating in anechoic abdominal fluid. The common findings of the electrocardiogram are low

voltage QRS complexes, deep Q wave with tachy-arrhythmia (Kumar and Srikala, 2014). Chronic inflammation of liver or chronic hepatitis and liver cirrhosis is clinically manifested in ascites cases, as a sequalae to an idiopathic origin, drug toxicity, portal hypertension, neurohormonal activation and hypoalbuminemia (Webster *et al.,* 2019). Origin of the hepatic disorder is assessed by a group of diagnostic tests, such as ultrasonography, radiography, serum biochemistry (AST, ALT, ALP) and biomarker (micro-RNA-122), while ultrasound-guided liver biopsy is indicated for confirmation of liver cirrhosis (Bhadesiya *et al.,* 2015). Ascites occurs due to increase in hydrostatic pressure (cirrhosis and congestive heart failure), reduced oncotic pressure (nephrotic syndrome) and increase in peritoneal fluid production. Ascites is asyndrome that arises as a complication to a primarydisease; therefore, investigation should be aimed at identifying the primary etiological factor. The treatment protocol of ascites is dependent on identification of the underlying pathogenesis (Meindel and Pohlman, 2013). The aim of the present study was to detect changes in hemogram and serum biomarker, ultrasonographic findings in ascites of hepatic origin.

Materials and Methods

The present work consists of sixteen clinical cases presented to Teaching Veterinary Clinical Complex and Department of Veterinary Clinical Medicine, Rajendranagar. Eight animals affected with ascites of hepatic origin were grouped as Group II with age (3-6 years), sex, breed, chief complaint, patient's regular diet, duration of the disease, history of other illness and post-treatment were recorded while control (Group I) comprised of 6 apparently healthy animals, subjected to various detailed clinical examinations, haemato-biochemical alteration (TLC, Hb, AST, ALT, GGT, TSP, Albumin, Globulin, A: G ratio, BUN, urea, Creatinine), radiography (lateral and ventrodorsal orthogonal view) and ultrasonography. The haematological examination revealed mild decrease in the Haemoglobin concentration, accompanied by leucocytosis and neutrophilia (Rakesh and Shanti, 1994 and Kumar, 2022). B-mode ultrasonography was conducted by M/s Esoate and a convex probe ultrasound machine (microconvex probe of 4.2-10 MHz) used to evaluate the altered function of hepatic parenchyma, gallbladder, biliary system and portal vasculature. Ultrasound guided abdominocentesis was done to collect the peritoneal fluid (Hall and German, 2005). Ascitic fluid was removed by abdominocentesis, inserting 18 gauze needle 2-3 cm caudal and towards left from the midline of umbilicus at 30 40-degree angle, to relieve abdominal distension to an extent, whichever case had the necessity.

Results and Discussion

The recorded clinical signs such as the persistent distended abdomen, lethargy, respiratory distress, pale to icteric mucus membranes and inappetence simulated with the studies conducted by Wadhwa *et al.,* (1995) and Dabas *et al.,* (2011). The average of the results obtained were classified into the controlled and diseased groups as depicted below.

The haematological examination revealed mild decrease in the Haemoglobin concentration, accompanied by leucocytosis and neutrophilia. The biochemical evaluation revealed increased values of SGPT and SGOT, hypoproteinaemia, hypoalbuminemia and therefore, significantly altering the values of albumin to globulin ratio. Serum blood urea nitrogen and creatinine values were unremarkable and well within the normal range. The radiographic examination documented the characteristic ground glass appearance of the abdomen, while the ultrasonography findings recorded anechoic spaces of ascitic fluid accumulation. The kidneys were normal in shape and size, with smooth capsule and cortex of uniform echogenicity. The USG of liver showed hyperechoic liver tissue along with hepatic congestion, irregularity of liver edge and hepatomegaly. The detailed biochemical analysis confirmed that ascites was of hepatic origin and the same results were observed by Kumar *et al.,* (2012).

The cases were treated with Inj. Dextrose Normal Saline @ 10 ml/kg b.wt, Inj. Amoxicillin & Cloxacillin @ 20 mg /kg b.wt, Inj. Furosemide @ 2 mg/ kg b.wt, Inj. Chlorpheniramine maleate @ 0.5 mg/kg b.wt and advised Syrup. Tefroliv forte (Silimarin) twice daily, with restricted salt diet. Ascites is usually characterized by abdominal swelling, respiratory distress, lethargy, anorexia, vomiting, weakness. Dyspnoea in ascites cases might be due to excessive fluid accumulation which exerts pressure on diaphragm resulting in its restricted movement (Samad, 2019). Increase in total leukocyte count and neutrophils may be due to granulomatous hepatitis, hepatic cirrhosis, hepatic abscess and hepatic neoplasia (Saravanan *et al.,* 2012). The rise in ALP indicates hepatocellular injury, while the decrease in albumin and total proteins might be attributed to the liver

damage while its major role of plasma protein synthesis, degradation and synthesis of other proteins might have been affected. The low serum albumin concentration can be explained by the decreased osmotic pressure in hypoalbuminemia conditions, resulting in an increased hydrostatic pressure causing fluid to escape from the vasculature into the body cavity. Hypoalbuminemia results from albumin loss, decreased production, or other inflammatory conditions (Center, 1989; Parker, 2002).

Higher SGPT and SGOT values were due to hepatic insufficiency with hepatic damage resulting into leakage of enzyme from hepatocytes into blood stream (Cornelius *et al.,* 1975) and hypoproteinaemia (Skardova, 1991). Serum blood urea nitrogen and creatinine values were unremarkable, indicating normal renal function. The detailed biochemical analysis confirmed that ascites was of hepatic origin and the same results were observed in the statistical studies conducted by Kumar *et al.,* (2012). Hypoalbuminemia rarely is the primary cause of ascites but definitely contributes to the continuation of the condition. Serum glutamic-pyruvate transaminase (SGPT) and serum glutamic-oxalacetic transaminase (SGOT) are leakage enzymes indicative of hepatocellular damage. Hepatocellular leakage is of multifactorial origins, including hypoxia, toxins, drugs, hepatitis, fatty change and degeneration due to metabolic disorders.

Table.1 Haematological examination details

Parameter	Reference	Control Group	Diseased Group
RBC	5.5-8.5× $10^{(6)}$ /uL	5.43±0.21	4.15±0.50
Hb	12-18 g/dL	11.17±0.34	8.58±0.73
PCV	37-55 %	35.06±1.88	27.53±2.32
WBC	6-17× 1000 /uL	15.98±0.36	17.56±3.22
Neutrophils	60-80%	73.17±0.60	81.72±3.78
Lymphocytes	12-30%	22.67±0.49	23.12±3.01
Basophils	3-10%	2.80±0.30	5.29±0.42***
Eosinophils	2-10%	1.83±0.21	1.60±0.20
Platelets	200-500 ×1000/uL	265.8±26.79	228.7±35.16

Table.2 Haemato-biochemical alteration details

Parameter	Reference	Control Group	Diseased Group
ALT	20-102 IU/L	46.83 ±2.93	68.05±10.81
ALP	20-156 IU/L	65.67±4.39	237.2±54.56
Total protein	5.5-7.5 g/dL	6.15±0.27	4.28±0.48
Albumin	2.2-4 g/dL	2.98 ±0.14	2.11±0.66***
Total bilirubin	0.2-0.6 mg/dL	0.75 ±0.11	1.54±0.38
Direct bilirubin	0.06-0.12 mg/dL	0.40±0.05	1.09±0.32
Indirect bilirubin	0.15-0.30 mg/dL	0.12 ±0.01	0.15±0.06
Creatinine	0.5-1.5 mg/dL	1.0±0.05	1.56±0.28
BUN	10-28 mg/dL	24.33±1.40	22.29±3.83
Urea	12.5-53.5 mg/dL	31.17±3.25	51.30±12.66

Lesions accompanying leakage of enzymes may be fatty change, 14 hepatocellular necrosis, fibrosis, or biochemical lesions. Levels of significance for these enzymes can be found in most clinical pathology references. It is important to remember that the magnitude of the increase in enzyme levels is proportionate to the damage, but no evaluation of the reversibility of the damage can be made from these levels. Therefore, persistence of high levels of these enzymes is a poor prognostic sign. If chronic-active hepatitis is diagnosed, the treatment will revolve around arresting the inflammation, correcting nutritional derangements, resolving fibrosis and resolving other associated complications. Corticosteroids will decrease

inflammation but may increase protein catabolism and increase ammonia production, which is undesirable. A high quality, low protein diet in numerous small feedings is recommended to diminish bacteria-induced conversion of excess protein into ammonia in the colon. Adequate energy required in the diet to minimize catabolism of proteins. Corticosteroids and colchicine may help resolve fibrosis. Antibiotics should be used to minimize bacterial infection via the portal circulation.

Author Contributions

Jatavath Jyothi: Investigation, formal analysis, writing—original draft. Rishika Benerji: Validation, methodology, writing—reviewing.

Data Availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

References

Bhadesiya C M, Jani R G, Parikh P V, Pandey A M, Rao N, Shai A. Hematobiochemistry and imaging study on ascites with hepatic and cardiac origin in a German shepherd pup. International Research Journal of Chemistry. 2015; 11:14-22.

Center, S.A. (1989). Pathophysiology and laboratory diagnosis of liver disease. In: Textbook of Veterinary Internal Medicine. S.J. Ettinger (edt.), W.B. Saunders & Company, Philadelphia.

Cornelius, L.M., Thrall, D.E., Halliwell, W.H., Frank, G.M., Kern, A.J. and Woods, C.B. 1975. Anomalous portosystemic anastomoses associated with chronic hepatic insufficiency in six young dogs. Journal of the American Veterinary Medical Association. 167(3): 220-228.

Dabas, V.S., Suthar, D.N., Chaudhari, C.F., Modi, L.C. and Vihol, P.D. 2011. Ascites of Spleenic Origin in a Mongrel Female Dog-A Case Report. Veterinary World. 4(8): 376.

Hall E J and German A J. Laboratory evaluation of hepatic disease," In BSAVA Manual of Canine and Feline Clinical Pathology, E. Villiers and L. Blackwood, Eds BSAVA, Gloucester, UK, 2nd edition; c2005. p. 184-206.

Hernaez R and Hamilton J P. Unexplained ascites. Clinical Liver Disease. 2016;7(3):53-56. https://doi.org/10.1002/cld.537

Kumar, V., Kumar, A., Varshney, A.C., Tyagi, S.P., Kanwar, M.S. and Sharma, S.K. (2012). Diagnostic imaging of canine hepatobiliary affections: A review. Vet. Med. International. https://doi.org/10.1155/2012/672107

Kumar, S. 2002. Thesis Abstract. Journal of Canine Development and Research. 2:78

Kumar S K, Srikala. Ascites with right heart failure in a dog: diagnosis and management. Journal of Advanced Animal Research. 2014;1(3):140-144. https://doi.org/10.5455/javar.2014.a15

Kumar R, Gupta D K, Gajbhiye S, Kumar S, Ramteke M, Pankaj D K, *et al.,* Occurrence of ascites in dogs. The pharma Innovation Journal. 2022;11(7S):1201-1204.

Meindel M and Pohlman L. Abdominal effusion in a dog. Cliniciansbrief.com; c2013. p. 96-98.

Parker, M. D. (2002). An unusual cause of abdominal distention in a dog. Vet. Med. 97: 189-195.

Rakesh and Shanti. 1994. Ascites in a bitch. Indian Journal of Veterinary Medicine. 18: 56-57.

Samad MA (2019). A systematic review of pre-clinical and clinical research reports on small ruminants published during the last six decades in the then East Pakistan and in Bangladesh. J. Vet. Med. OH Res. 1 (2): 111-183. https://doi.org/10.36111/jvmohr.2019.1(2).0010

Saravanan, M., Sharma, K., Kumar, M., Vijaykumar, H. and Mondal, D.B. (2012). Analysis of serum ascites albumin gradient test in ascitic dogs. Vet. World 5: 285-287.

Skardova, I. (1991). Occurrence of ascites abdominalis in dogs. J. Nutr. 121: S159-S160

Turkar S, Randhwa C S, Uppal S K. Ascites associated with Ancylostomiasis in a pup: A case report. Intas Poli. Veterinary. 2009;10(II):357-359.

Wadhwa, D. R. ; Randhawa, S. S. ; Nauriyal, D. C. ; Singh, K. B., 1995. Clinico-biochemical and therapeutic studies on brewer's grain toxicity in buffaloes. Indian J. Vet. Med., 15 (2): 87-89

Webster C R L, Center S A, Cullen J M. ACVIM consensus statement on the diagnosis and treatment of chronic hepatitis in dogs. J Vet Intern Med. 2019; 33:1173-1200. https://doi.org/10.1111/jvim.15467

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