Multimodal Diagnosis and Management of Canine Ascites: A Clinical Case Report

ABSTRACT

Ascites, the pathological accumulation of fluid within the peritoneal cavity, often results from an imbalance between fluid production and absorption. This case involved a seven-and-a-half-year-old neutered female Labrador retriever that presented with abdominal distension, inappetence, weakness, and lethargy. Clinical examination revealed respiratory distress, abdominal pain, pale mucous membranes, muffled heart sounds, and a positive fluid thrill upon abdominal palpation. Hematological evaluation showed normocytic normochromic anemia, neutrophilia, leukocytosis, and thrombocytopenia. Biochemical analysis revealed hypoalbuminemia, hypoglycemia, elevated liver enzymes (ALT, AST, ALP), and increased blood urea nitrogen (BUN) and creatinine levels, pointing to impaired liver and kidney function. Arterial blood gas analysis indicated hepatobiliary dysfunction, showing low pCO_2 , reduced bicarbonate (HCO_3) levels, and a negative base excess (BE), suggestive of metabolic acidosis with compensatory respiratory alkalosis. Radiographic imaging showed a ground-glass appearance and pleural effusion, while ultrasonography confirmed the presence of free anechoic fluid in the peritoneal cavity, rounded liver margins, a dilated hepatic portal vein, hyperechoic liver parenchyma, ill-defined corticomedullary junctions in the kidneys, and splenomegaly. Cytological analysis of the straw-colored ascitic fluid showed fibrin strands and white blood cells, with a serum-ascites albumin gradient (SAAG) of 2.4 g/dL, indicating portal hypertension as the underlying cause. The final diagnosis was ascites of hepatic origin. Treatment included diuretics, antibiotics, fluid therapy, liver supplements, and dietary modifications, including salt restriction and the provision of high-quality protein. This case underscores the importance of a comprehensive diagnostic approach, incorporating clinical, hematological, biochemical, and imaging findings to ensure timely intervention and effective management of canine ascites.

Keywords: Abdominocentesis, Imaging, liver, Portal hypertension, Therapeutics

1. Introduction

Ascites, described as the pathological accumulation of fluid (transudate or modified transudate) within the peritoneal cavity, indicates an imbalance between the production and resorption of intraperitoneal fluid [1]. It is a manifestation of underlying disease and can develop due to cirrhosis, peritoneal infection, carcinomatosis, congestive heart failure, or a combination (mixed ascites), vital organs like the liver, kidneys, and heart are predominantly involved in the development of ascites [2]. Cases are more frequently seen in dogs between the ages of five and seven years; however, cases in dogs younger than five and older than seven years have also been reported. There is breed predisposition for development of ascites, with higher incidence in Pomeranians, Labradors, Boxers, Dobermans, and mongrel breeds [3].

Evaluation of a patient with ascites should include a directed history, a focused physical examination, and diagnostic paracentesis with ascitic fluid analysis. An abrupt change in the dietary regimen is considered as one of the predisposing factors for developing ascites [4]. Imaging techniques such as ultrasound, radiography, and CT scans are beneficial in refining the diagnosis and determining the involvement of various organs [5,6]. Abdominal ultrasound is a valuable diagnostic tool for assessing ascites, the echogenicity of various organs, and the nature of the fluid, whether transudate or exudate. Abdominal paracentesis can differentiate ascitic fluid types: clear straw-colored fluid indicates modified transudate associated with cardiac failure or liver cirrhosis, pink discoloration suggests a medical emergency with potential bacterial infection, reddish fluid indicates hemorrhage due to large vessel rupture, trauma, or coagulopathies, and greenish discoloration is due to bile seepage into the peritoneal cavity [2]. Peritonitis is a typical sequel of ascites [7]. The management of ascites includes repeated large-volume abdominocentesis, liver supplements, liver-safe antibiotics, and a focus on low-sodium diets, high quality proteins and diuretics. Although ascites cases have been described in the literature, this study aims to describe various diagnostic methods that are important for veterinary clinicians for precise disease diagnosis and appropriate treatment.

2. Case Presentation

A seven-and-a-half-year-old, neutered female Labrador retriever, weighing 24 kg, was presented to the Teaching Veterinary Clinical Complex (TVCC) at Sher-e-Kashmir University of Agricultural Sciences and Technology, Jammu with complaints of abdominal distension, groaning while lying down, reduced food intake, and weakness for the past week. The dog had been properly dewormed and vaccinated on time. On clinical examination, the dog was dehydrated, and lethargic. The vital parameters revealed normal rectal temperature (101°F), labored breathing, elevated respiration rate, and pale conjunctival mucous membranes. On abdominal palpation, the dog exhibited pain and a fluid thrill was noted on abdominal ballottement. The femoral pulse was thready on palpation, and heart sounds were muffled on auscultation. Blood samples were collected for hematology (CBC) and biochemistry. Hematology revealed normocytic normochromic anemia, leukocytosis, neutrophilia, and thrombocytopenia. Biochemical analysis was performed using Chem-7 semi-automated clinical chemistry analyzer (Erba Mannheim®) which revealed hypoalbuminemia, hypoglycemia, and increased levels of ALT, AST, and ALP, along with elevated blood urea nitrogen [BUN], and creatinine levels (Table 1). To evaluate hepatobiliary disease, an arterial blood gas analysis was performed, revealing low bicarbonate (HCO_3 ; 11 mmol/L) and a negative base excess (BE; -10.05 mmol/L), indicative of metabolic acidosis. The pH was slightly alkaline (7.66), suggesting mild alkalosis, potentially metabolic or respiratory in origin. The low partial pressure of CO_2 (pCO_2 ; 16 mmHg) and partial pressure of O_2 (pO_2 ; 81 mmHg) indicated respiratory alkalosis, likely due to hyperventilation as a compensatory response to the metabolic acidosis. To examine the presence of abdominal fluid, freehand abdominocentesis was performed by placing the animal in lateral recumbency and using a 21-gauge needle punctured 3 cm right to midline with a

10 ml syringe to aspirate the fluid (Figure 1). The ascitic fluid was straw-colored. Cytology of the ascitic fluid revealed the presence of fibrin cells and WBCs. The serum-ascites albumin gradient value was 2.1 g/dL, indicating portal hypertension. The case was subjected to radiographic examination using a Heliophos D Siemens X-ray machine, which revealed a classic 'ground-glass appearance' of the abdomen, with pleural effusion and masking of abdominal cavity details (Figure 2). Abdominal ultrasonography was performed using a Chison i8VET diagnostic ultrasound machine in real-time B-mode with a 2.5–5.0 MHz convex probe. Sonographic findings revealed the presence of free anechoic fluid in the intraperitoneal space (Figure 3), rounded liver lobe margins, marked portal vessel dilatation, increased echogenicity and size of the liver (Figure 4), and splenomegaly with a homogeneous parenchyma (Figure 5) and an ill-defined corticomedullary junction (Figure 6). These findings indicated that the dog was suffering from liver disease, leading to hypoalbuminemia and contributing to the development of ascites. Additionally, portal hypertension was identified as a contributing factor to the ascites condition.

Table 1: Hemato-biochemical parameters of a Labrador bitch affected with ascites.

S. No.	Parameter	Value obtained	Ref. Range	Parameter	Value obtained	Ref. Range
1	Hb (g/dl)	6.2	12-18	AST (U/L)	72	18-56
2	PCV (%)	20.5	37-55	ALT (U/L)	113	17-95
3	RBC ($\times 10^6$ /µl)	3	5.5-8.8	GGT (U/L)	8.5	0-8
4	MCV (fl)	68	60-77	ALP (U/L)	118	7-115
5	MCH (pg)	20.6	21-26	TP (g/dl)	8.5	5.3-7.6
6	MCHC (%)	30.5	32-36	Albumin (g/dl)	1.93	3.2-4.2
7	RDW (%)	14.8	10.6-14.3	Globulin (g/dl)	2.57	1.9-3.7
8	Platelets ($\times 10^3$/µl)	135	186-545	A:G ratio	0.75	0.9-1.9
9	WBC ($\times 10^3$/µl)	19.2	6-17	Total bilirubin (mg/dl)	0.49	0-0.2
10	Neutrophil ($\times 10^3$/µl)	11.97	2.7 - 9.4	Direct bilirubin (mg/dl)	0.34	0-0.1
11	Lymphocytes ($\times 10^3$/µl)	4.65	0.9-4.7	Indirect bilirubin (mg/dl)	0.15	0-0.1
12	Monocytes ($\times 10^3$/µl)	0.31	0.1-1.3	Urea nitrogen (mg/dl)	29	9-26
13	Eosinophils ($\times 10^3$/µl)	0.37	0.1-2.1	Creatinine (mg/dl)	1.9	0.6-1.4
14	Basophils ($\times 10^3$/µl)	0	0-0.1	Uric acid (mg/dl)	1.07	0.1-0.4
15	Neutrophils (%)	65	42-54	Ca (mg/dl)	8.8	9.4-11.1
16	Lymphocytes (%)	49	9-47	P (mg/dl)	3.2	2.7-5.4
17	Monocytes (%)	5	2-12	Na (mEq/L)	155.2	143-150

18	Eosinophils (%)	6	1-18	K (mEq/L)	4.6	4.1-5.4
19	Basophils (%)	0	0-1	Cl (mEq/L)	116	106-114

Based on the history and clinical observations, the case was diagnosed as ascites of hepatic origin. Treatment was initiated with amoxicillin at 10 mg/kg administered intramuscularly twice daily, furosemide at 1 mg/kg intravenously twice daily, and fluid therapy with D10 (300 ml) and normal saline (500 ml) intravenously. The patient also received supportive medications such as liver supplement (Silymarin @ 10mg/kg twice daily), pantoprazole @ 1mg/kg intravenously once daily, ranitidine @ 2 mg/kg subcutaneously twice daily, and vitamin B complex injections (Eldervit® @ 2 ml intravenously once daily). The abdominal paracentesis was performed frequently for first seven days, to relieve the intra-abdominal pressure. The owner was advised to restrict salt intake, inclusion of high quality protein such as egg albumin and cottage cheese in the diet, and ensure adequate rest. The treatment continued for seven days, during which gradual recovery was observed, including improvement in appetite and normalization of hematological and biochemical parameters. After one month, the owner reported full recovery of the patient with a normal appetite and activity.

3. Discussion

Ascites, or abdominal dropsy, is the pathological accumulation of excess fluid in the peritoneal cavity [1,8,9], frequently linked to liver disease, hypoalbuminemia, and increased sodium and water retention [10]. Early diagnosis through a combination of hematology, biochemistry, and imaging techniques, including radiography, ultrasound, and CT scans, is critical for timely intervention and effective disease management. Given the multifactorial etiology of ascites, diagnosing the exact cause remains challenging. A combination of diagnostic tests is often necessary to differentiate ascites from other diseases. This case provided detailed diagnostic observations, particularly those detected via ultrasound and radiographic imaging, to identify the precise cause of ascites and guide treatment planning.

In the present case, the clinical signs observed were consistent with those described by Ettinger and Feldman [12] and Ghosh *et al*. [13]. Elevated AST and ALT levels likely reflect hepatocellular damage, where enzymes leak into the bloodstream due to hepatic injury. Decreased hemoglobin levels, along with leukocytosis and neutrophilia, align with findings reported by Rautray *et al*. [14] and Sunil [15]. The Serum-Ascites Albumin Gradient (SAAG) is essential for diagnosing the etiology of ascites, particularly for detecting portal hypertension. A SAAG value >1.1 g/dL is suggestive of portal hypertension [2,16], which was observed in this case (SAAG = 2.4 g/dL). Emerging diagnostic techniques, including platelet indices and leukocyte esterase reagent strips, are promising tools for diagnosing ascites [2].

Ascites can be classified based on the nature of the fluid as either transudate or exudate. Transudative ascites, as seen in this case, typically results from portal hypertension and hypoproteinemia, whereas exudative ascites often accompanies inflammatory or malignant conditions [9]. In this case, Grade III ascites (massive fluid accumulation with gross abdominal distension) was noted [11]. Ultrasound was useful in present case in distinguishing ascites from other conditions such as a ruptured bladder, diaphragmatic hernia, and urethral obstruction. Abdominal ultrasound can detect as little as 100 ml of peritoneal fluid. Radiographic imaging helps in the detection of pleural effusion, liver abnormalities, and organ displacement. Together, these imaging techniques are invaluable for evaluating the nature of fluid and the echogenicity of affected organs [5,6].

Arterial blood gas analysis is instrumental in detecting hepatobiliary dysfunction. In the present case, low bicarbonate (HCO_3 , 11 mmol/L) and a negative base excess (BE, -10.05 mmol/L) indicated metabolic acidosis. Although the pH remained slightly alkaline (7.66), suggesting mild alkalosis, of either metabolic or respiratory type. Low partial pressure of CO_2 (pCO_2 , 16 mmHg) and partial pressure of O_2 (pO_2 , 81 mmHg) suggested respiratory alkalosis, likely due to hyperventilation as a compensatory mechanism for metabolic acidosis, a pattern consistent with Kaneko *et al*. [19].

The development of ascites associated with hepatic disease further causes sodium retention by the kidneys. Systemic hypotension and increased renal sodium retention are common. This is partially due to decreased sodium delivery to the tubules and reduced glomerular filtration rate, as well as increased release of renin-angiotensin-aldosterone (RAAS), which increases sodium retention in the distal tubules, contributing to fluid retention, which further aggravates ascites. The excess fluid in the abdomen compresses the caudal vena cava, reducing venous return and perpetuating a cycle of fluid retention and ascites formation.

The goal of ascites treatment is to remove accumulated fluid and restore sodium balance until the underlying cause is addressed [11,17,18]. Diuretics like spironolactone and furosemide are commonly used to reduce intra-abdominal fluid. Fluid replacement therapy (@ 90 ml/kg) is also crucial to prevent hypovolemic shock during treatment [8,11].

4. Conclusion

This case study underscores the importance of a comprehensive diagnostic approach for the accurate diagnosis and management of canine ascites. Timely intervention with appropriate medical treatment, dietary adjustments, and supportive care can lead to significant improvement and recovery in affected dogs.

Acknowledgment

Not Applicable

Authors' Contributions: supervision, Analysis, Critical Review – S.S.J.; Design, Literature Search, Writing Manuscript – A.R.; Resources, Material, Data collection and/or Processing, Review – I.M. All authors read and approved the final manuscript.

Ethics

Not applicable

Conflict of Interests

The authors have no conflict of interest to declare.

Availability of data and materials

All available data have been shared in the manuscript.

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