JOURNAL OF AMERICAN MEDICAL SCIENCE & RESEARCH

Case Study
ISSN: 3041-5578
Journal homepage: https://medical.researchfloor.org/

Ascites in Dogs: A Comprehensive Study on Diagnosis and Therapeutic Management

Ankit Kaushik*, Ravinandan Verma, Satyavrat Singh, Alok Singh and Sonu Jaiswal

Department of Veterinary Medicine, Acharya Narendra Deva University of Agriculture and Technology, Kumarganj, Ayodhya, India

ABSTRACT

The present study documents five clinical cases of canine ascites presented at the Teaching Veterinary Clinical Complex (TVCC), ANDUAT. Dogs of different breeds and age groups were evaluated based on clinical history, physical examination, haematological and biochemical profiling, and ultrasonographic assessment. Among the cases, Labrador Retrievers were most affected, with age ranging between 3 months to 11 years. Common clinical signs included abdominal distension, limb oedema, inappetence, and lethargy. Haematological alterations included elevated TLC and lymphocytosis, while biochemical profiles showed elevated ALT, AST, creatinine, and blood urea nitrogen (BUN), along with marked hypoalbuminemia and reduced total protein. These findings correlated with underlying hepatic or renal aetiologies. Ultrasonography proved valuable in detecting the presence and extent of ascitic fluid and assessing hepatic parenchymal changes. Treatment was tailored based on the aetiology and involved the use of diuretics (Furosemide, Spironolactone), hepatoprotectives (Silymarin-based formulations), and supportive care including amino acids and nephroprotective. Therapeutic outcomes were favourable in all five cases, with clinical improvement observed within 7–15 days of initiating treatment. This case series underscores the diagnostic utility of combined clinical and laboratory evaluation and highlights the effectiveness of targeted medical management in canine ascites of varied aetiology.

Citation: Ankit Kaushik, Ravinandan Verma, Satyavrat Singh, Alok Singh and Sonu Jaiswal (2025). Ascites in Dogs: A Comprehensive Study on Diagnosis and Therapeutic Management.
DOI: https://doi.org/10.51470/AMSR.2025.04.01.46
Journal of American Medical Science and Research.

Received 20 April 2025
Revised 15 May 2025
Accepted 09 June 2025

Corresponding Author: ***Ankit Kaushik***
Email Address: ***kaushikwankit@gmail.com***

Keywords: *Canine ascites, Hepatic dysfunction, Renal insufficiency, Ultrasonography, Hematobiochemical analysis, Diuretics, Silymarin, Spironolactone, Nephroprotective.*

Introduction

Ascites, defined as the pathological accumulation of serous or serosanguineous fluid within the peritoneal cavity, is a common clinical manifestation in dogs and often signals systemic or organ-specific dysfunctions. The condition is most frequently associated with hepatic insufficiency, congestive heart failure, nephrotic syndrome, and hypoalbuminemia, but can also result from conditions such as protein-losing enteropathy, intestinal lymphangiectasia, neoplasia, or parasitic infestations like ancylostomiasis {1,2,3}.

Pathophysiology, ascites develops due to alterations in Starling's forcesincluding increased hydrostatic pressure (as seen in portal hypertension), decreased oncotic pressure (due to hypoalbuminemia), or impaired lymphatic drainage. Hepatic causes, particularly chronic hepatitis and cirrhosis, often lead to intrahepatic portal hypertension resulting in transudative ascites with low protein and cellular content, whereas conditions such as peritonitis or intra-abdominal tumours result in exudative fluid accumulation {4}.

Diagnostic evaluation involves a combination of clinical examination, complete blood count (CBC), serum biochemical profiling, ultrasonography, and ascitic fluid analysis. Specific biomarkers such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), total protein, albumin, and blood urea nitrogen (BUN) help identify hepatic or renal involvement {5,6}. The Serum Ascitic Albumin Gradient (SAAG) has emerged as a more reliable indicator than the traditional transudate-exudate classification, especially for identifying portal hypertension-related causes {7}.

Ultrasonography is particularly useful due to its sensitivity in detecting even small volumes of fluid and in assessing hepatic echotexture, renal morphology, and evidence of secondary complications. While ascites itself is not a disease but a consequence of underlying pathology, its management must be aetiology-driven. Therapies typically include diuretics, hepatoprotectives, and nephroprotective, along with supportive fluid and nutritional therapy {8,9}.

The present study compiles five clinical cases of canine ascites evaluated at the Teaching Veterinary Clinical Complex (TVCC), ANDUAT. Each case was analysed based on clinical history, physical signs, laboratory and imaging findings, and the response to therapeutic protocols. This study highlights the clinical diversity of ascites in dogs and the importance of a multidisciplinary diagnostic and treatment approach.

Clinical Case Summary of Ascitic Dogs (Pre-Therapy Evaluation)

Case	Breed	Age	MM	Temperature	Gross Evaluation	Clinical History
1	Spitz	11 yr	Not recorded	102.8 °F	Abdominal distension, painful right side, fluid thrill	Polyuria, dropping of masses during sleep, swelling in abdomen for 1 month
2	Labrador ♂	8 yr	Not recorded	103.4 °F	Bilateral abdominal distension, limb & scrotal oedema, reduced urination	Oedema in limbs & scrotum (18 days), gradual distension, oliguria
3	Rottweiler	3 yr	Pink	104.7 °F	Severe abdominal enlargement, tense abdomen, dehydration signs	Chronic distension (3 months), oliguria
4	GSD Puppy ♂	3 M	Pale	102.8 °F	Distended belly, hair loss, signs of malnutrition	Gradual abdominal distension (4 days), pica, inappetence, recent rabies bite treatment
5	Labrador ♀	5 yr	Pink-mild	100.7 °F	Teat discharge, mild dehydration, visible abdominal sagging	Distension (20 days), inappetence (2 mo), weight loss, maggot wound history, loose stools

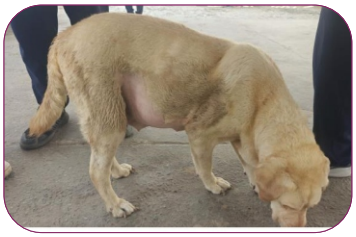
DOI: https://doi.org/10.51470/AMSR.2025.04.01.46

Spitz

Labrador

Rottweiler

GSD Puppy



Labrador

Materials and Methods

This clinical study was conducted on five canine cases of ascites presented to the Teaching Veterinary Clinical Complex (TVCC), Acharya Narendra Deva University of Agriculture and Technology (ANDUAT), Ayodhya. Dogs varied in breed, age, and sex, and were included based on clinical suspicion of abdominal effusion.

Clinical Examination

Each animal underwent a comprehensive evaluation involving detailed history and general clinical examination. Parameters recorded included rectal temperature, heart rate, respiratory rate, mucous membrane color, capillary refill time, and hydration status. Specific signs like abdominal distension, fluid thrill, teat discharge, scrotal oedema, and dyspnoea were recorded, following general diagnostic guidelines as described in standard texts {10}.

Haematological and Biochemical Evaluation

Blood was collected aseptically via cephalic or saphenous venipuncture into K_3EDTA tubes for haematological analysis and plain vacutainers for serum biochemistry. The haematological profile included haemoglobin concentration, TLC, DLC, PCV, MCV, MCH, MCHC, and platelet count, following standard veterinary haematology protocols {11}. Biochemical parametersALT, AST, total protein, albumin, BUN, creatinine, and bilirubinwere used to evaluate hepatic and renal function, as per established biochemical interpretation standards {12}.

Ultrasonographic Examination

Abdominal ultrasonography was carried out using a 3.5–5 MHz convex transducer, following established procedures {13}. Imaging focused on identifying ascitic fluid, evaluating hepatic echotexture, gallbladder integrity, and renal margins. Ultrasonography was preferred due to its sensitivity in detecting even small volumes of peritoneal fluid and for characterizing parenchymal abnormalities of the liver and kidneys.

Ascitic Fluid Collection and Evaluation

Abdominocentesis was performed in cases with marked abdominal distension. Fluid was aspirated aseptically using 22G needles for gross and biochemical analysis. Parameters such as color, clarity, protein content, and albumin were noted. While SAAG (Serum-Ascitic Albumin Gradient) is considered a valuable diagnostic marker for portal hypertension {1}, it was not applied in all cases due to logistical limitations.

Grouping of Animals

Based on clinical and laboratory findings, animals were retrospectively grouped by presumed aetiology. Three dogs were categorized under hepatic origin, and two under renal involvement, consistent with the biochemical and ultrasonographic profiles. The grouping followed approaches like those used in diagnostic classification {2}. Grouping was irrespective of breed, sex, or age, and each case was managed individually.

Table 1. Initial Biochemical Profile of Ascitic Dogs Presented to TVCC ANDUAT (Compared with Reference Ranges)

Parameter	Reference Range	Spitz (Dog 1)	Labrador (Dog 2)	Rottweiler (Dog 3)	GSD Puppy (Dog 4)	Labrador ♀ (Dog 5)
ALT (U/L)	10 – 100	41.03 ✓	17.92 ✓	29.55 ✓	42.59 ✓	13.27 ✓
AST (U/L)	10 – 50	24.08 ✓	0.11 ↓	105.27 ↑	18.88 ✓	19.74 ✓
Total Protein (g/dL)	5.5 – 7.5	6.03 ✓	5.63 ✓	5.41 ↓	3.67 ↓	5.54 ✓
Albumin (g/dL)	2.6 – 3.3	2.82 ✓	0.24 ↓	2.64 ✓	1.41 ↓	1.43 ↓
Blood Urea Nitrogen (mg/dL)	7 – 27	87.52 ↑	48.60 ↑	24.33 ↓	39.68 ↑	145.48 ↑
Creatinine (mg/dL)	0.5 – 1.8	1.52 ✓	1.18 ✓	1.14 ✓	0.27 ↓	4.11 ↑
Total Bilirubin (mg/dL)	0.1 – 0.6	—	0.24 ✓	—	1.59 ↑	0.19 ✓
Direct Bilirubin (mg/dL)	0.0 – 0.3	—	—	—	0.88 ↑	0.08 ✓

✓ = Within reference range ↑ = Above reference range ↓ = Below reference range

Reference ranges based on Jain (1993), Benjamin (1985), and Ettinger & Feldman (2017)

Haematological and Serum Biochemical Profile

In the current study of five ascitic dogs presented to TVCC, ANDUAT, the haematological findings indicated significant alterations in haemoglobin levels, packed cell volume (PCV), total erythrocyte count (TEC), and leukocyte distribution. Anaemia, characterized by reduced Hb and PCV, was observed in the GSD puppy (Hb: 10.3 g/dL, PCV: 28.6%), the female Labrador (Hb not recorded, but clinical signs indicated systemic debilitation), and more severely in the Rottweiler (Hb: 9.0 g/dL, PCV: 22.3%) and the Labrador male (Hb: 8.0 g/dL, PCV: 24.3%). This aligns with findings that ascitic dogs, particularly those with chronic liver disease or nephropathy, often display non-regenerative anaemia due to decreased erythropoiesis and systemic protein loss {1,2}.

Total erythrocyte count (TEC) was below the reference range in the Rottweiler and Labrador, correlating with reduced

DOI: https://doi.org/10.51470/AMSR.2025.04.01.46

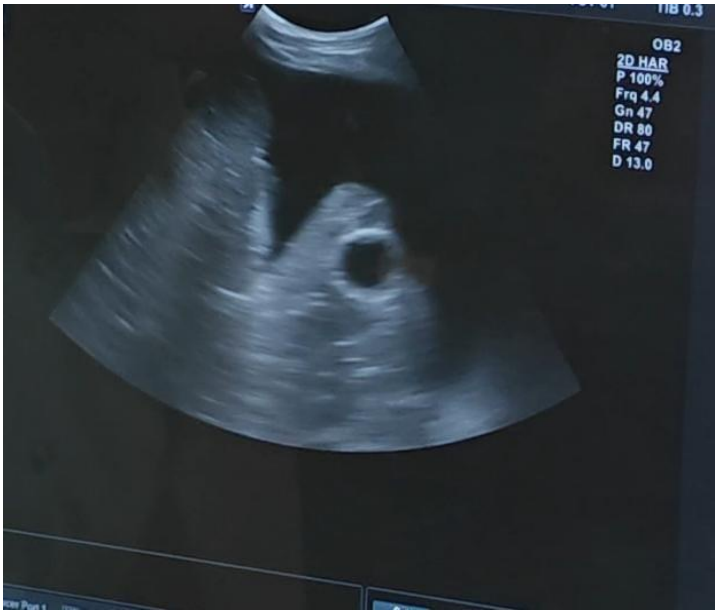
erythropoietin production and possible chronic disease anaemia, especially in renal-origin ascites {3}. In contrast, the Spitz (TEC: 5.18 million/µL, PCV: 43%) exhibited values within normal limits, possibly due to early-stage or subclinical hepatic involvement.

Leukogram evaluation revealed leucocytosis in the Spitz (TLC: 18,000/µL) and GSD puppy (TLC: 16,030/µL), with lymphocytic dominance in the Spitz (83%) and neutrophilia in the GSD puppy (27.1%) and Labrador (19.2%). Elevated TLC has been previously associated with systemic inflammation, hepatic damage, or secondary bacterial peritonitis {4}. Lymphocytosis, as seen in the Spitz, may reflect chronic immune stimulation or viral aetiology, while neutrophilic shifts often accompany bacterial infection, protein-losing enteropathy, or acute hepatic inflammation {1,2}.

Serum ALT and AST levels showed moderate elevation in the Rottweiler (ALT: 29.55 U/L; AST: 105.27 U/L) and GSD puppy (ALT: 42.59 U/L; AST: 18.88 U/L), suggesting hepatocellular injury or early hepatic fibrosis. ALT is hepatocyte-specific and released upon membrane leakage, while AST, found in both liver and muscle, rises significantly with necrosis or cirrhosis {5}. ALP, though not consistently measured in all cases, has been strongly linked to cholestasis and hepatic dysfunction, especially in chronic liver disease {6}.

Hypoproteinaemia and hypoalbuminemia were striking features in the Labrador male (Albumin: 0.24 g/dL), GSD puppy (1.41 g/dL), and female Labrador (1.43 g/dL). These findings indicate either hepatic synthetic failure or renal protein loss, both of which reduce oncotic pressure and promote fluid leakage into the abdominal cavity {1}. The Rottweiler, though moderately hypoalbuminemia (2.64 g/dL), maintained relatively better hepatic output, consistent with ultrasonographic evidence of mild hepatic fibrosis. Total protein also dropped below the reference range in these dogs, a common finding in advanced hepatic or renal pathology {7,8}.

Elevated blood urea nitrogen (BUN) and creatinine levels were most prominent in the female Labrador (BUN: 145.48 mg/dL; Creatinine: 4.11 mg/dL), indicative of renal azotaemia and reduced glomerular filtration. Similar renal-origin ascites cases have been documented where accumulation of nitrogenous waste correlated with fluid retention {9}. Mild to moderate increases in BUN were also observed in the GSD puppy (39.68 mg/dL) and Labrador male (48.60 mg/dL), supporting renal contribution to the ascitic pathology.



A:G ratio, though not calculated numerically in all cases, was inferred to be low in dogs with marked hypoalbuminemia, especially in the Labrador male and GSD puppy. Such imbalances further contribute to ascites and have prognostic value in chronic hepatic failure {10}.

Overall, the haematological and biochemical changes in these dogs correlate well with the degree of hepatic or renal involvement identified via clinical and ultrasonographic assessment. These trends are consistent with the patterns previously described in ascitic dogs {1,2,11}.

Radiographic and Ultrasonographic Findings

In the present study, radiographic evaluation of all five ascitic dogs revealed characteristic loss of abdominal detail with a ground-glass appearance suggestive of peritoneal effusion. This radiographic pattern, attributed to fluid-induced soft tissue opacity, aligns with classical descriptions of ascitic radiographs {1,2}, and further confirms the utility of plain radiography in identifying moderate-to-severe abdominal effusions.

Abdominal ultrasonography was found to be highly effective in both detecting free fluid and assessing organ architecture. All dogs exhibited anechoic or hypoechoic peritoneal fluid accumulation, displacing visceral organs from their normal anatomical positions. This observation concurs with previous findings documenting similar anechoic patterns in ascitic dogs with hepatic and renal pathologies {3,4}. The Spitz, Rottweiler, and Labrador male demonstrated varying degrees of hyperechoic hepatic parenchyma, with the Rottweiler showing focal echogenicity and irregular liver margins, indicating possible hepatic fibrosis or early cirrhosis. Such echogenic changes, including coarse stippling and marginal irregularities, have been reported as hallmark ultrasonographic features of chronic hepatic disease {5,6}.

The female Labrador, suspected to have renal-origin ascites, exhibited large-volume effusion and mild hepatic echogenicity, but her kidneys appeared normal in shape, size, and cortical echotexture like observations in diffuse hepatic disease with normal renal presentation {7}. Gallbladder distension was observed in both the GSD puppy and Labrador male, indicating biliary stasis or hepatobiliary compromise. In the GSD puppy, the presence of anechoic fluid with floating organs and mild hyperechoic liver texture suggested ongoing hepatic damage.

Improvement in ultrasonographic appearance following treatment was more pronounced in dogs with hepatic origin ascites. The Spitz and Rottweiler showed restoration of liver margins and reduced free fluid on follow-up scans, whereas in renal-origin cases, such as the female Labrador, the fluid persisted longer with slower hepatic recovery. These patterns reaffirm that ultrasonography not only aids in fluid detection but also enables assessment of hepatic structural recovery during treatment {8,6}.

In summary, ultrasonography proved to be a non-invasive, reliable, and sensitive modality in evaluating ascitic cases, helping distinguish hepatic versus renal involvement based on organ echotexture and fluid patterns. It was instrumental in both initial diagnosis and monitoring therapeutic progress, echoing the diagnostic and prognostic significance noted in previous studies {9,5}.

DOI: https://doi.org/10.51470/AMSR.2025.04.01.46

Table 2. Ultrasonographic Findings in Ascitic Dogs Presented to TVCC ANDUAT

Dog ID	Breed	Hepatic Echotexture	Gallbladder Findings	Kidney Appearance	Fluid Pattern	Suspected Aetiology
1	Spitz	Mildly hyperechoic; uniform	Normal	Normal	Anechoic free fluid	Early hepatic origin
2	Labrador ♂	Coarse; irregular margins	Mild distension	Not evaluated	Moderate anechoic	Hepatic fibrosis
3	Rottweiler	Focal hyper echogenicity; irregular margins	Normal	Normal	Moderate free fluid	Hepatic origin
4	GSD Puppy	Mildly hyperechoic	Slight distension	Normal structure	Anechoic; floating viscera	Renal contribution
5	Labrador ♀	Mild echogenicity	Not significant	Normal shape and size	Large-volume anechoic	Renal origin

Key Notes:

- Anechoic fluid was present in all dogs.
- Hepatic echogenicity and gallbladder distension were prominent in hepatic ascites.
- Normal kidneys helped rule out intrinsic renal disease in hepatic-origin cases.
- Floating viscera were observed especially in GSD Puppy, suggestive of high-volume free fluid

Therapeutic Management - The therapeutic management of ascites in all five dogs presented at TVCC ANDUAT was based on the presumed hepatic or renal origin and tailored according to clinical symptoms, laboratory findings, and ultrasonographic interpretation. In the Spitz, clinical signs such as abdominal swelling, pain, and elevated blood urea (87.52 mg/dL) were initially managed using antimicrobial, anti-inflammatory, and supportive agents. Subsequent therapy incorporating Livotas, Zipvit, Bestozyme, diuretics (Lasix), and nephroprotective (Urigo) led to clinical improvement. Addition of Amylcure DS and Dytor (furosemide + spironolactone) for 15 days, alongside fluid tapping, accelerated recovery, in agreement with findings that silymarin and diuretics are beneficial in managing hepatic ascites through hepatocellular repair and fluid mobilization {1}.

In the Labrador with scrotal and limb oedema, severe hypoalbuminemia (0.24 g/dL), and oliguria, oral supplementation with liver tonics (Livotas Pet), vitamin-mineral support (Ferrican, Amino Acid), and fluid-eliminating agents (Dytor Plus) provided effective relief by Day 15. This correlates with earlier reports that silymarin promotes hepatic regeneration and protects hepatocytes by stabilizing cellular membranes and acting as an antioxidant {2,3,4}.

In the Rottweiler, who had chronically elevated liver enzymes (AST: 105.27 U/L), stepwise addition of Cannitone LS, Amylcure, Chelidonium 30C, and injectables like Belamy and L-carnitine contributed to hepatic recovery and improved appetite. These results mirror previously observed benefits of herbal hepatoprotectives such as *Silybum marianum*, *Tribulus terrestris*, *Solanum nigrum*, and *Boerhaavia diffusa*, which possess anti-inflammatory, diuretic, and immunomodulatory effects useful in liver pathology {5,6}.

The 3-month-old GSD puppy, with hypoalbuminemia (1.41 g/dL), anaemia (Hb: 10.3 g/dL), and high bilirubin (1.59 mg/dL), responded well to a regimen of Hesta Liv, amino acid supplements, and Dytor Plus. Notable improvement by Day 10 was supported by reductions in abdominal girth and restoration of appetite, aligning with reports that supportive hepatic therapy and protein-rich nutrition enhance recovery in early-stage ascites {7}.

The female Labrador, diagnosed with renal-origin ascites based on highly elevated urea (145.48 mg/dL) and creatinine (4.11 mg/dL) but relatively normal ALT and AST, was treated with IV fluid therapy (RL and DNS), nephroprotective agents (Renogrit, Neeri KFT), and diuretics (Dytor Plus), resulting in improved hydration status and reduced abdominal distension. Herbal nephroprotectives with antioxidant and diuretic actions have previously demonstrated positive effects on renal ascites by promoting toxin clearance and maintaining fluid balance {6}.

Across all five cases, therapeutic success was attributed to a combination of targeted agents silymarin-based heptatonic, potent loop diuretics like furosemide, and herbal neuroprotective supported by nutritional and fluid therapy. Improvements were observed within 10–20 days, and the use of silymarin was particularly notable for its membrane-stabilizing, anti-lipid peroxidative, and hepatoregenerative properties {8,9,10}. The rationale for diuretic use lies in their renal handling, with furosemide acting on the loop of Henle to reduce sodium reabsorption and promote fluid excretion, as established in previous pharmacodynamic studies {11,12}.

All five dogs demonstrated complete clinical recovery with no recurrence of ascites during the follow-up period, confirming the efficacy and reliability of the chosen therapeutic protocols.

Thus, the outcomes support the therapeutic potential of combined silymarin and diuretic regimens in canine ascites, especially when guided by organ-specific biochemical markers and real-time imaging.

Discussion and Conclusion

Ascites, characterized by the accumulation of serous or serosanguineous fluid in the peritoneal cavity, represents a common yet diagnostically complex clinical condition in dogs. In the present series of five canine ascites cases, clinical, haematological, biochemical, and ultrasonographic evaluations were central to distinguishing the underlying aetiologiesprimarily hepatic and renal. This aligns with the consensus that ascites is a clinical syndrome rather than a disease per se, often secondary to chronic hepatic failure, nephropathy, or hypoalbuminemia {1}.

Clinically, all dogs exhibited hallmark signs such as abdominal distension, reduced appetite, lethargy, and in some, peripheral or scrotal oedema. The Labrador male and Rottweiler demonstrated marked hypoalbuminemia (0.24 g/dL and 2.64 g/dL respectively), which likely contributed to decreased plasma oncotic pressure and subsequent transudation of fluid, as also previously observed in ascitic dogs {2}. Concurrently, the GSD puppy and female Labrador displayed elevated urea and creatinine levels, suggesting renal dysfunction as a contributing or primary cause. These findings substantiate earlier reports that emphasized the role of renal retention of sodium and water, along with protein loss, in the pathogenesis of ascites {3}.

Ultrasonographic examination proved indispensable in confirming fluid accumulation and assessing hepatic and renal parenchymal architecture. All five dogs exhibited anechoic or hypoechoic fluid in the peritoneal cavity, with the Spitz and Rottweiler showing hyperechoic hepatic tissue and irregular margins, indicative of chronic hepatic insultfindings consistent with previous ultrasonographic observations in dogs with hepatic ascites {4}. These imaging features aided in correlating clinical and biochemical data for etiological differentiation, especially when serum ascitic albumin gradient (SAAG) estimation was logistically constrained {5}.

Therapeutically, aetiology-specific treatment yielded favourable outcomes. Dogs with hepatic origin ascites responded well to the administration of silymarin-based hepatoprotectives such as Livotas and Amylcure DS.

DOI: https://doi.org/10.51470/AMSR.2025.04.01.46

The therapeutic efficacy of silymarin lies in its multifaceted actions including hepatocellular membrane stabilization, antioxidant effects, enhancement of protein synthesis, and inhibition of lipid peroxidation {6}. Silybinin, the principal bioactive constituent of silymarin, has been shown to induce mitochondrial biogenesis and reduce hepatic oxidative stress and inflammatory mediators {7}. This is consistent with the clinical improvement observed in the Spitz, Rottweiler, and Labrador male within 15–20 days of initiating silymarin therapy.

The use of loop diuretics such as furosemide, in combination with aldosterone antagonists like spironolactone, was effective in mobilizing ascitic fluid, especially in cases of fluid overload and oliguria. This pharmacological synergy exploits different nephron sites to reduce sodium and water reabsorption and has been previously validated in the management of ascitic conditions {8,9}. Adjunct use of amino acid formulations and vitamin-mineral supplements further supported hepatic and systemic recovery, particularly in the GSD puppy and female Labrador where anaemia and debility were pronounced. The therapeutic combination used in these cases closely mirrors protocols employing multivitamin heptatonic containing tricholine citrate, selenium, inositol, and vitamin B12, which are reported to have antioxidant, lipotropic, and regenerative actions on hepatic tissues {10,11}.

In the renal-origin ascites case (female Labrador), management focused on nephroprotective (Renogrit, Neeri KFT) and cautious fluid and electrolyte therapy, which successfully restored appetite and reduced abdominal girth. This approach is consistent with previous recommendations emphasizing the use of supportive renal therapeutics and diuretics in non-hepatic ascitic conditions {3}.

Importantly, all five dogs in the study demonstrated complete clinical recovery without recurrence of ascitic signs during the follow-up period. This highlights the efficacy of aetiology-guided therapeutic regimens and underscores the value of individualized treatment planning in achieving successful outcomes. In summary, this case series reinforces the clinical relevance of a multimodal, organ-specific approach in the therapeutic management of canine ascites. Silymarin-based hepatoprotectives were observed to be particularly effective in hepatic ascites, while diuretics, nephroprotective agents, and supportive therapy played a critical role in renal-origin ascites. Ultrasonography not only facilitated the initial diagnosis but also helped in monitoring treatment efficacy. Thus, early identification of the underlying pathology, coupled with evidence-based medical management, can result in significant clinical improvement and favourable prognosis in dogs suffering from ascites.

Future Scope

While the present study offers valuable insights into the clinical and diagnostic patterns of ascites in dogs, it is not without limitations. One notable constraint was the unavailability of Serum-Ascitic Albumin Gradient (SAAG) evaluation in all cases, which is considered a superior method for distinguishing between portal hypertension and other causes of ascites {1}. Incorporating SAAG analysis across all cases could have strengthened diagnostic stratification, particularly in differentiating hepatic versus non-hepatic effusions. Additionally, the inability to consistently perform ascitic fluid cytology and bacterial culture limited a deeper understanding of potential infectious or neoplastic causes {2}.

Future studies should aim to incorporate molecular diagnostics, such as PCR-based assays for hepatotropic viruses, *Leptospira* spp., or other infectious agents, which can significantly enhance early and specific aetiological identification {3}. Moreover, longitudinal follow-up data would provide crucial information on recurrence rates, survival, and the comparative efficacy of hepatoprotective versus nephroprotective regimens {4,5}. The establishment of standardized clinical guidelinesincluding severity grading of ascites, criteria for therapeutic paracentesis, and monitoring indiceswill be instrumental in streamlining diagnosis and treatment protocols across veterinary centres. The integration of such guidelines can enable uniform, evidence-based approaches to canine ascites and improve both prognosis and patient care {6,7}.

References

1. Berman, L. B., & Ebrahimi, M. (1965). Pharmacological properties of furosemide. *The American Journal of Cardiology, 15*(4), 538–543.

2. Cornelius, L. M., Thrall, D. E., Halliwell, W. H., Frank, G. M., Kern, A. J., & Wood, C. B. (1975). Diagnostic significance of biochemical changes in hepatic insufficiency. *Journal of the American Veterinary Medical Association, 167*, 220–228.

3. Deepak, K. K., Raj, G. D., & Arulmozhi, A. (2015). Evaluation of hepatoprotective efficacy of multivitamin herbal formulations in dogs. *Indian Veterinary Journal, 92*(1), 44–46.

4. Jana, S., Das, P. K., Banerjee, D., Ghosh, D., Mukherjee, P., & Mukherjee, J. (2019). A case study on ascites of hepatic origin in a dog. *Indian Journal of Animal Health, 58*(1), 135–138.

5. Pandey, V., & Sahni, Y. P. (2011). Silymarin: A miracle herb in treatment of liver diseases. *International Journal of Current Microbiology and Applied Sciences, 8*(9), 1120–1137.

6. Pradhan, M. S., Dakshinkar, N. P., Waghaye, U. G., & Bodkhe, A. M. (2008). Successful treatment of ascites of hepatic origin in a dog. *Veterinary World, 1*(1), 23.

7. Randhawa, S. S., Dhaliwal, P. S., Kirti, S., & Singh, K. B. (1988). Ascites of hepatic origin in dogs. *Indian Journal of Animal Health, 12*, 165–166.

8. Runyon, B. A. (1994). The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. *Annals of Internal Medicine, 117*(3), 215–220.

9. Saller, R., Meier, R., & Brignoli, R. (2001). The use of silymarin in the treatment of liver diseases. *Drugs, 61*(14), 2035–2063.

10. Saravanan, M., Sarma, K., Kumar, M., Mahendran, K., & Mondal, D. B. (2013). Therapeutic management of ascites in dogs. *Indian Veterinary Journal, 90*(2), 110–111.

11. Seeff, L. B., Curto, T. M., Szabo, G., Everson, G. T., Bonkovsky, H. L., et al. (2008). Herbal product use by persons enrolled in the HALT-C trial. *Hepatology, 47*(2), 605–612.

DOI: https://doi.org/10.51470/AMSR.2025.04.01.46

12. Serviddio, G., Bellanti, F., Tamborra, R., Rollo, T., Romano, A. D., et al. (2014). Silybinin induces mitochondrial biogenesis in cirrhotic rats. *Clinical Science, 126*(8), 517–527.

13. Skardova, I. (1991). Decrease of total protein and albumin in the blood serum of ascitic dogs. *Veterinarstvi, 41*(5), 205–207.

14. Timmerman, L. A., Schultze, M. O., & Heyl, A. (1964). Pharmacology of furosemide. *Archives Internationales de Pharmacodynamie et de Thérapie, 149*, 273–285.

15. Trinchet, J. C., Coste, T., & Roulot, D. (1989). Protective effect of silymarin against liver damage. *Liver, 9*, 193–197.

DOI: https://doi.org/10.51470/AMSR.2025.04.01.46