*Archives of Razi Institute Journal*

***Volume 80, NO. 3, 2025***

**Journal homepage:** https://archrazi.areeo.ac.ir

**Case Study**

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

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**Article Info:**

**Received:** 6 August 2024
**Revised:** 26 October 2025
**Accepted:** 27 October 2024

**Keywords:**

Abdominocentesis, Imaging, Liver, Portal Hypertension, Therapeutics.

## 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 presenting 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, indicating 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), consistent with 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. The serum-ascites albumin gradient (SAAG) was 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 that combines clinical, hematological, biochemical, and imaging findings to enable timely intervention and effective management of canine ascites.

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**How to cite this article:** **Manzoor I, Rasool A, Jasvinder S.S. Multimodal Diagnosis and Management of Canine Ascites: A Clinical Case Report. *Archives of Razi Institute*. 2025;80(3):807-812.** DOI: 10.66224/ARI.2025.80.3.807

## 1. Introduction

Ascites, defined as the pathological accumulation of fluid, either transudate or modified transudate, within the peritoneal cavity, indicates an imbalance between intraperitoneal fluid production and resorption (1). It is a clinical manifestation of various underlying disease which can include cirrhosis, peritoneal infection, carcinomatosis, congestive heart failure, or a combination (mixed ascites). The involved vital organs like the liver, kidneys, and heart play a central role 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. Additionally, certain breeds show 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 as transudate or exudate. Abdominal paracentesis can differentiate the 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 common complication of ascites (7). The management of ascites typically involves repeated large-volume abdominocentesis, liver supplements, liver-safe antibiotics, and a focus on low-sodium diets, high quality proteins and diuretics. Although ascites have been well-documented, this study describe emphasizes 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 over 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 a normal rectal temperature (101°F), labored breathing, an 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, performed using Chem-7 semi-automated clinical chemistry analyzer (Erba Mannheim®), 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 of potentially metabolic or respiratory 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 ,and 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, along with pleural effusion and masking of abdominal cavity details

**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 ($\times10^6$/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 ($\times10^3$/µl) | 135 | 186-545 | A:G ratio | 0.75 | 0.9-1.9 |
| **9** | WBC ($\times10^3$/µl) | 19.2 | 6-17 | Total bilirubin (mg/dl) | 0.49 | 0-0.2 |
| **10** | Neutrophil ($\times10^3$/µl) | 11.97 | 2.7 - 9.4 | Direct bilirubin (mg/dl) | 0.34 | 0-0.1 |
| **11** | Lymphocytes ($\times10^3$/µl) | 4.65 | 0.9-4.7 | Indirect bilirubin (mg/dl) | 0.15 | 0-0.1 |
| **12** | Monocytes ($\times10^3$/µl) | 0.31 | 0.1-1.3 | Urea nitrogen (mg/dl) | 29 | 9-26 |
| **13** | Eosinophils ($\times10^3$/µl) | 0.37 | 0.1-2.1 | Creatinine (mg/dl) | 1.9 | 0.6-1.4 |
| **14** | Basophils ($\times10^3$/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 |

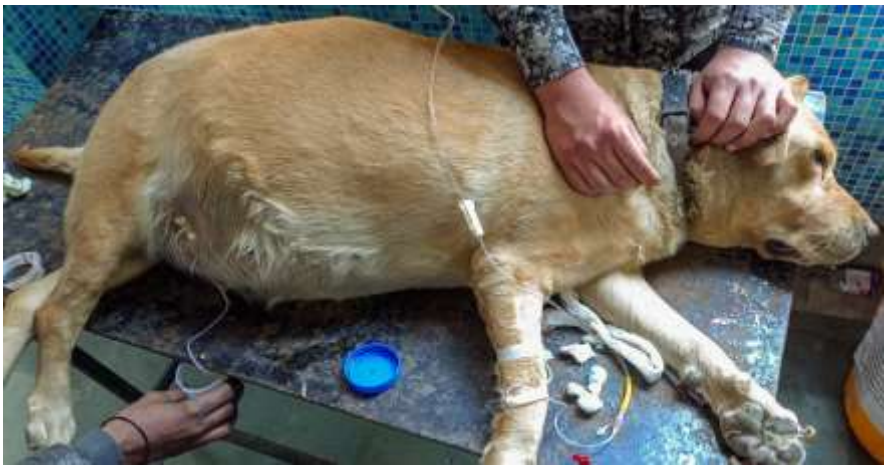

**Figure 1.** Abdominocentesis performed on a Labrador bitch with ascites, showing a distended abdomen.

(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. The 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.

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) administered intravenously. The patient also received supportive medications, including liver supplement (Silymarin @ 10mg/kg twice daily),

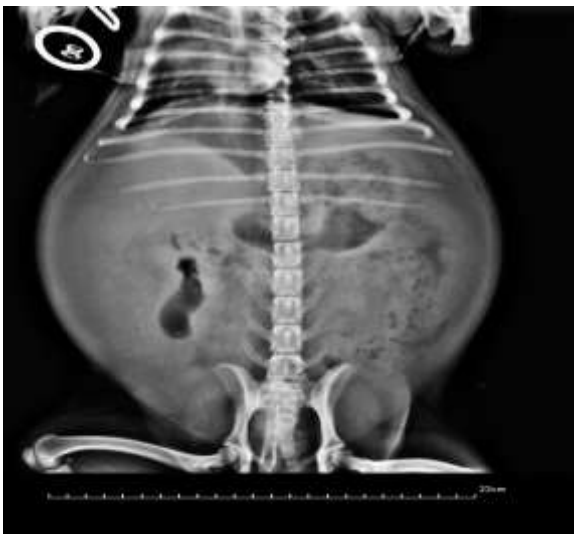

**Figure 2.** Ventro-dorsal radiographshowing the classic "ground glass appearance" of the abdomen, with obscured abdominal cavity details.

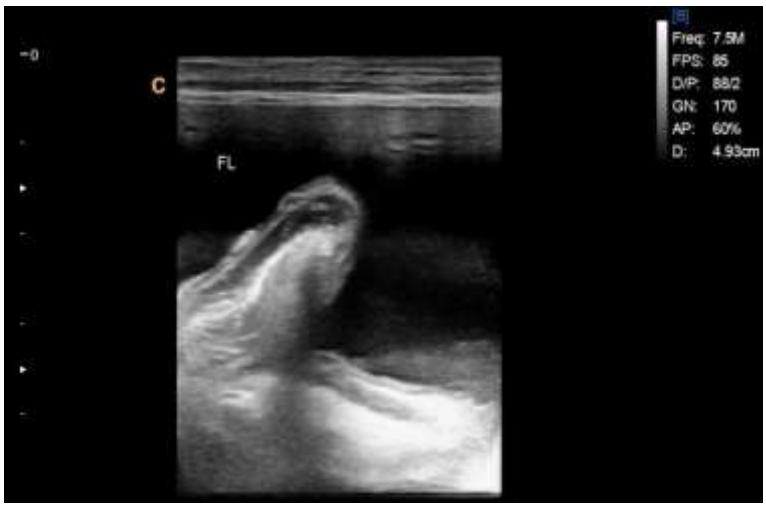


**Figure 3.** Sonogram depicting presence of anechoic fluid in the intraperitoneal space.

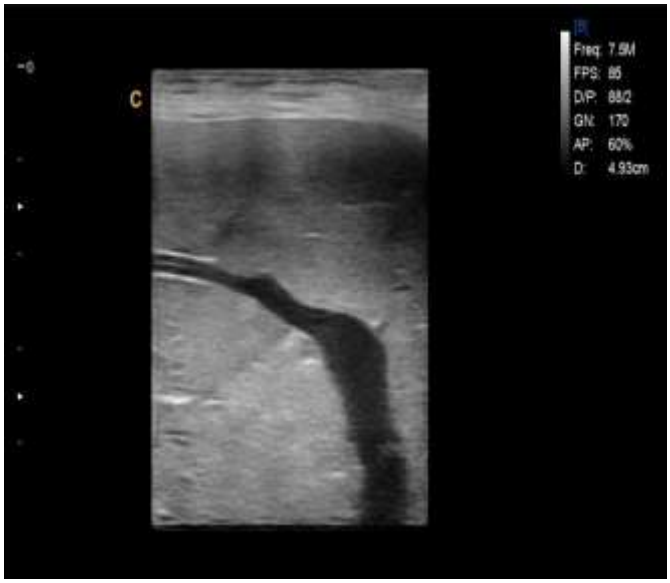


**Figure 4.** Sonogram of liver showing hypoechoic parenchyma with marked portal vessel dilatation.

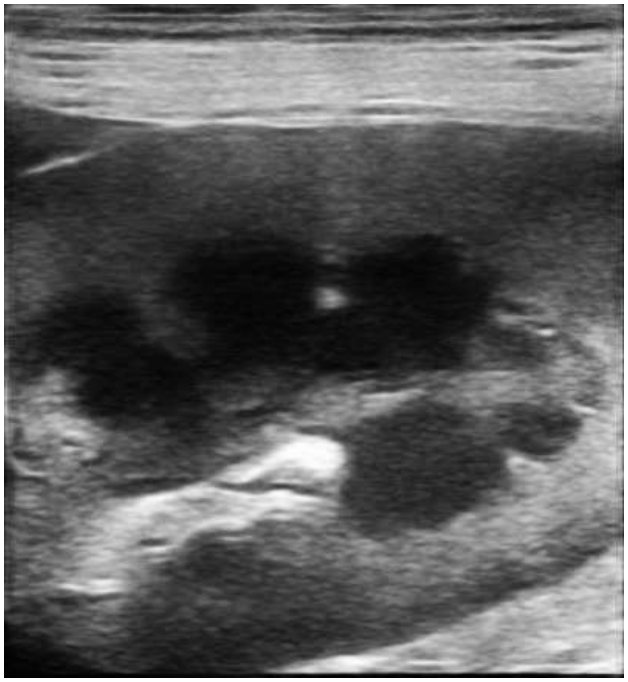

**Figure 5.** Sonogram showing an ill-defined cortico-medullary junction of the kidney.

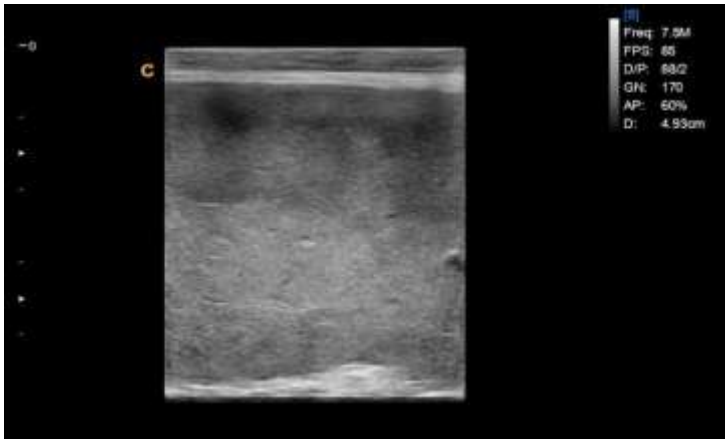


**Figure 6.** Sonogram showing splenomegaly with homogenous parenchyma.

pantoprazole @ 1mg/kg intravenously once daily, ranitidine @ 2 mg/kg subcutaneously twice daily, and vitamin B complex injections (Eldervit® @ 2 ml intravenously once daily). Frequent abdominal paracentesis was performed during the first seven days to relieve the intra-abdominal pressure. The owner was advised to restrict salt intake, include 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, with the patient exhibiting 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 of ascites often remains challenging. A combination of diagnostic tests is often necessary to differentiate ascites from other diseases. This case provided detailed diagnostic observations, particularly through ultrasound and radiographic imaging, to identify the precise cause of ascites and guide treatment planning (10). In the present case, the clinical signs observed were consistent with those described by Ettinger and Feldman (11) and Ghosh *et al*. (12). 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.* (13) and Sunil (14). 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 suggests portal hypertension (2, 15), 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. In this case, transudative ascites, typically result from portal hypertension and hyperproteinemia, 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 this 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), this suggested a mild alkalosis of metabolic or respiratory type.The 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 response to metabolic acidosis, a pattern consistent with Kaneko *et al*. (16).

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, reduced glomerular filtration rate and increased release of renin-angiotensin-aldosterone (RAAS), which increases sodium retention in the distal tubules. This contributes to fluid retention, further aggravating 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 (9, 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' Contribution

Study concept and design: SS. J, I.M, A.R.
Acquisition of data: A. R, I. M.
Analysis and interpretation of data: I.M, A. R.
Drafting of the manuscript: A. R, I. M.
Critical revision of the manuscript for important intellectual content: S.S. J, I. M, A. R.
Statistical analysis: N. A.
Administrative, technical and material support: S.S. J, I. M.

## Ethics

Not applicable

## Conflict of Interest

The authors have no conflict of interest to declare.

## Data Availability

All available data have been shared in the manuscript.

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