

## THERAPEUTIC MANAGEMENT OF ASCITES IN SPITZ DOG IN BANGLADESH WITH A BRIEF REVIEW ON CANINE ASCITES

M. A. Samad\*

Department of Medicine, Faculty of Veterinary Science, Bangladesh Agricultural University,  
Mymensingh-2202, Bangladesh \*E-mail: [vetmedbd@yahoo.com](mailto:vetmedbd@yahoo.com)

### ABSTRACT

**Background:** Ascites is one of the most important clinical syndromes, caused by multiple organ disorders, characterized by abdominal distension with accumulation of fluid of various colors and consistencies depending on the etiology that are encountered commonly in canine practice worldwide. Although it has been reported from different countries including India but it has not yet been documented from Bangladesh.

**Objectives:** To evaluate the successful therapeutic management of a clinical case of ascites in dog supported with its brief review for its appropriate application

**Materials and Methods:** A female Spitz dog two and half years old brought for treatment with the history of abdominal distension on 1<sup>st</sup> November 2009. Clinical examination, abdominocentesis and laboratory examination of ascitic fluid were used for the diagnosis of ascites in dog.

**Results:** Clinical examination revealed dyspnea, discomfort, lethargy, weakness, pale mucous membrane, normal rectal temperature 103.2 °F and distended abdomen with fluid thrill on palpation. Examination of ascitic fluid revealed clear white fluid (pure transudate) which is mainly hepatic origin resulting portal hypertension and hypoproteinaemia. Treatment with restricted sodium diet, antibiotic (amoxicillin), diuretic (furosemide; Lasix, Sanofi Aventis) and vitamin B-complex and C- vitamin supplement with regular monitoring assisted in successful recovery. The recovered dog survived for next five years up to 2014 and then died due to other reasons.

**Conclusions:** This clinical case record on canine ascites with successful treatment along with review especially on the methods of diagnosis and cause-wise treatment would certainly help the clinician for proper management of the clinical cases of canine ascites.

**Keywords:** Ascites, Spitz dog, Diagnosis, SAAG, Therapeutic management, Brief review

**Article Info:** Article ID No. © LEP: JVMOHR/00005/2019

Received: 1 April 2019    Revised: 2 May 2019    Accepted: 30 May 2019    Published: 30 June 2019

**Citation:** Samad MA (2019). Therapeutic management of ascites in an Indian breed of Spitz dog in Bangladesh with a brief review on canine ascites. *J. Vet. Med. OH Res.* 1 (1): 49-62



**Copy right** © 2019. The Author. Published by LEP. This is an open access article under the CC-BY-NC-ND License (<http://creativecommons.org/licenses/by-nc-nd/4.0/>)

## **INTRODUCTION**

Ascites (also known as abdominal effusion) is the abnormal accumulation of fluid in the peritoneal cavity both in humans and animals, especially more importance in dogs. The effusion is always a syndrome of diseases, associated with multiple causes and risk factors.<sup>1-4</sup> Clinically it is characterized by distended abdomen, anorexia, lethargy, dyspnea, weakness, discomfort and occasionally vomiting.<sup>2,4</sup> Ascites is one of the most commonly found clinical problems in dogs in India<sup>1,2,4-6</sup> and elsewhere<sup>7,8</sup> but it has not been reported from Bangladesh. This paper describes a successful therapeutic management of a clinical case of ascites in an Indian breed of Spitz dog for the first time in Bangladesh with a brief review on canine ascites for future perspective.

## **MATERIALS AND METHODS**

An Indian breed of Spitz dog (bitch) two and half years of age was brought to the Bangladesh Agricultural University (BAU) Veterinary Teaching Hospital (BAUVTH) from Mymensingh town for treatment with the complaint of distended abdomen for four months. History revealed that the dog was treated once in a month for four months at the Mymensingh Sadar Veterinary Hospital with oxytetracycline, antihistaminic, anthelmintic, Carmina Syrup (Herbal product; Hamdard) and antacid that caused only transient improvement and then the case was referred to the BAUVTH on 10<sup>th</sup> October 2009. The BAUVTH referred the case directly to me at the Department of Veterinary Medicine, BAU for treatment on 1<sup>st</sup> November 2009.

Passed treatment history also revealed that the dog was treated with ivermectin injection six months and albendazole orally three months earlier. Owner history revealed tedious and depress with massive voluminous abdomen with history of anorexia in the affected dog. Clinically examination revealed normal rectal temperature 103.2 °F but pale mucous membranes of eyes, dyspnea and tachycardia with fluid thrill (fluid wave) on tactile percussion. Microscopic examination of fecal sample found negative for parasitic infection. Based on the clinical findings (**Photo 1**) and abdominocentesis of abdominal fluid (**Photo 2**) it was diagnosed as a case of ascites.<sup>9</sup>

Approximately two liters of foamy acetic fluid were drained out from the abdomen (**Photo 3**) and the case was treated with amoxicillin (Fimoxyl<sup>®</sup> Sanofi Aventis) suspension @ 250 mg 8 hourly orally for 7 days for secondary bacterial infection, furosemide (Lasix<sup>®</sup> Sanofi Aventis) 40 mg table @ ½ Tablet (2.0 mg / kg body weight) orally daily for 7 days as diuretic, supportive treatment of B-complex (Beconex<sup>®</sup> Syp., Renata) and vitamin C (Ascason<sup>®</sup> Syp. Jayson) daily for 12 days.

## **RESULTS AND DISCUSSION**

This study recorded a clinical case of ascites in an Indian breed of Spitz female dog of two and half years of age with successful therapeutic management for the first time in Bangladesh. Comparatively higher incidence of ascites has been reported in Spitz dog (7 dogs) followed by Labrador Retrievers (2 dogs) and one non-descriptive dogs from two years of age in India.<sup>10</sup> The occurrence of ascites in dogs may be breed dependent with higher incidences in Pomeranian (33.35%) than in Labrador retriever (20%), Boxer (16.66%), Doberman pinscher



Photo 1. A Spitz bitch affected with ascites showing distended abdomen



Photo 2. Abdominocentesis in an ascites case of Spitz bitch- clear white fluid in the syringe.



Photo 3. Collection of about 2 liters of ascitic fluid from a bitch



Photo 4. Abdomen size of the bitch become normal after abdominocentesis but depressed

(13.37%), Mongrels (10%) and least in Alsatian (6.66%).<sup>6</sup> These findings are related with the availability of breeds in the investigated regions.

Ascites itself can physiologically interfere with respiration, cause general discomfort and disturb fluid and electrolyte metabolism. The clinical signs of anorexia, lethargy, weakness, dyspnea, discomfort, pale mucous membrane, rectal temperature 103.2 °F and distended abdomen with fluid thrill on palpation recorded in ascites affected dog in this study are in conformity with the earlier reports.<sup>6</sup>

### **Etiology and risk factors**

Ascites is always a sign of disease, associated with multiple causes and risk factors.<sup>11</sup> Generally two main causes of ascites are described which include cardiac problems and liver

disease (Table1) of which greater percentage caused by cardiac problems.<sup>7</sup> Multiple organ disorder, hypoproteinemia<sup>1,3</sup> and right side heart failure are the common causes associated with ascites in dogs. However, it can occur secondary to a number of diseases and pathological conditions (Table 1).

Table 1. Classification causes of canine ascites		
<p>A. Pathological conditions <sup>11</sup></p> <p>1. Venous hypertension</p> <ul style="list-style-type: none"> <li>• Cirrhosis of liver</li> <li>• Congestive cardiac failure</li> <li>• Constrictive pericarditis</li> <li>• Hepatic venous outflow obstruction</li> <li>• Acute portal vein thrombosis</li> </ul> <p>2. Hypoalbuminemia</p> <ul style="list-style-type: none"> <li>• Cirrhosis of liver</li> <li>• Nephrotic syndrome</li> <li>• Malnutrition</li> </ul> <p>3. Infections</p> <ul style="list-style-type: none"> <li>• Tuberculosis (human)</li> <li>• Parasitic (ancylostomiasis)</li> </ul>	<p>B. Organs affected<sup>7</sup></p> <p>1. Cardiac problems</p> <ul style="list-style-type: none"> <li>• Heartworms</li> <li>• Congestive cardiomyopathy</li> <li>• Right heart failure</li> <li>• Congenital pulmonary stenosis</li> </ul> <p>2. Hepatic diseases</p> <ul style="list-style-type: none"> <li>• Liver insufficiency</li> <li>• Chronic active hepatitis</li> <li>• Cirrhosis</li> <li>• Cholangitis</li> </ul> <p>3. Renal factors</p> <ul style="list-style-type: none"> <li>• Amyloidosis</li> <li>• Glomerulonephritis</li> </ul>	<p>C. Liver involvement<sup>12</sup></p> <p>1. Pre-hepatic causes</p> <ul style="list-style-type: none"> <li>• Portal vein thrombosis</li> <li>• Tuberculosis</li> <li>• Malnutrition</li> <li>• Hypoalbuminaemia</li> <li>• Strongyloidosis</li> <li>• Entamoeba</li> </ul> <p>2. Hepatic causes</p> <ul style="list-style-type: none"> <li>• Cirrhosis</li> <li>• Hepatitis</li> <li>• Portal hypertension</li> </ul> <p>3. Post- hepatic causes</p> <ul style="list-style-type: none"> <li>• CHF</li> <li>• LSHF</li> <li>• RSHF</li> <li>• CP</li> <li>• HWD</li> </ul>
<p>LSHF = Left-sided heart failure    RSHF = Right-sided heart failure</p>		

**Accumulation of ascitic fluid**

Normally, a small amount of lubricating fluid is found in the peritoneum which comes from the surrounding tissue and vessels. If the fluid exceeds the normal amount, an individual may have an effusion. The total amount of fluid that passes across the peritoneal membrane in a 24 hours period is about 80 ml / kg body weight in normal animals.

Ascitic fluid may accumulate rapidly or gradually depending upon the cause. Mild ascites may not produce any symptoms. Moderate ascites may just produce an increase in abdominal girth and weight gain. Large amounts of fluid can produce abdominal discomfort and hinder the mobility of the patient. Excessive fluid accumulation caused pressure on diaphragm and restriction of its movements can produce dyspnea. The causes, pathogenesis and characteristic of ascitic fluid are varied (Table 2)

The small size Indian breed Spitz bitch was severe affected with ascites with the accumulation of more than two liters transudate (**Photo 3**) in abdominal cavity that caused anorexia, lethargy, weakness, dyspnea and abdominal palpation revealed fluid thrill which are in conformity with the earlier reports.<sup>2,4,9,10</sup> The draining of two liters ascitic fluid reduced the abdominal distension to approximately normal size and shape (**Photo 4**). However, approximately 15 liter of colorless but slightly cloudy exudative ascitic fluid was drained out from a 10 months old Alsatian bitch with recurrent ascites.<sup>14</sup>

Table 2. Classification of ascites based on characteristic and formation of ascitic fluids <sup>13</sup>		
SNAscetic types	Pathogenesis	Major causes
① Normal fluid	Clear to slightly yellow	Naturally produced with specific gravity < 1.016
② Transudative ascites	Diseases that cause increased venous hydrostatic pressure with portal hypertension Total protein < 2.5g/dl	CHF, liver diseases or decreased COP e.g. protein losing enteropathy and nephropathy or severe hepatic failure (hypoalbuminaemia)
③ Exudative ascites	Form secondary to increased permeability of the peritoneal surface affected by inflammatory processes or neoplastic. Total protein > 2.5 g/ dl	Pancreatitis, bile peritonitis, malignant disease in peritoneum and septic peritonitis, Inflammatory disease conditions e.g. TB, neoplasm, pancreatitis, myxedema
④ Hemorrhagic ascites	Secondary to the pathological accumulation of blood in the peritoneal cavity.	Secondary to trauma, spontaneous ruptures pathological organs and coagulopathy.
CHF = Congestive heart failure		COP = Colloid osmotic pressure

### Diagnosis of ascites

Ascites is a syndrome of diseases therefore investigation should be aimed at identifying the primary underlying cause and risk factors.<sup>2,4</sup>

Standard diagnostic procedures of canine ascites include the physical examination, clinical examination, ultrasonography, computed tomography, serum ascites albumin gradient (SAAG), biochemical analysis such as triglyceride, urea and creatinine concentration and total protein. Novel diagnostic procedures such as platelet indices, leucocyte esterase reagent strip and tumor markers would aid in easy diagnosis of ascites.<sup>12</sup>

### Differential diagnosis

Ascites needs to be differentiated from abdominal distension due to other causes like gross obesity, gaseous distension, liquid, bowel obstruction, abdominal cysts, organomegaly or masses.

- Ascites with an elevated temperature indicates infectious or inflammatory condition.
- Auscultation reveal muffled heart sound which is consistent with pericardial effusion and cardiac tamponade.
- Heart murmurs or irregular heartbeats are suggestive of right-sided heart failure.
- An elevated heartbeat or tachypnea may result from dyspnea due to cranial displacement of the diaphragm into the thoracic cavity.
- Cardiovascular abnormalities are confirmed through the use of electrocardiograph and echocardiography.
- Intra-abdominal gas will give a sharp rebound to percussion and a higher pitched resonance when auscultated then fluid.

- The ascetic abdomen has a characteristic pear shaped and percussion of the abdomen will result in a sharp fluid rebound on the opposite site.
- A fluid thrill or wave may be demonstrated in cases of tense ascites.
- Ballottement through the abdominal wall may indicate a mass or enlargement of an organ.
- The presence of cardiac murmur on auscultation on the right side, 3<sup>rd</sup> to 5<sup>th</sup> intercostal space may indicate a tricuspid insufficiency due to cardiomyopathy or valvular fibrosis.
- Severe heartworm disease can cause right sided heart failure and delayed heart sounds such as late closure of the pulmonary valve may also indicate heartworms.
- Abdominal ultrasound scan is used to determine the abdominal content and aid in differentiating excess fluid accumulation from abdominal content and aid in differentiating excess fluid accumulation from abdominal masses and organ enlargement.

### **Abdominal paracentesis and analysis of ascetic fluid**

- Abdominal paracentesis is accompanied by advancing a needle into the most pendulous area of the abdomen while keeping a slight negative pressure in the syringe. This insures that fluid will be withdrawn as soon as the needle enters the peritoneal cavity.
- Abdominocentesis usually performed with the patient restrained in standing position or in left lateral recumbency and the area surrounding the umbilicus subjected to clipping and full surgical preparations. A site should be selected 2 and 3 cm caudal to the umbilicus and 2 to 3 cm left of the midline for sampling.
- Gently place the dog on a lateral decumbency exposing the larger part of the ventral abdomen.
- Swab the ventral abdomen less covered with hair coat, along the linea alba down to the ventral abdomen between the left and right hind limbs. Linea alba is the preferred site of paracentesis due to its less vascularization and less chance of contamination of the fluid with blood from puncture vessels and abdominal organs when approached through a different site of the abdomen.
- Puncture the linea alba using a 21 gauge needle and 10 ml syringe, and aspirate the fluid.
- Decant the content of the syringe into a clean and well-labeled tube for laboratory investigation.
- Fluid collected in EDTA should be submitted for total nucleated cell counts (TNCC), total erythrocyte count or PCV if the fluid is hemorrhagic or serosanguinous, cytology and further analysis as clinically indicated.
- Fluid collected in serum tubes can be used for analysis of albumin, bilirubin, creatinine, potassium, triglyceride, glucose, lactate and lipase levels.
- Fluid sample collected in sterile tubes can be stored for bacteria, mycoplasma and fungal culture. Anaerobic cultures are not refrigerated and should be processed within 24 hours of collection.

### **Physical examination of ascetic fluid**

The color of ascetic fluid ranges from clear fluid to yellowish, reddish and opaque with flakes of fibrin and debris depending on the etiology (Table 3).



SN	Color of fluid	Characteristics & composition	Major causes
a.	Clear opaque	Pure transudate free from contamination except with few invasions of mesothelial cells and tissue macrophages.	Portal hypertension, hepatic diseases, osmotic gradient deficit (hypoalbuminaemia), protein losing enteropathy, kidney impairment and albuminuria.
b.	Clear straw-colored	Modified transudate often characterized by the presence of fibrin cells and white blood cells such as neutrophils and lymphocytes.	Cases of long-standing ascites from various conditions including right-sided heart failure, cancerous growth and hepatic disease giving chance to invasion of fibrinogens.
c.	Pinkish discoloration	Purulent exudates, often turbid contain total protein > 2.0g/dl, > 6000 cells / $\mu$ l (neutrophils)	Evidence of 3+ bacterial infections
d.	Reddish discoloration	Cases of hemorrhages and collection of blood in the peritoneal cavity. Fluid contains high RBC and PCV > 20%	Trauma, coagulopathies and blood and blood vessel neoplasm. Peritoneal carcinoma, malignancy, pancreatitis, intestinal infraction and tuberculosis.
e.	Greenish/Blue discoloration	Rupture and seepage of bile into the peritoneal cavity.	Presence of bile with gallbladder problems.
f.	Milk-colored	Indicates chylous ascites (lipid-rich lymph in fluid)	Disease conditions such as carcinoma, lymphoma, tuberculosis or infection.

### Differences between transudate and exudate

The main differences between transudate and exudate are presented in Table 4.

SN	Parameters	Transudate	Exudate
①	Fluid produced / Secreted from:	Increased pressure in the hepatic, portal vein	Inflammation or malignancy
②	Protein content	Low (< 30 g / L)	High (> 30g/L)
③	Lactate dehydrogenase	Low	High
④	pH	High (> 7.3)	Low (< 7.3)
⑤	Glucose level	Normal	Low
⑥	White blood cell	Fewer cells	Large number

### Analysis of biochemical constituents

Biochemical constituents includes total protein, albumin, creatinine and urea, liver enzymes and coagulation profile would help in revealing cases of hypoalbuminaemia, hypoproteinaemia, hepatic and kidneys diseases.

**Total protein (hypoproteinaemia)**

Loss of protein can occur through the kidneys or intestinal tract or by a lack of production by the liver. Loss of protein through the kidneys can be pre-renal, renal or post-renal.

- a. Pre-renal proteinuria caused by increased hemoglobin (Hb), myoglobin or Bench-Jones proteins (from neoplastic plasma cells) in the urine.
- b. Renal proteinuria could result from functional or pathologic causes.
  - Functional proteinuria can result from heat, stress, seizures, fever or extreme exercise.
  - Pathologic proteinuria usually results in the highest and most persistent levels of proteinuria. Causes include glomerulonephritis and amyloidosis.
- c. Post-renal proteinuria could be the result of urinary tract infections, inflammation and hemorrhage (sources distal to the kidney).

**Serum ascites albumin gradient (SAAG)**

The diagnosis of ascites based on total protein concentration of < 2.5g/dl / > 2.5g/dl) in the ascitic fluid has an accuracy of only 56%.<sup>15</sup> However, the SAAG method is found as a more reliable tool in classification of ascites with efficacy ranging from 80 to 100%.<sup>16</sup> The transudative and exudative ascites were classified earlier based on the total protein concentration of the ascitic fluid but recently it has classified as ‘high gradient’ and ‘low gradient’ based on the SAAG.<sup>10,17,18</sup>

If the difference between serum albumin and ascitic fluid albumin is  $\geq 1.1$  g/dl it is called high gradient ascites, whereas if the difference is  $\leq 1.1$  g/dl it is termed as low gradient ascites (Table 5).<sup>16</sup>

Table 5. Estimation of SAAG in ascitic fluid (n = 10; Mean $\pm$ SE)			
SN Biochemicals	Serum	Ascitic fluid	
① Total protein (g/dl)	4.208 $\pm$ 0.245	0.770 $\pm$ 0.086	SAAG = Serum ascites albumin gradient
② Albumin (g/dl)	2.089 $\pm$ 0.195	0.296 $\pm$ 0.057	
③ Globulin (g/dl)	2.119 $\pm$ 0.259	0.473 $\pm$ 0.099	**SAAG $\geq 1.1$ g / dl = High gradient ascites
④ A : G ratio	1.212 $\pm$ 0.230	1.024 $\pm$ 0.378	
⑤ SAAG	1.793 $\pm$ 0.185**		SAAG $\leq 1.1$ g/ dl = Low gradient ascites

The SAAG is considered as a marker of portal hypertension and the use of this index to replace the exudates-transudate concept in ascitic fluid.<sup>19</sup>

The SAAG is calculated by subtracting the albumin concentration of the ascitic fluid from the albumin concentration of a serum specimen obtained on the same study.<sup>16</sup>

The SAAG  $\geq 1.1$ g /dl suggested high gradient ascites which is directly related with high portal hypertension pressure with an accuracy of 97-100%. Portal hypertension results in an abnormally increased hydrostatic pressure between the portal bed and the ascitic fluid.<sup>16,18</sup> High SAAG were noticed in cirrhotic liver, cardiac failure, portal vein thrombosis and hepatic failure.<sup>17</sup>



The SAAG  $\leq$  1.1g / dl suggested low gradient ascites which is directly related with normal portal pressure which are typically associated with disorders of vascular leakage and inflammation.<sup>20</sup>

Ascites from cardiac origin produces greater ( $>$  2.5g /dl) SAAG compared to cases of cirrhosis.

#### **Other biochemical analysis**

- Increased SGOT indicates hepatic insufficiency with extensive damage resulting into the leakage of enzymes from hepatic cell into blood stream.<sup>2,4,21</sup>
- The hypoglycemia is the indicative of hepatic insufficiency.<sup>2,4</sup>

#### **Triglycerides**

- An elevated triglycerides concentration in ascitic fluid  $>$  2.2 mmol/L indicates chylous ascites.<sup>22</sup>
- Chylous ascites is common in neoplastic cases and it may occur in 6% of cirrhosis.<sup>8</sup>

#### **Urea and creatinine**

- Elevated levels in urea and creatinine concentrations in ascitic fluid indicate pre-renal failure due to peritoneal absorption of urea (37). Urinary ascites is often associated with changes and urethra obstruction.<sup>23,24</sup>

#### **Hemato-cytologic examination**

- Hematology revealed significantly lower haemoglobin (Hb; 9.49 g/dl) and packed cell volume (PCV; 34.39%) whereas total leukocytic count (TLC) and differential leukocytic count (DLC) showed non-significant variation.
- Cytological examination of the fluid (direct smear or sediment) may indicate lymphosarcoma or sediment) may indicate lymphosarcoma or other neoplasia as the origin of the fluid.

#### **Bacterial culture**

- Cultured ascetic fluid should be subjected to sensitivity test to identify effective antimicrobiological agent in treatment.
- PCR for Mycobacterium tuberculosis offers a high sensitivity (94%) test compared to acid-fast stained and culture.<sup>22</sup>

#### **Differential diagnosis**

- Spontaneous bacterial peritonitis (SBP): fever, abdominal pain, abdominal tenderness, polymorphonuclear leucocyte (PMNL) count  $>$  250 cells / cumm.<sup>25</sup>
- Liver cirrhosis: portal hypertension, ascites, elevation of liver enzymes, SAAG  $>$  1.1g/dl.
- Portal hypertension: ascites, portal vein thrombosis, schistosomiasis.<sup>21</sup>
- Right sided hear failure: respiratory distress, cough, cyanotic tongue and syncope.<sup>11</sup>

#### **Treatment of ascites**

- Treatment of ascites is dependent on the identification of the underlying cause of ascites (Table 6).

Table 6. Reported primary causes of canine ascites			
SN Primary causes	Ref. No.	SN Primary causes	Ref. No.
<b>A. Cardiac disorders</b>			
① Congestive heart failure	32	② Right sided heart failure	11,26
<b>B. Liver and splenic disorders</b>			
① Portal hypertension	21	② Hepatic origin	2,5,28-30
③ Liver cirrhosis	14,30	④ Splenic origin	1
<b>C. Renal disorders</b>		<b>D. Infections</b>	
① Renal preservation	12	① Bacterial infection	25,31
		② <i>Babesia gibsoni</i>	27
		③ Ancylostomiasis	3

- Treatment efforts geared towards relieving manifesting symptoms and preventing progression of ascites.
- Paracentesis is applied to relieve abdominal tension on the diaphragm and enhance normal respiration. Repeated paracentesis is not required except in cases of failing treatment.<sup>25</sup>

If cardiac failure is the primary cause the standard treatment of diuretics and digitalis may help resolve the ascites as cardiac function is improved.

Cardiac drugs such as dopamine and digoxin can be used in cases of congestive heart failure in dogs.

- Dogs with right-side heart failure should be placed on cage rest and on sodium-restricted diet.<sup>26</sup>
- The administration of albumin dosed @ 1.5g /kg on the first day and 1.0 kg on the third day ensured renal preservation and reduced mortality.<sup>12</sup>
- Diuretics are used in addition to paracentesis to relieve ascites.
- Spironolactone @ 100 mg/ day or Furosemide @ 40 mg/ day
- Antibiotic treatment to ensure complete cure of the bacterial infection.
- Portal hypertension is managed by the use of antihypertensive medications: A drug such as metolazone (Mykron, Zaroxolyn) aids in the elimination of edema in congestive heart failure. It enhances sodium excretion by inhibition of sodium reabsorption from distal tubules, a function which is beneficial in renal conditions.

A three year old cross-bred bitch affected with ascites was treated with a combination of Lasix, Aldactone, D10, Hermin, Dexoma and Intacef upon regular monitoring assisted in successful recovery.<sup>9</sup>

Right heart failure due to ascites was confirmed in a female Labrador dog which was successfully managed with losartas, spironolactone, co-enzyme Q10 and tricholine citrate and sorbitol.<sup>11</sup>

Fifteen dogs of different breeds and age affected clinically by ascites were treated with dextrose 25% 100 ml IV for 5 days followed by furosemide (Lasix, M/S Aventis) @ 2 mg / kg bid PO daily for 10 days alone with supportive treatment of B-complex and vitamin C (Eldervit) @ 2 ml IM every alternate day for 12 days. Ceftriaxone sodium @ 25 mg / kg body weight and Astymin-3 40 ml IV OD for 5 days.<sup>6</sup>

An 8-year old female Labrador dog affected with ascites caused by hepatic congestion and right side heart failure was treated with angiotensin receptor blockers (losartas @ 25 mg, SID 90 days), aldosterone blockers (spironolactone @ 5 mg / kg BID, 30 days), syp sorbiline (tricholine citrate and sorbital) and nutraceutacle (co-enzyme Q-10, 1 tab for 90 days). Disappearance of abdominal distention and complete clinical recovery were recorded on day 90, no sign of recurrence was noticed within this time.

Successful treatment of a case of ascites of hepatic origin in a Doberman pensioner female dog 8 years of age with medicinal treatment including terramycin, Lasix, spiro lactone, doxycycline and tricholine citrate has been reported from India.<sup>2</sup> However, these types of medicinal treatment failed to recover the patient.<sup>11</sup>

The bitch was treated consecutively for 6 days IM with Furosemide (Lasix) @20 mg BID and Dexamethasone (Dexona) @ 2.2 mg OD; IV with 10% Dextrose (D10) @ 150 ml OD and Amino acid (Hermin injection) @ 50 ml OD; and oral with spironolactone (aldactone tablet) @ 12.5 mg BID. The animal was treated with Ceftriaxone (Intacef injection) @ 10 mg /kg bw IV for 5 consecutive days. The bitch was improved gradually as body weight declined day by day while treating. The bitch was represented on day 10<sup>th</sup> of treatment and it was found quite normal in gait with normal defecation and urination.<sup>11</sup>

Furosemide, a loop diuretic, helps in reduction of active absorption of sodium and chloride from the ultra at the luminal phase of the renal loop.

Aldactone, an aldosterone antagonist, increases sodium, calcium and chloride excretion and reduces excretion of potassium, hydrogen and ammonium ions by blocking aldosterone receptors in the renal tubule cells. Use of Hermin covered insufficiency of protein during ascites. Corticosteroids and antibiotics were used as supportive treatment to mitigate any inflammatory condition in the body.

Combined treatment of Lasix, Aldactone, D10, Hermin, Dexona and Intacef upon regular monitoring has been assisted successful recuperation of ascites.

Surgical treatment and removal of fluid will be the obvious choice for treating operable neoplasia and traumatic injuries leading to accumulation of bile, blood and urine in the peritoneal cavity.

Eggs, milk, lean meat, glucose and B complex vitamins are examples of high quality foods to be included in such a diet.

Removal of large quantities of fluid at any time is not generally recommended because of the albumin loss and possibility of circulatory collapse.

A summary of the different types of drugs used for the treatment of canine ascites with their indications and doses are presented in Table 7.

Table 7. Types of drugs used for the treatment of canine ascites with their indications and doses				
SN	Drugs used	Drug types	Indications	Dose (kg/bw)
①	Furosemide (Lasix <sup>®</sup> Sanofi)	Diuretics	Edema of cardiac, hepatic or renal	1-2 mg
②	Spirolactone (Inospiron <sup>®</sup> Incepta, Aldactone <sup>®</sup> Searle)	K-sparing diuretic	CHF, hypertension cirrhosis	3 mg
③	Metolazone (Metolaz <sup>®</sup> , Navana)	Diuretics (hypertension)	Edema, CHF, renal failure)	0.5 mg / dog daily
④	Digoxin (Cardoxin <sup>®</sup> Evsco; Lanoxin <sup>®</sup> GlaxoSmith)	Cardiac glycoside	CHF, dilated cardiomyopathy	0.0025-0.005mg / lb every 12 hours
⑤	Dopamine (Myomine <sup>®</sup> , Incepta)	Neurotransmitter	Cardiac shock, kidney disease	2-5 mcg/kg / min initially
⑥	Canine Albumin (Lyphilized 5g)	Albumin supplement	Hypoalbuminaemia or hypovolemic shock	450 mg/kg to rise blood albumin by 0.5/dl
⑦	Coenzyme Q10 (Ubiquinone, Ubidecarenone, Coenzyme Q)	Cardiovascular disease	Anti-oxidant- reduce damage of cells	@ 30 mg every 24-48 hours orally
⑧	Losartan potassium (Angilock <sup>®</sup> Square, Osartil <sup>®</sup> Incepta)	Angiotensin-II	Hypertension, CHF, renal arterial stenosis	25 mg/ daily orally
⑨	Tricholine citrate (Sorbiline <sup>®</sup> Susp., India (T citrate 0.55g + Sorbiline 7.15g)	Lipotropic action in liver	Hepatic disorders and asthma symptoms	1.0 TSF twice daily orally
⑩	Ceftriaxone (Ceftron <sup>®</sup> Vet Square)	Antibiotics	Bacterial infection	15-50 mg/kg bwt 12-24 hr im or iv injection
⑪	Astymin 3 injection (Tablets India Ltd.)	Nutritional Supplements	Vitamins, minerals & amino acids	@ 40 ml IV infusion daily for 5 days
⑫	Dexamethasone (Dexona <sup>®</sup> India, Dexa <sup>®</sup> Renata)	Corticosteroids	Swelling from inflammation	2.2 mg daily orally
⑬	Dextrose (D10) infusion (Baxter, India)	Glucose suppl.	Hypoglycemia	@ 150 ml orally or iv infusion
⑭	Amino acid injection (Aminosyn <sup>®</sup> , Freamine <sup>®</sup> FDA)	Protein & amino acid supplement	Hypoproteinaemia Hypoalbuminaemia	@ 50 ml/ dog iv

## CONCLUSIONS

Ascites is one of the most commonly recorded syndromes in pets of various age with multiple organs affected with multiple etiology and risk factors with high incidence in middle-aged dogs. Ascites is a common manifestation of a decompensate cirrhosis, cardiac diseases and several other etiologies and is best diagnosed through established standard procedures of

physical and clinical examinations, complete blood picture, cytology and various biochemical analyses. Treatment is centered on effective diagnosis of the etiology. However, ascitic fluid draining by abdominocentesis, diuretic, antibiotic (if required) and amino acids and vitamins supplement helped to complete recovery of the clinical case of ascites in dogs. It appears that the restricted sodium diet and treatment with diuretics only may not be adequate to eliminate the abdominal fluid but abdominocentesis is required in severe cases along with the restricted sodium diet and diuretics. Review findings especially diagnosis and treatment of ascites would be useful for the veterinary clinicians for application of therapeutic management of the canine ascites.

### CONFLICT OF INTEREST

There is no conflict of interests. Clinical management of a dog and no funding has been received for any part of this study.

### REFERENCES

01. Dabas VS, Suthhar DN, Chaudhari CF, Modi LC and Vihol PD (2011). Ascites of splenic origin in a Mongrel female dog- A case report. *Veterinary World* 4: 376-377
02. Pradhan MS, Dakshinkar NP, Waghaye UG and Bodkhe AM (2008). Successful treatment of ascites of hepatic origin in dog. *Veterinary World* 1: 23
03. Turkar S, Randhawa CS and Uppal SK (2009). Ascites associated with ancylostomiasis in pup: A case report. *Intas Polivet* 10: 357-359
04. Kumar A, Das S and Mohanty DN (2016). Therapeutic management of ascites in GSD female dog- case report. *International Journal of Science, Environment and Technology* 5: 654-657
05. Randhawa SS, Dhaliwal PS, Kirti S and Singh KB (1988). Ascites of hepatic origin in dogs. *Indian Journal of Animal Health* 12: 165-166
06. Saravanan M, Sarma K, Kumar M, Mahendran K and Mondal DB (2013). Therapeutic management of ascites in dogs. *Indian Veterinary Journal* 90: 110-111
07. Peden WM and Zenoble RD (1982). Canine ascites. *Iowa State University Veterinarian* 44: 12-17
08. Laterre PF, Dugernier T and Reyaert MS (2000). Chylous ascites: Diagnosis, causes and treatment. *Acta Gastroenterologica Belgica* 63: 260-263
09. Chutia T, Ghorai S, Sarma K, Das G, Behera SK, Konwar B, Ahmed FA and Lalrintluanga K (2016). Therapeutic management of ascites in a bitch – a case report. *International Journal of Livestock Research* 6: 71-73 doi:10.5455/ijlr.20161127031551
10. Saravanan M, Sarma K, Kumar M, Vijaykumar H and Mondal DB (2012). Analysis of serum ascites albumin gradient test in ascetic dogs. *Veterinary World* 5: 285-287
11. Kumar KS and Srikala D (2014). Ascites with right heart failure in a dog: diagnosis and management. *Journal of Advanced Veterinary and Animal Research* 1: 140-144
12. Nwoha RIO (2019). Review on ascites in pets [Online First], *IntechOpen*, Doi:10.5772/intechopen.84767
13. Zoia A, Drigo M, Piek CJ, Simioni P and Caldin M (2017). Hemostatic findings in ascetic fluid: A cross-sectional study in 70 dogs. *Journal of Veterinary Internal Medicine* 31: 43-50
14. Nottidge HO, Okewole EA, Taiwo VO, Emikpe B, Adedokun RAM and Oduye OO (2003). Liver cirrhosis associated with a non-responsive ascites in a 10 month old Alsatian dog. *African Journal of Biomedical Research* 6: 151-153

15. Sood R (2000). Ascites: diagnosis and management. *Journal of Indian Academy of Clinical Medicine* 5: 81-89
16. Burgess LJ (2004). Biochemical analysis of pleural, peritoneal and pericardial effusions. *Clinical Chemistry Acta* 34: 61-64
17. Das B, Acharya U and Purohit A (1998). Comparative utility of sero-ascites albumin gradient and ascetic fluid total protein for differential diagnosis of ascites. *Indian Pediatrics* 35: 542-545
18. Beg M, Husain S, Ahmed N and Aktar N (2001). Serum ascites albumin gradient in differential diagnosis of ascites. *Indian Academy of Clinical Medicine* 2: 51-54
19. Tarn AC and Lapworth R (2010). Biochemical analysis of ascetic (peritoneal) fluid: what should we measure? *Annals of Clinical Biochemistry* 47: 397-407
20. Mondal DB, Kumar M, Saravanan M and Sharma K (2012). Peritoneal fluid analysis in canine disease diagnosis. *Journal of Advanced Veterinary Research* 2: 307-313
21. Beker SG and Valencia-Parparcen J (1968). Portal hypertension syndrome. *The American Journal of Digestive Diseases* 13: 1047-1054
22. Oey RC, van Buuren HR and de Man RA (2016). The diagnostic work-up in patients with ascites: Current guidelines and future prospects. *The Netherlands Journal of Medicine* 74: 330-338
23. Peeters P, Colle II, Serinesael J and Verbeelen D (2001). Relapsing ascites and uremia due to urinary bladder leakage. *European Journal of Internal Medicine* 12: 60-63
24. Snauwaert C, Geerts A, Colle I and Van vlierberghe H (2012). *Ascites gastro-enterologica Belgica* 75: 45-48
25. Koulaouzidis A, Bhat S, Karagiannidis A, Tan WC and Linaker BD (2007). Spontaneous bacterial peritonitis. *Postgraduate Medical Journal* 83: 379-383
26. Moore CM and Van Thiel DH (2013). Cirrhotic ascites review: pathophysiology, diagnosis and management. *World Journal of Hepatology* 27: 251-263
27. Gonde S, Chhabra S, Singla LD and Bansal BK (2014). Peritoneal effusion in a dog due to *Babesia gibsoni* infection. *Case Reports in Veterinary Medicine*. Article ID 807141; <http://dx.doi.org/10.1155/2014/807141>
28. Raffan E, McCallum A, Scase TJ and Watson PJ (2009). Ascites is a negative prognostic indicator in chronic hepatitis in dogs. *Journal of Veterinary Internal Medicine* 23: 63-66
29. Regmi B and Shat MK (2017). A case study on ascites of hepatic origin and their proper management in a male German Shepherd dog. *International Journal of Applied Sciences and Biotechnology* 5: 555-558
30. Christopher MM and Van Thiel DH (2013). Cirrhotic ascites review: pathophysiology, diagnosis and management. *World Journal of Hepatology* 5: 251-263
31. Bray J (1996). Diagnosis and management of peritonitis in small animals. *In Practice* 31: 403-431
32. Alsaad KM, Alfaris AA and Lafta MH (2018). Ascites associated with congestive heart failure in a police dog (K9) (Diagnosis and Management). *Veterinary Science and Medicine* 1: 1-4