RESEARCH ARTICLE

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

Vinay R. Baria[1]*, Arshi A. Vagh[1], Avinash K. Bilwal[1], Jayendra R. Damor[2], Raisudin M. Sherasiya[1], Priyanshi V. Patel[1]

Abstract

The dogs (n=29) presented at Veterinary Clinical Complex, Kamdhenu University, Junagadh with complain of abdominal distention were screened for ascites. For the diagnosis, detailed history and clinical examinations were performed together with haemato-biochmeical analysis, ascitic fluid analysis and ultrasonography, and a suitable therapeutic protocol was used. Among the confirmed cases of ascites higher incidence was found in female dogs with Labrador retriever being most affected breed and age group of 4 to 8 years most affected. Dogs with ascites displayed notable haematological alterations; including significant decrease in TEC, PCV and lymphocyte levels, while significant increase in TLC. Dogs with ascites exhibited a significant elevation in ALT, ALP, creatinine and BUN levels. Additionally, highly significant reduction was observed in total protein, albumin, and A/G ratio. The ascitic fluid's SAAG (serum ascitic albumin gradient) values played a crucial role in determining the underlying cause of ascites. Ultrasonography emerged as a valuable imaging modality due to its ability to detect even small amount of ascitic fluid and identify accompanying alterations in echotexture of vital organs. The therapy of silymarin and supportive care proved effective in managing ascites caused by hepatic disorders. Additionally, the use of herbal nephroprotectives showed promising results in the recovery of ascites associated with renal disorders.

Key words: Ascites, Ascitic fluid analysis, Dogs, Serum ascitic albumin gradient (SAAG), Silymarin, Ultrasonography.

Ind J Vet Sci and Biotech (2024): 10.48165/ijvsbt.20.6.24

Introduction

Ascites is characterized by the accumulation of serous or sero-sanguinous fluid in the peritoneal space. There are various factors that can lead to the development of ascites. This condition is more prone to occur when diseases or pathological conditions affect the liver, heart, kidneys, spleen, peritoneum or intestines. These underlying issues can disrupt the normal fluid balance in the body, leading to the accumulation of fluid in the abdominal cavity. Major hepatic dysfunctions such as hepatic cirrhosis, hepatic neoplasms and chronic hepatitis contribute to the development of ascites. Additionally, conditions like congestive heart failure, protein-losing nephropathy, inflammatory bowel disease, intestinal lymphoma, intestinal lymphangiectasia (Dossin and Lavouè, 2011), caudal vena cava kinking (Pelosi *et al.*, 2012), peritonitis, haemorrhage, neoplasia and even ruptures of the gall bladder or urinary bladder can lead to ascites (Rutgers and Biourge, 2007).

Ascites is commonly associated with hypoalbuminemia, portal hypertension and salt and water retention. Renal disorders such as glomerulonephritis and nephrotic syndrome can also result in ascites (Ettinger and Feldman, 2000). Typical clinical signs of ascites include a gradual expansion of the abdomen, a partial to complete loss of appetite, lethargy, weight loss and dyspnea (Saravanan *et al.*, 2014). Various serum markers including bilirubin, alkaline phosphate, total protein, albumin levels, alanine amino transferase and aspartate amino transferase are valuable in diagnosing liver impairment (Tiwari *et al.*, 2011). Diagnostic tools such as complete blood count, serum biochemistry, urine analysis, ascitic fluid analysis, radiography, ultrasonography, electrocardiography and echocardiography can aid in determining cardiac and renal involvement alongside liver disorders. Ascitic fluid analysis performed after abdomenocentesis, involves determining specific gravity, total protein, albumin, nucleated cell count and the Serum Ascitic Albumin Gradient (SAAG). SAAG serves as a marker for portal hypertension, offering a valuable index that replaces the exudates-transudate concept in ascitic fluid (Tarn and Lapworth, 2010). An effective therapeutic approach is essential for treating ascites, aiming to eliminate

[1]Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Kamdhenu University, Junagadh, Gujarat, India

[2]Veterinary Clinical Complex, College of Veterinary Science and Animal Husbandry, Kamdhenu University, Junagadh, Gujarat, India

Corresponding Author: Vinay R. Baria, Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Kamdhenu University, Junagadh, Gujarat, India. e-mail: vinaybar98@gmail.com.

How to cite this article: Baria, V. R., Vagh, A. A., Bilwal, A. K., Damor, J. R., Sherasiya, R. M., & Patel, P. V. (2024). Ascites in Dogs: A Comprehensive Study on Diagnosis and Therapeutic Management. Ind J Vet Sci and Biotech. 20(6), 124-130.

Source of support: Nil

Conflict of interest: None

Submitted: 12/07/2024 **Accepted** 20/09/2024 **Published** 10/11/2024

causative factors, minimize fibrosis and control emerging complications. This article provides a comprehensive overview of canine ascites, exploring its incidence, diagnostic complexities, and therapeutic strategies.

Materials and Methods

The study was conducted at the Department of Veterinary Medicine, College of Veterinary Science and Animal Husbandry, Kamdhenu University, Junagadh (India) during the period from October 2022 to March 2023. Six healthy dogs which were presented for vaccination and/or routine check-ups were selected as the control group. Of the dogs (n=817) presented at Veterinary Clinical Complex, Kamdhenu University, Junagadh, those (n=29) presented with complain of abdominal distension and suspected for ascites were investigated based on their history, clinical signs and physical examinations. Confirmatory diagnosis was done based on haematological analysis, serum biochemical analysis, ascitic fluid analysis and ultrasonography.

For estimation of haematological and biochemical parameters, whole blood was collected from either saphenous or cephalic vein aseptically in K_3EDTA vial and clot activator vials on the day of presentation and again after 14th day following treatment. All the parameters were measured by automatic haematology analyser and automatic biochemical analyser. Ascitic fluid was collected by performing abdominocentesis for physical examination, biochemical analysis and cytology. Physical examination was done to evaluate colour and transparency of ascitic fluid. For biochemical analysis total protein and albumin of ascitic fluid was measured same as serum parameter on the day of collection. The Serum Ascitic Albumin Gradient (SAAG) was calculated by subtracting the ascitic fluid albumin concentration from that of the serum albumin concentration. For cytology, the fluid was centrifuged and a smear from sediment was prepared followed by staining with Field stain and observed under microscope.

The hairs on the abdomen extending halfway up the body wall over the right and left caudal intercostal spaces were clipped for ultrasonographic examination. Abdominal ultrasonography (B-mode) was performed by using convex probe of 3.5-5 MHz with coupling gel to ensure proper contact. Variations in echogenicity, structure, extent and other features were assessed by comparing them to the standard appearance of the liver, gall bladder and kidneys. The echogenicity of the detectable lesion observed in the grayscale 2-dimensional images were subjectively categorized as normal, increased (hyperechoic), decreased (hypoechoic) or absent (anechoic).

Among the confirmed cases of ascites, 18 dogs were divided into two main treatment groups. One group, categorized with hepatic origin comprised 12 dogs (Group 2), while the other group with renal origin included 6 dogs (Group 3). These groups were then compared to a control group of healthy dogs (Group 1) consisting of 6 dogs. The Group 2 dogs were given silymarin containing liver tonic orally for 30 days, while Group 3 dogs were suggested nephroprotective herbal tonic orally for 30 days. Also therapy with antibiotics (amoxicillin 20 mg/kg) intravenously or intramuscularly and diuretics (Furosemide 2 mg/kg) intramuscularly was given for 5 days. Supportive therapies like multivitamins, amino acids infusions, fluid therapy, oral haematinics, antacids, antiematics, protein supplements etc. were given as per the need. Salt free and protein rich diet was advised with complete rest. The efficacy of the treatment was determined by assessing the improvement in clinical signs, haemato-biochemical parameters and changes in ultrasonographic findings post-treatment.

Statistical analysis of data was done using Graphpad prism 9.0. All the data recorded were as mean± standard error (SE). Haemato-biochemical parameters of Group 2 and 3 were compared through one-way ANOVA followed by Tukey's test, while biochemical parameters of ascitic fluid of these groups were compared using unpaired t-test.

Results and Discussion

Incidence

Out of a total of 817 dogs brought to the small animal medicine OPD, Veterinary Clinical Complex, Junagadh during the study period, 29 dogs (3.55%) were suffering from ascites, which was in accordance with Singh *et al.* (2019[a]). The age wise higher incidence (Table 1) was observed in dogs within the age group of 4 to 8 years (41.38%, 12/29) and concurred with the reports of Saravanan *et al.* (2013) and Padhi *et al.* (2022). Among the 29 dogs diagnosed with ascites, 18 were female dogs (62.07%) and 11 were male (37.93%). Behera *et al.* (2017) and Padhi *et al.* (2022) reported a higher incidence of ascites in females. In contrast, Saravanan *et al.* (2014) and Singh *et al.* (2019[a]) documented a higher incidence of ascites in male dogs.

Table. 1: Age wise incidence of ascites in dogs (n=29)

Age of affected dogs	Number of dogs affected	Percentage of dogs affected
1-4 years	10	34.48
4-8 years	12	41.38
> 8 years	7	24.14
Total	**29**	**100**

In this study, Labrador Retrievers exhibited the highest incidence of ascites among various dog breeds (31.03%, 9/29) followed by German Shepherds (27.59%, 8/29), non-descript dogs (13.79%, 4/29), Saint Bernards and Rottweilers (6.89% each, 2/29), and Pomeranian, Golden Retriever, Pug and Great Dane (3.45% 1/29 each). The results of this study align with the results of Behera *et al.* (2017) and Padhi *et al.* (2022) who noted Labrador Retrievers as the breed most commonly affected by ascites. Saravanan *et al.* (2014) and Singh *et al.* (2019[a]) found a higher incidence in Spitz, while Prajapati *et al.* (2022) reported non-descript dogs as predominantly affected by ascites.

Out of these 29 dogs, 16 dogs had ascites due to hepatic disorder (55.17%), 7 had ascites due to renal disorder (24.14%), 4 had cardiac origin (13.79%) and 2 dogs had other etiology (6.89%). Behera *et al.* (2017) and Singh *et al.* (2019[a]) similarly observed higher incidence of ascites in dogs due to liver disorders. However, Ihedioha *et al.* (2013) and Padhi *et al.* (2022) reported a higher incidence of ascites linked to cardiac diseases.

Clinical Findings

From the 29 dogs with ascites, a detailed study was carried out on 18 dogs and according to etiology therapeutic management was done. In these 18 dogs with ascites various clinical signs were observed which were Abdominal distension 100% (18/18) (Fig. 1), Fluid thrill on percussion 100% (18/18), Lethargy 77.78% (14/18), Inappetance/anorexia 66.67% (12/18), Vomiting 55.55% (10/18), Pale mucous membrane 50% (9/18), Respiratory distress 44.44% (8/18), Melena 38.89% (7/18), Tachycardia 27.78% (5/18), Pedal edema and Jaundice both 22.22% (4/18), Ecchymosis and Emaciation both 16.67% (3/18), Polydipsia/polyuria and Epilepsy each 5.55% (1/18). Similar types of clinical signs were also reported by Saravanan *et al.* (2014) and Singh *et al.* (2019[a]).

The presence of fluid in dogs with ascites can cause discomfort, leading to heaviness, lethargy and reluctance to engage in physical activity. Discomfort from fluid accumulation may also result in reduced appetite and aversion to eating. In hepatic dysfunction, endotoxins from the gastrointestinal system can bypass the liver, reaching other body systems through the chemoreceptor trigger zone located in the fourth ventricle, potentially contributing to vomiting (Meyer and Rothuizen, 2013). Vomiting can also occur due to uraemia in renal disorders (Polzin, 2010). Pale mucous membrane observed in the study is linked to accelerated breakdown of red blood cells, commonly associated with liver dysfunctions and elevated bile acid levels in most liver disorders (Meyer and Rothuizen, 2013). Ascites leads to hypovolemia, triggering compensatory mechanisms to maintain adequate blood supply to organs by pumping blood at a faster rate leading to tachycardia. Icterus observed in this study may be attributed to the accumulation of bilirubin in the blood and extravascular spaces which can result from increased production, impaired clearance, compromised liver conjugation, and/or compromised bile flow. Ecchymosis results from hepatobiliary dysfunction-associated coagulation abnormalities (Watson and Bunch, 2009). Emaciation in liver disorders can be attributed to increased tissue catabolism and insufficient nutritional intake due to decreased appetite (Hess and Bunch, 2000).

Haematological and Serum Biochemical Parameters

Referring to the haematological data presented in the Table 2, there was significant decrease ($p<0.05$) in the mean values of PCV and TEC in group 2 dogs compared to healthy dogs, while there was highly significant decrease ($p<0.01$) in TEC of group 3 dogs compared to group 1. These findings were in accordance with Ihedioha *et al.* (2013) and Saravanan *et al.* (2013) in ascitic dogs. The fibrosis involving large portion of the liver mass due to chronic hepatic disorders could result in comparatively lower PCV, TEC and Hb concentration in dogs with ascites (Stockham and Scott, 2008). The reduction in TEC in ascites of renal origin may be due to reduction in erythropoietin hormone levels caused by renal insufficiency (Chakrabarti, 1997). There was a significant ($p<0.05$) increase in the mean total leukocyte counts and reduction in the mean lymphocyte percentage in group 2 compared to group 1. Saravanan *et al.* (2014) and Phom *et al.* (2019) also observed increase in neutrophils and decrease in lymphocytes, similar to our findings. This elevated TLC can be associated with various factors including hepatocellular damage, infection

Table. 2: Haematological parameters in healthy dogs and dogs with ascites on day 0 and 14 of treatment

Parameters	G 1 Healthy (n=6)	0 day		14 day	
		G 2 (n=12)	G 3 (n=6)	G 2 (n=12)	G 3 (n=6)
Hb (g/dL)	13.13±0.84	10.09±0.91*	10.10±1.49*	10.54±0.63	10.78±1.16
PCV (%)	41.69±2.21	30.63±2.91*	31.17±3.15*	35.33±2.38	31.44±2.99*
TEC ($x10^6/\mu L$)	7.46±0.25	5.18±0.51*	5.30±0.35**	5.80±0.34	5.31±0.16
TLC ($x10^3/\mu L$)	10.69±0.57	27.81±4.03**	20.27±3.63**	18.65±1.91	16.76±1.39
Neutrophils (%)	65.83±0.87	74.06±3.41	71.83±3.25	67.50±1.45	71.0±2.88
Lymphocyte (%)	27.83±1.85	17.53±2.74*	21.83±3.43*	25.25±1.18	21.50±2.79
Monocytes (%)	4.50±1.09	5.42±0.79	4.33±0.76	4.17±0.39	4.67±0.67
Eosionophils (%)	1.50±0.50	2.92±0.93	2.0±1.1	2.67±0.28	2.33±0.33
Basophils (%)	0.33±0.21	0.08±0.08	0.17±0.17	0.42±0.15	0.33±0.21
Platelets ($x\ 10^5/\mu L$)	3.58±0.60	2.16±0.37	1.98±0.37	2.36±0.30	2.27±0.26

*P<0.05 statistically significant, **p<0.01 highly significant from healthy control group.

or sepsis. Other haematological parameters showed non-significant changes (Table 2).

There was significant increase in serum ALT-ALP of group 2 and ALP of group 3 compared to group 1. Tantary *et al.* (2013) and Saravanan *et al.* (2014) documented similar findings. ALT, which is highly specific for the liver, is released into the bloodstream when there is an increase in the permeability of hepatocyte membranes or the presence of hepatobiliary dysfunctions (Lidbury and Steiner, 2013). ALP is a sensitive indicator for cholestasis with a sensitivity rate of 85% (Lidbury and Steiner, 2013). Elevated serum ALP level is found in both acute and chronic liver disorders, with more significant increase generally indicating cholestasis (Tennant and Center, 2008).

As per the Table 3, there was highly significant decrease ($p<0.01$) in serum total protein and albumin levels of both group 2 and 3. In dogs with hepatic disorders, impaired functions of liver cells and presence of fibrous tissue in liver results in reduced protein synthesis leading to hypoproteinemia and hypoalbuminemia, while in renal diseases loss of protein by damaged kidneys leads to reduced protein in blood stream (Stockham and Scott, 2008). Tantary *et al.* (2013), Saravanan *et al.* (2014) and Singh *et al.* (2019[b]) recorded significant decrease in total protein and albumin in dogs with ascites. Serum creatinine and BUN of dogs with renal disorder (Group 3) showed significant increase ($p<0.01$) compared to healthy group. These findings aligned with Ihedioha *et al.* (2013) and Phom *et al.* (2019). In renal disease, the impaired functioning of the kidneys results in a reduction of the glomerular filtration rate. So, the clearance of creatinine and urea from the blood is diminished leading to their elevated levels in the bloodstream. Other serum biochemical parameters showed non-significant changes (Table 3).

Ascitic Fluid Analysis

Most of the collected ascitic fluid samples were clear and transparent, although a small number of samples had a straw color. Among the biochemical parameters of ascitic fluid, total protein and albumin were non-significantly higher in group 3 than in group 2, while SAAG value of group 2 was significantly higher ($p<0.01$) compared to group 3 (1.28±0.11 vs. 0.65±0.06 g/dL). Upon microscopic examination, the majority of the samples displayed a small number of mesothelial cells with few lymphocytes and neutrophils.

Clear transparent fluid signifies the accumulation of pure transudate within the peritoneal cavity, uncontaminated except for occasional mesothelial cells and tissue macrophages. This form of ascites is evident in conditions like portal hypertension, hepatic diseases, reduced osmotic gradient (due to hypoalbuminemia), protein-losing enteropathy, renal dysfunction and albuminuria. Clear straw-colored fluid described as a modified transudate, typically contains fibrin cells as well as white blood cells like neutrophils and lymphocytes. This type of fluid is commonly observed in cases of prolonged ascites resulting from various conditions such as right-sided heart failure, malignancies and hepatic diseases which can lead to the infiltration of fibrinogens (Nwoha, 2019). The protein content in modified transudates varies between 2.5 to 5.0 g/dL (Mondal *et al.*, 2012). Based on this physical examination, presence of cells and protein content the ascitic fluid present study was categorised in transudate (66.67%, 12/18) and modified transudate (33.33%, 6/18). Patients with ascites due to portal hypertension, such as cirrhosis, congestive heart failure (CHF), massive liver metastasis, fulminant liver failure and portal vein thrombosis usually display SAAG value equal to or greater than 1.1 g/dL, while ascites not associated with portal hypertension such as peritoneal carcinomatosis, tuberculosis (TB), pancreatic ascites, biliary ascites, nephrotic syndrome and bowel obstruction or infarction is characterized by a SAAG value below 1.1 g/dL (Mondal *et al.*, 2012). Present findings of ascitic fluid were consistent with previous findings of Saravanan *et al.* (2014) and Phom *et al.* (2019).

Table. 3: Biochemical parameters in healthy dogs and dogs with ascites on day 0 and 14 of treatment

Parameters	G 1 Healthy (n = 6)	0 day		14 day	
		G 2 (n=12)	G 3 (n=6)	G 2 (n=12)	G 3 (n=6)
ALT (U/L)	41.55± 5.04	123.35±19.68*	66.48±17.72	81.14± 6.22*	56.11± 11.52
ALP (U/L)	65.90±5.88	336.33±76.41*	246.58±61.43*	149.24±31.84	110.74±24.27
Total protein (g/dL)	6.62±0.25	4.94±0.25**	4.43±0.26**	5.29±0.18	4.92±0.17**
Albumin (g/dL)	3.47±0.11	2.03±0.12**	1.66±0.11**	2.77±0.08	2.08±0.10*
Globulin (g/dL)	3.15±0.15	2.91±0.17	2.77±0.34	3.01±0.13	2.84±0.22
A/G ratio	1.11±0.03	0.71±0.05**	0.66± 0.11**	0.77±0.04	0.76±0.08
Creatinine (mg/dL)	0.87±0.09	1.12±0.09	2.88±0.55**	1.12±0.08	2.47±0.49**
BUN (mg/dL)	30.63±5.92	52.01±10.34	113.68±25.60**	45.02±6.38	80.42±16.44*
Total bilirubin (mg/dL)	0.24±0.04	1.92±0.80*	1.04±0.46	1.30±0.56*	0.76±0.28
Direct bilirubin (mg/dL)	0.06±0.02	0.81±0.46	0.45±0.30	0.54±0.32	0.37±0.23

*P<0.05 statistically significant, **p<0.01 highly significant from healthy control group.

Ultrasonography

Upon ultrasonographic examination, in all the cases with ascites presence of anechoic peritoneal fluid was detected. In group 2 dogs various findings were noted like diffuse hyperechoic parenchyma (Fig. 5), round liver margin, mixed echogenicity (Fig. 3), hypoechoic liver parenchyma, thickening of gall bladder wall (Fig. 6), hyper echoic bile ducts, presence of biliary sludge etc., which were also documented by Tantary *et al.* (2014), Phom *et al.* (2019) and Pandya *et al.* (2022) in dogs with ascites. Ultrasonographic examination of dogs in group 3 with renal disorders revealed the presence of anechoic fluid in the peritoneum, increased echogenicity of renal cortex, an increase in kidney size (Fig. 7), decreased kidney size or irregular shape and hyperechoic urinary bladder. These findings aligned with previously work done by Chutia *et al.* (2016) and Phom *et al.* (2019).

Therapeutic Management

Based on the laboratory parameters and ultrasound imaging, therapeutic management was done according to the system involved. After the 14 days of silymarin treatment (Group 2), improvement in clinical signs (Fig. 1, 2), haemato-biochemical parameters (Table 2, 3) was seen along with changes in ultrasonography (Fig. 3, 4, 5, 6). Singh *et al.* (2019[b]) and Dhillon *et al.* (2020) also documented recovery of liver disorder by using silymarin. Silymarin, derived from *Silybum marianum* (Milk thistle), has been utilized for centuries as a herbal remedy for treating liver disorders. Silymarin exhibits potent anti-inflammatory and antioxidant properties also promoting cellular repair and hepatic regeneration (Pandey and Sahni, 2011). Group 3 dogs treated with herbal nephroprotective and other supportive therapy also showed improvement in clinical signs, haemato-biochemical parameters (Table 2, 3) and ultrasonographic changes (Fig. 7, 8). This polyherbal nephroprotective contains *Boehavia diffusa*, *Crata evanurvala* and *Solanum nigrum* possess anti-inflammatory, diuretic and nephroprotective properties beneficial for the kidneys and other content like *Tribulus terrestris* exhibits anti-inflammatory, antiseptic and antioxidant effects (Pradhan and Roy, 2012).

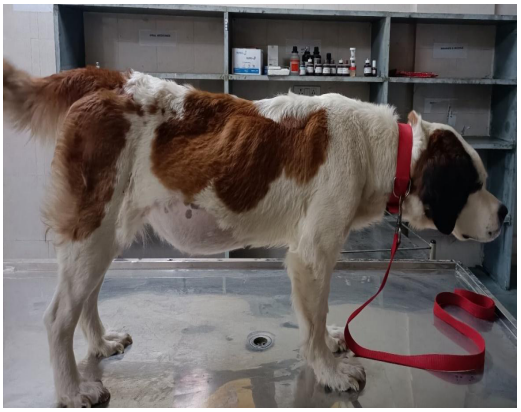

Fig. 1: Abdominal distension in 12 years old female Saint Bernard on day 0

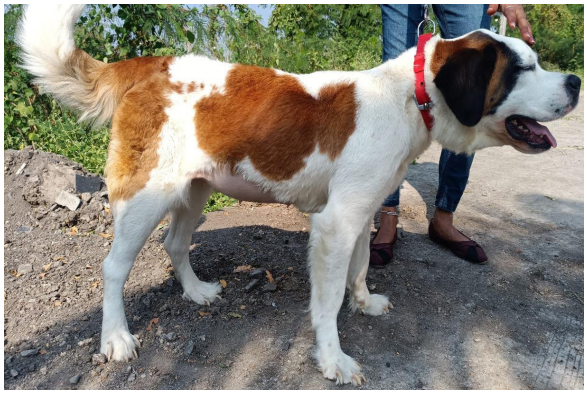

Fig. 2: Reduction in abdominal distension after 14 days

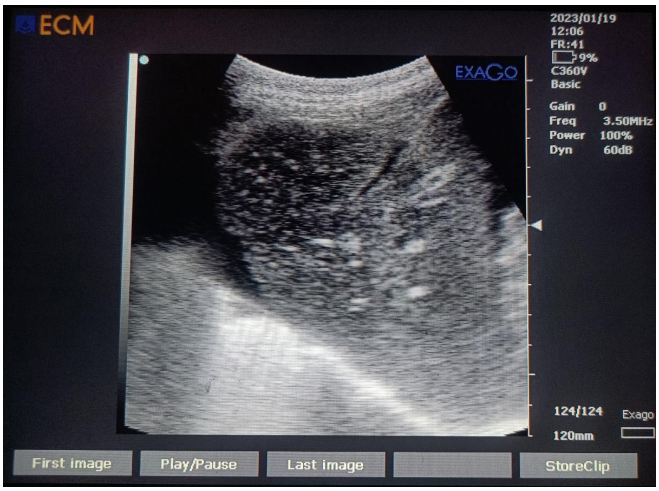

Fig. 3: Acute hepatitis - mixed echogenicity of liver parenchyma with anechoic peritoneal fluid at 0 day

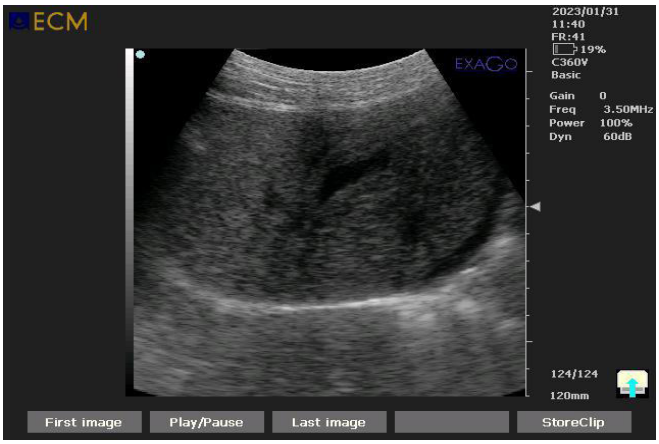

Fig. 4: Change in the echogenicity and reduction in peritoneal fluid after 14 days

Conclusion

Ascites in dogs is very difficult to manage without knowing its origin. Combine use of haemato-biochemical analysis, ascitic fluid analysis and ultrasonography provide essential information regarding cause of ascites. The therapy of silymarin and supportive care proved effective in managing

ascites caused by hepatic disorders. The use of herbal nephroprotectives showed promising results in the recovery of ascites associated with renal disorders. This study in general provides valuable insight regarding diagnostic procedure and therapeutic management of ascites in dogs.

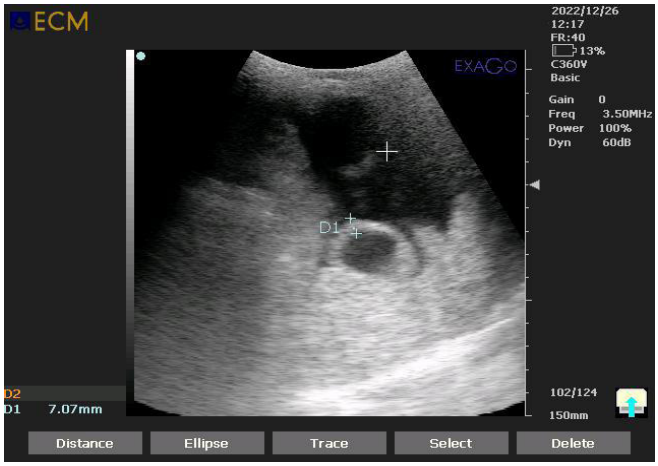

Fig. 5: Chronic hepatitis - hyperechoic irregular liver parenchyma and thickened gall bladder wall (7.07 mm)

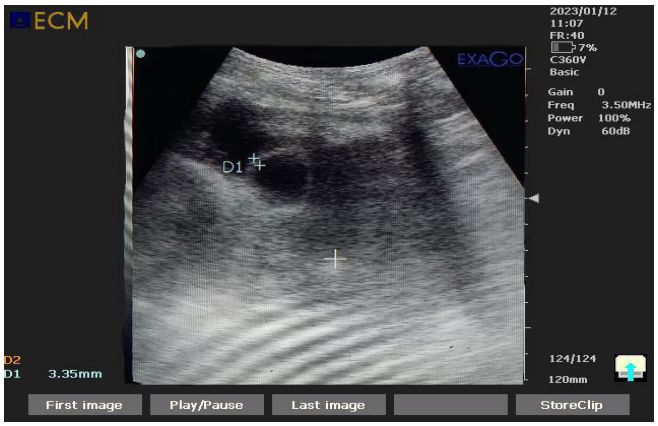

Fig. 6: Reduction in hyperechogenecity and gall-bladder wall thickness after 14 days

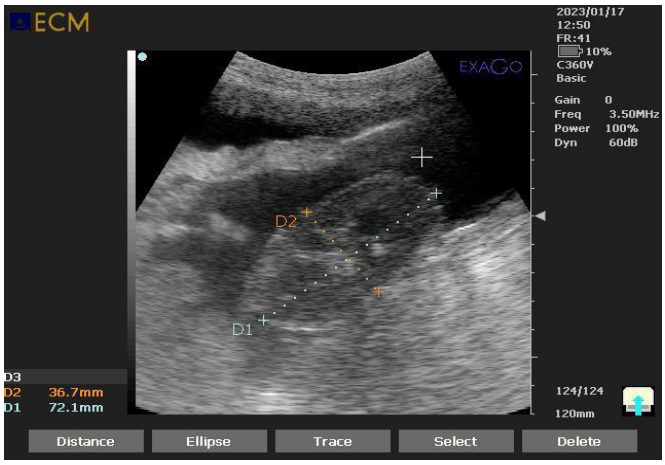

Fig. 7: Enlarged kidney with disrupted cortico-medullary junction and anechoic peritoneal fluid

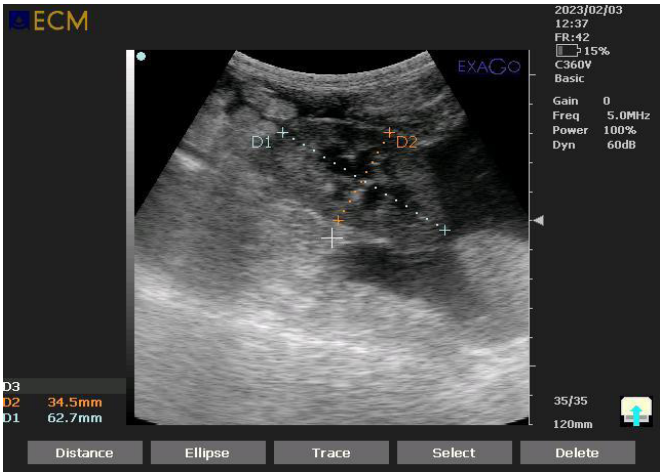

Fig. 8: Reduction in kidney size after 14 days

Acknowledgment

The authors extend their heartfelt gratitude to the Principal of the Veterinary College, Kamdhenu University, Junagadh, Gujarat for providing the essential facilities required for conducting this study.

References

Behera, M., Pandal, S.K., Nath, I., Panda, M.R., Kundu, A.K., Gupta, A.R., & Behera, S.S. (2017). Incidence of canine ascites in and around Bhubaneswar, Odisha, India. *International Journal of Science, Environment and Technology*, *6*(6), 3382-3392.

Chakrabarti, A. (1997). *Text book of Clinical Veterinary Medicine*, Kalyani Publishers, New Delhi, India, pp. 476-485.

Chutia, T., Ghorai, S., Sarma, K., Das, G., Behera, S. K., Konwar, B., Ahmed, F.A., & Lalrintluanga, K. (2016). Therapeutic management of ascites in a bitch- A case report. *International Journal of Livestock Research*, *6*(12), 71-73.

Dhillon, K.S., Kaur, S.J., & Singh, A. (2020). Ascites of hepatic origin in a female Beagle pup: Diagnosis, treatment and management. *Journal of Entomology and Zoology Studies*, *8*(2), 1942-1944.

Dossin, O., & Lavouè, R. (2011). Protein-losing enteropathies in dogs. *Veterinary Clinics of Small Animal*, *41*, 399-418.

Ettinger, S.J., & Feldman, E.C. (2000). *Textbook of Veterinary Internal Medicine*. W.B. Saunders Company, pp. 4604-4739.

Hess, P.R., & Bunch, S.E. (2000). Diagnostic approach to hepatobiliary diseases. In: Kirk's *Current Veterinary Therapy-Small Animal Practice*. 18th eds., W. B. Saunders and Co., Philadelphia.

Ihedioha, J.I., Anosa, V.O., & Esievo, K.A. (2013). Prevalence of and clinic-pathologic findings associated with ascites in dogs in Enugu State, Nigeria. *Comparative Clinical Pathology*, *22*(2), 185-193.

Lidbury, J.A., & Steiner, J.M. (2013). Diagnostic evaluation liver. In: *Canine and Feline Gatroenterology*. St. Louis, Missouri: Elsevier Health Sciences, pp. 863-875.

Meyer, H.P., & Rothuizen, J. (2013). History and physical examination. In: Washabau, R. J. & Day, M.J., 1st ed., *Canine and Feline Gastroenterology*, Saunders, St. Louis, MO, USA, pp. 849-958.

Mondal, D.B., Kumar, M., Saravanan, M., & Sharma, K. (2012). Peritoneal fluid analysis in canine disease diagnosis. *Journal of Advanced Veterinary Research*, *2*, 307-313.

Nwoha, R. (2019). Review on Ascites in Pets. In: S. O. Bekoe, M. Saravanan, R. K. Adosraku, & P. Ramkumar (Eds.), *Veterinary Medicine and Pharmaceuticals*, Intech Open.

Padhi, P., Patra, R.C., Senapati S.K., Panda, S., Das, D. ., & Biswal, S. (2022). Prevalence, clinico-pathological changes and therapeutic management of ascites in dogs. *The Pharma Innovation Journal*, *SP-11*(11), 742-746.

Pandey, G., & Sahni, Y.P. (2011). A review of hepatoprotective activity of silymarin. *International Journal of Research in Ayurveda and Pharmacy, 2*(1), 75-79.

Pandya, P.B., Vagh, A.A., Joseph, J.P., Bilwal, A.K., & Parmar, V.L. (2022). Assessment of hepatobiliary ultrasound score with hemato-biochemical alterations between healthy dogs and dogs with hepatobiliary dysfunctions. *The Pharma Innovation Journal*, *SP-11*(11), 933-937.

Pelosi, A., Prinsen, J.K., Eyster, G.E., Schall, W., & Olivier, N.B. (2012). Caudal vena cava kinking in dogs with ascites. *Veterinary Radiology and Ultrasound, 53*, 233-235.

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

Polzin, D.J. (2010). Chronic kidney disease. In: Ettinger, S. J. & Feldman, E. C. (Eds) *Textbook of Veterinary Internal Medicine*. St. Louis, Saunders Elsevier, pp. 1990-2021.

Pradhan, N.R., & Roy, S. (2012). Chronic renal failure in dogs and its management. *Indian Journal of Canine Practice*, *4*(2), 88-92.

Prajapati, A.S., Suthar, A.N., Chauhan, P.M., & Patel, K.D. (2022). Retrospective study of ascites in canines of north Gujarat region. *International Journal of Bio-resource and Stress Management*, *13*(9), 981-986.

Rutgers, C., & Biourge, V. (2007). Nutrition of dogs with liver disease. *Encyclopaedia of canine clinical nutrition*. pp.141-161.

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

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.

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

Singh, S., Shukla, S.K., Bhatt, P., & Singh, A.K. (2019[a]). Prevalence of ascites in canines in and around Tarai region of Uttarakhand. *Journal of Entomology and Zoology Studies, 7*(2), 1194-1197.

Stockham, S.L., & Scott, M.A. (2008). *Fundamentals of Veterinary Clinical Pathology*, Blackwell Publishing, Iowa, USA.

Tantary, H.A., Soodan, J.S., & Chirag, S. (2013). Diagnostic and therapeutic studies in ascites in ascites in dogs. *Veterinary Research, 6*(5-6), 111-114.

Tarn, A.C., & Lapworth, R. (2010). Biochemical analysis of ascitic (peritoneal) fluid: What should we measure. *Annals of Clinical Biochemistry, 47,* 397-407.

Tennant, B.C., & Center, S.A. (2008). Hepatic function. In: Kaneko, J.J., Harvey, J.W., & Bruss, M.L. 2nd eds., *Clinical Biochemistry of Domestic Animals*, Elsevier Publication, pp.379-412.

Tiwari, P., Varshney, J.P., & Hoque, M. (2011). Biliary obstruction in dogs: diagnosis and treatment. *Indian Journal of Veterinary Medicine, 21*(2), 115-116.

Watson, F.J., & Bunch, S.E. (2009). Diagnostic tests for the hepatobiliary system. In: S.J. Ettinger, *Small Animal Internal Medicine*. 4th eds., Elsevier Mosby St. Louis, Missouri, pp.496-519.