Journal of Entomology and Zoology Studies

Available online at www.entomoljournal.com

E-ISSN: 2320-7078
P-ISSN: 2349-6800
www.entomoljournal.com
JEZS 2020; 8(2): 1942-1944

Received: 25-01-2020
Accepted: 27-02-2020

Kanwarpal Singh Dhillon
Assistant Professor and Corresponding author, Department of Veterinary Medicine, Khalsa College of Veterinary and Animal Sciences (KCVAS), Amritsar, Punjab, India

Simran Jot Kaur
Executive Technical Manager, Paras Nutrition Private Limited, Moga, Punjab, India

Alamjit Singh
B.V.Sc. Student, Khalsa College of Veterinary and Animal Sciences, Amritsar, Punjab, India

Corresponding Author:
Kanwarpal Singh Dhillon
Assistant Professor and Corresponding author, Department of Veterinary Medicine, Khalsa College of Veterinary and Animal Sciences (KCVAS), Amritsar, Punjab, India

Ascites of hepatic origin in a female beagle pup: Diagnosis, treatment and management

Kanwarpal Singh Dhillon, Simran Jot Kaur and Alamjit Singh

Abstract
A three months old female Beagle puppy was presented with a history of enlarged abdomen, inappetence and weakness since two weeks. Clinical examination revealed fever, fluid thrill on palpation of abdomen, dyspnoea, dehydration, rough body coat, pale mucous membranes and tachycardia was evident. Haematological findings revealed anemia, neutrophilia along with thrombocytopenia. Serum biochemical parameters evinced increased levels of ALT (260 U/L), AST (186 U/L) and ALKP (425 U/L) but decreased in total protein (3.2 g/dl) and blood glucose (52 mg/dl) level suggesting hepatic involvement. Furthermore, abdominal ultrasonography revealed accumulation of fluid in abdominal cavity with hyperechoic hepatic structure. Abdominal radiograph revealed characteristic 'Ground glass' appearance over abdomen. The haemoprotozoal and fecal examination showed negative result. Hence, the puppy was diagnosed as ascites of hepatic origin resulting hypoproteinemia and the condition was successfully managed with fluid, antibiotics, diuretics and liver tonics along with protein rich and but salt free diet. The significance of diagnostic protocol, haematobiochemistry and treatment has been discussed.

Keywords: Beagle, pup, ascites, hepatic, diagnosis, treatment, management

1. Introduction
Increasing interest has been observed among pet owners to keep dogs as a household pet/companion in urban as well as rural Punjab. Dogs do suffer from several infectious disease conditions such as bacterial, fungal, viral, endo-ectoparasites, haemoprotozoan diseases etc. for which vaccination and deworming protocols have been adopted. However, there are number of non-infectious diseases such as vitamin and mineral deficiencies, traumatic injuries, fractures and many more systemic diseases and disorders which contribute to variable morbidities and mortalities and require due clinical attention [1].

Ascites in dogs is one of such non-infectious diseases, a frequently encountered entity at clinics. Ascites is commonly defined as the pathologic accumulation of fluid in the abdominal cavity due to escape of fluid between the parietal and visceral peritoneum from blood vessels, lymphatics, internal organs or abdominal masses [2] and is also one of the sequelae of various primary diseases [3]. It is usually a secondary sign of a number of hepatic, renal, cardiac and many other systemic diseases and disorders of dogs [4] and thus, it becomes necessary to rule out organ involvement in development of clinical ascites by use of biochemical tests as well as imaging studies. Present report deals with a case of ascites in a young pup with hepatic involvement with special reference to usefulness of chemical analysis of blood, serum and ascitic fluid as well as imaging studies.

2. History, Laboratory Investigation and Diagnosis
A three months old female Beagle pup was presented to Veterinary Clinical Complex, Khalsa College of Veterinary and Animal Sciences, Amritsar with a complaint of progressive abdominal distension, inappetence, and lethargic since two weeks. The animal was repeatedly treated by local veterinarians without success. History regarding deworming and vaccination was complete. Clinical examination revealed pyrexia (103.8°F), distended abdomen on both sides with palpable fluid thrill (Fig. 1), dyspnoea, dehydration, rough body coat, pale mucous membranes and tachycardia was evident. Abdomino-centesis was performed to ameliorate the difficult respiration and about 50 ml fluid was removed and evaluated. The fluid was a transudate (total protein 2.0 gm/dl) and had no diagnostic constituents in cytological examination. The fecal examination showed negative result. Haematological findings revealed anemia and neutrophilia

(Hb- 6.2 g/dl, TEC- 4.1 $\times 10^6$/µl, TLC- 8.2 $\times 10^3$/µl, N- 82/µl, L-16/µl, E- 2/µl) along with thrombocytopenia (PLT- 98 $\times 10^5$/µl). Blood smear examination for haemoprotozoa was negative. Serum biochemistry revealed higher levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALKP) were 260 U/L, 186 U/L and 425 U/L respectively, and decreased level of total protein and blood glucose were 3.2 g/dl and 52 mg/dl, respectively. Blood urea nitrogen (BUN), serum creatinine and total bilirubin levels were 22.4 mg/dl, 0.8 mg/dl and 0.6 mg/dl, respectively. Radiographic examination revealed the *ground-glass* appearance in abdominal (Fig. 2) and thoracic cavity with slight pneumonic changes in lungs. Ultrasonographic examination revealed large amount of anechoic fluid in abdominal cavity indicating ascites. No abnormal echogenicity was noticed with the abdominal organs such as spleen, kidney and bladder, except liver; where there was presence of hyperechoic to mixed echogenecity. The electrocardiographic findings revealed low voltage QRS complexes, which were suggestive of effusion in body cavities.

3. Treatment and Discussion

Based on the history, clinical-haemato-biochemical examinations, radiographic and ultrasonographic findings, the case was diagnosed as ascites of hepatic origin and treated accordingly. Treatment was instituted with Inj. Dextrose 10% @10 ml/kg IV BID ×7 days, Inj. Ceftriaxone @25mg/kg IM BID ×5 days, Tab Lasilactone 50 (Frusemide 20mg + Spironolactone 50mg) @¼ tab/5kg PO BID ×7 days and Inj. Tribivet (Vitamin B_1, B_6 and B_{12}) @0.5 ml IM OD for a week along with supportive therapy with syrup Livotas pet (Liver tonic) @1 tsf. PO BID for 14 days. The owner was advised to feed chicken soup and egg white twice a day for 14 days along with restricted dietary sodium intake. Subsequently, reduction in abdominal distension was observed from 3rd day of treatment and the animal became completely healthy by 10th day (Fig. 3). The haemato-biochemical estimations revealed almost normal levels of ALT, AST and total protein with increase in haemoglobin levels (10 gm/dl). The owner was advised continue to feed chicken soup, egg white and liver tonic for next ten days and follow up thereafter a week. After ten days owner informed that the pup has completely recovered without any recurrence and thriving well.

Low levels of Hb, TEC and platelets were in correlation with findings of Bhatt *et al.* [5], Ihedioha *et al.* [4] and Saravanan *et al.* [6]. Increased ALT and AST indicates hepatic insufficiency with extensive damage resulting into the leakage of enzymes from hepatic cell into blood stream was in correlation with findings of Vijayakumar [2], Bhatt *et al.* [5] and Jain *et al.* [7]. Normal concentrations of BUN and creatinine indicate normal function of kidney. The hypoglycemia and hypoproteinaemia is the indicative of hepatic insufficiency [8,9]. Microscopic examination of ascitic fluid did not reveal presence of epithelial cells or pus cells. Similar findings were reported by Ihedioha *et al.* [4] and Jain *et al.* [7]. Direct examination of faecal sample revealed negative results were in correlation with findings of Chakrabarti *et al.* [10]. Lateral abdominal radiograph revealed characteristic 'Ground glass' appearance due to presence of ascitic fluid. Abdominal ultrasonography revealed presence of anechoic (black) free floating abdominal fluid was in correlation with findings of Saravanan *et al.* [6]. Caudal lobe of liver showed hyperechoic foci suggestive of hepatic involvement in development of clinical ascites. Kidneys did not show any ultrasonographic changes. Low voltage QRS complexes with reduced R wave amplitude was considered as significant electrocardiographic abnormality of ascites dogs, as described by Ettinger and Feldman [1].

Dextrose 10% has been well established in checking dehydration, depleting liver glycogen and preventing tissue catabolism. Diuretic combination of spironolactone and furosemide had better efficacy than Furosemide alone [11]. Egg albumen was given as protein supplements that have high biological value [12]. The animal was given sodium free diet to check sodium retention or secondary hyperaldosteronism [13]. Ascites, a non-infectious disease though itself does not create much problem, but the disease process in any vital organs like liver, heart, kidney causing ascites becomes a serious and major health problem in canine population. Any delay in treatment and management may lead to various complications and ultimately may lead to death.

Fig 1: Distended abdomen

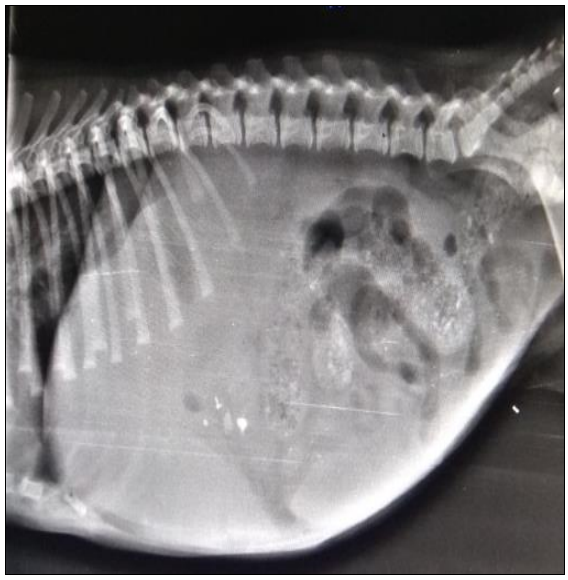

Fig. 2: X-ray of abdomen showing "glass ground appearance"

Fig 3: Recovered pup after treatment

4. Conclusion

It is hereby concluded that anamnesis, clinical-haemato-biochemical examinations, radiographic and ultrasonographic findings, provide confirmation of the ascites due to hepatic origin and treated accordingly

5. Acknowledgements

The author is thankful to the Principal, KCVAS, Amritsar for providing necessary facilities to carry out this study.

6. References

1. Ettinger SJ, Feldman EC. Diseases of the Dog and Cat. In: Textbook of Veterinary Internal Medicine. 6th Edn, WB Saunders, Philadelphia, 2005, 137-145, 632-636.
2. Vijayakumar, G. Therapeutic management of ascites in dogs. Intas Polivet. 2002; 3:179-184.
3. Mishra B. Survey study of gastro-intestinal disorders in hospital population of dogs with special reference to epidemiology, clinico-pathology and therapeutics aspects of ascites. M.V.Sc. Thesis, College of Veterinary Science & Animal Husbandry, Anand Agricultural University, Anand, Gujarat, 2015.
4. Ihedioha JI, Anosa VO, Eseivo KAN. Prevalence of clinicopathologic findings associated with ascites in dogs in Enugu State, Nigeria. Comparative Clinical Pathology. 2013; 22:185-193.
5. Bhatt P, Singh GD, Dabas YPS. Ancylostomosis associated ascites in a dog and its therapeutic management. Intas Polivet. 2011; 12:104-106.
6. Saravanan M, Mondal DB, Sharma K, Mahendram K, Sasikala V. Utility of Serum Ascites Albumin Gradient in dogs as indicator of hepatobiliary disorders induced ascites. Indian Veterinary Journal. 2014; 91:33-35.
7. Jain S, Shakkarpude N, Chandra N, Soni A. Hematobiochemical changes and therapeutic aspects of ascites with hepatic involvement in dogs. Environment & Ecology. 2013; 31:1205-1206.
8. Skardova I. Occurrence of ascites abdominalis in dogs. The Journal of Nutrition. 1991; 121:159-160.
9. Pradhan MS, Dakshinkar NP, Waghaye UG, Bodkhe AM. Successful treatment of ascites of hepatic origin in dog. Veterinary World. 2008; 1:23.
10. Chakrabarti A, Amin R, Samanta TK. Clinicotherapeutic studies on ascites in dog. Indian Journal of Veterinary Medicine. 1994; 142:187.
11. Varshney JP. Ibid. 2001; 21:87. (Original not seen. Cited by Ghosh CK, Sarkar S, Chakrabarti A. Hepatic disorder related canine ascites - A case report. Intas Polivet. 2008; 9:177-179.
12. Mosier JE, Bradley WB. The Liver. In: Canine Medicine by EJ Catcott. 4th Edn, American Veterinary Publications, California, 1979, 404.
13. Wyllie R, Arasu TS, Fitzgerald JF. Ascites: Pathophysiology and management. The Journal of Pediatrics. 1980; 97:167.