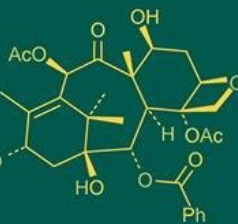
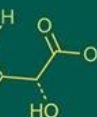
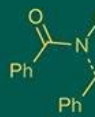
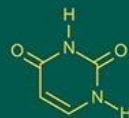


International Journal of Advanced Biochemistry Research



ISSN Print: 2617-4693
 ISSN Online: 2617-4707
 NAAS Rating (2025): 5.29
 IJABR 2025; 9(12): 956-959
www.biochemjournal.com
 Received: 15-11-2025
 Accepted: 18-12-2025

All authors' names and
 affiliations are given below,
 after the references.

Diagnosis and therapeutic management of ascites of hepatic origin in a Labrador dog: A case report

K Padmaja, Arukontham Deepika, P Suman, K Ramesh, Kasthuri Dhileep, T Rachana, S Vijaya Lakshmi, B Vignatha and Ch. Shiva Krishna

DOI: <https://www.doi.org/10.33545/26174693.2025.v9.i12l.6695>

Abstract

A one and half year old male Labrador dog was presented with the history of inappetance, lethargy, weakness and distended abdomen since 1 month. Physical examination revealed fluid thrill upon tactile percussion. Clinical examination revealed pale mucous membrane, dyspnea, dehydration, rough body coat and tachycardia. Haematological findings revealed anemia. Serum biochemical parameters evinced increased levels of ALT (566.1 U/L) and AST (120 U/L) but decreased in total protein (3.1 g/dl) and blood glucose (52 mg/dl) level suggesting hepatic involvement. Hepatosonogram revealed anechoic fluid in abdominal cavity with hyperechoic hepatic parenchyma and rounded borders. Skiagram revealed characteristic 'Ground glass' appearance over abdomen. Ascitic fluid analysis revealed moderately transudate fluid with SAAG less than 1.1 g/dl. The above clinical signs paved way for the etiology of ascites which could be of hepatic origin. Therapeutic management included S-adenosyl methionine and milk thistle for 30 days and was treated with antibiotic, diuretics and supportive therapy along with protein rich and salt restricted diet. Post treatment serum hematobiochemical parameters were back to normal along with clinical improvement.

Keywords: Percussion, Abdominocentesis, SAAG, Ascitic fluid analysis

Introduction

Ascites is a serious, possibly life-threatening medical condition and a frequently encountered entity in dogs. It is a common clinical manifestation in dog as a result of fluid accumulation in the peritoneal cavity due to escape of fluid between the parietal and visceral peritoneum from blood vessels, lymphatics, internal organs or abdominal masses. It is also one of the sequelae of various primary diseases (Ettinger *et al.*, 2005) [3]. Which include chronic hepatic failure, congestive heart failure, nephritic syndrome, malnutrition, ancylostomiasis and protein losing enteropathy in canine (Randhawa *et al.*, 1980) [9]. It has become necessary to rule out organ involvement in development of clinical ascites by use of biochemical tests as well as imaging studies. Ascites can be treated after diagnosis of underlying cause (Turkar *et al.*, 2009) [9].

Case history and clinical observations

A one and half year old female Labrador was presented to Veterinary Clinical Complex, College of Veterinary Science, Rajendranagar, Hyderabad with a major complaint of abdominal distension, inappetance, lethargy and weakness. Vaccination and deworming were upto date. Physical examination revealed fluid thrill upon tactile percussion and pear shaped abdomen (Fig. 2), heart rate was 120 BPM, respiratory rate was 24 BPM and temperature was 102° F. Clinical examination revealed pale mucous membrane, dyspnea, dehydration and rough body coat. A series of investigations were ZAA advised to rule out the etiology of ascites. Hematological examination (table 1) revealed normal parameters except for hemoglobin (9.03 g/dl) which is less than normal suggesting anemia. Serum biochemistry (table 2) revealed elevated levels of ALT (566.1 U/L) and AST (120 U/L) and decreased total proteins, Serum albumin and glucose levels suggesting hepatic damage. Creatinine and BUN levels were normal suggestive of no renal involvement. Fecal sample examination revealed no parasitic ova's. Skiagram of abdomen revealed ground glass appearance (Fig. 3)

Corresponding Author:
P Suman
 Department of Veterinary
 Medicine, College of Veterinary
 Science, Rajendra Nagar,
 Hyderabad, Telangana, India

suggestive of grade 2 ascites with gas filled intestines floating in ascitic fluid. Sonogram revealed hyperechoic liver parenchyma with rounded borders floating in anechoic fluid (Fig. 4) with no change in echogenicity in other organs.

Abdominocentesis (Fig. 5) was done to relieve stress on diaphragm and ascetic fluid was subjected to various physical, biochemical and microscopic examination to determine the etiology of ascites. Physical examination of ascitic fluid revealed yellow colour (Fig. 6) Biochemical constituents of ascitic fluid, which include Total Protein 2.65 grams/dl, Albumin 1.20g/dl, Specific gravity 1.015 and pH 7.6 was estimated. Cytology of the ascitic fluid revealed few mesothelial cells and neutrophils indicating moderately transudate fluid. SAAG was estimated which is a marker of portal hypertension. SAAG was calculated which is less than 1.1 g/dl suggesting low gradient ascites and suggesting inflammation and vascular leakage. Based on the above physical, clinical, biochemical, radiographic and sonographic examination the above case was diagnosed as ascites due to hepatic origin.

Treatment and Discussion

The animal was treated with Inj. DNS 10ml/kg bwt IV for 3 days, Amoxicillin @ 12.5 mg/kg bwt IV for 5 days, inj. Chlorpheniramine maleate @ 1ml IM 3 days, inj. Furesamide @ 2 mg/kg bwt for 7 days, inj. Pantoprazole @ 0.4 mg/kg IV for 3 days, inj. Astymin 1ml/kg bwt IV for 7 days, and Inj. Toxol 1ml/kg bwt IM for 3 days, with an additional liver supportive therapeutic regimens include Susp. SAME pet @ 20 mg/kg bwt PO, Milk thistle @ 6 mg/kg bwt PO for 30 days. The owner was advised to give the animal egg white and chicken soup for 1 month along with salt restricted diet. Reduction in abdominal distension was seen from day 3 (Fig. 7, 8) of treatment and the animal became apparently healthy by day 10. The hemato biochemical parameters were back to normal. The owner was advised to continue egg protein and chicken soup.

Table 1: Haematological parameters in dogs affected with Ascites

S. No	Parameters	Before Treatment	After Treatment
1	Hemoglobin (g/dl)	9.03	11.23
2	Total erythrocyte count (x106/ μ l)	4.11	4.55
3	Total leucocyte count (x103/ μ l)	28.02	16.35
4	PCV (%)	34.4	40.45
5	Neutrophils (%)	81	67
6	Lymphocytes (%)	20	29
7	Monocytes (%)	01	01
8	Eosinophils (%)	02	01

Table 2: Biochemical parameters in dogs affected with Ascites

S. No	Parameters	Before treatment	After Treatment
1	ALT (U/L)	566.1	123
2	AST (U/L)	120	101
3	ALP (U/L)	211	89
4	Total bilirubin (mg/dl)	1.23	0.98
5	Total Protein (g/dl)	3.11	6.2
6	Albumin (g/dl)	2.03	2.98
7	Blood glucose (mg/dl)	56	102
8.	BUN (mg/dl)	13.03	18.97
9.	Creatinine (mg/dl)	1.02	1.09



Fig 1: Marked abdominal distention



Fig 2: Pear shaped abdomen



Fig 3: Skiagram of lateral view of abdomen showing ground glass appearance with gas filled intestines



Fig 4: Hepatosonogram showing hyperechoic liver parenchyma with rounded liver borders



Fig 5: Abdominocentesis



Fig 6: Yellow colour ascitic fluid-modified transudate



Fig 7: Reduced distension of abdomen after treatment (day 10)



Fig 8: Reduced distension of abdomen after treatment (Top line view)

Low levels of Hb correlation with findings of (Bhatt *et al.*, 2011) [1] (Ihedioha *et al.* 2013) [4] and Saravanan *et al.* 2014) [8]. 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, 2002, Bhatt *et al.* 2011, and Jain *et al.* 2013) [10, 1, 5]. BUN and creatinine were normal indicate normal function of kidney. Hypoglycemia and hypoproteinaemia was indicative of inappetence/anorexia complemented by malabsorption from the intestines, decreased gluconeogenesis and glucogenolysis. Microscopic examination of ascitic fluid did not reveal presence of epithelial cells or pus cells. Similar findings were reported by (Ihedioha *et al.* 2013 and Jain *et al.* 2013) [4, 5]. Direct examination of faecal sample revealed no parasitic eggs which were in correlation with findings of (Chakrabarti *et al.* 1994) [2]. Skiagram of lateral abdomen revealed characteristic 'Ground glass' appearance due to presence of ascitic fluid. Abdominal ultrasonography revealed presence of free floating anechoic abdominal fluid which was in correlation with findings of (Saravanan *et al.* 2014) [8]. Hepatic sonogram revealed hyperechoic liver suggestive of hepatic involvement in development of clinical ascites. Kidneys did not show any ultra-sonographic changes. Dextrose 10% has been well established in checking dehydration, depleting liver glycogen and preventing tissue catabolism, and antibiotics were given to prevent any secondary bacterial infection. Egg albumin was given as protein supplements that have high biological value (Mosier JE, *et al.*, 1979) [7]. The animal was given sodium free diet to check sodium retention or secondary hyperaldosteronism (Wyllie R *et al.*, 1980) [11]. Ascites is a clinical sign, not a disease, and it can occur due to multiple non-infectious as well as infectious causes. Finding the etiology is important in treating ascites. Delay in treatment may leads some other complication and may even lead to death some times.

Conclusion

In ascites effected dog, invesitigations like Physical, clinical, hematological, serobiochemical, radiography, ultrasonographic examination paves way for the diagnosing the origin of ascites as hepatic and was treated accordingly.

Reference

1. Bhatt P, Singh GD, Dabas YPS. Ancylostomosis associated ascites in a dog and its therapeutic management. *Intas Polivet.* 2011;12:104-106.
2. Chakrabarti A, Amin R, Samanta TK. Clinicotherapeutic studies on ascites in dog. *Indian J Vet Med.* 1994;14(2):187-187.
3. Ettinger SJ, Feldman EC. Diseases of the dog and cat. In: Ettinger SJ, Feldman EC, editors. *Textbook of Veterinary Internal Medicine.* 6th ed. Philadelphia: WB Saunders; 2005. p. 137-145, 632-636.
4. Ihedioha JI, Anosa VO, Eseivo KAN. Prevalence of clinicopathologic findings associated with ascites in dogs in Enugu State, Nigeria. *Comp Clin Pathol.* 2013;22:185-193.
5. Jain S, Shakkarpude N, Chandra N, Soni A. Hematobiochemical changes and therapeutic aspects of ascites with hepatic involvement in dogs. *Environ Ecol.* 2013;31:1205-1206.
6. Mishra B. Survey study of gastro-intestinal disorders in hospital population of dogs with special reference to

- epidemiology, clinico-pathology and therapeutic aspects of ascites. M.V.Sc. Thesis. Anand: College of Veterinary Science & Animal Husbandry, Anand Agricultural University; 2015. p. 1-150.
7. Mosier JE, Bradley WB. The liver. In: Catcott EJ, editor. Canine Medicine. 4th ed. California: American Veterinary Publications; 1979. p. 404-404.
 8. Saravanan M, Mondal DB, Sharma K, Mahendram K, Sasikala V. Utility of serum ascites albumin gradient in dogs as an indicator of hepatobiliary disorder-induced ascites. Indian Vet J. 2014;91:33-35.
 9. Turkar S, Randhawa CS, Uppal SK. Ascites associated with ancylostomiasis in a pup: a case report. Intas Polivet. 2009;10:357-359.
 10. Vijayakumar G. Therapeutic management of ascites in dogs. Intas Polivet. 2002;3:179-184.
 11. Wyllie R, Arasu TS, Fitzgerald JF. Ascites: pathophysiology and management. J Pediatr. 1980;97:167-167.

Author's Details**K Padmaja**

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

Arukontham Deepika

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

P Suman, K Ramesh

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

Kasthuri Dhileep

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

T Rachana

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

S Vijaya Lakshmi

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

B Vignatha

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India

Ch. Shiva Krishna

Department of Veterinary Medicine, College of Veterinary Science, Rajendra Nagar, Hyderabad, Telangana, India