RESEARCH ARTICLE

Clinical, Haemato-Biochemical and Ultrasonographic Evaluation of Ascites of Hepatic Origin in Dogs

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Abstract

This study evaluated ascites of hepatic origin in dogs through haemato-biochemical profiling, ultrasonography and peritoneal fluid analysis to establish reliable diagnostic indicators. Twenty-four dogs with confirmed hepatic ascites presented to the Veterinary Clinical Complex, Veterinary College, Shivamogga (January-June 2025) were included in the study. Clinical signs included abdominal distension (100%), anorexia (83.3%), epigastric pain (50%) and diarrhoea (45.8%). Haematology revealed significant reductions in haemoglobin, packed cell volume and erythrocyte count, with leukocytosis, while platelet counts remained unchanged. Biochemical analysis showed marked elevations in ALT, ALP, GGT, LDH and C-reactive protein, with non-significant decrease in total protein, albumin and globulin. Ascitic fluid was typically clear, low-protein transudate with serum-ascites albumin gradient (SAAG) >1.1 g/dL, confirming hepatic involvement. Cytology revealed mesothelial cells and leukocytes. Ultrasonography consistently demonstrated peritoneal effusion, hepatomegaly, altered echogenicity and gallbladder wall changes. The integration of clinical, laboratory and imaging findings provides a comprehensive diagnostic framework for characterizing hepatic ascites in dogs, enhancing accuracy in differentiating it from other etiologies.

Key words: Ascites in dogs, Hepatic disease, Serum Ascites Albumin Gradient (SAAG), Ultrasonography

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Introduction

Ascites, the abnormal accumulation of fluid within the peritoneal cavity, is a common clinical manifestation of several underlying systemic disorders in dogs, particularly hepatic diseases. Hepatic-origin ascites often results from portal hypertension, hypoalbuminemia or hepatic insufficiency, leading to fluid transudation into the abdominal cavity. Identifying the primary hepatic cause is crucial for accurate diagnosis, prognosis and therapeutic management. Conventional diagnostic approaches combining haemato-biochemical profiling, ultrasonography and peritoneal fluid analysis provide valuable insights into the pathophysiological mechanisms involved (Runyon *et al.,* 1992; Alleman, 2003). Haemato-biochemical evaluations reveal hepatic functional status and associated systemic effects, while ultrasonography serves as a non-invasive, reliable imaging tool for detecting hepatic parenchymal changes and fluid accumulation. Analysis of ascitic fluid further aids in differentiating transudate, modified transudate and exudate, thereby assisting in determining the underlying etiology. Despite advances in veterinary diagnostics, ascites of hepatic origin remains a diagnostic and therapeutic challenge due to its multifactorial nature and variable clinical presentation. Hence, the present study was undertaken to assess ascites of hepatic origin in dogs through comprehensive evaluation of haemato-biochemical alterations, ultrasonographic findings and peritoneal fluid characteristics to enhance diagnostic accuracy and understanding of the disease pathogenesis.

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Materials and Methods

The study was conducted at the Veterinary College, Shivamogga (Karnataka, India), from January to June 2025. Dogs presented with abdominal distension, fluid thrill and clinical suspicion of hepatic disease were screened. Cases with cardiac, renal, or other systemic causes of ascites were excluded through clinical, biochemical and ultrasonographic assessments. Twenty-four dogs with confirmed hepatic-origin ascites and six clinically healthy dogs served as controls. Blood samples were collected for haematological parameters like haemoglobin, packed cell volume, erythrocyte and leukocyte counts using Exigo® haematology (Boule Medical, AB Sweden), and biochemical analysis for ALT, ALP, GGT, LDH, total protein, albumin, globulin, bilirubin, creatinine and C-reactive protein using Robonik® Prietest Touch plus biochemical analysers (Unitron Bio-medicals).

Peritoneal fluid was obtained via abdominocentesis for physical, biochemical and cytological evaluation. The Serum Ascites Albumin Gradient (SAAG) was calculated as the difference between serum and ascitic fluid albumin (Runyon *et al.,* 1992). Ultrasonography was performed using a SONORAY®D50 PLUS machine (Sonoray Medical Systems Chennai) with a 3-8 MHz convex probe to evaluate hepatic parenchyma, gallbladder and peritoneal fluid characteristics (Mannion, 2006; Burk and Feeney, 2003).

The data obtained were subjected to statistical analysis as per the methods described by Snedecor and Cochran (1994) using SPSS 22.00 version.

Results and Discussion

The clinical signs of hepatic-origin ascites in dogs are presented in Fig. 1. Ascitic dogs exhibited major clinical signs including abdominal distension (100%), which could be due to involvement of the liver and hepatic portal hypertension leading to seepage of fluid in to peritoneal cavity. Anorexia was recorded in 83.33 % of ascitic dogs with hepatic involvement which might be due to the compromised detoxification capacity of the liver (Rothuizen, 2009). Epigastric pain (50.0%) was associated with hepatitis and hepatomegaly due to the location of the liver in epigastric area (Jain *et al.*, 2013). Diarrhoea (45.8%) was attributed to malabsorption of fats from reduced bile production, altered gut microbiota and secondary infections and reduced detoxification capacity of the diseased liver. These findings were consistent with the reports of Saravanan *et al.* (2014a), Phom *et al.* (2019) and Singh *et al.* (2019), who documented distended abdomen, anorexia, epigastric pain, diarrhoea and icterus in dogs with ascites of hepatic origin.

Haematological findings revealed significant anaemia, with mean haemoglobin (8.9±0.47 g/dL), PCV (26.33±1.38%) and erythrocyte counts (4.46±0.21 x10^6/µL) markedly lower

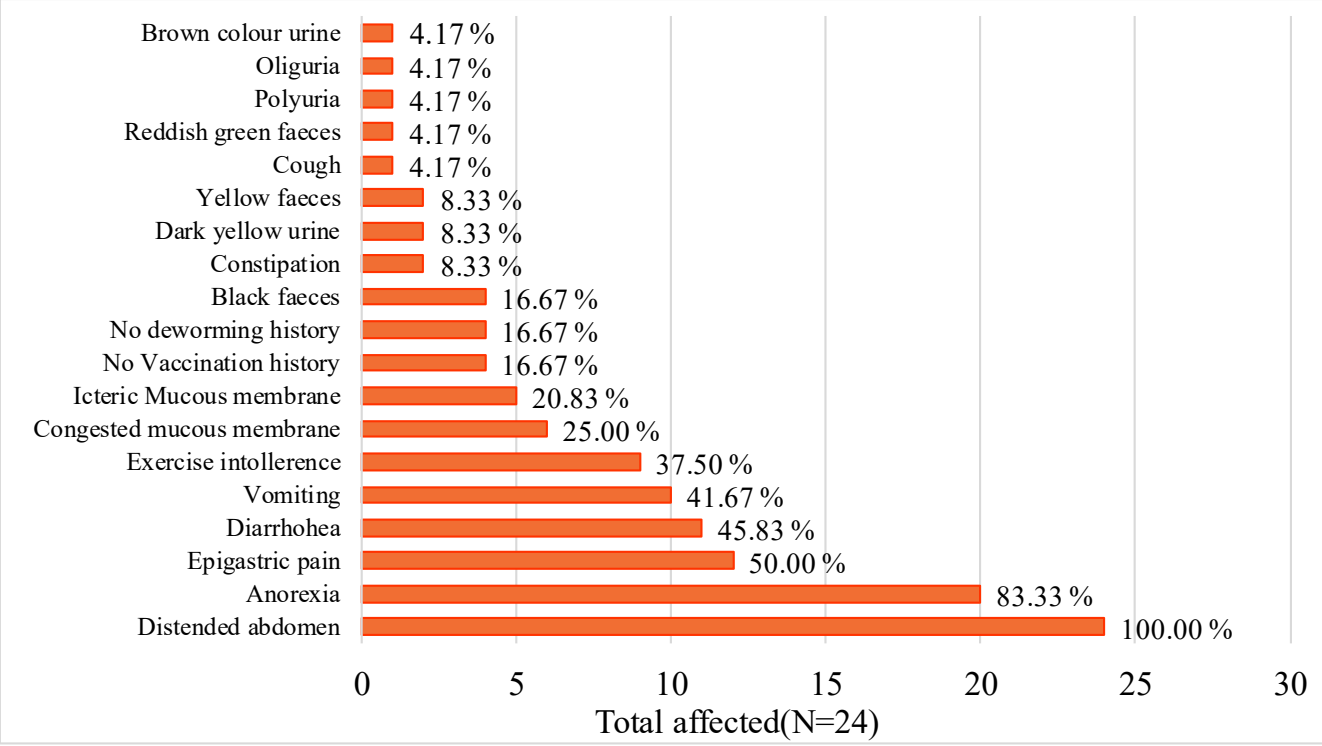

Fig. 1: Clinical signs of dogs with ascites of hepatic origin

with significantly increased leucocyte counts (20.10±1.85 x10^3/µL) than controls (Table 1). It's mainly due to increased erythrocyte transit time through the spleen as a result of decreased portal blood flow and increased red blood cell fragility as a result of elevated bile acid levels (Hassan *et al*., 2022) and also the reduced nutrient uptake and reduced availability of micronutrients from liver could be the cause of anaemia (Bush, 2002). Leukocytosis indicated ongoing persistent inflammatory process in hepatobiliary dysfunction (Bhatti, 2020). However other haematological parameters were within normal range of healthy dogs.

Table 1: Haematological findings in ascitic dogs with hepatic involvement

Clinical observations	Healthy control (n=6)	Ascitic dogs (n=24)
Haemoglobin (g/dL)	12.82±0.67	8.90±0.47*
Packed cell volume (%)	39.13±1.91	26.33±1.38*
TEC (x10^6/µL)	6.19±0.25	4.46±0.21*
TLC (x10^3/µL)	11.40±0.71	20.10±1.85*
Platelet count (x10^3/µL)	253.50±9.19	251.79±22.91
Lymphocytes (%)	20.50±2.30	20.64±1.28
Monocytes (%)	9.50±0.60	10.18±0.53
Granulocytes (%)	70.10±2.66	69.35±1.67
MCV (fL)	63.48±1.38	57.88±0.93
MCH (pg)	20.95±0.44	19.38±0.38
MCHC (g/dL)	32.62±0.31	33.44±0.32

*Significant at p≤0.05 when compared to apparently healthy dogs.

Biochemical profiles demonstrated elevated hepatic enzymes like ALT (160.08 ± 14.23 U/L), ALP (321.85 ± 18.63 U/L), GGT (50.75 ± 4.84 U/L) and LDH (310.85 ± 26.43 U/L) (Table 2), which could be attributed to increased hepatocellular membrane permeability caused by direct hepatic injury and progressive necrosis of liver tissue, leading to the release of these enzymes into circulation (Kumar *et al.,* 2003; Cocker *et al.,* 2017) suggesting hepatocellular damage and cholestasis. Serum total protein and albumin were reduced non-significantly, reflecting impaired hepatic synthesis. Higher CRP levels suggest more extensive inflammation or necrosis within hepatic tissue (Craig *et al.,* 2016). The other serum biochemical parameters were within normal range of healthy dogs.

Table 2: Serum biochemical findings in ascitic dogs with hepatic involvement

Clinical observations	Healthy control (n=6)	Ascitic dogs (n=24)
ALT (U/L)	32.60±3.40	160.08±14.23*
ALP (U/L)	145.09±7.63	321.85±18.63*
GGT (U/L)	25.61±3.15	50.75±4.84*
LDH (U/L)	171.08±6.76	310.85±26.43*
C-Reactive protein (mg/L)	0.32±0.05	1.54±0.05*
Total protein (g/dL)	6.44±0.35	5.15±0.20
Albumin (g/dL)	2.61±0.13	2.26±0.09
Globulin (g/dL)	3.82±0.28	2.89±0.14
A:G Ratio (A:G)	0.70±0.05	0.805±0.04
Total bilirubin (mg/dL)	0.21±0.02	0.604±0.086
Creatinine (mg/dL)	0.84±0.11	0.99±0.07

*Significant at p≤ 0.05 when compared to apparently healthy dogs.

Ascitic fluid examination revealed it a clear, low-protein transudate with a mean SAAG of 1.79 ± 0.08 g/dL, confirming hepatic origin (Table 3, 4). Cytological analysis identified mesothelial and inflammatory cells without bacterial contamination. Ultrasonographic findings included hyperechoic hepatic parenchyma, hepatomegaly, gallbladder wall thickening and peritoneal effusion, supporting hepatic etiology (Fig. 2-5). These findings agreed with Saravanan *et al.* (2014a,b), Phom *et al.* (2019) and Patel *et al.* (2022), who described similar biochemical and imaging alterations in dogs with hepatic ascites. The ultrasonographic diagnosis of causes of ascites due to hepatic origin is depicted in Figure 6. Thus, integration of haemato-biochemical markers, ultrasonography and SAAG provides a reliable, non-invasive diagnostic protocol for assessing ascites of hepatic origin in dogs.

Table 4: Biochemical findings of Ascitic fluid in dogs with hepatic involvement

Biochemical parameters	Mean ± SE
Total protein (g/dL)	0.966±0.103
Albumin (g/dl)	0.421±0.060
SAAG (g/dL)	1.835±0.075
Total nucleated cell count	214.08±27.30

Table 3: Physical examination of Ascitic fluid in dogs with hepatic involvement

Sl. No	Physical Parameters		No of animals affected (N=24)	Per cent
1	Colour	Transparent	22	91.67
		Yellowish	2	8.33
2	Consistency	Watery	24	100
3	Odour	Odourless	24	100
4	Specific gravity		Range between 1.135 and 1.339	
5	pH		Range between 6.5 to 7.5	

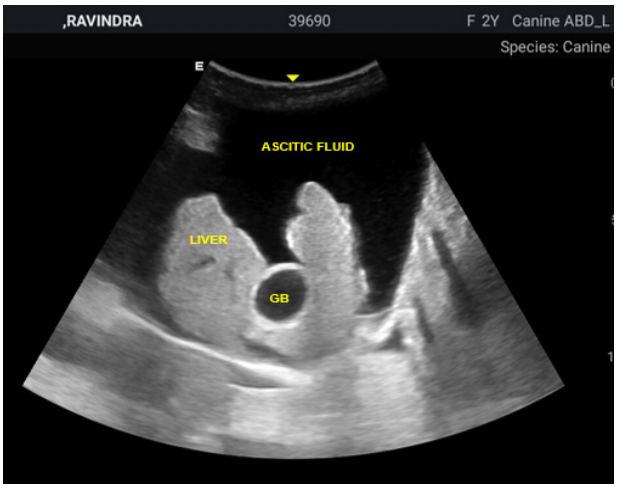

Fig. 2: Hyperechoic gall bladder wall with hyperechoic irregular borders of liver indicative of Cholecystitis associated with hepatitis.

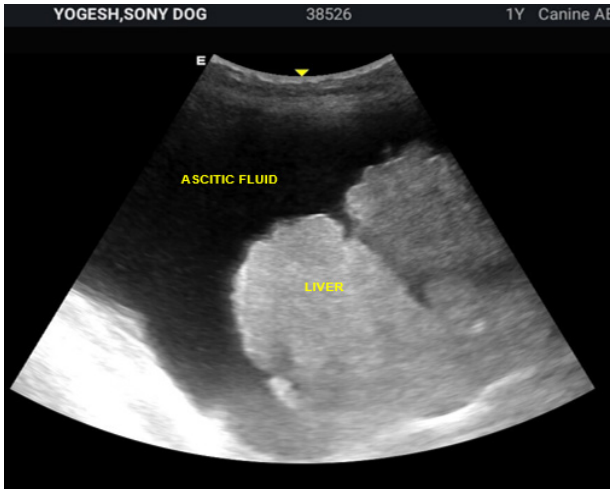

Fig. 3: Diffuse hyperechoic and irregular borders of the liver lobes with altered echotexture indicative of cirrhotic liver.

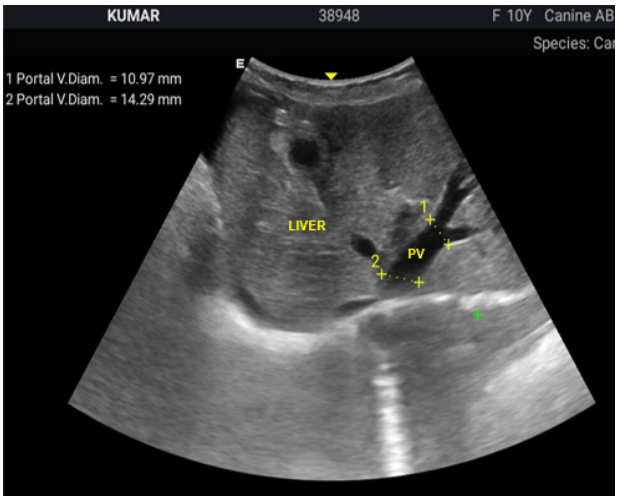

Fig. 4: Increased in diameter of the portal veins indicate presence of portal hypertension.

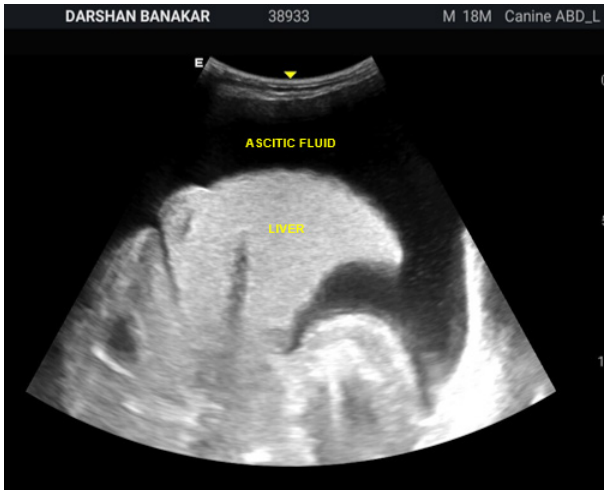

Fig. 5: Hyperechoic liver lobes with defused fatty infiltration indicative of hepatitis with fatty liver.

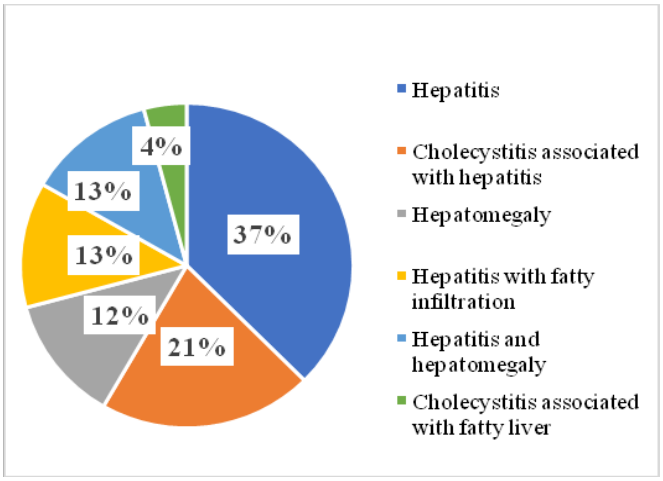

Fig. 6: Ultrasonographic diagnosis of ascites due to hepatic origin

Conclusion

The study established that ascites of hepatic origin in dogs is characterized by distinct clinical, haemato-biochemical and ultrasonographic profiles. A combination of elevated hepatic enzymes, anaemia, hypoalbuminemia, a high SAAG value (>1.1 g/dL) and characteristic ultrasonographic findings serve as strong diagnostic indicators. Integrating these parameters enhances diagnostic accuracy, enabling early differentiation of hepatic ascites from other causes and improving clinical management and prognosis in ascitic dogs.

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References

Alleman, A.R. (2003). Abdominal, thoracic and peritoneal effusion. *Veterinary Clinics of North America - Small Animal Practice, 33,* 89-118.

Bhatti, A.K. (2020). Investigations on ascites in dogs. *Ph.D. Thesis.* Himachal Pradesh Krishi Vishva Vidyalaya, Palampur-176062, India.

Burk, R.L., & Feeney, D.A. (2003). *Small Animal Radiology and Ultrasonography*. WB Saunders, Chapter 3, pp. 249-476.

Bush, B.M. (2002). *Interpretation of Laboratory Results for Small Animal Clinicians*. Blackwell Science Ltd., Oxford, UK.

Cocker, S., Richter, K., Ettinger, S.J., Feldman, E.C., & Cote, E. (2017). Diagnostic evaluation of the liver. *Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat*. 8th edn., St Louis, MO: Elsevier, pp. 1611-1621.

Craig, S.M., Fry, J.K., Rodrigues Hoffmann, A., Manino, P., Heilmann, R.M., Suchodolski, J.S., Steiner, J.M., Hottinger, H.A., Hunter, S.L., & Lidbury, J.A. (2016). Serum C-reactive protein and S100A12 concentrations in dogs with hepatic disease. *Journal of Small Animal Practice, 57*(9), 459-464.

Hassan, M.S.M., Abdel-Saeed, H., Abd El Aziz Ahmed, K., & Abdou, O.M. (2022). Clinical and hemato-biochemical studies on canine anaemia. *Advances in Animal & Veterinary Sciences*, *10*(4), 838-844.

Jain, S., Jyotsana, S., Naresh, C., & Ashish, S. (2013). Haemato-biochemical changes and therapeutic aspect of ascites with hepatic involvement in dogs. *Environment and Ecology, 31*(2C), 1205-1206.

Kumar, S.K., Anil Ahuja, A.A., Bihani, D.K., & Gahlot, A.K. (2003). Studies on canine ascites. *Journal of Canine Disease Research, 3*(2), 9-17.

Mannion, P. (2006). *Diagnostic Ultrasound in Small Animal Practice*. 1st Edn., Blackwell Publishing Company, UK, pp. 51-80.

Patel, M.D., Parikh, P.V., & Barot, H.M. (2022). Clinical, ultrasonographic and haemato-biochemical assessment of liver and gall bladder affections in dogs. *Indian Journal of Veterinary Surgery, 18*(1), 65-71.

Phom, O.K., Sarma, K., Arya, R.S., Behera, S.K., Konwar, B., Saikia, B., Chaudhary, J.K., Rajesh, J.B., Prasad, H., & Das, H. (2019). Ascites and hepato-renal syndrome in cirrhosis in dogs. *Journal of Entomology & Zoological Studies,7*(5), 313-321.

Rothuizen, J. (2009). Important clinical syndromes associated with liver disease. *Veterinary Clinics: Small Animal Pracrtice, 39*(3), 419-437.

Runyon, B.A., Montano, A.A., & Akriviadis, E.A. (1992). The serum-ascites albumin gradient is superior to the exudates-transudate concept in the differential diagnosis of ascites. *Annals of Internal Medicine*, *117*(3), 215-220.

Saravanan, M., Mondal, D.B., Sarma, K., Mahendran, K., Vijayakumar, H., & Sasikala, V. (2014a). Comprehensive study of haemato-biochemical, ascitic fluid analysis and ultrasonography in the diagnosis of ascites due to hepatobiliary disorders in dog. *Indian Journal of Animal Sciences, 84*(5), 503-506.

Saravanan, M., Mondal, D.B., Sharma, K., Mahendran, K., & Sasikala, V. (2014b). Utility of serum ascites albumin gradient in dogs as indicator of hepatobiliary disorders induced ascites. *Indian Veterinary Journal, 91*(4), 33-35.

Singh, S.K., Shukla, S.K., Bhatt, P., & Singh, A.K. (2019). Clinico-therapeutic studies on ascites in canines. *International Journal of Current Microbiology & Applied Sciences*, *8*(9), 1120-1137.