The Pharma Innovation Journal 2022; SP-11(7): 4591-4594

www.ThePharmaJournal.com

The Pharma Innovation

**ISSN (E): 2277-7695**
**ISSN (P): 2349-8242**
**NAAS Rating: 5.23**
TPI 2022; SP-11(7): 4591-4594

www.thepharmajournal.com
Received: 01-04-2022
Accepted: 04-05-2022

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# Biochemical studies in ascites of dogs

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**Abstract**
The comparative evaluation was carried out to study the alterations in biochemical profile in ascitic dogs (N=30) in four phases. Phase-1 i.e., pre-treatment phase ($0^{th}$ day of diagnosis) and post/during treatment phases of ascites i.e., phase-2 ($3^{rd}$ day of treatment), phase-3 ($7^{th}$ day of treatment) and phase-4 ($14^{th}$ day of treatment) were included in our study, respectively. Biochemical profile Serum Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), Direct bilirubin, creatinine and blood urea nitrogen (BUN) concentration showed a highly significant ($P<0.01$) increase while a highly significant ($P<0.01$) decrease in serum glucose, cholesterol and albumin was observed in pre-treatment phase of ascites (phase-1). Whereas Total protein and globulin concentration showed a non-significant ($P>0.05$) alteration during pre-treatment and treatment /post-treatment phases of ascites in our study.

**Keywords:** Dog, ascites, 4 phases, biochemical studies

## Introduction
Ascites refers to the abnormal collection of serous fluid within the peritoneal cavity in both humans and animals and it occurs mainly due to liver disorders (Samad, 2019) [19]. The word ascites derived from Greek word “askos” means “bag” and “ites” means “like a”. Ascites is a transudate fluid accumulation in the abdomen that is most commonly found in dogs and cats, but also in sheep and other animals. Many owners mistakenly believe their female dog is pregnant and fail to seek medical attention for the ascites condition. It is divided into three types based on the pathophysiology of its formation: exudate, transudate, and haemorrhagic ascites (Zoia *et al.*, 2017) [24].
True ascites is a build-up of serous or serosanguinous fluid in the peritoneal cavity. Symptoms include abdominal distension, dyspnoea, lethargy, anorexia, vomiting, weakness, and discomfort. Ascites is always a sign of a serious illness. It can disrupt fluid and electrolyte metabolism, restrict breathing, and cause general discomfort (Peden and Zenoble, 1982) [14].
Ascites is not a disease in and of itself, but just a condition or clinical sign that develops as a result of another primary disease condition. Chronic liver failure, congestive heart failure, nephritic syndrome, malnutrition, ancylostomiasis and protein-losing enteropathy in dogs, high parasitism, and abdominal neoplasia of various origins have all been linked to it as a result of normal functions or pathological conditions (Dabas *et al*., 2011) [14].
Hepatic, renal, and cardiovascular insufficiencies, hormonal and metabolic diseases, liver tumours, and carcinomas are all linked to ascites. Congestive heart failure is the other common cause of ascites, followed by cirrhotic liver disease, chronic active hepatitis, and kidney injury, each of which is marked by unique blood chemistry and haematological abnormalities (Chaturvedi *et al*., 2013[3], Ihedioha *et al.*, 2013) [8]. In dogs, ascites has been well established as a result of cardiac failure and ventricular hypertrophy (Mukherjee *et al*., 2017) [12]. Aim the ascites is one of the most important clinical problems in dogs in India. The major role of clinico-hematobiochemical profile is very important in early and accurate diagnosis of ascites in affected dogs, which may be beneficial for improving the health condition and helping in the treatment in different phases of ascites.

## Materials and Methods
The present study was conducted on 30 ascetic dogs, were brought for the treatment at Government Veterinary Polyclinic hospital, Jayanti Market, MI Road, Jaipur and other nearby hospitals. Blood samples (5 ml each) were collected aseptically from cephalic/saphenous vein of affected dogs, directly into the Serum Separating Tubes (SST) in order to analyse different biochemical parameters, respectively. Phase-1 i.e., pre-treatment phase ($0^{th}$ day of diagnosis)

and post/during treatment phases of ascites i.e., phase-2 (3rd day of treatment), phase-3 (7th day of treatment) and phase-4 (14th day of treatment) were included in our study, respectively. The biochemical parameters i.e., The liver function test AST (aspartate aminotransferase/SGOT and ALT (alanine aminotransferase/SGPT), The Renal function test (Blood Urea Nitrogen and Creatinine), Serum metabolites (Total Protein, Albumin, Globulin, Glucose, Direct Bilirubin and Cholesterol) were estimated by using Automated Blood Biochemistry Analyzer (TurboChem100) by using Jeev Diagnostic Kits.

## Results and Discussion

Details of various biochemical parameters recorded at different phases of ascites in thirty dogs.

| Parameters | Pre-treatment | Treatment /Post-treatment Phase | | |
|---|---|---|---|---|
| | Phase- 1 (0 Day/Diagnosis of Ascites) | Phase -2 (3rdDay of Treatment) | Phase-3 (7thDay of Treatment) | Phase – 4 (14thDay of Treatment) |
| AST (U/I) | 95.09±0.99$^{d}$ | 65.45±0.98$^{c}$ | 35.28±0.99$^{b}$ | 28.11±0.85$^{a}$ |
| ALT (U/I) | 124.97±0.97$^{d}$ | 111.46±0.98$^{c}$ | 56.82±0.94$^{b}$ | 34.43±0.97$^{a}$ |
| BUN | 35.75±0.99$^{d}$ | 33.16±0.94$^{c}$ | 28.55±0.91$^{b}$ | 21.20±0.79$^{a}$ |
| Creatinine | 2.36±0.16$^{c}$ | 1.63±0.12$^{b}$ | 1.43±0.10$^{b}$ | 1.01±0.05$^{a}$ |
| Total Protein | 4.99±0.21 | 5.18±0.21 | 5.22±0.39 | 5.31±0.23 |
| Albumin | 1.07±0.07$^{a}$ | 1.24±0.10$^{ab}$ | 1.35±0.09$^{b}$ | 1.69±0.08$^{c}$ |
| Globulin | 3.92±0.16 | 3.94±0.13 | 3.85±0.26 | 3.62±0.07 |
| Glucose | 58.13±0.98$^{a}$ | 64.01±0.98$^{b}$ | 70.14±0.75$^{c}$ | 87.69±0.94$^{d}$ |
| Direct Bilirubin | 1.08±0.07$^{c}$ | 0.96±0.04$^{c}$ | 0.79±0.03$^{b}$ | 0.47±0.02$^{a}$ |
| Cholesterol | 104.56±0.99$^{a}$ | 107.93±0.97$^{b}$ | 121.19±0.96$^{c}$ | 145.57±0.92$^{d}$ |

Note: Comparison has been done between different phases.
Mean ± SE bearing different superscripts differed significantly ($p \leq 0.05$).

### Aspartate aminotransferase/AST/SGOT (U/L)

The mean ± SE values of AST (U/L) were measured as 95.09±0.99, 65.45±0.98, 35.28±0.99, and 28.11±0.85 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. AST recorded with highly significant ($p<0.01$) difference during the periodic intervals of ascites in dogs. This finding of our study was well in agreement with Lakshmi and Padmaja (2021) [10], Prebavathy *et al*. (2020) [16], Elhiblu *et al*. (2015) [7], Saravanan *et al*. (2014) [21], Ihedioha *et al.* (2013) [8], Kumar *et al.* (2013) [9] and Sanjeeta *et al*. (2013) [20] in ascites with canine hepatic disorders. A non-significant ($p>0.05$) increase in the mean values of AST (95.37±9.47 IU/L) was found by Lakshmi *et al.* (2017) [11] in dogs with right sided heart failure. It might be due to severe heart diseases being able to cause pre-renal azotemia, and electrolyte imbalances.

### Alanine aminotransferase/ALT/SGPT (U/L)

The mean ± SE values of ALT (U/L) were measured as 124.97±0.97, 111.46±0.98, 56.82±0.94, and 34.43±0.97in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed that concentration of serum ALT varied highly significantly ($p \leq 0.01$) during ascites in dogs. The serum ALT was well in accordance with Lakshmi and Padmaja (2021) [10], Prebavathy *et al*. (2020) [16] in ascites associated with canine hepatic disease, Bera and Lodh (2019) [2], Phom *et al*. (2019) [15] in dogs with ascites and hepato-renal syndrome in cirrhosis, Roopali *et al*. (2018) [18] in dogs canine Ehrlichiosis, Elhiblu *et al*. (2015) [7] in dog with liver cirrhosis, Saravanan *et al*. (2014) [21] and Kumar *et al.* (2013) [9] in ascitic dogs, It could be due to hepatocellular dysfunction, hepatitis, hepatic trauma, anaemia, and toxaemia. An increased ALT level might be caused by hepatocellular necrosis and inflammation with injured hepatocytes, multi-organ dysfunction particularly hepato-renal syndrome (HRS), liver, and kidney problems. Liver is the major site of metabolism therefore; elevated AST and ALT levels might rather be due to decrease in catabolism and/or by increase extracellular leakage of injured hepatocytes. As AST and ALT are markers of the hepato-cellular function.

### Blood Urea Nitrogen (mg/dl)

The mean ± SE values of BUN (mg/dl) were measured as 35.75±0.99, 33.16±0.94, 28.55±0.91, and 21.20±0.79 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. The estimated values of BUN were more in Phase-1, Phase-2 and Phase-3 than normal range. The serum concentration of BUN recorded with highly-significant ($p<0.01$) increase in phase-1 (pre-treatment phase), which was well in agreement with Prebavathy *et al*. (2020) [16] and Phom *et al*. (2019) [15] in ascites and hepato-renal syndrome in cirrhosis and Roopali *et al*. (2018) [18] in canine Ehrlichiosis. It might be due to immune complex mediated glomerulonephritis. Bera and Lodh (2019) [2] also reported a significant increase in BUN in (ascites associated with canine hepatic disorders. It might be due to renal abnormalities, resulting into urinary tract retention caused by any obstruction.

### Creatinine (mg/dl)

The mean ± SE values of creatinine (mg/dl) were measured as 2.36±0.16, 1.63±0.12, 1.43±0.10, and 1.01±0.05 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed that concentration of serum creatinine varied highly significantly ($p \leq 0.01$) during ascites in dogs. The higher serum creatinine level in Phase-1 (pre-treatment phase) found in ascitic dogs in the present study was similar to Prebavathy *et al*. (2020) [16], Singh *et al*. (2019) [22], Bera and Lodh (2019) [2] in ascites related to canine hepatic disorders. Roopali *et al*. (2018) [18] also observed a significant increase in creatinine level in canine Ehrlichiosis. It might be due to immune complex mediated glomerulo-nephritis or caused by renal abnormalities and urinary tract retention due to obstruction.

### Total Protein (g/dl)

The mean ± SE values of total serum protein (g/dl) were measured as 4.99±0.21, 5.18±0.21, 5.22±0.39, and 5.31±0.23 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed a non-significant ($P \geq 0.05$) differences on serum concentration of total protein during ascites in dogs. During the treatment of ascites in dogs.

The lower serum protein level in Phase-1 (pre-treatment phase) was found in ascitic dogs in our study which was well in agreement with Dhillon *et al.* (2020) [6], Bera and Lodh (2019) [2], Phom *et al.* (2019) [15], Alsaad *et al.* (2018) [1], Lakshmi *et al.* (2017) [11], Elhiblu *et al.* (2015) [7], Saravanan *et al.* (2014) [21] and Nottidge *et al.* (2003) [13] in various ascites associated with hepato-cardiac-renal diseases in dogs. Hypoproteinaemia might be an indication of hepatic insufficiency; Rao *et al.* (2021) [17] observed anaemia and protein loss in ascites in dogs associated with Ancylostomiasis. It might be occurred due to failure of protein and glucose synthesis in liver dysfunction as well as increasing catabolic activity in liver.

**Albumin (g/dl)**

The mean ± SE values of serum Albumin (g/dl) were measured as 1.07±0.07, 1.24±0.10, 1.35±0.09 and 1.69±0.08 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed that concentration of serum albumin varied highly significantly ($p \leq 0.01$) during ascites in dogs. The lower serum albumin level in Phase-1 (pre-treatment Phase) was found in ascitic dogs in the present study, which was well in agreement with Prebavathy *et al.* (2020) [16] in canine hepatic disease, Roopali *et al.* (2018) [18] in canine Ehrlichiosis, Elhiblu *et al.* (2015) [7], Alsaad *et al.* (2018) [1], Saravanan *et al.* (2014) [21], Lakshmi *et al.* (2017) [11] in ascites with right side heart failure and Dayrell-Hart *et al.* (1991) [5] in hepatotoxicity of phenobarbital in dogs. Low serum albumin concentration was observed due to liver disease indicated a diffuse and chronic hepatopathies in ascitic dogs. In hypoalbuminemia, the osmotic pressure is decreased, resulting in increased hydrostatic pressure causing fluid to escape from the vasculature into the body cavity.

**Globulin (g/dl)**

The mean ± SE values of serum globulin (g/dl) were measured as 3.92±0.16, 3.94±0.13, 3.85±0.26 and 3.62±0.07 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed a non-significant ($P \geq 0.05$) variations on serum concentration of globulin during ascites in dogs. Globulin recorded in agreement with Lakshmi and Padmaja (2021) [10], Prebavathy *et al.* (2020) [16] and Bera and Lodh (2019) [2] in ascites associated with canine hepatic disorders, Roopali *et al.* (2018) [18] in canine Ehrlichiosis. Hyperglobulinemia might be occurred due to chronic liver diseases. A higher globulin concentration could be due to a committed B cell response to the infective organism's continuous antigenic stimulation, increased gamma globulin fraction synthesis associated with increased systemic immune reactivity against portal antigens, or secondary to antibody production indicating a long-term infection.

**Glucose (mg/dl)**

The mean ± SE values of glucose (mg/dl) were measured as 58.13±0.98, 64.01±0.98, 70.14±0.75 and 87.69±0.94 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Analysis of variance (ANOVA) revealed that concentration of serum Glucose varied highly significantly ($p \leq 0.01$) during ascites in dogs. The lower serum glucose level in Phase-1 (pre-treatment Phase) was found in ascitic dogs in our study which was well in accordance with Lakshmi and Padmaja (2021) [10], Dillon *et al.* (2020) [6], Bera and Lodh (2019) [2] and Kumar *et al.* (2013) [9] in ascites associated with canines' hepatic disorder and Elhiblu *et al.* (2015) [7] in ascitic dogs with liver cirrhosis. Hypoglycaemia was seen in chronic hepatitis patients, which could be attributable to inappetence and anorexia, as well as malabsorption from the gut. Hypoglycaemia in patients with hepatic disorders was caused by decreased glycogenolysis and gluconeogenesis, as well as hyperinsulinemia due to decreased hepatic metabolism.

**Direct Bilirubin (mg/dl)**

The mean ± SE values of direct bilirubin (mg/dl) were measured as 1.08±0.07, 0.96±0.04, 0.79±0.03 and 0.47±0.02in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. Direct bilirubin recorded with highly-significant ($p<0.01$) difference in various phases of ascites, which was well in accordance with Lakshmi and Padmaja (2021) [10], Bera and Lodh (2019) [2], Phom *et al.* (2019) [15] and Saravanan *et al.* (2014) [21] in Dogs with ascites and hepato-renal syndrome in cirrhosis. An increased bilirubin i.e., hyperbilirubinemia in ascites could be due to a disruption in the imbalance between bilirubin synthesis, metabolism, and excretion.

**Cholesterol (mg/dl)**

The mean ± SE values of cholesterol (mg/dl) were measured as 104.56±0.99, 107.93±0.97, 121.19±0.96, and 145.57±0.92 in Phase-1, Phase-2, Phase-3 and Phase-4, respectively. The concentration of cholesterol varied highly significantly ($p \leq 0.01$) during ascites in dogs. The lower serum cholesterol level in Phase-1 (pre-treatment Phase) was found in ascitic dogs in our study, which was similar to Bera and Lodh (2019) [2], and Srivastava and Syed (2013) [23] in ascites with canine hepatic disorders. They reported low level of cholesterol i.e., hypocholesterolaemia which might be attributed to decrease in synthesis or absorption from gut or excessive conversion of cholesterol into bile acids. Hypocholesterolaemia is might be due to anaemia, hyperthyroidism, cancer, liver disease, critical illness, severe stress, malabsorption or malnutrition, acute or chronic infection, chronic inflammation, and the use of certain medicines.

**Conclusion**

Based on obtained findings in pre-treatment phase of ascites (phase-1), it can be concluded that ascites affects biochemical parameters in dog. It has a profound impact on health status of dog. This study can serve as a diagnostic means for the detection of ascites when correlated with the history and clinical signs.

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