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Multimodal assessment and management of ascites in dogs: A study on clinical parameters, laboratory findings, imaging features and therapeutic response#

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Abstract

Ascites, the abnormal accumulation of fluid within the peritoneal cavity, is an important clinical manifestation in dogs, reflecting diverse underlying etiologies. The present study was conducted in dogs with ascites presented to University Veterinary Hospitals, Mannuthy and Kokkalai, between September 2024 and May 2025 Ascites was attributed to hepatic etiology in 46.67 per cent, cardiac etiology in 40 per cent and hypoproteinaemia 13.33 per cent of cases. Clinical signs reported cases were abdominal distension, anorexia, weight loss, weakness, vomiting, melena and respiratory distress. Haematology revealed anaemia in both hepatic and cardiac cases. Elevated gamma-glutamyl transferase (GGT), creatinine and total bilirubin were observed in cardiac cases. Ascitic fluid obtained was protein-rich or protein-poor transudate. Echocardiography confirmed systolic dysfunction in dilated cardiomyopathy (DCM), whereas ultrasonography in hepatic cases revealed microhepatica, irregular margins and regenerative nodules. Dogs with cardiac causes of ascites responded favourably to combination therapy with pimobendan, diuretics and angiotensin converting enzyme (ACE) inhibitors. Dogs with cirrhosis and portal hypertension were treated with a multimodal regimen including carvedilol, diuretics, amino acid supplementation and liver supportives. The study emphasizes the importance of systematic diagnostic evaluation and tailored multimodal management in canine ascites.

Keywords: *Ascites, cirrhosis, DCM, portal hypertension*

Ascites, the pathological accumulation of fluid within the peritoneal cavity, reflects serious underlying diseases. Its development is multifactorial, with portal hypertension being the predominant mechanism, especially in hepatic disorders. Cardiovascular diseases, including right-sided heart failure, dilated cardiomyopathy, pulmonary stenosis and heartworm infestation frequently responsible for ascites formation. Hypoproteinaemia from liver diseases, renal amyloidosis, glomerulonephritis, or protein-losing enteropathies may also predispose to fluid accumulation. Abdominal neoplasia, traumatic haemorrhage and urinary bladder rupture are also less common causes of ascites.

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(Pratibha *et al*., 2022). The pathophysiology of ascites formation involves an imbalance of hydrostatic and oncotic pressures, increased vascular permeability and impaired lymphatic drainage. In hepatic disorders, intrahepatic fibrosis elevates portal venous pressure and together with systemic vasodilation, neurohormonal activation, and renal sodium and water retention, creates a self-perpetuating cycle of ascites formation (Buob *et al*., 2011). Diagnosis of ascites in dogs requires a systematic approach, integrating clinical assessment with laboratory and imaging techniques. Ultrasonography is the most sensitive modality for detecting even small fluid volumes and provides concurrent evaluation of hepatic and cardiac pathology. Evaluation of protein concentration, total nucleated cell count and calculation of serum ascites albumin gradient provide important clues regarding the underlying cause. A clear understanding of the underlying pathophysiology and judicious use of diagnostic tools are crucial for effective case management and prognosis.

Materials and methods

Dogs of all breed, age and gender presented to University Veterinary Hospitals at Mannuthy and Kokkalai from September 2024 to May 2025 with clinical signs and complaints of a distended abdomen was studied. Twenty-five cases presented with distended abdomen were screened initially by ultrasonography for fluid accumulation. Among 25 cases, 15 dogs with presence of fluid accumulation were randomly selected for the present study and detailed clinical examination of the dogs was conducted. Six apparently healthy dogs served as control.

Data including age, breed, gender, body weight, onset and duration of clinical signs, diet of the animal, urination characteristics, exercise intolerance and previous treatment history were recorded. Abdominal girth (cm) was measured on the day zero and on subsequent reviews. Body condition was scored on the day of presentation according to body condition score (BCS) of World Small Animal Veterinary Association (WSAVA) nutritional committee on a scale from one to nine. All the dogs were subjected to detailed clinical examination and temperature (°F), pulse (rate/min), respiratory (rate/min) were recorded.

Right lateral view of abdomen and thorax were attained, detailed ultrasonographic examination of abdomen and echocardiography was performed (Mattoon and Nyland, 2015). Haematology, serum biochemistry, faecal sample, wet film, peripheral blood smear examination and urinalysis was performed.

Ascitic fluid was collected by single-site centesis according to Bexfield and Lee (2014). Physical characteristics was evaluated (Cote *et al*., 2024) and quantitative evaluation of total protein, albumin and creatinine in fluid. Cytology was done to detect abnormal cells. Ascitic fluid collected in an EDTA vial was subjected to nucleated cell count by using Neubauer's haemocytometer.

Serum ascites albumin gradient was calculated by subtracting ascitic fluid albumin from serum albumin level as per Shahed and Rahman (2016). Blood pressure was measured non-invasively. Electrocardiogram of the animals with ascites were recorded using 12 12-channel ECG recorder. Ascites diagnosed with each etiology were treated following standard treatment protocols. Animals after initial stabilization were reviewed at seven-day interval for two weeks. Statistical analysis of data obtained was carried out by independent student's t test, paired sample t test, repeated measures ANOVA and Friedman test using SPSS version 24.0.

Results and discussion

Of the 15 cases selected with ascites, six were diagnosed with ascites due to cardiac causes (40 per cent), seven due to cirrhosis (46.67 per cent) and two due to hypoproteinemia (13.33 per cent). In the present study, ascites was most common in Labrador Retrievers(33.33 per cent), followed by Doberman and Pomeranian breeds (13.33 per cent each), while German Shepherd, Rottweiler, Lhasa Apso, Pug, Non-descript and Dachshund accounted for 6.66 each. Females (60 per cent) were more frequently affected than males. Ascites was more in mature adult dogs (2–6 years), followed by senior, adolescent and geriatric animals. These results contradict with Baria *et al*. (2024), who observed a higher incidence in elderly and geriatric animals and Kumar *et al*. (2022), who reported peak occurrence in dogs aged 6 to 9 years. In terms of heterogeneity in causes of ascites, it would be improper to relate the prevalence of ascites to age of dogs.

The predominant presenting complaints were abdominal distension, anorexia, weakness, vomiting, weight loss, melena, and respiratory distress, consistent with earlier reports (Webster *et al*., 2019; Bekoe *et al*., 2020). Clinical signs such as exercise intolerance, cough, retching, and limb oedema was also observed in ascites cases due to cardiac etiology (Martin *et al*., 2009). Distended abdomen (100 per cent) and dyspnoea (73.33 per cent) were the primary clinical sign seen in ascitic animals followed by enlarged lymph node, cachexia, weak pulse, pale mucous membrane, limb oedema. Body condition score (BCS) of majority of dogs affected with cirrhosis or cardiac disease had a low BCS (2 and 3) with visible ribs, lumbar vertebrae, pelvic bones, and muscle wasting, likely due to reduced intake, malabsorption, and hypermetabolism (Plauth and Schutz, 2002).

No statistically significant difference was observed in rectal temperature between diseased and healthy dogs, consistent with the findings of Baria *et al*. (2024). A significant increase in pulse (122.2 ± 5.13) and

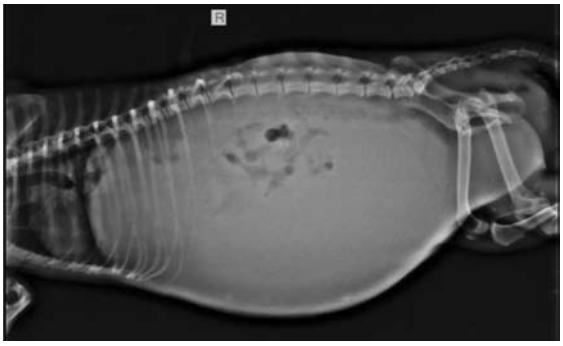

Fig.1: Abdominal radiograph of ascitic dog with ground glass appearance

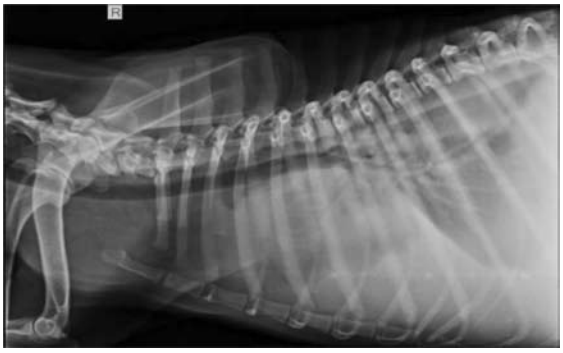

Fig. 2: Thoracic radiograph of ascitic dog with DCM and pleural effusion

respiratory rates (37.67 ± 1.42) was recorded in affected animals when compared to healthy control (101.16 ± 4.00), (29.00 ± 0.89) respectively . The increased respiratory rate may be attributed to diaphragmatic compression from ascitic fluid (Tilley *et al*., 2024). Although the pulse rate of diseased dogs remained within the upper physiological range of 70–180 beats/min (Tilley *et al*., 2024), its significant elevation compared to controls may be related to systemic hypertension noted in most cases. Additionally, muffled heart and lung sounds were identified in three dogs with pleural effusion, consistent with descriptions by Cote *et al*. (2024). Cardiac murmurs were also detected in dogs with DCM, aligning with the observations of Wess *et al*. (2017). Routine faecal sample examination was negative for ova of gastrointestinal parasites and blood smear was negative in all cases.

Abdominal radiographs of ascitic dogs revealed a characteristic ground-glass appearance (Bekoe *et al*., 2020) (Figure 1). Thoracic radiographs of pleural effusion cases showed obscured cardiac silhouette, increased thoracic opacity and scalloped ventral lung margins, consistent with Thrall (2018) (Figure 2). In dogs with DCM, dorsal tracheal displacement, cardiomegaly, pulmonary oedema, increased sternal contact and interstitial patterns were common (Martin *et al*., 2009; Saini *et al*., 2023; Umesh, 2025). These findings support the pathophysiology described by Thrall (2018); wherein

Table 1. Haematological parameters of cardiac group compared to healthy control on day zero

Parameters	Diseased dogs on day 0 (n=6)	Healthy control (n=6)	t- value	p- value
TEC (10^6 / µL)	4.68± 0.26	6.13 ± 0.25	3.97**	0.003
Hb (g/dL)	10.97 ± 0.87	14.28 ± 0.41	3.46**	0.006
VPRC (%)	30.42 ± 2.73	41.25 ± 1.45	3.51**	0.006

¨Significant at 0.01 level

Table 2. Haematological parameters of cardiac group on day zero compared with day seven and day 14

Parameters	Day 0 (n=3) Median IQR (g/dL)	Day 7 (n=3) Median IQR (g/dL)	Day 14 (n=3) Median IQR (g/dL)	χ^2 value	p value
TEC (10^6 / µL)	4.45 (3.59-5.44)	4.54 (4.50-4.92)	4.93 (4.37-5.05)	0.00	1.00
Hb (g/dL)	10.80 (7.10-12.90)	10.60 (9.00-11.50)	11.30 (10.80-11.60)	2.36	0.31
VPRC (%)	25.60 (19.50-36.90)	27.20 (26.30-33.50)	29.60 (26.50-34.40)	2.00	0.37
TLC (10^3 / µL)	24.00 (6-25.80)	11.30 (9.60-36.40)	14.30 (8.80-16.90)	0.67	0.72
Granulocyte (%)	72.00 (70.80-83.90)	73.40 (67.50-80.50)	83.90 (79.90-84.50)	3.82	0.15
Lymphocyte (%)	23.20 (13.69-25.10)	22.70 (16.10-26.90)	13.00 (12.60-16.40)	4.67	0.09
Monocyte (%)	4.10 (2.50-4.80)	3.90 (3.40-5.60)	3.10 (2.90-3.70)	2.67	0.26
Platelet (10^3 / µL)	174 (141-272)	394 (173-835)	336 (171-380)	2.00	0.37

elevated left ventricular end-diastolic pressure leads to pulmonary venous hypertension and subsequent interstitial to alveolar pulmonary oedema. Increased vertebral heart scores (VHS) in DCM cases supported previous reports (Vishnurahav *et al*., 2018), with values above 10.7 considered strongly indicative of cardiac disease (Tilley *et al*., 2024).

Ascites due to cardiac etiology

In the present study, six cases of ascites were attributed to cardiac causes, comprising five cases of dilated cardiomyopathy (DCM) and one case of valvular insufficiency.

Haematology revealed a statistically significant reduction in erythrocyte count, haemoglobinand volume of packed red cells (VPRC) in diseased dogs compared with healthy controlson day zero (Table 1). Anaemia in congestive heart failure (CHF) may result from multiple mechanisms including reduced erythropoietin production due to concurrent renal dysfunction, cachexia associated increased inflammatory cytokines (TNF-α, IL-1, IL-6) impairing erythropoiesis and iron utilization. An improvement in anaemia was evident by day 14 (Table 2), suggesting a positive response to therapy.

Serum biochemistry revealed no significant differences in alanine aminotransferase (ALT), alkaline phosphatase (ALP), blood urea nitrogen (BUN) and direct bilirubin between diseased and healthy dogs on day zero; however, gamma glutamyl transferase (GGT), creatinine, and total bilirubin were significantly elevated in affected animals on day zero (Table 3). Elevated GGTand bilirubin might be due to hepatic congestion and oxidative stress in cardiac disease (Chong *et al*., 2022). Significant serum creatinine elevation in present study may result from reduced renal perfusion, renin angiotensin aldosterone system (RAAS) activation (Chong *et al*., 2022). Mean serum total protein and globulin did not differ significantly, whereas serum albumin was significantly lower in diseased dogs compared to healthy control on day zero(Table 4), in contrast with the findings of Amaravathi *et al*. (2019). Hypoalbuminaemia in CHF may result from hepatic congestion with impaired synthesis, reduced nutrient absorption due to systemic congestion and cachexia (Biancucci *et al*., 2024). In present study, although serum total protein, albumin and globulin values increased in diseased animals, the differences between day zero and day seven, day 0,7 and 14 were not statistically significant.

Ascitic fluid obtained in all cases were blood tinged (Ihedioha *et al*., 2013) with total protein and nucleated cell count (NCC) below 20 g/L and 5000 cells/ mm^3, consistent with the characteristics of a protein rich transudate(Villiers and Ristic, 2016). Cytological analysis of the fluid revealed presence of lymphocytes, few neutrophils and RBCs as per Alleman (2003). A mild, non-significant rise in fluid total protein and albumin was noted by day seven, which was contrast to progressive decline reported by Kumar *et al*. (2024). These variations may be related to transient vascular leakage, low-grade inflammation, or sampling variability. Uroabdomen was excluded, as fluid creatinine values did not exceed twice the serum creatinine concentrations (Bohn, 2016).

No significant difference in systolic, diastolic or mean arterial blood pressure values between diseased and healthy dogs was obtained (Umesh, 2025). Electrocardiographic findings of diseased dogs (Table 5) revealed a statistically significant increase in heart rate, aligning with findings of Umesh (2025) which can be attributed to sympathetic and neurohormonal responses to reduced cardiac output in DCM (Cote *et al*., 2024). The P wave duration was significantly prolonged, indicating left atrial enlargement as described in DCM (Varshney, 2020). The PR interval was significantly elevated though within the normal range of 0.08–0.12 sec (Varshney, 2020), possibly due to atrial dilation or conduction system changes (Fuentes *et al*., 2010). The QT interval prolongation, though within the reported normal range of 0.15–0.25 sec (Fuentes *et al*., 2010), suggested delayed ventricular repolarization associated with myocardial remodelling and electrical instability in DCM.

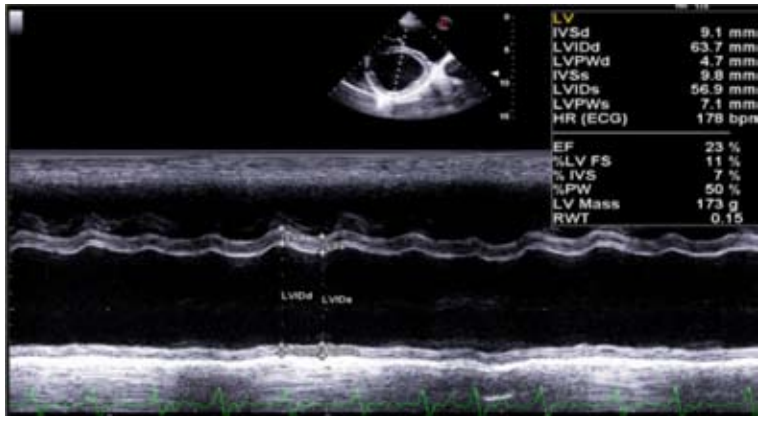

Fig. 3:M mode echocardiographic parameters of an ascitic dog with DCM

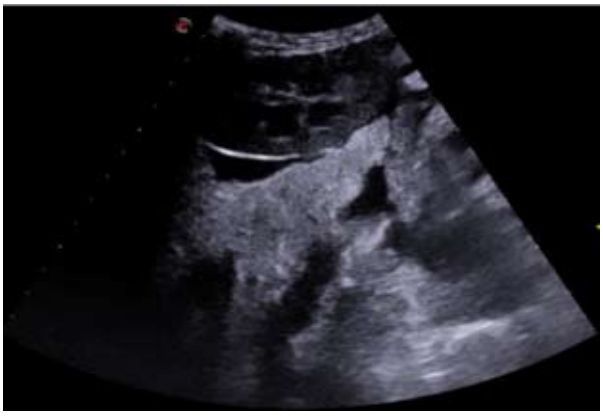

Fig. 4: Liver with irregular border, microhepatica and anechoic peritoneal fluid

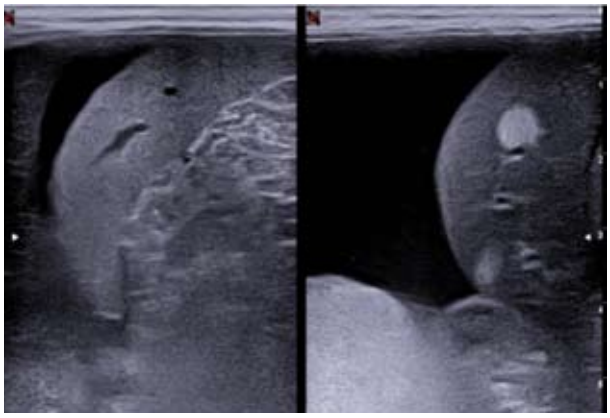

Fig. 5: Liver withmixed echogenicity and multiple hyperechoic nodules

Two-dimensional echocardiography revealed presence of pleural effusion in three cases.A significant increase in mean left atrium to aortic root (LA/Ao) ratio in diseased animals compared to control (Table6) consistent with the observations made by Umesh (2025). This elevation reflects chronic left atrial pressure and volume overload secondary to systolic dysfunction in DCM (Tilley *et al*., 2024). Ejection fraction (EF), fractional shortening (FS) and E-point septal separation (EPSS) of diseased group showed significant difference from healthy control in present study (Figure 3), (Table7). Diseased animals exhibited markedly reduced EF and FS values, aligning with findings of Umesh (2025). Reduced EF and FS in DCM result from impaired contractility, ventricular dilatation, remodelling. In DCM, ventricular dilatation and hypokinesis prevent normal anterior mitral leaflet motion, leading to elevated EPSS indicative of systolic dysfunction (Tilley *et al*., 2024). Ultrasonography revealed grade III ascites, hepatic congestion and renal congestion. Reduced perfusion due to CHF was manifested as proteinuria, haematuria, and low urine specific gravity (Littman, 2011).

In the present study, dogs with CHF were treated with a combination of pimobendan @ 0.25 mg/kg orally twice daily, diuretics and an ACE inhibitor, forming the primary therapeutic regimen. Pimobendan enhanced systolic function and reduced afterload and delayed the progression of CHF (McCauley *et al*., 2020).

Ascites due to cirrhosis

Of the fifteen cases selected seven cases were diagnosed as ascites due to cirrhosis (46.67 per cent) and one case among seven had an intraperitoneal mass with haemorrhagic effusion.

Haematology revealed a significant reduction in total erythrocyte count, haemoglobin concentration and VPRC when compared to healthy control (Table8), similar to the findings of Regmi and Shah (2017). Anaemia in dogs with cirrhosis or portal hypertension result from impaired hepatic production of hepcidin and other iron-regulating proteins disrupting iron homeostasis (Webster *et al*., 2019). In this study, diseased dogs exhibited a significant increase in leucocyte counts, likely due to reduced hepatic clearance of bacteria and endotoxins in cirrhosis due to portal hypertension, predisposing to bacteraemia or endotoxemia. Significant increase in neutrophils could be attributed to increased leucocytes.

Table 3. Serum biochemical parameters of cardiac group compared with healthy control on day zero

Parameters	Diseased dogs on day 0 (n=6)	Healthy control (n=6)	t value	p value
GGT (IU/L)	11.02 ± 2.79	1.33 ± 0.33	3.44**	0.006
Total Bilirubin (mg/dL)	1.10 ± 0.17	0.37 ± 0.04	4.26*	0.02
Creatinine (mg/dL)	1.47 ± 0.18	0.87 ± 0.10	2.85*	0.02

*** Significant at 0.01 level, * Significant at 0.05 level*

Table 4. Serum total protein and albumin of cardiac group compared with healthy control on day zero

Parameters	Diseased dogs on day 0 (n=6)	Healthy control (n=6)	t- value	p- value
Total protein (g/dL)	4.59 ± 0.50	5.56 ± 0.26	1.70	0.12
Albumin (g/dL)	2.39 ± 0.23	3.04 ± 0.16	2.39*	0.04
Globulin (g/dL)	2.20 ± 0.40	2.52 ± 0.30	0.62	0.55

**Significant at 0.05 level*

Table 5. Electrocardiographic findings of cardiac group and healthy control

Parameters	Diseased dogs (n=6)	Healthy control (n=6)	t-value	p-value
HR (bpm)	167 ± 10.92	101 ± 11.91	5.52**	0.00
P duration (sec)	0.05 ± 0.004	0.04 ± 0.00	2.23*	0.04
PR interval (sec)	0.12 ± 0.01	0.09 ± 0.007	2.58*	0.03
QT interval (sec)	0.17 ± 0.09	0.12 ± 0.04	4.34*	0.04

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

Table 6. LA/Ao of cardiac group compared with healthy control

Parameter	Diseased animals (n=6)	Healthy control (n=6)	t-value	p-value
LA/Ao	2.55 ± 0.23	1.12 ± 0.05	6.12**	0.00

*** Significant at p ≤ 0.01*

Table 7. Comparison of echocardiographic parameters between cardiac group and control

Parameters	Diseased dogs(n=6)	Healthy control (n=6)	t-value	p-value
EF (%)	38.33 ± 7.73	61.17 ± 2.08	2.85*	0.03
FS (%)	19.5 ± 4.51	31.68 ± 1.54	2.55*	0.02
EPSS (mm)	10.73 ± 1.68	3.27 ± 0.33	6.19**	0.00

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

Table 8. Haematological parameters of cirrhosisgroup compared to healthy control on day 0

Parameters	Diseased dogs on day 0 (n=7)	Healthy control (n=6)	t- value	p- value
TEC (10^6 / µL)	4.38 ± 0.51	6.13 ± 0.25	2.92*	0.014
Hb (g/dL)	9.62 ± 1.02	14.28 ± 0.41	3.96**	0.002
VPRC (%)	28.04 ± 2.92	41.25 ± 1.45	3.83**	0.003
TLC (10^3 / µL)	15.79 ± 1.56	10.47 ± 1.32	2.55*	0.03
Granulocyte (%)	81.74 ± 1.79	71.74 ± 1.87	3.84**	0.003
Lymphocyte (%)	11.47 ± 2.40	23.55 ± 1.87	3.86**	0.003

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

Serum biochemistry revealed significant differences in ALT and GGT and no significant difference in ALP, BUN, direct bilirubin, total bilirubin and creatinine value(Table 9). Significantly elevated ALT remaining within the normal range, indicated reduced hepatocyte turnover in advanced hepatic diseases (Villiers and Ristic, 2016). Statistically significant elevation of GGT in diseased dogs may be attributed to progressive hepatocellular damage and declining functional hepatic parenchyma, which preferentially elevates GGT and ALP over ALT in chronic liver disease (Webster *et al*., 2019). Serum albumin of diseased animals showed significant decrease when compared to healthy control on day zero while serum total protein and globulin remain non-significant (Table 10), (Ihedioha *et al*., 2013). Hypoalbuminemia in cirrhosis is mainly due to impaired hepatic synthesis due to hepatocellular loss, malnutrition and cytokine-mediated suppression and is considered as a late marker of hepatic synthetic failure (Webster *et al*., 2019).

All ascitic fluid samples obtained were clear and the colour ranged from colourless to yellow (Cote *et al*., 2024) and one case with cirrhosis and an intraperitoneal mass had ascitic fluid with VPRC of 3 per cent, indicating haemorrhagic effusion (Villiers and Ristic, 2016). Ascitic fluid analysis revealed protein poor transudates in four cases with NCC ranging between 300-850cells/$mm^3$and protein-rich transudates in two cases with NCC 400 and 3100 cells/mm^3.

The systolic, diastolic and mean arterial pressure did not show any significant difference in diseased group on day zero when compared to healthy control. Despite the known association between portal hypertension in cirrhosis and vasodilation in both systemic and splanchnic circulation (Buob *et al*., 2011), the blood pressure readings obtained in this study were inconsistent and difficult to interpret reliably. Electrocardiography revealed significant elevation in heart rate and QT interval of diseased animals and healthy control, while p amplitude, p duration, PR interval, R amplitude and QRS duration remained non-significant (Table 11). Significant increase in heart rate can be due to hyperdynamic circulatory syndrome linked to portal hypertension in dogs with cirrhosis (Buob *et al*., 2011). QT interval was significantly prolonged, though its precise mechanism in liver disease remains uncertain Toma *et al*., 2020). Echocardiography in cirrhotic animals revealed no significant differences in mean LA/Ao ratio, EF (%), FS (%), or EPSS (mm) between diseased and control animals.

Table 9. Serum biochemical parameters of cirrhotic group compared to healthy control on day 0

Parameters	Diseased dogs on day 0 (n=7)	Healthy control (n=6)	t value	p-value
ALT (IU/L)	49.38 ± 13.19	16.83 ± 1.35	2.45*	0.049
GGT (IU/L)	12.29 ± 2.96	1.33 ± 0.33	3.67**	0.01

***Significant at 0.01 level, *Significant at 0.05 level*

Table 10. Serum total protein and albumin of cirrhosisgroup compared with healthy control on day 0

Parameters	Diseased dogs on day 0 (n=7)	Healthy control (n=6)	t- value	p- value
Total protein (g/dL)	4.97 ± 0.45	5.56 ± 0.26	1.07	0.31
Albumin (g/dL)	2.14 ±0.22	3.04 ± 0.16	3.23**	0.008
Globulin (g/dL)	2.61 ± 0.46	2.52 ± 0.30	0.16	0.87

*** Significant at p ≤ 0.01*

Table 11. Electrocardiographic findings of cirrhosisgroup and healthy controlon day 0

Parameters	Diseased dogs (n=7)	Healthy control (n=6)	t-value	p-value
HR (bpm)	133.57± 9.32	101 ± 11.91	2.94*	0.013
QT interval (sec)	0.19 ± 0.15	0.12 ± 0.04	3.97**	0.002

**Significant at p ≤ 0.05 and ** Significant at p ≤ 0.01*

Ultrasonography revealed characteristic alterations in hepatic architecture, including irregular hepatic margins, microhepatica and a mixed echogenic parenchyma. Multiple hyperechoic hyperplastic nodules and well-circumscribed mixed echogenic lesions within the liver were also evident. Such findings are consistent with chronic hepatocellular injury and regenerative nodular changes described in cirrhosis, wherein ongoing hepatocellular necrosis and fibrosis contribute to architectural distortion and parenchymal heterogeneity (Webster *et al*., 2019). Projection of the gallbladder beyond the liver margin observed in some cases may be attributable to hepatic volume loss associated with chronic parenchymal contraction. In case with haemorrhagic effusion, the presence of echogenic peritoneal fluid and cystic intraperitoneal mass raises concern for neoplastic growth, warranting further investigation.

Urinalysis revealed haematuria and proteinuria, indicating renal compromise probably due to portal hypertension associated with cirrhosis. The consistently low urine specific gravity indicated polydipsia and polyuria associated with portosystemic shunting and advanced hepatic dysfunction (Nelson and Couto, 2014).

In the present study, dogs diagnosed with cirrhosis portal hypertension were managed using a multimodal therapeutic regimen consisting of carvedilol at 0.5 mg/kg orally twice daily, diuretics (2–4 mg/kg twice daily), fluid therapy, parenteral amino acid supplementation (Astymin/ Hermin @ 2 ml/kg), antibiotics and liver supportives, in addition to dietary sodium restriction. Despite the comprehensive management, two dogs succumbed before the day-seven review, which could be attributed to the advanced stage of disease at presentation and poor hepatic reserve. However, the remaining cases showed significant clinical improvement, with notable reduction in abdominal girth and better overall activity on successive reviews, when integrated into a multimodal therapeutic approach.

Ascites due to hypoproteinemia

Among the 15 cases of ascites, two dogs (13.33 per cent) were diagnosed with hypoproteinaemia as the underlying cause. Haematological evaluation on day zero revealed mild anaemia in one dog. Serum biochemical parameters, including ALT, ALP, GGT, total and direct bilirubin, BUN and creatinine, were within the reference range in both animals. However, serum albumin concentrations were markedly reduced at day zero (1.49 g/dL and 1.23 g/dL, respectively). A gradual and significant improvement in albumin levels was observed in subsequent reviews, suggesting recovery of protein balance following therapy.

Ascitic fluid obtained from both animals was clear and colourless, with total protein concentrations below 20 g/L, consistent with a low-protein transudate. Nucleated cell counts were 650 and 750 cells/mm^3, while cytological examination revealed low cellularity without abnormal cells, ruling out neoplasia or septic causes. Fluid creatinine values were less than twice the serum creatinine concentrations, excluding uroabdomen. Ultrasonography demonstrated anechoic peritoneal fluid accumulation, while echocardiography and electrocardiography revealed no abnormalities. Blood pressure measurements were comparable in both cases, with systolic, diastolic, and mean arterial pressures averaging 124/79/92 mmHg and 124/78/95 mmHg, respectively, indicating hemodynamic stability. Urinalysis revealed specific gravity <1.005 in both dogs, suggestive of impaired urine concentration ability. A fall in serum albumin may result from insufficient dietary protein or energy intake, impaired hepatic synthesis,

increased renal or gastrointestinal losses, or redistribution mechanisms (Gatta *et al*., 2012). Since no overt disease process could be demonstrated in these animals, inadequate dietary protein intake appeared to be the most likely explanation for the hypoproteinaemia.

Therapeutically, both animals were managed with intravenous furosemide at 2 mg/kg daily, intravenous amino acid supplementation (Astymin @ 2 ml/kg on alternate days), oral protein supplementation, dietary sodium restriction, and adjustment of dietary protein levels. On successive evaluations, a marked reduction in abdominal distension and improved activity were observed, accompanied by resolution of ascites. This suggests that nutritional correction played a pivotal role in restoring protein balance and contributed to clinical recovery.

Conclusion

Ascites in dogs arises from diverse etiologies, with cirrhosis and cardiac diseases being predominant causes, followed by hypoproteinaemia. Comprehensive evaluation integrating clinical, laboratory, imaging and ascitic fluid analysis is critical for accurate diagnosis and differentiation of portal hypertensive from non-portal hypertensive causes. Ascites due to cardiac disease responded well to standard triple therapy with pimobendan, diuretic, and ACE inhibitors; cirrhotic dogs with portal hypertension benefited from multimodal therapy including carvedilol, highlighting its potential role in veterinary medicine. However, prognosis remains guarded in advanced hepatic disease with poor hepatic reserve. Early recognition, timely intervention and proper therapeutic protocols are essential for improving clinical outcomes in canine ascites.

Conflict of interest

There are no conflicts of interest reported by the authors.

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