



Case Study

A Case Study on Ascites of Hepatic Origin and Their Proper Management in a Male German Shepherd Dog

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Abstract

A male German shepherd dog of 11 months was presented to HART Clinic, Pokhara with the history of abdominal swelling, respiratory distress, lethargy, anorexia and weakness. Physical examination revealed dyspnea, pale mucous membrane, and undulating movement (thrills) of fluid on tapping the abdomen. Fecal sample collected for assessing the severity of endoparasites which was found negative. The hematological study showed an increase in numbers of neutrophils, while there were decreased erythrocytes and hemoglobin concentration. The biochemical analysis resulted in an elevated SGOT, SGPT level but decrease in total protein level. Ascitic fluid collected from abdominal paracentesis on examination revealed transudate fluid with serum-albumin ascetic gradient (SAAG) >1.1 gm/dl suggesting ascites due to portal hypertension (96% accuracy) caused by Liver cirrhosis. The dog was diagnosed as ascites of hepatic origin resulting portal hypertension and hypoproteinemia. The abdominocentesis was performed to drain the ascetic fluid followed by albumin and normal saline administration. The dog was further treated with antibiotic, diuretic, amino acid and liver tonics along with protein rich but salt free diet. The dog showed remarkable improvement with gradual reduced in abdominal distention and normalization of the appetite after 7 days of treatment.

Keyword: ascites; dog; biochemical; SAAG; hepatic

Introduction

Ascites, referred as accumulation of serous fluid in peritoneal cavity, has been attributed to chronic hepatic failure, congestive heart failure, nephritic syndrome, malnutrition, ankylostomiasis and protein losing enteropathy in canine. It results in abdominal swelling, dyspnea, lethargy, anorexia, vomiting, weakness, discomfort. Ascites is always a sign of disease; therefore investigation should be aimed at identifying the primary

underlying problem (Pradhan *et al.*, 2008; Kumar *et al.*, 2016).

Generally, the ascetic fluid has been evaluated for diagnosis of ascites. In particular, it involves the collection of abdominal fluid to analyze the bacterial presence, protein makeup, and bleeding. Besides, the urine analysis to diagnose urinary loss of protein that may be due to the diseases like amyloidosis and glomerulonephritis. Proteinuria causes hypoproteinemia resulting into ascites.

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Radiography and ultrasonography could be performed to determine the nature of abdominal fluid effusion.

A diagnostic evaluation of an animal presented with ascites may include a complete blood count (CBC), biochemical evaluation, abdominal paracentesis and biochemical and cytologic analyses of the fluid obtained, radiographs, biopsy and organ function tests (Peden & Zenoble, 1982; Satish Kumar & Srikala, 2014). In this case study, we used hematological and serum biochemical report along with clinical signs as diagnostic tools of ascites. Ascites fluid analysis and SAAG were used to confirm the origin of ascites.

Case History & Observation

A German shepherd dog of eleven months was brought to the HART Clinic as outdoor patient for the clinical treatment. Its weight was 18 kg. The dog showed the symptoms of swollen abdomen, discomfort, dyspnea, anorexia since 5 days. There was a symmetrical enlargement of abdomen assuming a pear shape appearance, mucous membranes were pale in color and tachycardia was evident. On tapping the abdomen there was undulating movements (Thrills) of the fluid (Fig. 1). The temperature was 102.4°F.



Fig. 1: General clinical examination

Materials and Methods

Fecal sample was collected per rectum and examined to reveal out the severity of endoparasitic infestation. The blood was taken from radial vein for the hematological and sero-biological analyses. Abdominal paracentesis was performed to obtain the fluid for the biochemical and cytological analyses (Fig. 2).



Fig. 2: Abdominocentesis

The ascetic fluid was drained aseptically for the immediate relief from dyspnea and discomfort (Fig. 3). The alamin (albumin) and Normal saline (NS) was concurrently administered for 3 consecutive days to prevent the hypervolemia and hypoalbuminemia.



Fig. 3: Draining of ascetic fluid

Result

The fecal examination showed negative result. Neutrophilia, hypoalbuminemia, hypoglycemia and elevated level of liver enzymes were found on the hemato-biochemical examination (Table 1; Table 2). Similarly, the ascetic fluid analysis (Table 3) revealed that the fluid was slightly reddish, watery, transudate with PMN=42cells/mm³ <250 cells/mm³ and SAAG = 31.6 = 1.4 > 1.1 gm/dl.

Table 1: Hematological Report

Hematology	Units	Reports	Normal Range
WBC	10 ³ /mm ³	11.5	6.0-17.0
RBC	10 ⁶ /mm ³	5.3	5.5-8.5
Hb	g/dl	9.6	12.0-18.0
PCV	%	36	37.0-55.0
Neutrophils	%	85	60-77
Lymphocytes	%	23	12-30
Eosinophils	%	1	2-10
Basophils	%	0	0-1
Monocytes	%	1	3-10

Abbreviations: WBC, White Blood Corpuscles; RBC- Red Blood Corpuscles; PCV, Pack Cell Volume; Hb, Hemoglobin

Table 2: Biochemical Report

Biochemistry	Units	Report	Normal Range
Blood Glucose	mg/dl	55	60-125
<i>Liver Function Test:</i>			
Total Protein	g/dl	4.8	5.1-7.8
Albumin	g/dl	2.2	2.6-4.3
Globulin	g/dl	2.6	2.3-4.5
A:G ratio		0.8	0.75-1.9
Total Bilirubin	mg/dl	0.8	0-0.4
Direct Bilirubin	mg/dl	0.3	0.0-0.1
Indirect Bilirubin	mg/dl	0.5	0-0.3
SGOT/AST	IU/L	62	5-55
SGPT/ALT	IU/L	128	5-60
Alkaline Phosphatase	IU/L	810	10-150
<i>Renal Function test:</i>			
BUN	mg/dl	24	7-27
Creatinine	mg/dl	0.7	0.4-1.8

Table: Serum Biochemical Analysis of Ascitic Dog (Reference Range: from Hitachi Chemistry Analyzer model 747 IDEXX Veterinary services); Abbreviations: A: G, Albumin: Globulin; SGOT/AST, Serum Glutamic Oxaloacetic Transaminase or Aspartate Aminotransferase; SGPT/AST, Serum Glutamic Pyruvic Transaminase or Alanine Aminotransferase; BUN, Blood Urea Nitrogen

Table 3: Ascitic Fluid Examination Report

Parameters	Results
Color	Watery (slightly reddish)
Volume	1.5 ml
Appearance	Clear
TLC	42 cells/mm ³
Neutrophils	55%
Lymphocytes	45%
Sugar	79 mg/dl
Protein	2.1 gm/dl
Albumin	0.8gm/dl
Globulin	1.3 gm/dl

Abbreviation: TLC, Total Leukocyte Count

Differential Diagnosis

Spontaneous Bacterial Peritonitis (SBP): fever, abdominal pain, abdominal tenderness, polymorphonuclear leucocyte (PMNL) count >250 cells/mm³ (Koulaouzidis *et al.*, 2007)

Liver Cirrhosis: portal hypertension, ascites, elevation of liver enzymes, SAAG >1.1 gm/dl

Portal Hypertension: ascites, portal vein thrombosis, Schistosomiasis (Beker & Valencia-Parparcén, 1968)

Right Sided heart Failure: respiratory distress, cough, cyanotic tongue, and syncope (Satish Kumar & Srikala, 2014)

Tentative Diagnosis

Liver Cirrhosis (SAAG = 3-1.6 = 1.4 >1.1 gm/dl)

Treatment

Abdominal fluid was drained every 24 hourly followed by normal saline and albumin administration intravenously to compensate the fluid and protein loss from abdominocentesis.

- 1) Furosemide Tab (40 mg)
Sig: 2 tabs, BID, PO x 5 days followed by 1 tab, BID, PO x 5 days
- 2) Livoferol Pet liq.
Sig: 15 ml, BID, PO x 10 days
- 3) Hepatosyl /Urodeoxycholic Acid (UDCA), 300 mg
Sig: 1 tab, BID, PO x 15 days
- 4) Megapen (Ampicillin + Cloxacillin), 250mg
Sig: 1 tab, BID, PO x 5 days

The owner was advised to feed cooked buffalo or goat liver daily with the restriction in salt and set the follow up date for next week.

Discussion

Increased SGOT indicates hepatic insufficiency with extensive damage resulting into the leakage of enzymes from hepatic cell into blood stream (Pradhan *et al.*, 2008; Kumar *et al.*, 2016; Beker & Valencia-Parparcén,

1968). Normal concentrations of BUN and creatinine indicate normal function of kidney. The hypoglycemia is the indicative of hepatic insufficiency (Pradhan *et al.*, 2008; Kumar *et al.*, 2016). Hematological study revealed slight decrease in Hb concentration and increase in neutrophils which is similar to the report of Pradhan *et al.* (2008). Similarly, analysis of the ascetic fluid showed that the fluid was transudate and there was no bacterial infection as PMN <250 cells/mm³ (Koulaouzidis *et al.*, 2007). SAAG can be used as a screening test in ascetic due to chronic liver disease (Satish Kumar & Srikala, 2014; Bhadesiya *et al.*, 2015). SAAG = 3-1.6 = 1.4 >1.1 gm/dl indicates high gradient ascites which is due to portal hypertension (96% accuracy) and the portal hypertension may be due to Liver cirrhosis (Beg *et al.*, 2001). Similarly, another research by Uddin *et al.* (2013) found SAAG 97% accurate in identifying the cause of ascites.

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Conflict of Interest

The authors declare that there is no conflict of interest.

References

- Beg M, Husain S, Ahmad N, & Akhtar N (2001) Serum/Ascites Albumin Gradient in Differential Diagnosis of Ascites. *Journal, Indian Academy of Clinical Medicine*, 2(1 and 2), 51–54. Retrieved from <http://medind.nic.in/jac/t01/i1/jact01i1p51.pdf?pagewanted=all>
- Beker SG, & Valencia-Parparcén J (1968) Portal hypertension syndrome. *The American Journal of Digestive Diseases*, 13(12), 1047–1054. DOI: [10.1007/BF02233549](https://doi.org/10.1007/BF02233549)
- Bhadesiya CM, Jani RG, Parikh PV, Pandey AM, Rao N, & Shai A (2015) Haematobiochemistry and imaging study on ascites with hepatic and cardiac involvement in a german shepherd pup. *International research journal of chemistry*, 11, 14–22. Retrieved from <http://www.petsd.org/wp/wp-content/uploads/2015/09/2>
- Koulaouzidis A, Bhat S, Karagiannidis A, Tan WC, & Linaker BD (2007) Spontaneous bacterial peritonitis. *Postgraduate Medical Journal*, 83(980), 379–83. DOI: [10.1136/pgmj.2006.056168](https://doi.org/10.1136/pgmj.2006.056168)
- Kumar A, Das S, & Mohanty DN (2016) Therapeutic management of ascites in GSD female dog. *International Journal of Science, Environment and Technology*, 5(2), 654–657. Retrieved from <http://www.ijset.net/journal/914.pdf>
- Peden WM, & Zenoble RD (1982) Canine Ascites. *Iowa State University Digital Repository*, 44(1). Retrieved from http://lib.dr.iastate.edu/iowastate_veterinarian
- Pradhan MS, Dakshinkar NP, Waghaye UG, & Bodkhe AM (2008) Successful treatment of Ascites of hepatic origin in Dog. *Veterinary World*, 1(1), 23. Retrieved from

<http://www.veterinaryworld.org/2008/January/Ascites.pdf>

Satish Kumar K, & Srikala D (2014) Ascites with right heart failure in a dog: diagnosis and management. *Vet. Anim. Res*, 1(3), 140–144. DOI: [10.5455/javar.2014.a15](https://doi.org/10.5455/javar.2014.a15)

Uddin MS, Hoque MI, Islam MB, Uddin MK, Haq I, Mondol G, & Tariquzzaman M (2013) Serum-ascites albumin gradient in differential diagnosis of ascites. *Mymensingh Medical Journal : MMJ*, 22(4), 748–54. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/24292307>