CLINICO-THERAPEUTIC STUDIES ON CANINE ASCITES

By

Apurva

(J-19-MV-589)

Thesis submitted to Faculty of Postgraduate Studies
In partial fulfillment of requirements
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IN
VETERINARY MEDICINE

Division of Veterinary Medicine

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CERTIFICATE-I

This is to certify that the thesis entitled **"Clinico-Therapeutic Studies on Canine Ascites"** submitted in partial fulfillment of the requirements for the degree of **Master of Veterinary Sciences** in **Veterinary Medicine,** Sher-e-Kashmir University of Agricultural Sciences and Technology of Jammu, is original work and has similarities with published work not more than minor similarities as per UGC norms of 2018 adopted by the University. Further the level of minor similarities has been declared after checking the manuscript with URKUND software provided by the University

The work has been carried out by **Ms. Apurva** Registration No. **J-19-MV-589** under my supervision and guidance. No part of the thesis has been submitted for any other degree or diploma. It is further certified that help and assistance received during the course of thesis investigation have been duly acknowledged.

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This is to certify that the thesis entitled "**ClinicoTherapeutic Studies on Canine Ascites**", submitted by **Ms. Apurva**, Registration No. **J-19-MV-589,** to the Faculty of Veterinary Sciences and Animal Husbandry, Sher-e-Kashmir University of Agricultural Sciences and Technology, Jammu, in partial fulfillment of the requirements for the degree of **Masters of Veterinary Medicine,** was examined and approved by the advisory committee and external examiner(s) on 6th Jan. 2022.

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Needless to say, all omissions and errors are mine.

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ABSTRACT

Title of Thesis	:	**Clinico-Therapeutic Studies on Canine Ascites**
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Abstract

The present study was conducted, to study the prevalence and clinical findings associated with ascites in dogs presented at Small Animal OPD of Teaching Veterinary Clinical Complex, Faculty of Veterinary Sciences and Animal Husbandry, R. S. Pura campus of SKUAST- Jammu and also in and around Jammu district, during the study period (August 2020-June 2021). Out of a total 2550 presented cases only 21 dogs were found to be affected with ascites making overall prevalence of 0.82 per cent (21/2550). Age wise prevalence of canine ascites was found to be higher in the age group of 2-5 years. Females were found more susceptible to ascites than males. The prevalence of ascites was highest in Labrador, followed by Spitz, German shepherd and least in Golden retrievers. Abdominal distension and fluid thrill were recorded in all ascetic dogs along with other clinical signs such as anorexia, inappetence, pale mucous membrane, tachycardia, respiratory distress and sub cutaneous edema of limbs. The 21 ascitic cases were classified into three causative types: hepatic origin, renal origin and involvement of both cardiac and hepatic system. The haematological changes observed were significantly ($p<0.05$) lower levels of haemoglobin concentration, PCV, TEC values in all the ascitic cases whereas significantly ($p<0.05$) higher values of TLC, absolute neutrophilic and lymphocytic count was observed in dogs with ascites of hepatic origin. The biochemical changes observed in dogs with ascites of hepatic origin involved significantly ($p<0.05$) higher serum activity of ALT, AST and ALP, significantly lowered levels of serum total protein and albumin and BUN. Renal origin ascites had significantly ($p<0.05$) high serum activity of ALT, AST, ALP, low serum total protein, albumin, very high serum levels of creatinine and BUN. Dogs with ascites of both cardiac and hepatic involvement had significantly ($p<0.05$) very high serum ALT, ALP activity and lower total protein levels. The ascitic fluid was transudate and modified transudate in nature. The SAAG values were significantly ($p<0.05$) higher in dogs with ascites due to cardiac and hepatic involvement compared to the ascites of hepatic or renal origin. Altered haematobiochemical parameters were restored by the 45th day of treatment. The present study will support the diagnosis and therapeutic management of canine ascites.

Keywords: Ascites, Dogs, Prevalence, Haemato-biochemistry, Ascitic fluid Characteristics, SAAG, Radiography, Ultrasonography, ECG.

Signature of Major Advisor

Signature of the Student

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ABBREVIATIONS

%	Per cent
@	At the rate of
µ	Micron
µl	Microliter
º F	Degree Fahrenheit
ACE	Angiotensin Converting Enzyme
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
ANOVA	Analysis of Variance
AST	Aspartate amino transferase
BID	Bis In Die
BUN	Blood urea nitrogen
cm	Centimeters
cu mm	Cubic Millimeters
CHF	Congestive Heart Failure
d	Day
dl	Deciliters
DLC	Differential leukocytic count
DMRT	Duncan Multiple Range Test
DNS	Dextrose normal saline 5%
D 10%	Dextrose intravenous infusion 10%
ECG	Electrocardiography
EDTA	Ethylene diamine tetra acetic acid
Et al.	Et Alia
F	Female Sex
Fig.	Figure
gm	Grams
GGT	Gamma Glutamyl Transferase
GLDH	Glutamate Dehydrogenase
Hb	Hemoglobin
hr.	Hour
IFCC	International Federation of Clinical Chemistry

Inj.	Injection
I/M	Intra muscular
IU	International unit
I/V	Intra venous
Kg	Kilogram
kVp	Kilovoltage Peak
L	Liters
L	Lymphocytes
M	Male Sex
mAs	Milliampere-Seconds
max	Maximum
mg	Milli gram
mEq	Milli Equivalents
mHz	Mega hertz
min	Minute
ml	Milliliter(s)
N	Neutrophils
PCV	Packed cell volume
pH	Negative molar concentration of hydrogen ions
PO	Per os
RBC	Red blood cells
Rpm	Revolutions per minute
SAAG	Serum Ascitic Albumin Gradient
SE	Standard error
SGOT	Serum Glutamate Oxaloacetate Transaminase
SGPT	Serum Glutamate Pyruvate Transaminase
SPSS	Statistical Package for the Social Studies
TB	Tuberculosis
TEC	Total Erythrocyte Count
Tid	Ter in Die
TLC	Total Leukocyte Count
TP	Total plasma protein
TVCC	Teaching Veterinary Clinical Complex
USG	Ultrasonography

Chapter-I

Introduction

CHAPTER-I

INTRODUCTION

Dog *(Canis lupus familiaris)* has been domesticated by mankind for over 30,000 years. Domestication of dogs as primary species by humans was due to the loyal and companionship behaviour of this animal. Since ancient times, dogs have been tamed and harnessed by humans. They have served man in various forms such as hunting companion, in police work, detection of explosives, cadaver detection, rescue operations, animal assisted therapy, herding etc. As dogs are closely associated to human lives and have become an inseparable part of their family, it is always a priority of the pet owner to keep them healthy by providing well timed care and fitness management. The maximum numbers of diseases which have an effect on the health of the dogs are associated with gastrointestinal system that stands subsequent to the dermatological conditions. Thus, it becomes the responsibility of a veterinarian to apprehend the ailment and offer the correct therapy to the dogs brought for treatment, as these pets are family member to their owners.

Ascites is the clinical condition which has multiple etiological factors. It is not one of the commonly observed clinical conditions in the daily canine practice; however it gains attention of the owners because of its aesthetic appearance and the ignorant notion of spread out in humans. Ascites is derived from Greek word "ASKOS" which means bag or sac and physicians knew about the disorder since the time of Hippocrates (circa 400BC), when only treatment known was paracentesis (Reynolds, 2000). Immoderate accumulation of fluid in the abdominal cavity is known as hydroperitoneum or Ascites (Kumar *et al*., 2005). On physical examination, the dogs suffering from ascites are presented with a visibly bulged abdominal region and hollow flank along with the presence of fluid thrills on percussion (Moore *et al.*, 2003). As the bulged abdomen can be easily recognised along with the presence of fluid thrill which makes the diagnosis process easy, treatment must be aimed at etiological factors responsible for it. Many scientists have stated the incidence of ascites in dogs similar to the condition observed in humans (Selgas *et al*. 2008; Biecker 2011).

Ascites is a symptom in place of an ailment. Accumulation of fluid inside body cavity results when the rate of filtration of fluid into area is more than the rate of fluid resorption from that area. The primary event which leads to development of ascites is an alteration in one or more of the starling forces that governs the fluid movement across the membranes. Imbalance in the starling forces results from the increased sinusoidal pressure which increases the permeability of serum protein to interstitial space. This drives the protein rich transudate from the liver capsule to the abdominal cavity. Renal retention of sodium and fluid along with the aldosterone release aggravates the ascites formation. Decreased plasma osmotic pressure due to the hypoalbuminemia also leads to ascites formation (Sevelius, 1995).

Any intense involvement of coronary heart, liver, kidneys and lungs produce ascites by disturbing the starling forces. Among the various etiological factors which attributed for occurrence of the ascites, Hepatic (Raffan *et al.,* 2009; Buob *et al.,* 2011; Tantary *et al.,* 2013), Cardiac (Peden and Zenoble, 1982; Antran *et al.,* 2005; Kumar *et al*., 2011), Renal (Richter, 1996; Sood, 2000; Gines *et al.,* 2004) and Hypoproteinemia (Bexfield and Watson 2009; Saravanan *et al.,* 2012; Kamalakar *et al.,* 2016) had commonly been observed in dogs.

Common pathophysiological changes that are accountable for ascites development are associated with the etiologies of ascites which includes: organomegaly (Hepatomegaly, splenomegaly), abdominal effusions (Pre, intra and post hepatic reasons), neoplasia and infections. Common cardiac issues which end up into ascites encompass heartworms, congestive cardiomyopathy, congestive heart failure and congenital pulmonary stenosis. Hepatic illnesses which lead to ascites are liver insufficiency, chronic active hepatitis, cirrhosis and cholangitis. Other reasons are hypoproteinemia because of renal loss of protein in diseases like glomerulonephritis, amyloidosis, neoplasm, ruptured urinary bladder and haemorrhage from trauma (Peden *et al.,* 1982). Animals with abdominal distension, vomition, inappetence, pale or yellow mucous membrane and breathing distress must be suspected for ascites. The predominant clinical symptoms related with ascites are gradual expansion of abdomen, partial to

complete lack of appetite, lethargy, weight reduction and dyspnoea (Randhawa *et al.*, 1988).

On the basis of accumulation of the peritoneal fluid in abdomen ascites can be grade into three grades: Grade 1, grade 2 and grade 3. Small amount of excess fluid accumulation which can be detected only by ultrasonographic examination is graded as Grade 1 ascites, whereas moderate fluid accumulation with symmetrically distended abdomen is graded as Grade 2 ascites. Grade 3 ascites is the most commonly presented form at clinics manifested by huge accumulation of peritoneal fluid in abdomen along with marked abdominal distention (Moore *et al*., 2003).

On the basis of the nature of gathered fluid in peritoneal cavity ascites may be categorised aetiologically into two types broadly- transudative and exudative fluid (Kumar *et al.* 2005). The possible causes of transudative ascites in animals include one or more of the following factors - increased vascular hydrostatic pressure, vascular damage, increased tissue oncotic pressure, decreased vascular oncotic pressure (usually colloidal pressure), or blockage of lymph drainage, while exudative ascites is associated with inflammations and cancers of the peritoneum (Reynolds, 2000; Milne *et al.*, 2001; Kumar *et al.*, 2005).

Runyon *et al*. (1992) proposed a recent system of ascitic fluid classification which is based on the amount of albumin in the ascitic fluid compared to the serum albumin: known as Serum Ascites Albumin Gradient (SAAG). The SAAG is calculated by subtracting the albumin levels of ascitic fluid from that of the serum albumin levels. When the SAAG is >1.1 g/dl, the ascites is attributed to portal hypertension and if it's far < 1.1 g/dl it is attributed to different reasons which includes malignancies, pancreatitis etc where portal pressure remains normal (Runyon *et al.*, 1992).

The accurate technique and approach determines the nature of primary problem within the affected patient stricken by ascites (Ettinger and Feldman, 2000). Various diagnostic techniques like simple radiography (Kumar *et al.*, 2011; Bhadesiya *et al.*, 2015), ultrasonographic examination (Kumar and Srikala 2014; Bhadesiya *et al.*, 2015), have emerged as famous to diagnose ascites cases. Holistic technique of mixing all

different supportive techniques encompass cytological examination of peritoneal fluid, haemato-biochemcal examination of blood/serum samples and ascitic fluid, electrocardiographic investigation throw light on development and diagnosis of the clinical signs.

The treatment of ascites should be carried out on the basis of primary cause of the ascites cause via the means of administering drugs (Dabas *et al.,* 2011; Saravanan *et al.,* 2013; Kumar *et al.,* 2016). Abdominocentasis is endorsed in sufferers displaying intense breathing misery (Peden and Zenoble, 1982; Turkar *et al.,* 2009; Kumar *et al.,* 2016). Diuretics like frusemide and low sodium diets are used to deal with ascites. In case of low potassium levels, potassium sparing diuretics are used like spiranolactone is used. Aldosterone mediated sodium and water resorption in renal tubules may be decreased by inhibiting rennin angiotensin aldosterone system via way of means of the usage of drugs which includes ACE inhibitors, angiotensin receptor blocker. An excessive quality, low protein weight loss program along with the no salt diet is suited in small feeds and antibiotics for bacterial infection (Peden and Zenoble, 1982).

There is a dire need to undertake prompt diagnosis and timely management of canine ascites. Combined clinico-haemato bio- chemical analysis of ascitic fluid samples and data is essential for establishing quick diagnosis. With regards to the therapeutic management of canine ascites, it is essential to determine its origin. The present study was planned with the following objectives in view:

1. To study the prevalence of canine ascites
2. To study the clinical appraisal and haemato-biochemical changes in canine ascites
3. To formulate the suitable therapeutic regimen in canine ascites

Chapter-II

Review of Literature

CHAPTER-II

REVIEW OF LITERATURE

Since ancient times accumulation of fluid in abdominal cavity is known and it was Celsus who is credited with first describing the technique of paracentesis for aspirating fluid from peritoneal cavity, using a bronze tube with a flanged collar to drain fluid in 20 B.C.

2.1 PREVALENCE

Nottidge *et al.* (2003) examined a 10 month old Alsatian dog at Veterinary Teaching Hospital, Ibadan, Nigeria, showing the symptoms of inappetence and abdominal distension.

Nestor *et al.* (2004) in Illinois carried out a prospective study related to abdominal effusion of neoplastic and non-neoplastic origin of ascites in 15 dogs.

Tyagi *et al.* (2004) stated a case of old male non-descript dog with distended ventral abdomen in the college of Veterinary Clinics, Palampur, Himachal Pradesh.

Pradhan *et al.* (2008) detected one female Doberman pinscher aged eight years displaying the symptoms of inappetence and symmetrical expansion of abdomen in the Department of Clinical Veterinary Medicine, Nagpur.

Gualtieri *et al.* (2009) observed a case of sudden abdominal distension in the Department of Veterinary Medicine, Milano, Italy.

Turkar *et al.* (2009) examined a case of 2 month old male German shepherd puppy in the Department of Veterinary Clinical Medicine, Ludhiana, with complaint of abdominal distension.

Dixit *et al.* (2010) found 82 dogs to be suffering from primary hepatopathies and 58 were suffering from secondary hepatopathies in a prospective study conducted on 140 dogs all showing symptoms of hepatopathies and ascites.

Routray *et al.* (2010) examined eight year old Labrador bitch with depraved appetite, weakness, dyspnoea, distended ventral abdomen and also reported another case of enlargement of lower abdomen in seven and half year non-descript male dog in the Department of Veterinary Medicine, Bhubaneswar, Odisha.

Bhagat *et al.* (2011) reported eight cases of dogs suffering from ascites with difficult walking and dyspnoea in the Department of Clinical Medicine, Pantnagar.

Ihedioha *et al.* (2012) study revealed that the prevalence of canine ascites in Enugu State of Nigeria was 0.78% and also no significant difference was observed from the different clinics.

Pathak *et al.* (2012) recorded a five year old Labrador dog with abdominal distension and anorexia at Teaching Veterinary Clinical Complex, Ludhiana.

Saravanan *et al.* (2012) in I.V.R.I. Izatnagar, U.P. screened two thousand seven hundred fifty four dogs, of which ten (0.36%) dogs were suffering from ascites.

Tantary *et al*. (2013) reported that 16 dogs were suffering from ascites out of 49 dogs making prevalence of 32.65%.

Upadhyay *et al.* (2014) Found the prevalence of canine ascites in Jabalpur (MP) from December 2006 to July 2007 was 0.5%.

Kashyap *et al.* (2015) reported a case of ascites in 5 years old Saint Bernard dog at TVCC, Arawali Veterinary College, Sikar, Rajasthan with the complain of inappetence and abdominal distension along with dyspnoea and tachycardia. Clinical examination revealed normal rectal temperature, laboured respiration and tachycardia.

Kumar *et al.* (2016) examined a 9 year old German shepherd female dog presented with the history of inappetence and enlargement of abdomen and was diagnosed as ascites of hepatic origin resulting hypoproteinemia at TVCC, Bhubaneswar Odisha.

Padhi *et al.* (2016) reported the overall prevalence of ascites in dogs was 0.34% at Bhubaneswar, Odisha.

Behera *et al.* (2017) conducted research to study the incidences of ascites in dogs in Bhubaneswar, Odisha and found out prevalence to be 0.59%.

Dixit *et al.* (2018) screened dogs for ascites and found 31 dogs to be affected with ascites of different organ origin indicating the overall incidence of ascites to be 8.03 per cent.

Singh *et al.* (2019) reported that according to hospital records, total prevalence of ascites was 2.9% out of which1.4% was due to hepatopathy.

Samad (2019) observed female Spitz dog of two and half years old with the history of abdominal distension, on examination of ascitic fluid revealed clear white fluid (pure transudate) which is mainly hepatic origin resulting from portal hypertension and hypoproteinemia.

Dhillon *et al.* (2020) examined three months old female Beagle pup which was presented with a history of enlarged abdomen, inappetence and weakness since two weeks. Clinical examination revealed fever, fluid thrill on palpation of abdomen, dyspnoea, dehydration, rough body coat, pale mucous membranes and tachycardia was evident. The puppy was diagnosed with ascites of hepatic origin resulting hypoproteinemia.

Ghosh *et al.* (2020) reported a case of ascites in a two and half months old Doberman pinscher male pup, which was confirmed to be suffering from ascites due to babesiosis after proper physical and haemato-biochemical examination.

2.1.1 Age and Sex Wise Prevalence

Nottidge *et al.* (2003) reported a ten month old Alsatian pup with clinical symptoms of depressed appetite and distended abdomen at Veterinary Teaching Hospital, Ibadan, Nigeria.

Tyagi *et al.* (2004) studied a case of non-descript male dog aged eleven and half years with the symptoms of bilateral distension of ventral abdomen.

Pradhan *et al.* (2008) recorded symmetrical enlargement of abdomen in a female Doberman aged eight years.

James *et al.* (2008) mentioned incidence of ascites in dogs ranged in between the age limit from 16 weeks to 9 years (mean, 3.12 years; median 2.0 years), Twelve (70.5%) dogs were 4 years of age or younger at time of presentation. He also reported incidences of ascites in dogs and found one sterilised male, six intact males, six sterilised females and four intact females to be suffering from ascites of different origins.

Gabriel (2009) observed that Young American and English Cocker spaniel male dogs were at risk of chronic hepatitis with rapid progression of cirrhosis, chronic idiopathic and females were predisposed to chronic idiopathic and copper associated hepatitis.

Gleich *et al.* (2009) encountered a four month old intact mixed-breed male puppy suffering from ascites due to infectious canine hepatitis virus infection.

Gualtieri *et al.* (2009) observed two year old male Labrador retriever dog which showed the symptoms of sudden abdominal distension and was diagnosed for vascular hepatic haematoma.

Turkar *et al.* (2009) detected abdominal distension in a two month old German shepherd pup.

Routray *et al.* (2010) encountered distended ventral abdomen and enlarged abdomen in an eight years old Labrador bitch and a non-descript male dog of seven and half years, respectively.

Das (2012) found more number of cases (48%) of ascites due to hepatic origin is in the age group of more than 5 years, 28% in the age group of 4-5 years and 24% less than 4 years of age and sex wise prevalence was more in male (68%) than in female (32%).

Pathak *et al.* (2012) examined a five years old Labrador showing the symptoms of abdominal enlargement.

Saravanan *et al.* (2012) recorded ascites in five dogs aged between 4-5 years followed by three dogs between 2-4 years and two dogs above 5 years. He also mentioned that the ascites was higher in males (60%) as compared to females (40%).

Saravanan *et al.* (2014) mentioned that overall sex wise distribution shows that male dogs (54.2%) had higher prevalence of ascites when compared to females.

Ihedioha *et al.* (2013) reported highest prevalence in dogs of 3 years and above age (71.4%) followed by the dogs in the age group 1- 2.9 years (21.4%) and least in dogs <1 year of age (7.1 %).

Kumar *et al.* (2014) observed eight years old female Labrador was presented with the history of persistent distended abdomen for over a period of time.

Upadhyay *et al.* (2014) mentioned age wise prevalence of ascites reported that the ailment was 8.1% in the age group of less than 1 year, 10.52 % in the age group of 1-5 year and 12.35% in the age group >5 year. Sex wise prevalence was higher in female (60.71%) than male (39.28%).

Kashyap *et al.* (2015) reported a case of ascites in 5 years old Saint Bernard dog with the complaint of inappetence and abdominal distension along with dyspnoea and tachycardia.

Zoia *et al.* (2017) mentioned a study of 70 dogs with abdominal distention, where age wise prevalence was found in the range between 1–15 years, sex wise there were 49 males (44 sexually intact and 5 neutered) and 21 females (13 sexually intact and 8 spayed) suffering from ascites.

Kumar *et al. (*2016) reported a nine year old German shepherd dog with the history of distended abdomen, dyspnea and dehydration which was later diagnosed to be suffering from ascites of hepatic origin.

Padhi *et al.* (2016) reported that dogs aged 3 years and above (50%) were highly susceptible to ascites, followed by 1-2.9 years (41.67%) age group, and lowest prevalence was recorded in <1 year age group (8.33%) and ascites was more in females (75%) than male dogs (25%).

Behera *et al.* (2017) studied the incidences of canine ascites in and around Bhubaneswar and found age wise incidence were slightly higher in > 6 years (36.21%) age group followed by 0-3 years (34.48%) and 3-6 years (29.31%) and sex wise ascites was predominantly higher in females (58.63%) in comparison to males (41.37%)

Sharma *et al.* (2017) reported the complaint of marked enlargement of abdomen, difficult breathing, off feed and irregular and decrease urination in a 5 months old non-descript pup.

Alsaad *et al.* (2018) mentioned about a male Mullinoise breed K9 dog aged 5.7 years with the history of open mouth breathing, lethargy, reduced feed intake and gradual abdominal distention which was later confirmed to suffer from ascites due to Congestive Heart Failure.

Dixit *et al.* (2018) observed age wise incidence of ascites and found 14 dogs were in the age between 5-10 yrs, followed by 8 dogs in the age between 1-5 years, 6 dogs were >10 years, and 3 dogs <1 year of age. Sex wise incidence revealed to be 70.96% in male dogs and 29.04% in female dogs.

Singh *et al.* (2019) carried out study to record the canine ascites prevalence in and around Tarai region Uttarakhand. Highest prevalence was observed in >5 years old dogs. Male dogs have shown higher prevalence as compared to female dogs.

Dhillon *et al. (*2020) reported a three months old female Beagle pup was presented with a history of enlarged abdomen, inappetence, weakness, abdominal distension, dyspnoea and dehydration. The pup was diagnosed with ascites of hepatic origin resulting in hypoproteinemia.

Ghosh *et al.* (2020) discussed a case study of two and half months old male Doberman pinscher pup presented with the history of persistent fever, progressive abdominal distension, inappetence, vomition, and depression was confirmed to be suffering from ascites.

2.1.2 Breed Wise Prevalence

Nottidge *et al.* (2003) observed a ten month old Alsatian puppy with complaint of depressed appetite and distended abdomen at Veterinary Teaching Hospital, Ibadan, Nigeria.

Tyagi *et al.* (2004) detected a case of ascites in a non-descript male dog aged eleven and half years showing bilateral distension of ventral abdomen at College of Veterinary Science, Palampur, Himachal Pradesh.

Pradhan *et al.* (2008) observed symmetrical enlargement of abdomen in one Doberman bitch of eight years old.

Gabriel *et al.* (2009) reported hereditary copper associated hepatitis in various breeds of dogs i.e., Bedlington terrier, West Highland white terrier, Sky Terrier, Doberman, Dalmatian and Labrador, whereas Young American and English Cocker spaniel breeds were at risk of chronic hepatitis with rapid progression to cirrhosis. Chinese Shar Pei breed dogs were predisposed to hepatic amyloidosis.

Gualtieri *et al.* (2009) reported sudden abdominal distension in a two year old Labrador breed male dog with vascular hepatic haematoma.

Turkar *et al.* (2009) identified a case of abdominal distension in a two month old German shepherd male pup.

Routray *et al.* (2010) detected distended ventral abdomen in an eight years old Labrador bitch along with enlargement of lower abdomen in a seven and half years old non-descript male dog.

Pathak *et al.* (2012) observed a five year old Labrador dog showing the symptoms of abdominal enlargement.

Saravanan *et al.* (2012) found in his study that the incidence of ascites was more in spitz breed followed by Labrador and non- descript dogs.

Das (2012) found in his study that occurrence of ascites due to hepatic insufficiency is more in large-sized breeds viz. Labrador, Doberman and German shepherd.

Ihedioha *et al.* (2013) found Alsatian-Rottweiler crosses had a significantly ($p<0.05$) higher prevalence (50%) than all other breeds.

Saravanan *et al.* (2014) reported that Spitz dogs had more incidences of ascites followed by Labrador Retrievers.

Kashyap *et al.* (2015) reported a case study of ascites in 5 years old Saint Bernard dog with complaint of inappetence and abdominal distension along with dyspnoea and tachycardia.

Zoia *et al.* (2017) in his study found eighteen of the dogs were crossbred, and 52 were purebred (13 German shepherds, 5 Cane Corsos, 5 Labrador retrievers, 3 Boxers, 3 Cocker spaniels, 3 Yorkshire terriers, 2 Maltese, 2 Siberian huskies, and 1 of each of the following 16 breeds: American Staffordshire terrier, Beagle, Bernese mountain dog, Border Collie, Doberman, Fox Terrier, Great Dane, Greyhound, German Pinscher, Rottweiler, Toy Poodle, West Highland White Terrier, and Whippet) which were suffering from one or the cause of ascites.

Kumar *et al.* (2016) reported a case study of a nine year old German shepherd female dog presented to the TVVC, Bhubaneshwar, Odisha with the history of inappetence and enlargement of abdomen. The bitch was diagnosed as ascites of hepatic origin resulting from hypoproteinemia.

Regmi and Shah (2017) reported a male German shepherd dog of 11 months, which was presented with the history of abdominal swelling, respiratory distress, lethargy, anorexia and weakness.

Behera *et al.* (2017) discussed that the breed wise incidence was found to be higher in Labrador retriever (41.37%) followed by German spitz (18.9%), German shepherd (17.24%), Dalmatian (5.17%), Golden retriever, Rottweiler (3.44%), Dachshund (3.44%), Boxer (1.72%), Cocker spaniel (1.72%), Pomeranian (1.72%)

Alsaad *et al*. (2018) reported a male, Mullinoise breed K9 dog, 23 kg weight and of 5.7 years old, had been brought with history of acute illness of 1-4 days' duration, Dyspnea with open-mouth breathing sometimes, lethargy, reduced food intake, an obvious distended abdomen developed gradually, reduced exercise tolerance, coughing, and occasional weight loss with fainting only at exercise sometimes. Congestive heart failure was suspected for cause of ascites.

Dixit *et al.* (2018) mentioned breed wise incidence of ascites in dogs indicating higher incidence 58.06% in Mongrels (non-descript dogs) followed by Spitz 25.80%.

Samad (2019) reported 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 (13.37%), Mongrels (10%) and least in Alsatian (6.66%).

Singh *et al.* (2019) revealed that out of 18 cases of ascites, seven were in the dogs of breed Spitz showing highest prevalence of 38.88% among the breeds followed by five in Labrador retriever (27.77%), two each of German shepherds and Mongrel (11.11%) and one each of Golden Retriever and Doberman pinscher (5.55%).

Dhillon *et al.* (2020) reported a three months old female Beagle pup with a history of enlarged abdomen, inappetence and weakness since two weeks. The pup was diagnosed as ascites of hepatic origin resulting hypoproteinemia.

2.1.3 Etiology

Parker (2002) correlated acute hypoalbuminic conditions albumin loss, decreased production, inflammatory conditions as factors for ascites in dogs which could be denoted by decreased A/G ratio.

Hall (2005) stated that decreases in serum albumin levels leads to formation of a pure transudate in the extracellular space and thus resulting in ascites in small animal patients because albumin was responsible for plasma colloid osmotic pressure.

Antran *et al*. (2005) reported that in congestive heart failure, systemic congestion occurs due to impaired blood flow returning to the heart from the body. Blood backs up and fluid leaks through the parts of the body result in ascites in dogs.

Ettinger and Feldman (2005) reported decreased production of plasma proteins due to liver cirrhosis, which in turn results in ascites in dogs.

James *et al.* (2008) carried out a retrospective analysis of 17 dogs with ascites due to pre-sinusoidal portal hypertension, and found idiopathic hepatic fibrosis or canine chronic hepatitis as the primary cause in the majority of cases.

Bexfield and Watson (2009) studied ascites in dogs and found to be caused by portal hypertension and in some animals due to hypoalbuminemia.

Hou and Sanyal (2009) stated that some of the common etiological factors for the development of ascites were cirrhosis, portal hypertension, increased intrahepatic resistance to blood flow and are compounded by splanchnic vasodilation in dogs.

Raffan *et al*. (2009) stated that ascites was considered to be a common complication of chronic hepatitis in dogs.

Turkar *et al.* (2009) reported a case of ascites in 2 months old German shepherd due to ancylostomiasis with complaint of abdominal distension, loose faeces and weakness.

Bhatt *et al.* (2011) reported a case of ancylostomiasis associated ascites and its successful management with fenbendazole, lasix and other supportive therapy.

Buob *et al*. (2011) reported to consider portal hypertension, increased vascular resistance in the portal circulation and increased portal venous blood flow as etiological factors for ascites in dogs.

Kumar *et al*. (2011) confirmed the cause for ascites in dogs to be congestive heart failure after performing 2D echocardiographic study of heart which revealed dilated right ventricle both on B and M modes, mitral and tricuspid valve insufficiency recorded and colour Doppler.

Das (2012) diagnosed 41.6% dogs had ascites due to hepatic origin, among 60 ascitic dogs he examined.

Saravanan *et al.* (2012) mentioned that discrimination among the ascites of hepatic origin than the other ascites was easy and better on the basis of serum-ascites albumin gradient in dogs. SAAG >1.1 g/dl is suggestive of the presence of portal hypertension due to chronic liver disease and findings correlated with ultrasonography of hyperechoic/ cirrhotic ascites dog and SAAG test can be used as a screening test in ascites due to chronic liver disease.

Ihedioha *et al.* (2013) reported that most of the ascites cases were largely due to congestive heart failure (50% of cases); other causes were cirrhotic liver disease (14.3%), chronic active hepatitis (21.4%) and kidney disease (14.3%).

Srivastava and Syed (2013) mentioned most common causes of ascites in dogs were malnutrition, nephrotic syndrome, ancylostomiasis, protein losing enteropathy and abdominal neoplasia of viscera conditions.

Tantary *et al*. (2013) attributed hepatic origin was the most common etiological factor for ascites in dogs.

Kashiide *et al.* (2014) reported canine peritoneal larval cestodiasis in dogs with ascites confirmed by abdominal ultrasound and X-ray imaging, wherein a number of mesocestoides parasites observed in the ascitic fluid.

Bota *et al*. (2016) reported that Yorkshire Terriers dogs were predisposed to protein losing enteropathy, and this was found in more than 40 % of cases of ascites. This condition was characterised by abdominal effusion, histologically by crypt lesions, lacteal dilation and inflammation.

Kamalakar *et al*. (2016) opined that hypoalbuminemia which was due to reduced plasma and tissue oncotic pressure was most common cause of ascites in dogs.

Beher *et al.* (2017) reported that out of the 58 cases, cardiac origin in 7, hepatic origin in 21, renal origin in 9, both renal and hepatic origin in 9, genital origin in 6 and parasitic origin in 6 (10.34%) instances had been evident.

Zoia *et al*. (2017) reported exudative ascites in animals which was due to conditions like pancreatitis, bile peritonitis, malignant disease of peritoneum and septic peritonitis in dogs.

Dixit *et al*. (2018) mentioned that out of 31 dogs suffering from ascites, 18 (58.06%) dogs showed hepatic origin.

Ghosh *et al* (2020) reported a case study ascites in a two and half months' old Doberman pinscher male pup, which was suffering from ascites due to babesiosis infection.

2.2 DIAGNOSIS

2.2.1 Clinical Signs

Vijaykumar *et al.* (2002) diagnosed ascites in dogs after physical palpation of abdomen by tapping on one side while placing palm of other hand flat on opposite side and felt a fluid wave moving across the abdomen.

Nottidge *et al.* (2003) reported dry, rough and lustre less hair coat in a 10 months old Alsatian pup with depressed appetite, bilateral mucopurulent ocular discharge and congested ocular mucous membrane.

Tyagi *et al.* (2004) recorded normal heart rate, respiration rate, rectal temperature and presence of fluid thrill on percussion in a non-descript dog showing bilateral distension of ventral abdomen.

Kruth (2005) suggested clinical examination for diagnosis of abdominal effusion by abdominal palpation in conditions like hepatomegaly, splenomegaly. Tachypnea also occurs in animals suffering from excessive abdominal fluid accumulation. He also suggested the use of diagnostic approach like jugular distention or pulsation, murmurs and arrhythmias to diagnose ascites due to cardiac origin.

Leduc and Troyer (2007) reported ascites as a complicating feature of many diseases of the liver and peritoneum and commonly lead to dyspnoea.

Pradhan *et al.* (2008) encountered the clinical symptoms of inappetence, symmetrical enlargement of abdomen, pale mucous membrane, dyspnoea, tachycardia, and presence of fluid thrill on tactile percussion and temperature of 103°F in Doberman dog with ascites.

Gualtieri *et al.* (2009) diagnosed a case of canine vascular hepatic haematoma in a dog with sudden abdominal distension.

Palma *et al.* (2009) encountered the symptoms of cardiac insufficiency in one dog and icterus mucous membrane and abdominal distention in another dog with hepatic capilariasis.

Turkar *et al.* (2009) observed poor health condition, rough hair coat, dullness, depression, laboured breathing, sub-normal body temperature and bilateral distension of ventral abdomen with presence of fluid thrill on percussion in a German shepherd pup suffering from ascites.

Dixit *et al.* (2010) examined one hundred dogs suffering from hepatopathies including eighty dogs and fifty eight were primary and secondary hepatopathies, respectively with the clinical signs of decreased appetite, anorexia, vomition, weight loss, ascites and bilateral hind limb oedema.

Routray *et al.* (2010) examined an eight Labrador bitch showing the symptoms of decreased appetite, weakness, dyspnoea, distended ventral abdomen and clinical examination revealed body temperature to be 102.6°F, pulse and respiration rates were 86/ minutes and 26/minutes, respectively. They also examined another non-descript male seven and half years old dog with symptoms of inappetence, weakness, extreme depression and clinical examination revealed body temperature to be103°F.

Bhagat *et al.* (2011) studied eight cases of dogs with symptoms of distended abdomen, difficult on walking, dyspnoea, arching back, with body temperature between 102 - 104°F, pulse rate (110/min), respiration rate (30-42/min) and heart rate (120/min).

Kumar *et al*. (2011) stated that the clinical signs of ascites in dogs were loss of appetite, vomiting, respiratory distress, lethargy, and persistent distended abdomen and in few cases distended jugular vein, cough, cyanotic tongue, seizers and syncope.

Saravanan *et al.* (2012) studied the cases of ascites in a dog and on clinical examination revealed presence of fluid thrill on palpation.

Pai *et al.* (2012) reported symptoms found on the physical examination of ascitic dogs were irregular pulse, present in 37.5% of the cases dilated Cardiomyopathy, femoral pulse was weak in 75% of the cases, pale mucous membrane was seen in 25% of the cases, thoracic auscultation revealed tachycardia (25%) and pulmonary crackles (75%), Pulse deficit was present in 50% of the cases, Ascites was confirmed by tactile percussion.

Pathak *et al.* (2012) observed the symptoms of abdominal enlargement and found blood tinged fluid in the peritoneum after paracentesis of abdominal cavity in a Labrador dog.

Das (2012) observed abdominal distension in all the cases. Other clinical signs were anorexia, pale mucous membrane and pyrexia in 76%, 54% and 64% cases, respectively.

Meindel and Pohiman (2013) observed clinical signs like weakness, lethargy, distended abdomen, jaundice mucous membranes, normal temperature and increased heart and respiratory rates on physical examination of ascitic dog.

Saravanan *et al*. (2013) recorded the clinical signs like anorexia, lethargy, accumulation of fluid in the abdomen in dogs suffering with ascites.

Tantary *et al*. (2013) observed anaemia, weight loss, diarrhoea and jaundice as clinical signs in dogs with ascites and hepatic encephalopathy.

Kumar and Srikala (2014) mentioned some of the common clinical signs like abdominal distention, respiratory distress, lethargy, cyanotic tongue and cough in a dog with ascites due to right heart failure.

Bhadesiya *et al*. (2015) reported enlarged abdomen, respiratory distress, thickening of skin folds, reduced feed intake, reduced urine output, normal defecation as clinical signs in a dogs suffering from ascites.

Elhiblu *et al*. (2015) observed clinical signs like inappetance, halitosis, melena, hematochezia, polyuria, dehydration, icterus, weight loss and abdominal distension in dogs with cirrhosis of liver and ascites.

Kashyap *et al.* (2015) reported a case of ascites in 5 years old Saint Bernard dog with complaint of inappetence and abdominal distension along with dyspnoea and tachycardia.

Kumar *et al.* (2016) mentioned the history of inappetence and enlargement of abdomen in 9 year old German shepherd dog and on physical examination of ascitic dogs dyspnea, dehydration and slightly pale mucus membrane, rough, lustreless body coat and abdominal distention along with hollowness at the flank region were observed. Undulating movements of the fluid were present on taping the abdomen.

Regmi and Shah (2017) examined a male German shepherd dog of 11 months with the history of abdominal swelling, respiratory distress, lethargy, anorexia and weakness. Physical examination revealed dyspnea, pale mucous membrane, and undulating movement (thrills) of fluid on tapping the abdomen revealing ascites.

Sharma et al. (2017) reported the physical examination of 5 month old ascites suffering non descript dog which revealed rectal temperature to be 101.2 F, pale mucous membrane, melena, increase pulse and heart rate with low amplitude, respiratory distress, increase capillary refill time along with Dry, rough and lustreless body coat, sunken eyes and prominent rib cage. Abdomen was enlarged and undulating fluid movements present on abdominal palpation.

Lakshmi *et al.* (2018) reported that ascitic dogs were presented with the clinical signs of anorexia, ascites, jaundice, pale mucous membranes, vomition, lethargy, polyuria and polydypsia or other manifestations suggestive of hepatobiliary disorders were selected.

Phom *et al.* (2019) stated that abdominal distensions, fluid wave test, inappetence, dyspnoea, exercise intolerance, pale mucous membrane, icterus, melena, vomition, peripheral edema and tachycardia had been the predominant scientific symptoms and symptoms discovered in ascites cases.

Singh *et al.* (2019) found in his study that all the 18 dogs showed abdominal distension and fluid thrill on percussion followed by clinical signs such as inappetence (15/18), pale mucous membrane (14/18), lethargy (14/18), respiratory distress (8/18), diarrhea (5/18), vomiting (4/18) and limb edema (1/18).

Dhillon *et al.* (2020) mentioned clinical examination of a three months old female Beagle pup revealed fever, fluid thrill on palpation of abdomen, dyspnoea, dehydration, rough body coat, pale mucous membranes and tachycardia was evident.

2.2.2 Haematological Study

Vijaykumar (2002) recorded decrease in RBCs level in anemia. Neutrophilia along with leukocytosis was commonly observed in patients suffering from systemic infection.

Nottidge *et al*. (2003) observed progressively decrease in PCV, Hb and RBC values during management and observed anaemia of normochromic, normocytic and non-regenerative nature. Decreased liver enzymes were suggestive of progressive liver deterioration in dogs.

Tyagi *et al.* (2004) studied the haematological status of non-descript male dog suffering with ascites due to hepatic neoplasm which revealed the presence of mild anaemia, haemoglobin was 10.0gm%, PCV was 30.0% and there was neutrophilia.

Pradhan *et al.* (2006) found Hb 7.91gm%, total leukocyte count 13500/cu mm and DLC count revealed neutrophil 84%, lymphocyte 13%, eosinophils 2% and monocytes 1% in a haematological study of an eight years Old Doberman bitch.

Pradhan *et al.* (2008) noticed slight decrease in Hb levels, leukocytosis with increase in neutrophil count in ascitic dogs.

Turkar *et al.* (2009) studied a case of two month old male German shepherd pup with a complaint of abdominal distension and haemogram values revealed normochromic normocytic anaemia along with neutrophilia.

Routray *et al.* (2010) reported two ascitic cases in dogs due to hepatitis. In one case, the haematology revealed the Hb 7.6gm%, total erythrocytes count $2.4x10^6$/ cu mm; total leucocytes count $12.0x10^3$/ cu mm, neutrophil 81%, lymphocyte 14%, Monocytes 2% and eosinophils 3%. In another case, the haematology statuses were Hb 8.0%, total erythrocyte count $2.8\times l0^6$/cu mm, total leukocyte count $13.5\times l0^3$/cu mm, neutrophil 84%, lymphocyte 13%, eosinophils 2% and monocytes 1%.

Das (2012) found significant decrease in Hb, PCV and TEC values whereas significant increase in TLC values and neutrophil counts.

Chaturvedi *et al.* (2013) reported marked decrease in haemoglobin concentration and RBC count in ascitic dogs with normocytic, hypochromic and non-degenerative type of anaemia. Higher TLC values resulting in neutrophilia, lymphopenia, eosinophilic and monocytic granulopenia were also reported in dogs.

Meindel and Polhman (2013) recorded mild lymphopenia and moderate thrombocytopenia in complete blood count of an ascitic dog.

Tantary *et al.* (2013) observed anaemia, thrombocytopenia and increased clotting time in ascitic dog.

Saravanan *et al.* (2014) reported that significant decrease in HB, PCV, RBC count, TLC values, neutrophil and basophile count whereas decreased lymphocytic count in ascitic dog.

Tantary *et al.* (2014) mentioned non-significant decrease in mean value of HB, PCV, total erythrocyte count and platelet count in acute hepatitis and significant decrease in values in chronic hepatitis as compared to healthy dogs.

Padhi (2016) reported that Hb and PCV value in ascetic dogs with renal disorder showed significant decrease, whereas significant increase was observed in neutrophil percentage along with significant decrease in platelet count was in all groups when

compared to control. RBCs values recorded were significantly lower in dogs suffering from renal failure as well as ascites due to both heart and liver involvement.

Elhiblu *et al.* (2015) reported the mean values of Hb, PCV, lymphocytes, MCV, MCH and platelets were significantly lower in liver cirrhosis group than control group, while TLC, neutrophils and MCHC were significantly higher than the control group. Anemia was observed in all the cases, thrombocytopenia in two and thrombocytosis in one case. Toxic changes in neutrophils were observed in four animals (mild to moderate in three and severe in one). Left shift was mild to moderate in four and marked in two animals.

Regmi and Shah (2017) reported a haematological study of dogs suffering from ascites which showed an increase in neutrophils count, while there was decrease in erythrocytes count and haemoglobin concentration.

Sharma *et al*. (2017) observed haematological findings of an ascitic dog which revealed values of different parameters such as haemoglobin 7.4 gm%, Total Red Blood Cells 5×10^6/ µL, PCV 25%. Total Platelet count 557×10^6/µL, Total Leukocyte Count 30.3×10^3 /µL and DLC- neutrophils 79%, eosinophils 1%, monocytes 3% and lymphocytes 17%.

Phom *et al.* (2019) revealed that the haemato-biochemical analysis revealed anemia, neutrophilic leukocytosis, hypoproteinaemia, hypoalbuminemia, hyponatremia, hypochloraemia, hyperbilirubinemia, increased alanine aminotransferase (ALT), alkaline phosphatase (ALP), blood urea nitrogen (BUN) and creatinine level in all the ascitic dogs.

Singh *et al.* (2019) reported that in haematological analysis of dogs suffering from ascites there was significant decrease in haemoglobin concentration, PCV, lymphocyte and TEC values. No significant change was found in eosinophilic and basophilic count.

Dhillon *et al.* (2020) reported haematological findings which revealed anemia, neutrophilia along with thrombocytopenia.

2.2.3 Biochemical Study

Vijaykumar (2002) reported low levels of Albumin and cholesterol with normal or high globulin levels in liver diseases along with elevated levels of AST, ALT, ALP, Bilirubin and bile acid in renal impairment along with elevated levels of BUN and creatinine.

Nestor *et al.* (2004) conducted prospective study of 15 dogs for biochemical parameter in abdominal effusion of neoplastic and non neoplastic origin and found the dogs with neoplastic group had significant lower glucose concentration and higher lactate level in their abdominal fluid compared to the dogs in the non-neoplastic group indicating low glucose and high lactate in abdominal effusion.

Pradhan *et al.* (2008) reported clinical pathological study of eight years old doberman female where the biochemical analysis of serum revealed blood glucose 70 mg/dl, serum urea 82 mg/dl, serum creatinine 0.48 mg/dl and SGOT levels 134.1 U/L.

Das (2012) found significant increase in ALT, AST and non significant difference in A/G ratio and decrease in blood glucose, serum total protein, serum albumin and serum globulin in ascetic dogs due to hepatic insufficiency.

Saravanan *et al*. (2012) reported increased serum- ascites albumin gradient more than 1.1 g/dl which is suggestive of high gradient ascites, portal hypertension resulted in an abnormally increased hydrostatic pressure between portal bed and the ascitic fluid in dogs.

Chaturvedi *et al*. (2013) recorded noticeable decrease in serum albumin, globulin, A/G ratio and total proteins and marked increase in ALT and AST levels concluded that ascites due to severe liver damage in dogs.

Ihedioha *et al.* (2013) reported that dogs with chronic liver disease had a significantly lower serum activity of ALT, AST and serum urea levels, and also significantly higher serum activity of ALP, while dogs suffering from chronic active hepatitis had a contrasting significantly higher serum activity of ALT, AST and ALP when compared to the controls. Dogs with congestive heart failure were characterised by

significantly higher serum AST activity, while dogs with kidney disease were characterised by significantly higher serum levels of creatinine and urea.

Meindel and Pohlman (2013) recorded mild increased ALP levels 269 IU/L, moderately increased ALT levels 218 IU/L and total bilirubin 2.3 mg/dl whereas mild decrease in total protein levels 4.6 g/dl and globulin levels 1.9 g/dl in ascitic dog.

Tantary *et al*. (2013) diagnosed ascites which was caused by hypoproteinemia and hypoalbuminemia in dogs.

Saravanan *et al*. (2014) recorded significant increase in ALT, AST, ALP, total bilirubin and prolonged prothrombin time and decrease in total protein and albumin level in ascitic dogs.

Bhadesiya *et al*. (2015) reported increased levels of liver specific enzymes in ascites cases which correlated with ultrasonographic changes on liver parenchyma, i.e., indicative of hepatic involvement in dogs.

Kamalakar *et al*. (2016) observed increased levels of ALT, AST, ALP, GGT, creatinine and bilirubin and concluded that ascites in dogs was of hepatic origin.

Padhi (2016) reported higher AST, ALT and ALP activities in the dogs suffering from ascites of liver, kidney and heart and liver involvement as compared to control. Total protein and albumin levels were significantly lower in all the ascitic dogs. Levels of BUN and creatinine were significantly higher in the ascitic dogs with renal involvement.

Regmi and Shah (2017) reported the biochemical analysis which resulted in an elevated SGOT, SGPT level but decrease in total protein level.

Sharma *et al.* (2017) mentioned the biochemical findings in a 5 month old pup which revealed blood sugar 44mg/dl, total protein 3.4gm/dl, albumin 1.25gm/dl, ALT 155 IU/L, AST 128 IU/L, serum creatinine 0.2mg/dl, BUN 5mg/dl, GGT 11 IU/L, ALP 150 IU/L, total bilirubin 0.1mg/dl and serum cholesterol 73mg/dl.

Phom *et al.* (2019) mentioned the haemato-biochemical analysis that revealed anemia, neutrophilic leukocytosis, hypoproteinaemia, hypoalbuminemia, hyponatremia,

hypochloraemia, hyperbilirubinemia, increased alanine aminotransferase (ALT), alkaline phosphatase (ALP), blood urea nitrogen (BUN) and creatinine level in all the ascitic dogs.

Singh *et al.* (2019) stated that major serum biochemical changes observed in ascitic dogs were hypoproteinemia, hypoalbuminea and hypoglobulinemia with increased ALT, AST, ALP, GGT, TLC, BUN and creatinine levels.

Dhillon *et al.* (2020) observed serum biochemical parameters evinced increased levels of ALT (260 IU/L), AST (186 IU/L) and ALP (425 IU/L) but decreased in total protein (3.2 g/dl) and blood glucose (52 mg/dl) level suggesting hepatic involvement.

2.2.4 Analysis of Ascitic Fluid

Vijaykumar (2002) reported that ascitic fluid analysis is vital as specific diseases have specific disease features which facilitates in differential diagnosis of the cause of the ascites.

Alleman (2003) discovered in a normal animal, the peritoneal space consists of small amount of fluid (less than 1 mg/kg body weight), which moistens the opposing surface and serves to lessen friction among the abdominal organs. And that the effusions are abnormal or increased accumulation of fluid in any of the body cavity, which was lined by mesothelial cells.

Mondal *et al.* (2012) stated that ascitic fluid can be used for the diagnosis of various diseases such as clear yellow in ruptured bladder, yellow and turbid acute diffuse peritonitis, red tinged in infarction of gut wall, milk coloured peritoneal fluid may indicate carcinoma, lymphoma, tuberculosis or infection, blood stained fluid due to traumatic tap, peritoneal carcinoma, and ascitic fluids with homogenous blood stained indicate malignancy, pancreatitis, intestinal infarction and tuberculosis, bloody fluid may indicate tumour or trauma, bile stained fluid may indicate gall bladder problems.

Jain *et al.* (2013) concluded that presence of complete transudate type of abdominal fluid was indicative of ascites of non-infectious origin in nature in dogs.

Vijayakumar *et al.* (2013) found no epithelial cells, pus cells in ascitic fluid examination and gram staining of fluid did not reveal presence of bacteria in dogs with ascites.

Jain *et al.* (2014) suggested that cytological examination of aspirated ascitic fluid as transudate or exudate based on protein content in the fluid in dogs.

Kumar and Srikala (2014) observed colourless, transparent and odourless peritoneal fluid with an average protein content of 2.8 g/dl, cell matter of 1350 cells/c. mm and specific gravity to be 1.010 which have been suggestive transudate nature in dogs.

Saravanan *et al.* (2014) analysed ascitic fluid which revealed clear/transudate in 68 dogs, clear/ straw coloured in 2 dogs and clear reddish yellow in 2 dogs. Cytological examination of fluid revealed mesothelial cells, lymphocytes, monocytes and neutrophils. Various parameters like specific gravity 1.014, total nucleated cell count 388.9 cells/ cu mm, total protein 1.96 g/dl and SAAG 1.20 g/dl were suggestive of transudative ascites.

Bhadesiya *et al.* (2015) opined that examination of ascitic fluid provide useful information on status of the condition as well as prognosis of the condition in dogs.

Elhiblu *et al.* (2015) mentioned that fine-needle aspiration biopsy of liver and cytological examination of the ascitic fluid was not fruitful in the diagnosis of liver cirrhosis in dogs.

Kumar *et al.* (2016) examined specific gravity, colour and cytology of ascitic and confirmed it as transudate with clear, pale yellow colour, protein levels 2.2 g/dl and specific gravity of 1.014 in a dog.

Zoia *et al.* (2017) suggested ascitic fluid of dogs has evidence of coagulation activation and fibrinogenolytic/fibrinolytic activity and that this phenomenon occurred independent of the underlying mechanism that leads to the formation of ascites.

Sharma *et al.* (2017) reported that the peritoneal fluid examination revealed pure transudate fluid with specific gravity of 1.015 with clear appearance and absence of any

leucocytes, blood cells and protein whereas traces of glucose were present which is typically associated with hepatobiliary disorders.

Phom *et al.* (2019) examined the ascitic fluid and found the values of specific gravity 1.027, total leukocyte count (8054 cells/ml), total protein (3.5 g/dl), albumin (0.1 g/dl) and SAAG (0.60 g/dl). Cytological ascitic fluid revealed the presence of few mesothelial cells, lymphocytes, monocytes and neutrophils in maximum of the ascitic dogs.

2.2.5 Serum Albumin Ascitic Gradient (SAAG)

Beg *et al.* (2001) and Burgess (2004) stated that >1.1 g/dl of SAAG are at once associated with high portal pressure.

Burgess (2004) advised ascites additionally may be categorized as "high gradient" and "low gradient" based on the Serum ascites albumin gradient (SAAG). If it is >1.1g/dl, it is called high gradient and low gradient ascites if <1.1 g/dl.

Tarn and Lapworth (2010) found that oncotic pressure gradient between the vascular bed can be reflected by SAAG and elevated gradient (≥1.1 g/dl) usually being associated with increased portal pressure, whereas a low gradient (<1.1 g/dl) is associated with conditions where ascites is not related to portal hypertension

Mondal *et al.* (2012) reported Serum ascitic albumin gradient is calculated by subtracting the albumin concentration of the ascetic fluid from the albumin concentration of a serum specimen obtained on the same day.

Saravanan *et al. (*2012) found mean±SE of serum ascites albumin gradient (SAAG) are 1.793±0.185. SAAG >1.1 g/dl is suggestive of the presence of portal hypertension and can be used as a screening test in ascetic due to chronic liver disease.

Saravanan *et al.* (2014) analysed ascitic fluid which revealed clear/transudate in 68 dogs, clear/ straw coloured in 2 dogs and clear reddish yellow in 2 dogs. Cytological examination of fluid revealed mesothelial cells, lymphocytes, monocytes and neutrophils. Various parameters like specific gravity 1.014, total nucleated cell count 388.9

cells/cmm, total protein 1.96 g/dl and serum ascites albumin gradient 1.20 g/dl were suggestive of transudative ascites.

Regmi and Shah (2017) reported that on analysis of ascitic fluid collected from abdominal paracentesis on examination revealed transudate fluid with serum-albumin ascetic gradient (SAAG) >1.1 gm/dl suggesting ascites due to portal hypertension (96% accuracy) caused by Liver cirrhosis.

Phom *et al.* (2019) said the ascitic fluid evaluation found out the values SAAG to be 0.60 g/dl.

Samad (2019) said that the diagnosis of ascites based on total protein concentration of <2.5g/dl or >2.5g/dl in the ascitic fluid has an accuracy of only 56%. However, the SAAG method is found as a more reliable tool in classification of ascites with efficacy ranging from 80 to 100%

2.2.6 Radiography

Vijay Kumar *et al.* (2002) studied the radiography of dog with ascites and found that hazy opaque abdominal cavity with a classic ground glass appearance is due to excessive fluid density and recognition of specific abdominal organ may not be possible.

Tyagi *et al.* (2004) performed abdominal radiograph of a male nondescript dog of eleven and half years of age with intermittent abdominal distension and found presence of diffused increased radio density in abdomen as ground glass appearance with appreciable changes.

Baumwart *et al.* (2005) Congestive heart failure was diagnosed in 24 of the 48 (50%) dogs on the basis of thoracic radiographic abnormalities in the dogs which showed abdominal distention.

Kumar *et al.* (2011) stated that ground glass appearance of abdomen and heart with increased sternal contact (cardiac resting) were the radiographic findings of abdomen and thorax of the affected dogs confirming that ascites was cardiac origin.

Kumar *et al.* (2012) studied the hepatic diseases in dog, the hepatic damage and dysfunction and identified possible primary causes of secondary liver diseases. Radiographic examination evaluates different hepatic affection.

Das (2012) observed ground glass appearance in 15(60%) dogs and hepatomegaly in (40%) dogs among 25 dogs with ascites due to hepatic insufficiency.

Pai *et al.* (2012) Radiographic examination revealed generalised cardiomegaly (75%), tracheal elevation (100%), pulmonary congestion (75%) and pericardial effusion (12.5%) in dogs showing abdominal distension.

Meindel and Polhman (2013) recorded radiograph which revealed diffuse fluid opacity in the abdomen of dog and concluded as accumulation of fluid in the abdomen.

Bhadesiya *et al.* (2015) described characteristic ground glass appearance due to presence of ascitic fluid, however difficult to rule out radiographic changes in other organs due to presence of ascitic fluid in the abdominal cavity.

Kumar *et al.* (2016) observed ground glass appearance in abdominal cavity in ascitic dog due to presence of fluid and enlargement of heart in thoracic cavity due to cardiac involvement.

Phom *et al.* (2019) found ground glass appearance with increased in vertical heart size (VHS) in radiographic examinations of ascitic dogs.

Dhillon *et al.* (2020) reported abdominal radiograph revealed characteristic ‘Ground glass’ appearance over abdomen in ascitic pup.

Gupta *et al.* (2020) reported that sensitivity of radiography were 57.14% in the animals with hepatic affections and the common findings were ground glass appearance of abdominal cavity in cases of ascites, mid abdominal soft tissue opacity pushing all intestines caudally, extension of liver lobes well beyond rib cage indicating hepatomegaly and increased opacity of liver on lateral radiographs.

2.2.7 Ultrasonography

Vijaykumar (2002) discussed the importance of Ultrasonographic examination of liver, spleen, pancreases, kidney, bladder and abdomen, as it helps to determine the cause of ascites.

Buob *et al*. (2011) explained that ultrasonographic evaluation of the hepatic parenchyma and biliary tract may help to anatomically localize the cause of Portal Hypertension.

Chaturvedi *et al.* (2013) reported revealed ground glass appearance of abdomen and masking of abdominal cavity details in ultrasonography examination of dog affected with ascites.

Kumar and Srikala (2014) carried out abdominal ultrasonographic examination and found viscera floating in anechoic effusion with engorged and distended hepatic vasculars with hyperechoic to mixed echogenicity which is indicative for hepatic disease resulting in ascites in dogs.

Saravanan *et al.* (2014) found liver, kidneys, spleen, intestine and urinary bladder suspended in ascitic fluid, on ultrasonographic examination in 72 dogs.

Bhadesiya *et al.* (2015) observed presence of free-floating fluid in abdominal cavity with anechoic to hyperechoic echogenicity. Also hyper echoic foci were present on the caudal lobe of liver suggestive of hepatic involvement in development of clinical ascites. Presence of anechoic fluid aids in better visualization of hepatic parenchyma on ultrasonography in dogs.

Elhiblu *et al.* (2015) observed ascites due to cirrhotic liver had increased echogenicity, rounding and irregularities of liver margins and microhepatica so called bright liver.

Bota *et al*. (2016) reported abdominal ultrasound were useful in detecting the condition in Yorkshire Terriers dogs which were predisposed to protein losing

enteropathy, and this was characterised by abdominal effusions, histologically by crypt lesions, lacteal dilation and inflammation.

Phom *et al.* (2019) carried out ultrasonography examination of ascitic dogs and found mild to extensive anechoic areas suggestive of fluid accumulation along with floating of intestines and internal viscera. Isoechoic kidney cortex and medulla; enlarged renal pyramids with anechoic echo texture, focal hyperechoic and loss of echogenicity of hepatic parenchyma with increased size and distended gall bladder were the other findings.

Dhillon *et al.* (2020) reported ultrasonographic examination revealed large amount of anechoic fluid present in abdominal cavity indicating ascites. No abnormal echogenicity was noticed with the abdominal organs except liver; where there was presence of hyperechoic to mixed echogenicity.

Gupta *et al.* (2020) reported sensitivity of ultrasonography were 100% in detecting hepatic abnormality. Major ultrasonographic findings were loss of normal echo texture, increased echogenicity of liver, roundening of hepatic margins, mixed echogenicity, hepatomegaly, presence of nodules, micro hepatica and presence of anechoic cavities separating liver lobes and other organs indicating ascites.

2.2.8 Electrocardiography

Vijaykumar (2002) recorded changes such as tall P wave in Lead II, deep S wave in lead I, II, aVf, right axis deviation, which were suggestive of right ventricular enlargement and deep Q wave in lead II suggestive of atrial ventricular arrhythmia.

Kumar *et al.* (2011) studied ECG of dogs with congestive heart failure for two years and found different abnormalities of amplitude and duration of P, Q, R, S, T and QRS waves, TS coving, ventricular premature complex, ventricular tachycardia, atrial standstill, junctional premature complex with II degree AV block, ventricular bigeminy, bundle branch block, low voltage QRS complex and atrial fibrillation.

Varshney *et al.* (2011) found 57 dogs suffered from congestive heart failure and the ECG findings were sinus rhythm, sinus tachycardia, sinus bradycardia, sinus arrest,

atrial fibrillation/ flutter, atrial premature complex, atrial tachycardia, 'p' mitrale (> 0.4 second), Ta wave, tall 'R' (.3.0 mV in large and .2.5 in small breed), broad QRS (.0.05 sec), low voltage complexes, ventricular escape complex, ventricular tachycardia, ventricular premature complex, ST segment changes, variable R-R interval (> 2 normal R-R interval), change in polarity of T-wave, prolonged QT interval, 'R' altemans.

Pai *et al.* (2012) reported most commonly encountered abnormalities in ECG of dogs affected with ascites due to dilated cardiomyopathy are sinus tachycardia (25%), atrial fibrillation (12.5%), ventricular premature complexes (25%) and ventricular pre – excitation (12.5%).

Kumar *et al.* (2014) found low voltage QRS complexes in electrocardiogram of an ascetic dog with right heart failure, which were suggestive of effusion in body cavities.

Padhi (2016) reported that the common changes observed in ECG were short R-R interval, prolonged QT interval, ST segment elevation, low voltage QRS complex and right axis deviation.

Sreehari (2017) found the common electrocardiographic changes in animals presented with ascites conditions were sinus arrhythmias whereas few animals showed R-wave peaking, prolonged R-R interval, variations in R-wave amplitude.

Phom *et al.* (2019) carried out ECG in all the cases of ascites to find out cardiac involvement and found sinus tachycardia, atrial standstill (absence of P wave) and prolonged QRS duration, alternating amplitude of R wave and decreased QRS amplitude, notching of R waves, fragmented QRS (f QRS) and Osborn wave in different cases indicating cardiac origin ascites.

2.3 THERAPEUTIC MANAGEMENT

Vijaykumar (2002) described to follow treatment schedule for ascites according to primary cause of disease such as use of Diuretics (Spironolactone (1-2 mg/kg PO q 12 hr) or frusemide (2-4 mg/kg PO 12hr)) antibiotics and antimicrobials (metronidazole (7.5 mg/kg PO q 8 hr), ampicillin (20 mg/kg PO q 8 hr) neomycin (20mg/kg PO q hr)), Fluid therapy (lactate ringer's or 0.9% saline with potassium chloride (20-30 mEq/L)) and B

complex. Glucose is added if hypoglycaemia is present. Hepatoprotectant or regenerator like Silymarin, a hepatoprotectant, antioxidant, membrane stabilizer, prostaglandin synthetase inhibitor at the dose rate 10 mg/kg PO bid.

Nottidge *et al.* (2003) treated a 10 months old Alsatian pup suffering from asites due to liver cirrhosis with frusemide @ 3 mg/kg BW BID by intravenous route. Nearly 200 ml of peritoneal fluid was drained daily through paracentesis. Oxytetracyclin was administration IM @ 5 mg/kg BW. Livjivan a herbal liver tonic was administered orally at a dose rate of 2 tsp. daily to improve liver function. The puppy died.

Tyagi *et al.* (2004) carried out treatment of 11.5 years old male non descript dog showing symptoms of abdominal distention with inj. Ampicillin-Cloxacillin 500 mg IM bid, Inj. Dexamethasone 8 mg IM sid and Beekom-L 1 ml IM sid for 7 days. This was followed by daily abdominocentesis along with administration of Inj. of frusemide 2 ml IM and Beekom-L for another 7 days. The dog died after treatment.

Kruth (2005) reported therapeutic management of ascites in dogs and cats due to cardiac failure which is based on improving cardiac performance and animals with portal hypertension due to right-sided heart failure or due to liver disease should be managed with diuretic therapy like frusemide and spironolactone @ 1mg/kg twice daily, and sodium-restricted diet is indicated in case of ascites with increased total body sodium.

Pradhan *et al.* (2008) therapeutically managed Doberman pinscher bitch of 8 years old age suffering from ascites of hepatic origin with Fructodex 200 ml IV, Inj. Terramycin 10 mg/kg body weight and Inj. Lasix 400 mg IM for five consecutive days and advised Tab Aldactone 100mg (spironolactone), Tab Doxy 100 mg (Doxycycline) and Susp. Sorbiline (Tricholine citrate 0.55 gm, sorbiline 7.15 gm) 1 tsf bid and Aminorich granules 1 tsf b.i.d and treatment continued for 10 days. Recovery in dog was observed in 15 days.

Bexfield and Watson (2009) advised for the use of spiranolactone which is potassium sparing aldosterone antagonist for the treatment of ascites due to portal hypertension. They also suggested for the use of spiranolactone and frusemide combination for speedy recovery.

Turkar *et al.* (2009) examined and treated a 2 months old male German shepherd pup with the complaint of abdominal distension, loose faeces and weakness with intravenous administration of dextrose (10%) on day 1. The dog was treated with pyrantel pamoate at the dose rate 5 mg/kg body weight PO, once daily for 3 days. Frusemide @ 2 mg/kg b.wt PO BID, Ampicillin @ 20 mg/kg b.wt PO BID for 5 days, drops Astymin-C 6 drops PO BID and powder proteinex 1/2 tsp PO BID for 7 days. Abdominal size reduced by day 3 and complete recovery was observed by day 10.

Routray *et al.* (2010) studied 2 cases of ascites; a. 8 years old female Labrador and B. 7.5 years old male non descript dog with the distended abdomen. Both the dogs were treated with ceftriazone with tazobactum injection (intacef Tazo 562.5 mg) once daily for five days, combination of spironolactone and frusemide 50 mg at dose rate of one tablet twice daily for 20 days. Liver protective injection (Inj. Neohepatex) was administered @2ml IM on alternate day for three doses. Liv 52 syrup at 2 tsf thrice daily and proteinex powder was fed at the dose rate of one tsf thrice daily orally in aqueous suspension for three weeks. Common salt restricted diet was advised for dog along with complete rest. Labrador dog recovered in 25 days and male dog showed complete recovery by 35^{th} day.

Dabas *et al. (*2011) reported a case of ascites of splenic origin in mongrel dog. Treatment was carried out with inj. Ceftriaxone @500mg, frusemide @2 mg/kg, inj. pheneramine maleate @1 ml and ketoprofen daily for 1 week and the animal showed complete recovery.

Kumar *et al.* (2011) studied cardiac cases and among 34 dogs, 11 dogs were confirmed to suffer from ascites due to Congestive Heart Failure. They suggested the use of losartan potassium, spiranolactone, tricholine citrate, sorbitol and ubiquinone for treating ascites.

Peddle *et al.* (2012) reported that torsemide is equivalent to frusemide at controlling clinical signs of dogs and is likely to achieve greater diuresis vs. frusemide.

Das (2012) carried out treatment of ascites in dogs due to hepatic insufficiencies with frusemide @2mg/kg b.wt. IM for 10 days. Meloxicam was given parenterally @0.2

mg/kg b.wt to dogs showing pyrexia along with broad spectrum antibiotics (Ceftriaxone sodium+tazobactum) parenterally @10 mg/kg for 3-5 days. Anorectic dogs also treated with Neohepatex injection @2ml IM on alternate day for five occasion followed by oral liver tonic (Liv-52 syrup) till return to normal appetite.

Malini *et al.* (2013) successfully treated a case of eosinophilic ascites in the setting of Toxocara infection in a post-partum patient with albendazole 400 mg twice daily for 5 days and other supportive therapy.

Saravanan *et al. (*2013) used dextrose 25% 100ml daily for 5 days, frusemide 2 ml/kg BID PO for 10 days, inj. ceftriaxone 25 mg/kg for 5 days, inj.astymin-3 40 ml/day and B-complex and vitamin-c 2ml/ day I/M for 12 days for treatment of ascites in dogs and improvement in attitude and appetite along with reduction in abdominal fluid accumulation was observed within 10 days. Ultrasonography on day 20 revealed improved liver status.

Kashiide *et al.* (2014) used Praziquantel @5mg/kg BW via S/C route for the treatment of canine peritoneal larval cestodiasis at an interval of 14 days and observed and dog was completely cured.

Kashyap *et al.* (2015) advocated conventional therapeutic management of ascitic dog is to remove the extra peritoneal fluid by paracentesis @1ml/Kg/day along with the supportive treatment such as reduced salt in diet, B-complex, liver tonics, protein rich biscuits and fluid and electrolyte therapy with DNS and RL, vitamin-E as antioxidant, vitamin-K to control bleeding and low copper and salt was given to prevent the recurrence and aggravation of condition and animals recovered in 21 days.

Manoj and Dhana Laksmi (2015) reported that effective treatment of ascites of hepatic origin can be carried out using the combination of diuretics along with protein supplementation.

Kamalakar *et al.* (2016) concluded that combination of spiranolactone and frusemide along with fluid and electrolyte therapy in dog proved to be the best in regression of ascites in 12 days.

Kumar *et al.* (2016) reported that after administering diuretics, liver tonics along with supportive therapy complete recovery of dog was observed without any recurrence or other complications.

Regmi and Shah (2017) reported that the dog showed remarkable improvement with gradual reduced in abdominal distention and normalization of the appetite after 7 days of treatment with antibiotic, diuretic, amino acid and liver tonics along with protein rich but salt free diet.

Sharma *et al*. (2017) suggested the management for ascites due to liver dysfunction using Inj. Ceftriaxone @25mg/kg b.wt. IV bid, Inj. frusemide @2 mg/kg b.wt. IV bid, Inj. 50% Dextrose @1ml/kg IV bid along with Inj. Tribivet 1 ml IV o.d, Inj. Ascorbic acid 100 mg IV o.d, Plasma Volume Expander (Haemaccel) @10 ml/kg b.wt I/V bd, syrup Livoferrol 0.5 TSF p.o bid, Cap. Evion 250 mg 1 cap p.o o.d. Paracentesis was done on alternate day for 3 times to remove half of the total fluid from the abdomen. Animal showed improvement within 5 days of treatment as the abdominal distension started to decreases.

Alsaad *et al.* (2018) carried out treatment of animal suffering from ascites associated with Congestive Heart failure with prescribed medical treatment which includes oral dose of Nebivolol 1mg daily (A Beta-blockers and blood pressure regulator), Ringer's solution I.V infusion, frusemide 4mg IM for 5 days. General health, physical activity and appetite improved within 2-3 days of treatment.

Singh *et al.* (2019) carried out therapeutic study and divided into three groups, namely G I, G II and G III comprising of 6 animals in each group. The dogs in group G1 were dosed with Silymarin- 7mg/ml PO, G2 group were treated with syrup containing Tricholine Citrate, Vitamin B12, Inositol, Methyl donors, Selenium, Vitamin E and Biotin in a base enriched with liver extracts whereas group G3 were given Alfalfa, Avena Sativa, Ashwagandha, Acid Phosphoricum, China, Hydrastis Canadensis, Five Phos, total medication 6.35%, in syrup base. 2tsf of each drugs were given to dogs of particular group for 45 days along with the symptomatic and supportive therapy. Improvement was observed with all 3 drugs but better results were observed with the use of Silymain.

Samad (2019) reported treatment of 2.5 years old female Spits dog with restricted sodium diet, antibiotic (amoxicillin), diuretic (frusemide; Lasix, Sanofi Aventis) and vitamin B-complex and C-vitamin supplement with regular monitoring assisted in successful recovery of ascites in dogs.

Dhillon *et al.* (2020) carried out treatment of 3 months old female beagle pup with Inj. Dextrose 10% @10 ml/kg IV BID×7 days, Inj. Ceftriaxone @25mg/kg IM BID×5 days, Tab Lasilactone 50 (Frusemide 20mg + Spironolactone 50mg) @¼ tab/5kg PO BID×7 days and Inj. Tribivet (Vitamin B1, B6 and B12) @0.5 ml IM OD for a week along with supportive therapy with syrup Livotas pet (Liver tonic) @1 tsp. PO BID for 14 days. Protein rich diet was advised for the pup along with the restriction of dietary sodium intake. Subsequently, reduction in abdominal distension was observed from 3rd day of treatment and the animal became completely healthy by 10th day.

Ghosh *et al.* (2020) reported a case study ascites in a two and half months' old Doberman pinscher male pup, which was suffering from ascites due to babesiosis infection. Treatment with babesiacidal, antibiotics, diuretics, hepatobiliary drugs resulted in uneventful recovery.

Chapter-III

Materials and Methods

CHAPTER-III

MATERIALS AND METHODS

3.1 LOCATION OF STUDY

The present study entitled "**ClinicoTherapeutic Studies on Canine Ascites**" was carried out as per the envisaged objectives of the study i.e., the haemato-biochemical changes and therapeutic management of dogs suffering from ascites at Teaching Veterinary Clinical Complex (TVCC), Faculty of Veterinary Sciences and Animal Husbandry, R. S. Pura campus of SKUAST -Jammu, during the period from August, 2020 to June, 2021.

3.2 SELECTION OF ANIMALS

The dogs with the complaint of fluid filled abdomen, breathing distress, weight reduction and pale to yellowish mucous membrane brought to Small Animal OPD of Teaching Veterinary Clinical Complex, F.V.Sc and A.H., Sher-e- Kashmir University of Agricultural Sciences and Technology, R. S. Pura, and the other sources in and around Jammu district had been screened for the study purpose. Only the ones with grossly observable ascites i.e., grade 3 ascites (Moore *et al*., 2003) had been marked and further investigated with the aid of using physical examination, radiographic examination, ultrasonographic examination and haemato-biochemical study for ruling out the reason of ascites.

3.2.1 Inclusion Criteria

1. Cases with abdominal distension
2. Cases with developing ascites detected on ultrasonographic examination
3. Cases with fluid thrill
4. Dogs with anorexia, breathing distress, weight reduction

3.2.2 Exclusion Criteria

1. Pregnant animals
2. Animals with bladder rupture
3. Animals with intra-abdominal mass
4. Obese animals with no intra- abdominal fluid
5. Pyometra

3.2.3 Anamnesis

History associated with age of the animal, breed, sex, preceding vaccination records, period of disease, any records associated with previous sickness affecting organs like liver or kidney, preceding medicinal drug and sort of feeding had been collected.

3.3 EPIDEMIOLOGICAL STUDY

The occurrence of the canine ascites with respect to age, gender, breed and etiology had been studied. The following physiological parameters had been studied and the values were recorded:

3.3.1 Rectal Temperature

Rectal temperature recorded with the aid of usage of a digital or mercury thermometer and expressed as degree of Fahrenheit.

3.3 2 Pulse Rate

Pulse rate was assessed with the aid of palpating femoral arterial pulse and expressed as rate per minute.

3.3 3 Respiratory Rate

Respiratory rate was recorded by counting the number of coastal/ abdominal wall movements during inspiration and expirations and expressed as breaths per minute.

3.3 4 Conjunctival Mucous Membranes

Conjunctival mucous membranes of dogs were examined and recorded the colour (roseate or congested or pale or yellow or cyanotic) as it reflects the general condition of the animals as well as certain disease conditions, jaundice, plant poisoning and anaemic conditions

3.4 CLINICAL EXAMINATION

Palpation, percussion and auscultation had been completed to recognise the cause of abdominal distension (gross obesity, gas, liquid, organomegaly, bowel obstruction, intussusceptions, hernia etc). Fluid thrill test were executed. Additionally, auscultation of heart was carried out to detect any abnormality in rate and rhythm.

3.5 COLLECTION OF SAMPLES

3.5.1 Blood

Aseptic measures had been accompanied to gather blood from affected dogs by the usage of sterilized syringe and needle. Five ml of blood was accrued from cephalic vein out of which one ml was collected in EDTA vaccutainer (lavender top) for haematological examine and left over was retained in clot activator vaccutainer (red/yellow top) for serum biochemistry examine. Tests had been executed at the same day of series.

3.5.2 Faecal Sample

Faecal samples had been accrued from the rectum of dogs.

3.5.3 Peritoneal Fluid

Before collection of peritoneal fluid, urinary bladder was emptied (Hall, 2005). By performing abdominocentesis five ml ascetic fluid was accrued, placing 18 gauze needles 2-3 cm caudal to the umbilicus and 2-3 cm left of the midline at 30-40 degree angle (Alleman, 2003; Mc Grothy and Doust, 2004; Rudloff, 2005). Fluid was collected in EDTA vial, clots activator vial and sterile tube as per the requirement.

3.6 HAEMATOLOGICAL STUDIES

Two millilitres of blood samples were collected into sterilized vials containing ethylene diamine tetra acetate (EDTA) for the estimation of the following haematological parameters.

3.6.1 Haemoglobin

The haemoglobin estimation was done by using acid-haematin method described by (Jain, 1986) and expressed in grams percentage (g %).

3.6.2 Packed Cell Volume

The packed cell volume values were estimated as per procedure described by (Jain, 1986) and expressed in percentage.

3.6.3 Total Erythrocyte Count

Total erythrocyte count was determined by dilution method (Jain, 1986) and expressed as millions per microliter.

3.6.4 Total Leukocyte Count

Total leukocyte count was determined by dilution method (Jain, 1986) and expressed as thousands per microliter.

3.6.5 Differential Leukocyte Count

Differential Leukocyte count was determined by using Leishman staining method and expressed as percentage (Jain, 1986).

3.7 BIOCHEMICAL STUDIES

Various changes in the biochemical parameters pre and post treatment periods like 0, 15 and 45 days were recorded. The following parameters were evaluated in serum samples collected from different groups. The blood sample collected in clot activator vial was allowed to stand for 20 minutes in slanting position at room temperature. The centrifuged at 2500- 3000 rpm for 10 minutes for serum separation. Biochemical parameters estimated were:

Liver function tests: Alanine aminotransferase (ALT)*/* serum glutamate Pyruvate transaminase (S.G.P.T.), aspartate aminotransferase (AST) / serum glutamate oxaloacetate transaminase (S.G.O.T.), alkaline phosphatise (ALP), serum total protein, and serum albumin.

Kidney function test: Blood urea nitrogen (BUN), serum creatinine.

3.7.1 Serum Alanine Amino Transferase (ALT) Activity

The serum ALT activity was determined by the IFCC method, Kinetic for in vitro determination of ALT in serum or plasma. (Reitman and Frankel, 1957) using an ALT test kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.7.2 Serum Aspartate Amino Transferase (AST) Activity

The serum AST activity was determined by the IFCC method, Kinetic for in vitro determination of AST in serum or plasma (Reitman and Frankel, 1957) using an AST tests kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.7.3 Serum Alkaline Phosphatase (ALP) Activity

The serum ALP activity was determined by the Phenolphthalein monophosphate method for the in vitro determination of ALP in serum or plasma (Klein *et al.*, 1960), using an ALP test kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.7.4 Total Protein

The serum total protein was determined by the Biuret method, End Point for quantitative in vitro determination of total protein in serum, plasma or urine (Lubran, 1978), using a total protein test kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.7.5 Serum Albumin

The serum albumin was determined by the Bromocresol green dye method for the in vitro determination of albumin in serum or plasma (Doumas *et al.,* 1972), using albumin test kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.7.6 Serum Creatinine

Serum creatinine was determined using modified Jaffe method (Blass *et al*., 1974) for the in-vitro determination of creatinine in serum, plasma or urine, using creatinine test kit **(**Trans Asia Bio medicals, Ltd. H.P., India).

3.7.7 Blood Urea Nitrogen

Serum Urea Nitrogen was determined by the GLDH- Urease method, Initial Rate (Tiffany *et al.,* 1972) for the in vitro determination of urea in serum or plasma using urea nitrogen test kit (Trans Asia Bio medicals, Ltd. H.P., India).

3.8 FAECAL SAMPLE EXAMINATION

The collected samples of dogs were examined for presence of parasitic ova by direct smear method and/or floatation technique method by using saturated salt solution of specific gravity 1.18-1.19.

3.9 PHYSICAL EXAMINATION OF THE ASCITIC FLUID

The fluid obtained by abdominal paracentesis was physically examined for its colour, consistency, transparency and odour if any.

3.9.1 Cell Counts on the Ascitic Fluid

The total cell count on the ascitic fluid was carried out by the haemocytometer method (Schalm *et al.,* 1975). Ascitic fluid (0.02ml) was pipetted into a small test tube containing 0.38ml of diluting fluid to make a 1:20 dilution of the ascitic fluid sample. The diluted sample was loaded on to the Neubauer counting chamber and all cells on the four corner squares were counted using a light microscope at ×10 objective. The number of cells counted for each ascitic fluid sample was multiplied by 50 to obtain the total cell count per microliter of the ascitic fluid.

3.9.2 Determination of the Total Protein and Albumin of the Ascitic Fluid

The ascitic fluid sample that was added to a plain test tube was centrifuged at 3000 rpm for 10 minutes and the supernatant was used to determine the ascitic fluid total protein and albumin. The ascitic fluid total protein and albumin were then determined by

the Biuret Method and bromocresol green method respectively, as earlier described for serum samples above, using the total protein and albumin test kits (Trans Asia Bio medicals, Ltd. H.P., India).

3.9.3 Calculation of Serum - Ascites Albumin Gradient (SAAG)

The SAAG was calculated by subtracting the albumin concentration of the ascitic fluid from that of the serum albumin concentration (Runyon *et al.*, 1992).

3.10 ELECTROCARDIOGRAPHIC STUDIES

Electrocardiography was carried out in the dogs suspected for ascites due to any cardiac changes on basis of clinical examination, auscultation and radiography. At the time of recording ECG all the dogs were manually handled without any anesthesia, in standard body position restrained in right lateral recumbency on a table. The skin and electrodes were moistened by the coupling gel before attaching the electrodes to the body. ECG was recorded using BPL Cardiart 6208 view three channel ECG Machine on ECG paper with a pace of 50 mm per second so each small box on horizontal axis equals 0.02 seconds.

3.11 RADIOLOGICAL STUDIES

Animals had been subjected to traditional lateral and ventrodorsal thoracic and belly radiographs to visualise the thoracic and belly visceral organs and come across any abnormalities associated with the dimensions and relative function to discover the viable etiological agent. About 40-60 kVp, 8-16 mAs had been used for radiography, relying upon the dimensions and breed of the animal and all other radiographic elements had been stored constant.

3.12 ULTRASONOGRAPHIC STUDIES

Ultrasonographic exam become performed through the usage of actual time B-mode ultrasonographic exam of the stomach become in transverse and sagittal planes duly getting ready the place through clipping the hair and making use of transmission gel

or coupling medium. 3.5, 5.5 and 7.5 MHz probes had been used for sonating the place and recorded the findings.

3.13 THERAPEUTIC MANAGEMENT

The ascitic dogs had been categorised into causative types - coronary heart related, liver related, kidney related and others on the idea of Clinical Symptoms, Physical Examination findings, Radiography and Haematobiochemical analysis. The affected dogs had been treated with drugs as given in Table 1. along with fluid therapy using dextrose 10% and normal saline solution for intravenous infusion. Symptomatic treatment of affected dogs as per requirement included: proton pump inhibitor (Pantoprazole; Pantop 40® by Cipla Ltd, India), antiemetics (Metoclopramide; Perinorm® by Ipca Laboratories Ld, India, Ondansetron: Emeset® by Cipla Ltd, India), multivitamin supplements (Vitcofol-C® by FDC Ltd, India, Eldervit-12® by Elder Pharmaceuticals Ltd, India). Dietary changes were brought about by prescribing salt free diet and high value protein sources diet such as cheddar cheese, egg albumin. Also intravenous protein supplementation was carried out using Alamin SN® by Albert David, India, Haemaccel® by Abbott Healthcare Pvt Ltd, India. Mixed origin ascitic cases were treated accordingly.

Table 1: Therapeutic management of canine ascites in different groups as per origin

S.No.	Causative Type	Treatment
1.	Cardiac Origin	Diuretics (combination of Frusemide and Spironolactone; Fruselac® by Lupin Ltd, India, Frusemide; Ridema® by Vetoquinol, India) Cardiac Glycosides (Pimobendan: Pimocard® by Corise Healthcare Pvt Ltd, India), ACE inhibitors (Ramipril; Cardiopet® by Corise Healthcare Pvt Ltd, India)
2.	Hepatic Origin	Diuretics (combination of Frusemide and Spironolactone; Fruselac® by Lupin Ltd, India, Frusemide; Ridema® by Vetoquinol, India), Liver tonics (Sylibion by Micro Labs Ltd), Antibiotics therapy (Ampicillin and Cloxacillin; Ampoxin by Unichem Laboratoties Ltd).
3.	Renal Origin	Diuretics (combination of Frusemide and Spironolactone; Fruselac® by Lupin Ltd, India, Frusemide; Ridema® by Vetoquinol, India), Antibiotics therapy (Ampicillin and Cloxacillin; Ampoxin by Unichem Laboratoties Ltd, India), ACE inhibitor (Ramipril; Cardiopet® by Corise Healthcare Pvt Ltd, India).

Affected dogs were reassessed for haematological and biochemical parameters after an interval of 15 and 45 days post treatment to check the response to treatment and clinical improvement.

3.14 STATISTICAL ANALYSIS

Statistical analysis of the data, collected in the course of present study was analyzed by two-way ANOVA for haematobiochemical parameters; significance was determined within and between groups by Duncan Multiple Range Test (DMRT). Whereas ascitic fluid parameters were analysed by one-way ANOVA and the significance was determined between groups by DMRT, using SPSS v. 16.0.

Chapter-IV

Results

CHAPTER-IV

RESULTS

In the present study, an attempt was made to study the haematobiochemical changes and therapeutic management of canine ascites in and around R.S. Pura, Jammu during the period of August 2020 to June 2021.

4.1 DETAILS OF SCREENING FOR CANINE ASCITES

A total of 2550 dogs comprising 1602 males and 948 females were screened for canine ascites. Ascites was recorded only in 21 out of 2550 dogs undertaken from different sources in the present study (Table 2).

Table 2: Details of screened animals

Source of Dogs	No. of Dogs Screened	Age Wise				Sex Wise	
		0-2 years	2-5 years	5-10 years	10-15 years	Male	Female
Small Animal OPD of TVCC, F.V.Sc. and A.H, R.S Pura	2488	884	609	746	249	1564	924
Others Sources (in and around Jammu District)	62	23	14	18	62	38	24
Total	2550	907	623	764	311	1602	948

4.1.1 Overall Prevalence of Canine Ascites

During the study period (August 2020 to June, 2021) a total of 2550 dogs presented for treatment at Teaching Veterinary Clinical Complex, R.S. Pura , Jammu and from the other sources in and around Jammu district were undertaken. Only 21 dogs (0.82 per cent) were found positive (Table 3 and Fig 1).

Table 3: Overall Prevalence of canine ascites

Total No. of Dogs Screened	No. of Ascitic Cases	Percent (%)
2550	21	0.82

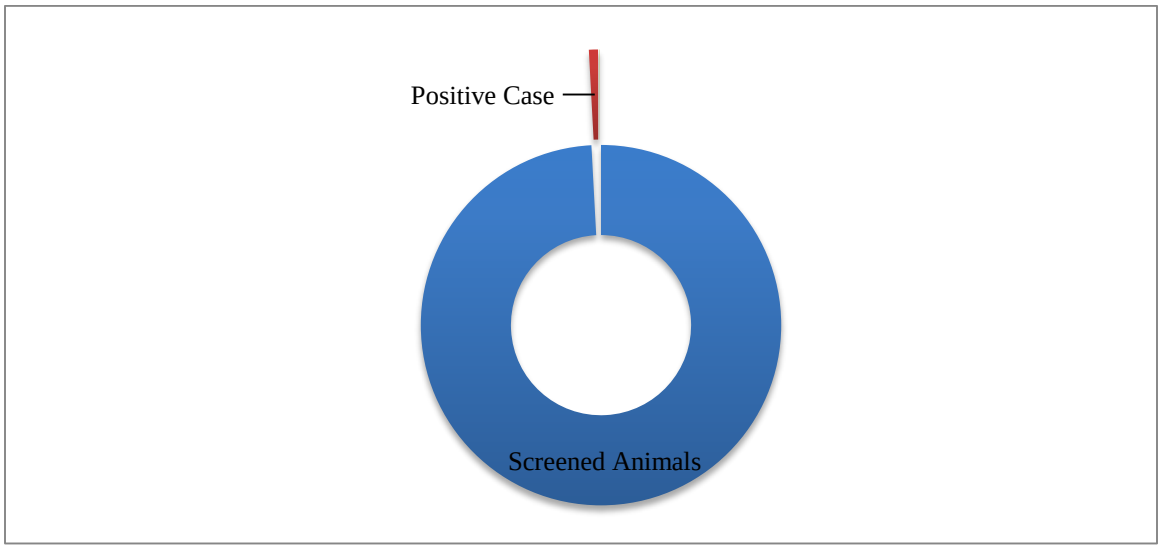


Fig.1: Prevalence of canine ascites

4.1.2 Age Wise Prevalence of Canine Ascites

The screened dogs were grouped according to the age into four groups ranging from 0-2 years, 2-5 years, 5-10 years and 10-15 years. Highest prevalence was recorded among the 2-5 years age group (42.85%) followed by 5-10 years (28.57%) and lowest prevalence of ascites in dogs were recorded in 0-2 and 10-15 years age group sharing equal percentage of 14.28% (Table 4, Fig 2).

Table 4: Age wise prevalence of canine ascites

S. No.	Age Group	No. of Ascitic cases	Percent (%)
1.	0-2 Yrs	3	14.28
2.	2-5 Yrs	9	42.85
3.	5-10 Yrs	6	28.57
4.	10-15 Yrs	3	14.28

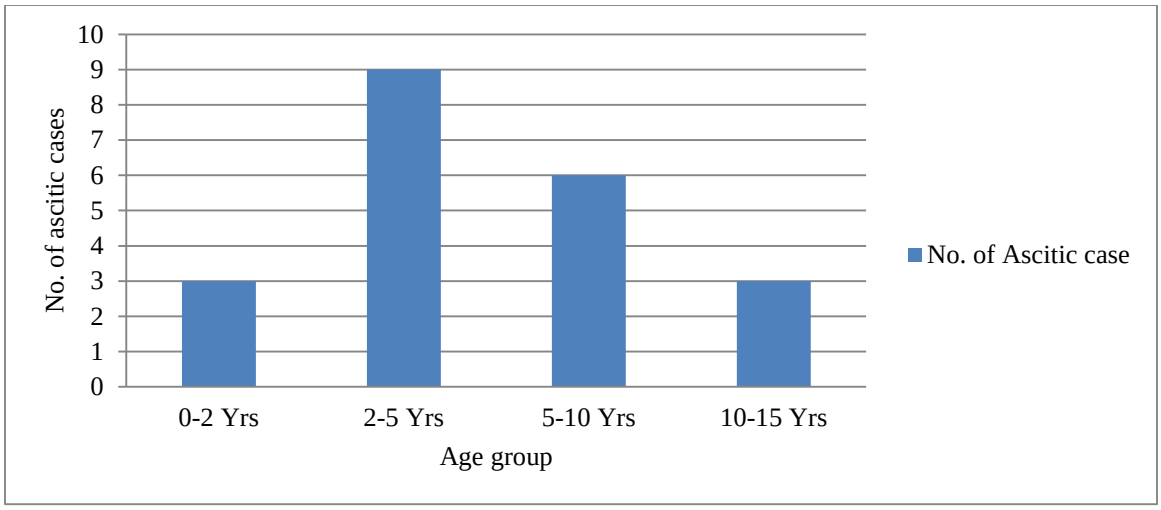


Fig.2: Age wise prevalence of canine ascites

4.1.3 Sex Wise Prevalence of Canine Ascites

Twenty one dogs affected with ascites comprised of nine males (42.85%) and twelve females (57.14%) as presented in Table 5, Fig. 3. It was observed that the prevalence of ascites in dogs was higher in females as compared to males.

Table 5: Sex wise prevalence of canine ascites

S. No.	Sex	No. of Ascitic cases	Percent (%)
1.	Male	9	42.85
2.	Female	12	57.14

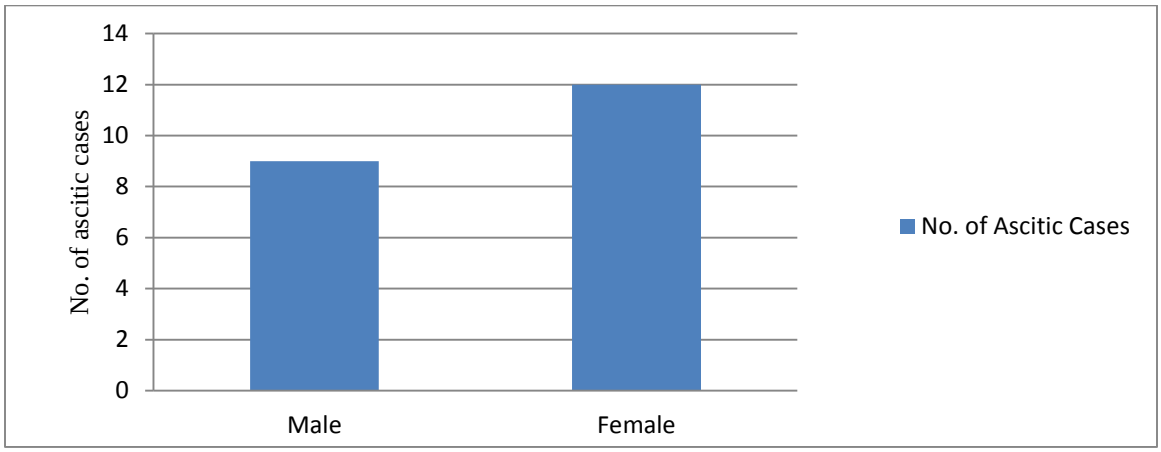


Fig. 3: Sex wise prevalence of canine ascites

4.1.4 Breed Wise Prevalence of Canine Ascites

Among 21 positive cases of ascitic cases 10 (47.61%) were Labrador, 3(14.28%) were German shepherd, 6 (28.57%) were Spitz and 2 (9.52%) were Golden retriever. The prevalence was highest in Labrador breed and lowest in Golden retriever (Table 6, Fig. 4).

Table 6: Breed wise prevalence of canine ascites

S. No.	Breed	No. of Ascitic Case	Percent (%)
1.	Labrador	10	47.61
2.	German Shepherd	3	14.28
3.	Spitz	6	28.57
4.	Golden Retriever	2	9.52

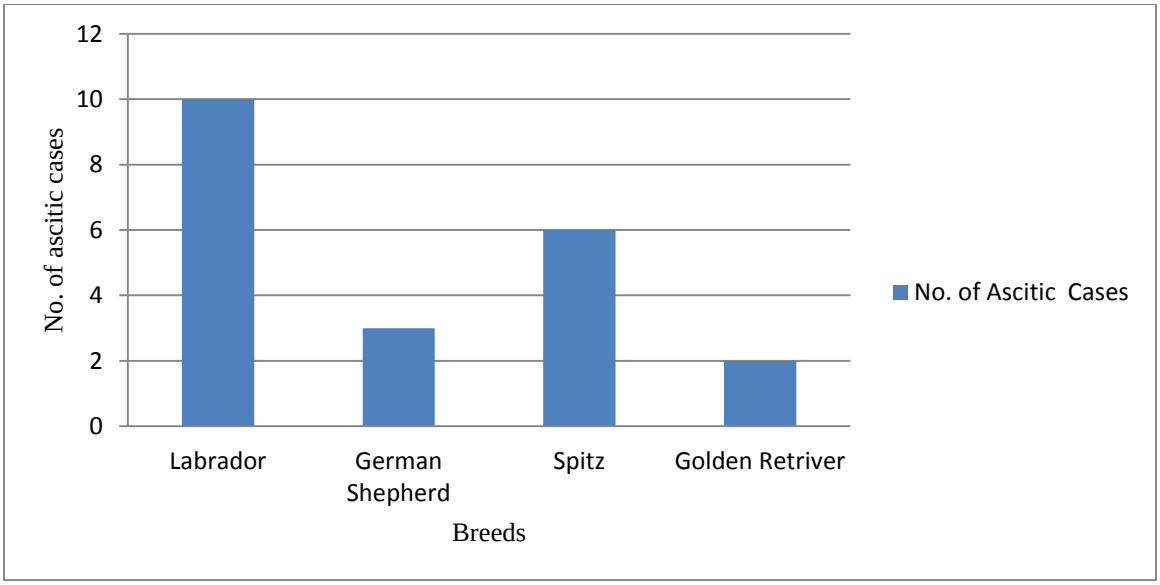


Fig.4: Breed wise prevalence of canine ascites

4.1.5 Etiology Wise Prevalence of Canine Ascites

Ascitic cases detected were classified on the basis of etiologies into following types: hepatic origin, renal origin and both cardiac and hepatic origin on the basis of clinical symptoms, physical examination and haematobiochemical analysis, radiographic

and ultrasonographic examinations. Out of 21 ascitic cases the number of cases detected in each etiological group was seven (33.33%) (Table 7, Fig 5).

Age wise, ascites of hepatic origin was maximally recorded among 2-5 years (42.85%) age group followed by 0-2 years (28.57%) age group whereas minimum cases were recorded in the age group of 5-10 and 10-15 years sharing equal percentage of 14.28%. Renal origin ascitic cases were recorded maximally in the age group of 5-10 years (57.14%) followed by 2-5 years (25.57%) and 0-2 years age group (14.28%). Maximum number of ascitic cases of both cardiac and hepatic origin was observed in the age group of 2-5 years followed by 10-15 years (28.57%) and 5-10 years (14.28%) (Table 8, Fig 6).

Sex wise, ascites of hepatic origin and both cardiac and hepatic origin were recorded more frequently in females 57.14% and 71.42%, respectively whereas ascites of renal origin was more common in males (42.85%) (Table 9, Fig 7).

Table 7: Etiology wise prevalence of canine ascites

S. No.	Etiology	No. of Ascitic case	Percent (%)
1.	Hepatic origin	7	33.33
2.	Renal origin	7	33.33
3.	Cardiac + Hepatic	7	33.33

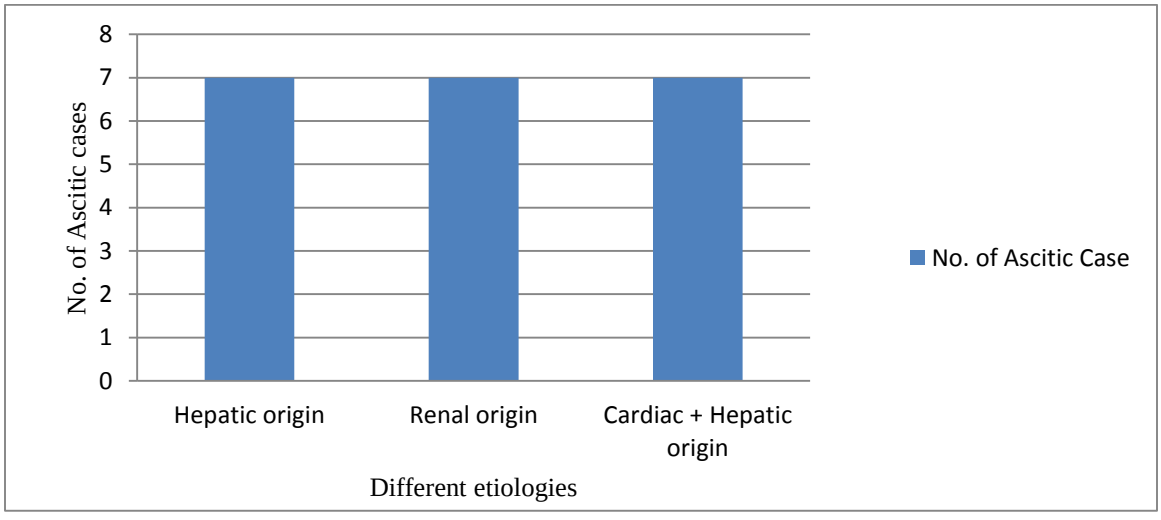


Fig. 5: Etiology wise prevalence of canine ascites

Table 8: Age wise prevalence of canine ascites of different etiologies

Age Group	Hepatic Origin		Renal Origin		Cardiac + Hepatic Origin	
	No. of Animals	Percent (%)	No. of Animals	Percent (%)	No. of Animals	Percent (%)
0-2 Yrs	2	28.57	1	14.28	0	0
2-5 Yrs	3	42.85	2	28.57	4	57.14
5-10 Yrs	1	14.28	4	57.14	1	14.28
10-15 Yrs	1	14.28	0	0	2	28.57

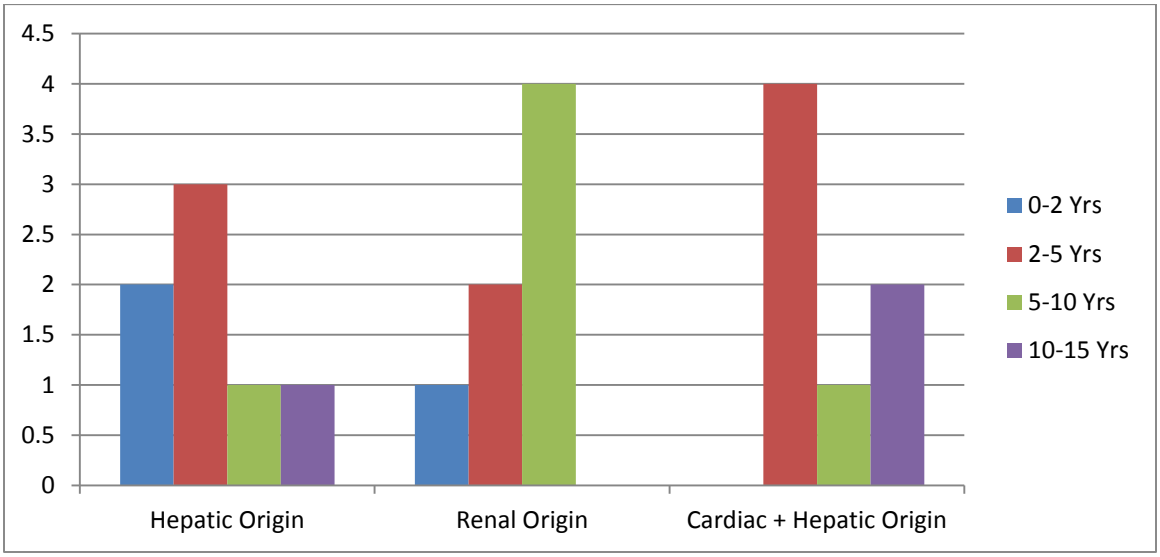


Fig. 6: Age wise prevalence of canine ascites of different etiologies

Table 9: Sex wise prevalence of canine ascites of different etiologies

S. No.	Types of Ascites	Males		Females	
		No. of dogs	Percent (%)	No. of dogs	Percent (%)
1.	Hepatic Origin	3	42.85	4	57.14
2.	Renal Origin	4	57.14	3	42.85
3.	Cardiac and Hepatic Origin	2	28.57	5	71.42

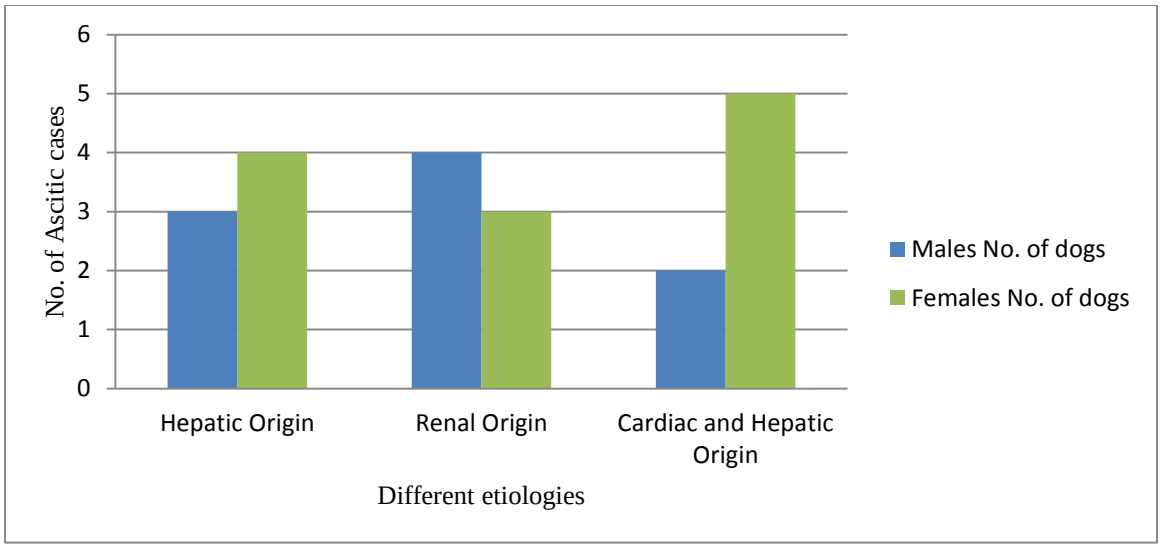


Fig. 7: Sex wise prevalence of canine ascites of different etiologies

4.2 CLINICAL SIGNS

The general clinical and physical signs exhibited by the dogs suffering from ascites included abdominal distension which was observed in all the 21 dogs (100%) (Fig. 9, 10 and 11). Out of 21 affected dogs, 8 (38.09%) were anorectic, 10 (47.61%) were showing inappetence, while 3 (14.28%) were having normal appetite. 13 (61.90%) dogs had pale mucous membrane, 4 (19.04%) dogs had slight yellow to moderate yellow mucous membrane and 4 (19.04%) had pale pink mucous membrane (Fig.12, 13 and 14). Pyrexia was observed in 6 (28.57%) dogs.

Other clinical signs observed were reduction in body weight, weakness and dullness. The dogs exhibiting the above clinical signs were subjected to abdominal palpation for confirmatory diagnosis of ascites. The abdominal palpation was performed with assisted abdominal pressure on one side and presence of fluid .thrill on opposite side. All the dogs were found positive for fluid thrill test. Respiratory distress was present in 10 (47.61 %) dogs and 9 (42.85%) dogs were showing signs of tachycardia. Sub cutaneous edema of limbs was shown by 6 (28.57 %) dogs (Fig 8). Abdominal ultrasonography was performed in 15 dogs. Liver size was subjectively assessed to be small (n=5), normal (n=8) and enlarged (n=2). Kidney size was subjectively assessed to be small (n=5), normal (n=7) and enlarged (n=3).

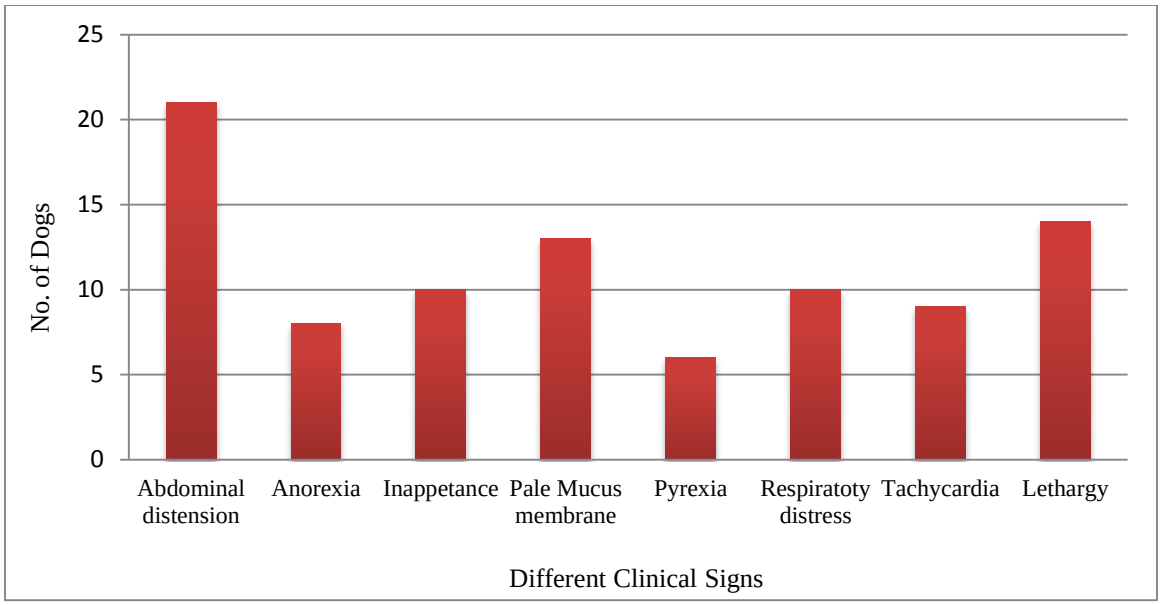


Fig.8: Different clinical signs observed in canine ascites

4.3 HAEMATOLOGICAL PARAMETERS

The hematological values recorded in the dogs affected with ascites due to different etiologies are presented in Table 10, Fig 15, 16, 17 and 18. A significant difference was observed in all the hematological parameters between the apparently healthy and diseased animals.

4.3.1 Hemoglobin

Significant ($p<0.05$) decrease was observed in the Hb concentration among the ascitic dogs of different origin when compared to healthy dogs. The mean value of Hb concentration in apparently healthy dogs was 11.95±0.20 gm % and in dogs affected with ascites of hepatic, renal and both cardiac and hepatic origin were 7.54±0.86 gm %, 7.38±0.18 gm % and 9.97±0.22 gm %, respectively (Table 10, Fig 15).

4.3.2 Packed Cell Volume (PCV)

Significant ($p<0.01$) decrease was observed in the PCV value in the ascitic dogs of different origin when compared to healthy dogs. The mean PCV value in apparently healthy dogs was 34.75±0.19 percent whereas in dogs affected with ascites of hepatic origin was 28.24±2.34 per cent, renal origin was 24.25±1.46 per cent and both cardiac and hepatic origin was 30.08±0.25 per cent (Table 10, Fig 15).

4.3.3 Total Erythrocyte Count (TEC)

Significant ($p<0.05$) decrease was observed in the TEC value in the ascitic dogs of different origin when compared to healthy dogs. The mean TEC value in apparently healthy dogs was 5.92±0.14x106/µl and in dogs affected with ascites of hepatic, renal and both cardiac and hepatic origin were 4.35±0.47 $x10^6$/µl, 3.84±0.38 $x10^6$/µl and 3.57±0.25$x10^6$/µl, respectively (Table 10, Fig 15).

4.3.4 Total Leukocyte Count (TLC)

Significant ($p<0.05$) increase was observed in the TLC value in the ascitic dogs of hepatic origin whereas non-significant ($p>0.05$) increase was observed in the ascites of renal and both cardiac and hepatic origin when compared to healthy dogs. The mean TLC value in apparently healthy dogs was 11.68±0.21$x10^3$/ µl and the dogs affected with ascites of hepatic origin was 17.17±2.69 $x10^3$/ µl, renal origin was 15.92±2.15 $x10^3$/ µl and both cardiac and hepatic origin was 7.47±0.67 x103/ µl (Table 10, Fig 15).

4.3.5 Differential Leukocyte Count

4.3.5.1 Neutrophil

The neutrophilic count in apparently healthy dogs was 66.85±4.10 percent and in ascitic dogs of hepatic, renal and both cardiac and hepatic origin were 71.51±1.75, 78.71±4.13 and 86.14±2.69 per cent respectively (Table 10). Significant ($p<0.05$) increase was observed in the neutrophil count in the ascitic dogs of renal and both cardiac and hepatic origin whereas non-significant ($p>0.05$) increase was observed in the ascitic dogs of hepatic origin compared to healthy dogs.

4.3.5.2 Lymphocytes

The lymphocytic count in apparently healthy dogs was 24.14±1.60 percent and in ascitic dogs of hepatic, renal and both cardiac and hepatic origin were 22.57±1.10, 18.57±3.63 and 10.71±2.10 percent respectively (Table 10). Significant ($p<0.01$) decrease was observed in the lymphocyte count in the ascitic dogs of renal and both cardiac and hepatic origin whereas non-significant ($p>0.01$) decrease was observed in ascites of hepatic origin when compared to healthy dogs.

4.3.5.3 Monocytes

The monocytes count in apparently healthy dogs was 5.42±0.36 percent and in ascitic dogs of hepatic, renal and both cardiac and hepatic origin were 3.14±0.34, 3.42±0.64 and 3.57±0.52 per cent respectively (Table 10) (Fig 13, 14, 15 and 16). Significant ($p<0.05$) decrease was observed in the monocytes count in the ascitic dogs of different origin when compared to healthy dogs.

Table 10: Hematological alterations in dogs suffering with canine ascites due to different etiologies

S.No.	Parameters		Control	Hepatic	Renal	Cardiac + Hepatic
1	Hb (gm %) *		11.95 ± 0.22^{C}	7.54 ± 0.86^{A}	7.39 ± 0.19^{A}	9.97 ± 0.23^{B}
2	PCV (%) **		34.75 ± 0.18^{C}	28.24 ± 2.34^{AB}	24.25 ± 1.46^{A}	30.09 ± 0.26^{B}
3	TEC($x10^6$/µl) *		5.93 ± 0.14^{B}	4.36 ± 0.47^{A}	3.84 ± 0.38^{A}	3.57 ± 0.26^{A}
4	TLC($x10^3$/ µl) *		11.69 ± 0.20^{AB}	17.17 ± 2.70^{C}	15.93 ± 2.16^{BC}	7.47 ± 0.68^{A}
5	DLC %	N *	66.86 ± 4.00^{A}	71.51 ± 1.76^{AB}	78.71 ± 4.14^{BC}	86.14 ± 2.69^{C}
		L**	24.14 ± 1.71^{C}	22.57 ± 1.11^{BC}	18.57 ± 3.63^{B}	10.71 ± 2.10^{A}
		M *	5.43 ± 0.38^{B}	3.14 ± 0.34^{A}	3.42 ± 0.65^{A}	3.57 ± 0.53^{A}

*Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.05$

**Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.01$

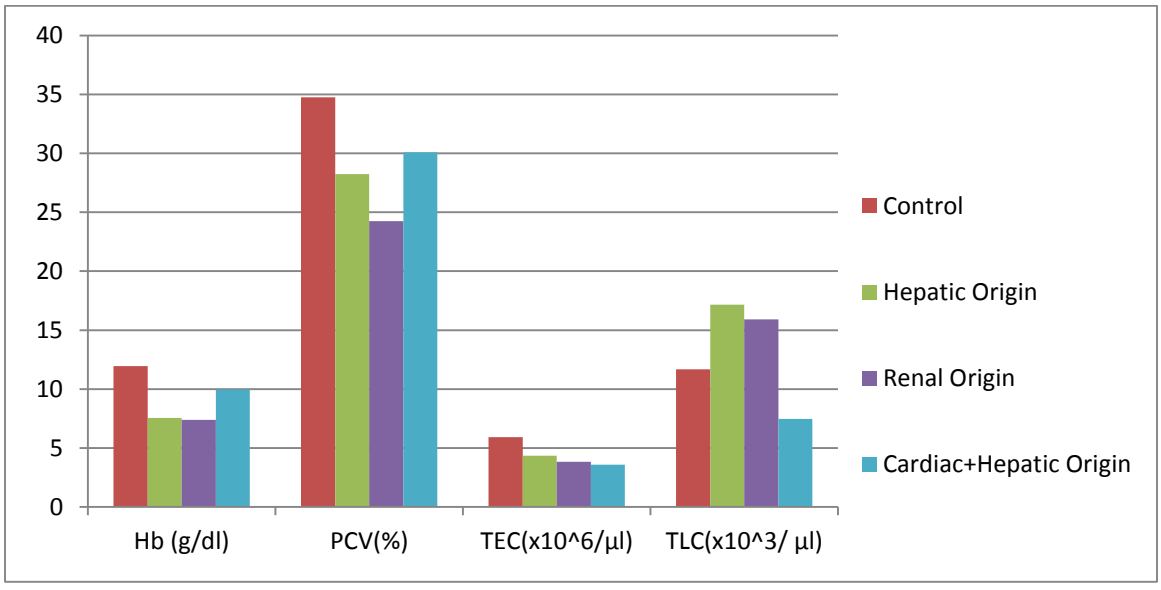


Fig. 9: Hematological alterations in dogs suffering with canine ascites due to different etiologies

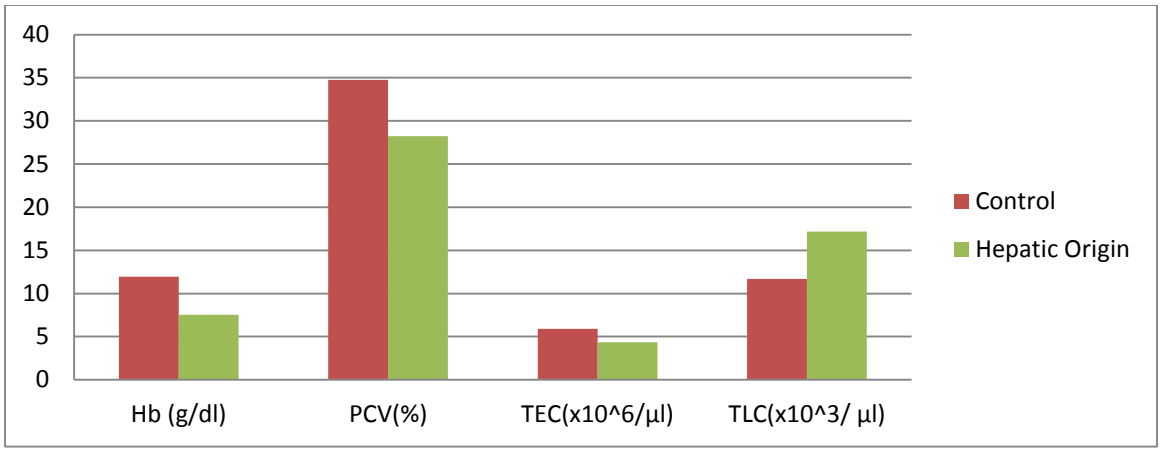


Fig. 10: Hematological alterations in dogs suffering with canine ascites due to hepatic origin

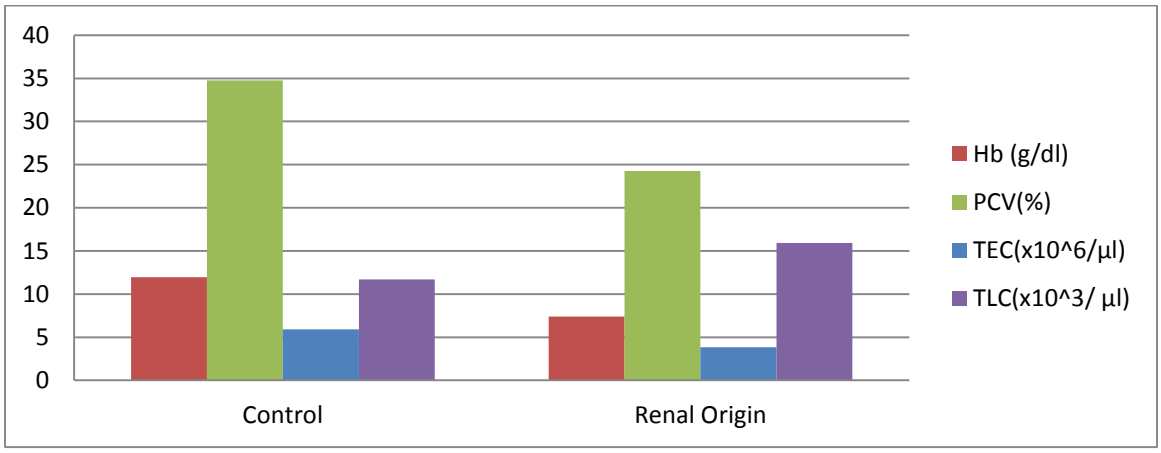


Fig. 11: Hematological alterations in dogs suffering with canine ascites due to renal origin

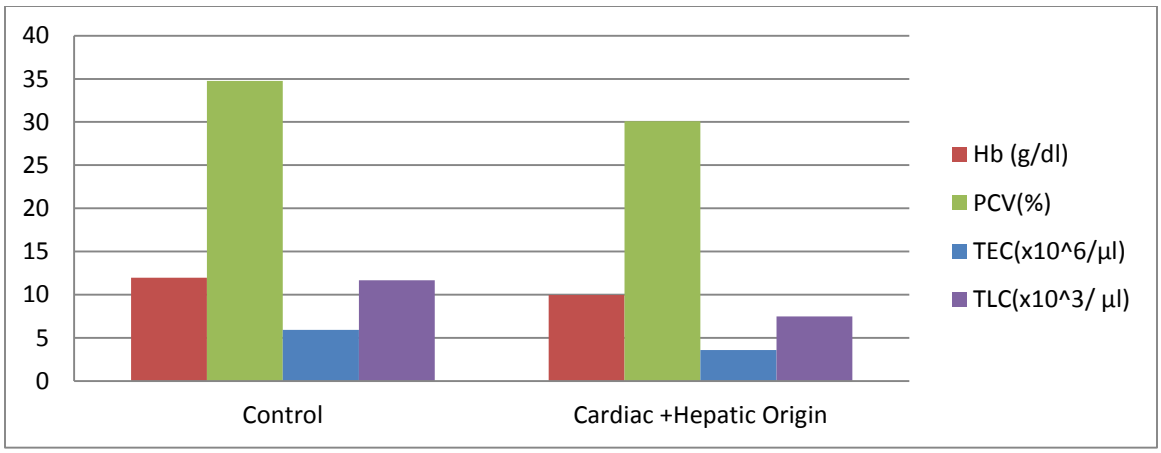


Fig. 12: Hematological alterations in dogs suffering with canine ascites due to both cardiac and hepatic origin

4.4 BIOCHEMICAL PARAMETERS

The biochemical parameter analysis of ascitic dogs of different origin was carried out and significant ($p<0.05$) decrease was observed in total protein and albumin content when compared to normal healthy control. ALT, AST, ALP, BUN and creatinine showed significant ($p<0.05$) difference when compared to healthy controlled. (Table 11, Fig 19, 20 and 21).

4.4.1 Total Plasma Protein

The mean value of total plasma protein of apparently healthy dogs was 6.34±0.18 g/dl and that of ascitic dogs of hepatic, renal and both the cardiac and hepatic origin were 4.53±0.24 g/dl, 5.71±.83 g/dl and 4.34±0.14 g/dl, respectively (Table 11, Fig 19). Significant ($p<0.05$) decrease was observed in the plasma total protein values of ascites of hepatic and both cardiac and hepatic origin whereas non-significantly ($p>0.05$) low total protein values were recorded in the ascites of renal origin when compared to healthy dogs.

4.4.2 Albumin

The mean value of albumin of apparently healthy dogs was 2.98±0.16 g/dl whereas in ascitic dogs of hepatic, renal origin and both the cardiac and hepatic origin were 1.73±0.13 g/dl, 2.40±0.43 g/dl and 2.55±0.15 g/dl, respectively (Table 11, Fig 19). Significant ($p<0.05$) decrease was observed in the albumin values of ascites of hepatic origin whereas non-significantly ($p>0.05$) lower values were observed in the renal and both cardiac and hepatic origin ascites when compared to healthy dogs.

4.4.3 ALT

The mean value of ALT of apparently healthy dogs was 47.85±4.39 IU/L and in ascitic dogs of hepatic origin was 160.14±13.86 IU/L, renal origin was 135.14±31.84 IU/L and both the cardiac and hepatic origin was 125±10.33 IU/L (Table 11, Fig 20). Significant ($p<0.05$) increase was observed in the ALT levels of ascites due to different etiologies when compared to healthy dogs.

4.4.4 AST

The mean value of AST of apparently healthy dogs was 32.57±2.83 IU/L and in ascitic dogs of hepatic origin was 185.14±57.97 IU/L, renal origin was 147.57±26.23 IU/L and both the cardiac and hepatic origin was 83.14±5.23 IU/L (Table 11, Fig 20). Significant ($p<0.05$) increase was observed in the AST levels of ascites of renal and hepatic origin whereas non-significantly ($p>0.05$) increased values were observed in ascites of both cardiac and hepatic origin when compared to healthy dogs.

4.4.5 ALP

The mean value of ALP of apparently healthy dogs was 63.20±1.77 IU/L) and in ascitic dogs of hepatic, renal and both the cardiac and hepatic origin were 562.14±176.05 IU/L, 272.14±52.98 IU/L and 578.71±59.39 IU/L, respectively (Table 11, Fig 20). Significant ($p<0.01$) increase was observed in the ALP levels of ascites due to different etiologies when compared to healthy dogs.

4.4.6 BUN

The mean value of BUN of apparently healthy dogs was 20.42±1.06 mg/dl and the ascitic dogs of hepatic origin was 15.42±3.30 mg/dl, renal origin was 63.14±27.83 mg/dl and both the cardiac and hepatic origin was 21.71±0.68 mg/dl (Table 11, Fig 21). Significant ($p<0.05$) increase was observed in the BUN levels of ascites of renal origin when compared to healthy dogs.

4.4.7 Creatinine

The mean value of creatinine of apparently healthy dogs was 1.29±0.06 mg/dl and the ascitic dogs of hepatic origin was 0.85±0.26 mg/dl, renal origin was 4.16±2.10 mg/dl and both cardiac and hepatic origin was 1.03±0.05 mg/dl (Table 11, Fig 21). Significant ($p<0.05$) increase was observed in the creatinine levels of ascites of renal origin when compared to healthy dogs.

Table 11: Biochemical alterations in dogs suffering with canine ascites due to different origin

S. No.	Parameters	Control	Hepatic	Renal	Cardiac + Hepatic
1	Total protein (g/dl) *	6.34 ± 0.19^{B}	4.53 ± 0.24^{A}	$5.71\pm.83^{B}$	4.34 ± 0.14^{A}
2	Albumin (g/dl) *	2.98 ± 0.15^{B}	1.73 ± 0.13^{A}	2.40 ± 0.43^{B}	2.55 ± 0.15^{B}
3	ALT (IU/L) *	47.85 ± 4.37^{A}	160.14 ± 13.86^{B}	135.14 ± 31.84^{B}	125 ± 10.33^{B}
4	AST (IU/L) *	32.57 ± 2.80^{A}	185.14 ± 57.97^{B}	147.57 ± 26.23^{B}	83.14 ± 5.23^{A}
5	ALP (IU/L) **	63.20 ± 1.79^{A}	562.14 ± 176.05^{C}	272.14 ± 52.98^{B}	578.71 ± 59.39^{C}
6	BUN (mg/dl) *	20.42 ± 1.04^{A}	15.42 ± 3.30^{A}	63.14 ± 27.83^{B}	21.71 ± 0.68^{A}
7	Creatinine (mg/dl) *	1.29 ± 0.08^{A}	0.85 ± 0.26^{A}	4.16 ± 2.10^{B}	1.03 ± 0.05^{A}

*Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.05$

**Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.01$

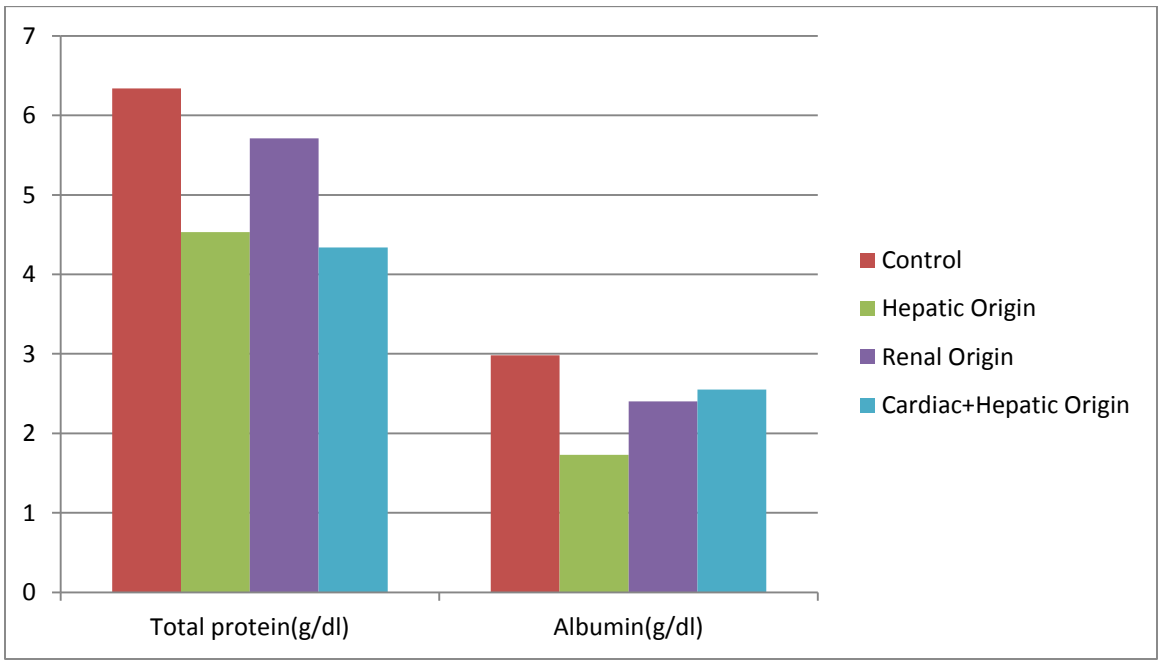


Fig. 13: Biochemical profile (Protein Parameters) of Ascitic Dogs

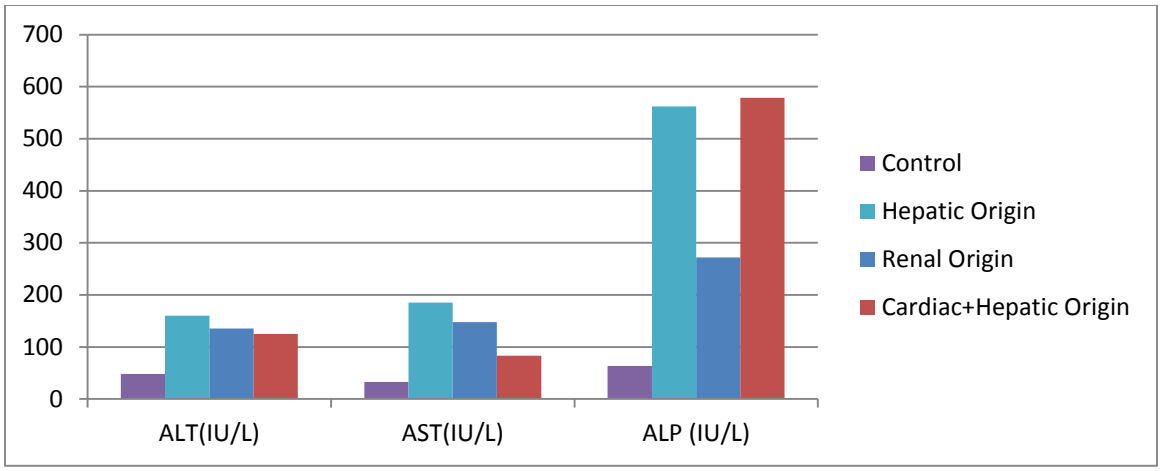


Fig.14: Biochemical profile (Enzymatic Parameters) of Ascitic Dogs

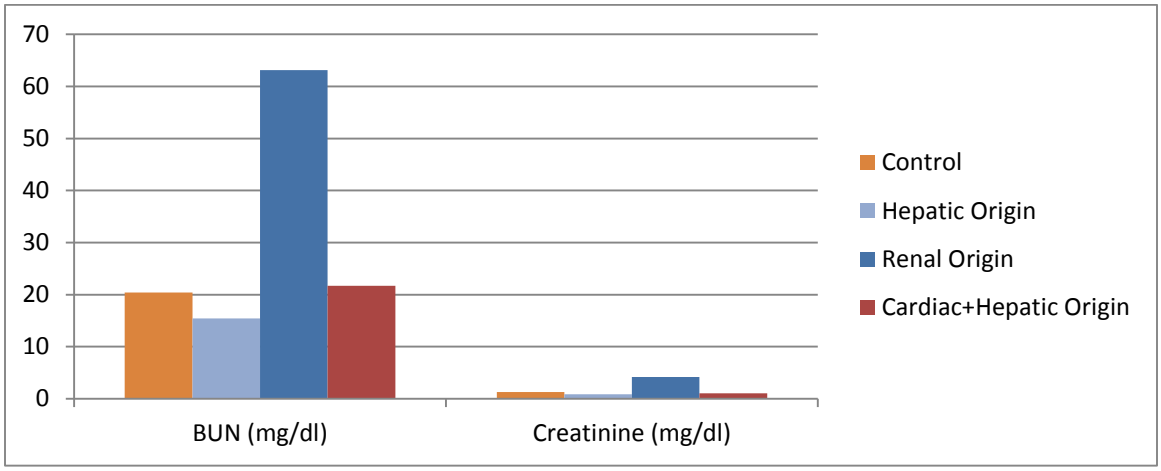


Fig.15: Biochemical profile of (KFT Parameters) of Ascitic Dogs

4.5 THERAPEUTIC STUDIES

The animals were divided into three groups based on clinical, haematobiochemical, radiographic and ultrasonographic examinations as follows:

- **Hepatic ascites group:** Diuretics, Liver tonics, IV Colloids, Supportive therapy.
- **Renal ascites group:** Diuretics, Antibiotics therapy, ACE inhibitor, Dietary management, Supportive therapy.
- **Cardiac and Hepatic ascites group**: Diuretics, Cardiac Glycosides, ACE inhibitors, Liver tonics, Supportive therapy.

Effect of therapeutic management on hematological and biochemical parameters were recorded on 15th and 45th day post treatment.

4.5.1 HEMATOLOGICAL STATUS

The hematological values post treatment were recorded in the dogs affected with ascites due to different etiologies are presented in Table 12a and 12b, Fig 16, 17 and 18. A significant difference was observed in all the hematological parameters between the days within the groups.

4.5.1.1 Hemoglobin

The mean Hb values of ascitic dogs due to hepatic origin were 7.45±0.86 gm%, 9.05±0.80 gm% and 11.44±0.64 gm% on day 0, 15th and 45th, respectively. In ascitic dogs of renal origin, the mean Hb values were 7.38±0.18 gm%, 8.63±0.25 gm%, and 10.82±0.42 gm% on day 0, 15th and 45th, respectively. Similarly, the Hb values of dogs with ascites of both cardiac and hepatic origin were 9.97±0.22 gm%, 10.56±0.25 gm% and 11.17±0.91 gm% on day 0, 15th and 45th. Significant ($p<0.05$) improvement was observed in Hb concentration in hepatic and renal origin on day 45 whereas non-significant ($p>0.05$) difference was observed in ascites of both Cardiac and Hepatic origin (Table 12a, Fig 16, 17 and 18).

4.5.1.2 Packed Cell Volume

Mean ± SE value of PCV in dogs with ascites of hepatic origin was 28.24±2.34 percent on day 0 which showed significant ($p<0.01$) increase on day 45 (34.57±0.92 percent). The mean values of PCV in ascites due to renal origin on day 0 was 24.25±1.46 percent which showed significant ($p<0.01$) increase on day 45 (37.58±1.00 percent) whereas, in dogs with ascites of cardiac and hepatic origin showed non-significant difference in the values of PCV on day 0 (30.08±0.25 percent) and 33.03±0.46 percent on day 45 (Table 12a, Fig 16, 17 and 18).

4.5.1.3 Total Erythrocyte Count

The mean TEC value of ascitic dogs due to hepatic origin on day 0 was 4.35±0.47 $\times 10^6/\mu l$ which showed significant ($p<0.05$) increase on day 45th (6.23±0.72$\times 10^6/\mu l$).

Mean ±SE value of TEC in ascites of renal origin was $3.84\pm0.38\times10^6$/µl on day 0 which showed significant ($p<0.05$) increase to $6.37\pm0.31\times10^6$/µl on day 45th. The mean TEC value of dogs with ascites of cardiac and hepatic origin was $3.57\pm0.25\times10^6$/µl on day 0 and which showed significant ($p<0.05$) increase on day 45th ($5.09\pm0.21\times10^6$/µl) (Table 12a, Fig 16, 17 and 18).

4.5.1.4 Total Leukocytes Count

The mean TLC value of ascitic dogs due to hepatic origin were 17.17 ± 2.70 x10^3 / µl, 11.64 ± 0.88 x10^3 / µl and 12.57 ± 2.85 x10^3 / µl on day 0, 15th and 45th, respectively. In ascitic dogs of renal origin, the mean TLC value was 15.93 ± 2.16 x10^3 / µl, 13.56 ± 2.30 x10^3 / µl and 11.81 ± 2.58 x10^3 / µl on day 0, 15th and 45th, respectively. Similarly, the mean TLC value dogs with ascites of both cardiac and hepatic origin were 7.47 ± 0.68 x10^3/ µl, 8.04 ± 0.61 x10^3/ µl and 10.42 ± 0.40 x10^3/ µl on day 0, 15th and 45th, respectively. The TLC value significantly ($p<0.05$) decreased in hepatic group on day 15th whereas non-significant ($p>0.05$) difference was observed on day 45th (Table 12a, Fig 16, 17 and 18).

4.5.1.5 Differential Leukocytes Count

4.5.1.5.1 Neutrophils

The mean Neutrophil count of dogs with ascites of renal origin showed significant ($p<0.05$) decrease in values from 78.71 ± 4.14 percent on day 0 to 64.71 ± 3.01 percent on 45th day. Similarly, dogs with ascites of cardiac and hepatic origin showed significant ($p<0.05$) decrease in values of neutrophil count from 86.14 ± 2.69 percent on day 0 to 68.86 ± 1.87 percent on day 45th (Table 12b).

4.5.1.5.2 Lymphocytes

The mean Lymphocyte count of dogs with ascites of hepatic origin showed significant ($p<0.01$) decrease in values from 22.57 ± 1.11 percent on day 0 to 15.29 ± 1.98 percent on 45th day. Similarly, dogs with ascites of cardiac and hepatic origin showed significant ($p<0.01$) increase in values of lymphocyte count from 10.71 ± 2.10 percent on day 0 to 23.14 ± 1.18 percent on day 45th (Table 12b).

4.5.1.5.3 Monocytes

The mean monocyte count of dogs with ascites of cardiac and hepatic origin showed significant ($p<0.05$) increase in values from 3.57±0.53 percent on day 0 to 5.14±0.34 percent on day 45th, respectively whereas other groups showed non-significant ($p>0.05$) increase in the monocyte count (Table 12b).

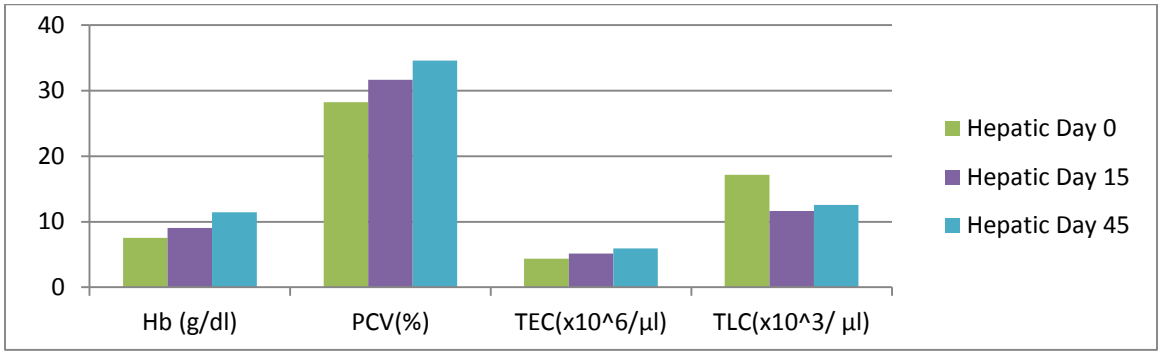


Fig. 16: Effect of Treatment on hematological parameters in canine ascites of hepatic origin

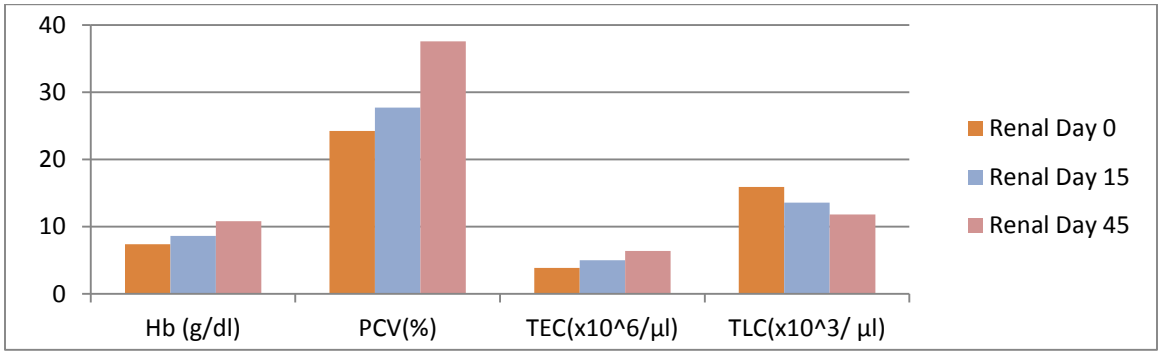


Fig. 17: Effect of Treatment on hematological parameters in canine ascites of renal origin

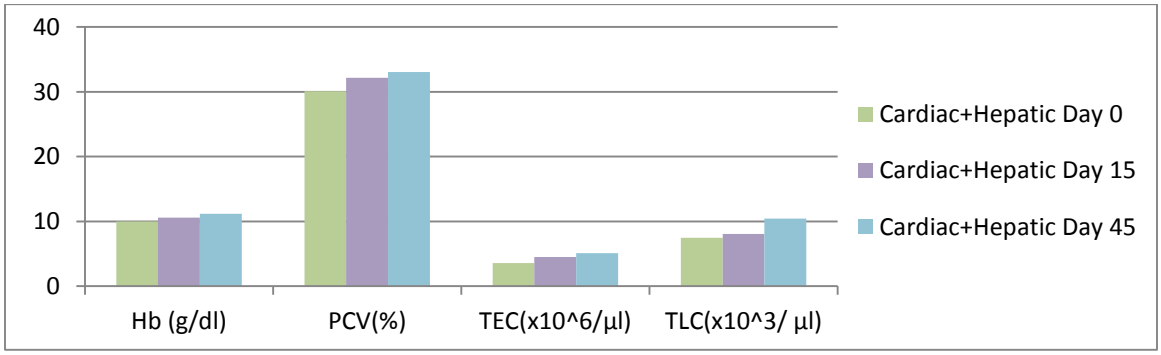


Fig. 18: Effect of Treatment on hematological parameters in canine ascites of both cardiac and hepatic origin

Table 12(a): Effect of treatment on hematological parameters in canine ascites (Mean±SE)

Groups	Hb (gm %) *			PCV (%) **			TEC (x10^6/ µl) *			TLC (x10^3/ µl) *		
	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45
Hepatic	7.54±0.86^{aA}	8.91±0.81^{aA}	10.66±1.16^{b}	28.24±2.34^{aAB}	32.73±2.25^{bB}	38.11±3.33^{cB}	4.36±0.47^{A}	5.17±0.64^{abAB}	6.23±0.72^{bAB}	17.17±2.70^{bC}	11.64±0.88a^{AB}	12.57±2.90^{ab}
Renal	7.39±0.19^{aA}	8.63±0.26^{aA}	10.83±0.42^{b}	24.26±1.46^{aA}	27.71±1.78a^{A}	37.59±1.00^{bAB}	3.84±0.38^{aA}	4.99±0.50^{bAB}	6.37±0.31^{cB}	15.93±2.16^{BC}	13.56±2.30^{B}	11.81±2.58
Cardiac+ Hepatic	9.97±0.2^{B}	10.56±0.25^{B}	11.17±0.19	30.09±0.26^{B}	32.16±0.44^{B}	33.03±0.46^{A}	3.57±0.26^{aA}	4.16±0.21^{abA}	5.09±0.21^{bA}	7.47±0.68^{A}	8.04±0.61^{A}	10.43±0.40

*Different superscript A, B, C indicate significant difference between columns at p<0.05 and different superscript a, b, c indicate significant difference between rows at p<0.05

**Different superscript A, B, C indicate significant difference between columns at p<0.01 and different superscript a, b, c indicate significant difference between rows at p<0.01

Table 12(b): Effect of treatment on hematological parameters in canine ascites (Mean±SE)

Groups	Neutrophils (%) *			Lymphocytes (%) **			Monocytes (%) *		
	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45
Hepatic	71.57±1.76^{AB}	68.29±1.99^{A}	64.43±2.10	22.57±1.11^{bBC}	21.14±1.62^{bAB}	15.29±1.98^{aA}	3.14±0.34^{A}	3.57±0.37^{A}	3.29±0.42^{A}
Renal	78.71±4.14^{bBC}	70.29±3.56^{abAB}	64.71±3.01^{a}	18.57±3.63^{B}	20.86±0.94^{AB}	23.57±0.90^{B}	3.43±0.65^{A}	3.86±0.55^{A}	4.14±0.40^{AB}
Cardiac + Hepatic	86.14±2.69^{bC}	77.57±1.61^{bB}	68.86±1.87^{a}	10.71±2.10^{aA}	17.29±1.17^{bA}	23.14±1.18^{cB}	3.57±0.53^{aA}	4.71±0.52^{abAB}	5.14±0.34^{bB}

*Different superscript A, B, C indicate significant difference between columns at p<0.05 and different superscript a, b, c indicate significant difference between rows at p<0.05

**Different superscript A, B, C indicate significant difference between columns at p<0.01 and different superscript a, b, c indicate significant difference between rows at p<0.01

4.5.2 BIOCHEMICAL STATUS

The biochemical parameter analysis of ascitic dogs of different origin was carried out post treatment and significant differences were observed in the values of total protein albumin content, ALT, AST, ALP, BUN and creatinine when compared within days between the groups (Table 13a and 13b, Fig 19, 20, 21, 22, 23, 24, 25, 26 and 27).

4.5.2.1 Total Plasma Protein

The mean total plasma protein (TPP) level in hepatic group ranged from 4.53±0.24 g/dl, 5.24±0.18g/dl and 6.02±0.28 g/dl on day 0, 15th and 45^{th,} respectively. In renal group, the mean TPP ranged from 5.71±0.83 g/dl, 5.87±0.45 g/dl and 6.48±0.26g/dl on day 0, 15th and 45th, respectively. Likewise the mean TPP values of cardiac and hepatic group ranged from 4.34±0.14 g/dl 4.60±0.11 and 4.94±0.09 g/dl on day 0, 15th and 45th, respectively. Significant (P<0.05) increase was observed in TPP value of hepatic group day 0 to 45th day (Table 13a, Fig. 19, 20 and 21).

4.5.2.2 Albumin

Mean ±SE value of Albumin in hepatic group was 1.73±0.13 g/dl on day 0 which showed significant (p<0.05) increase on day 45 (3.03±0.30 g/dl). The mean values of albumin in renal group on day 0 was 2.41±0.44 g/dl which showed significant (p<0.05) increase on day 45 (3.78±0.31 g/dl) whereas, cardiac and hepatic group showed non-significant difference in the values of albumin on day 0 (2.55±0.15 g/dl) and 3.14±0.12 on day 45 (Table 13a, Fig. 19, 20 and 21).

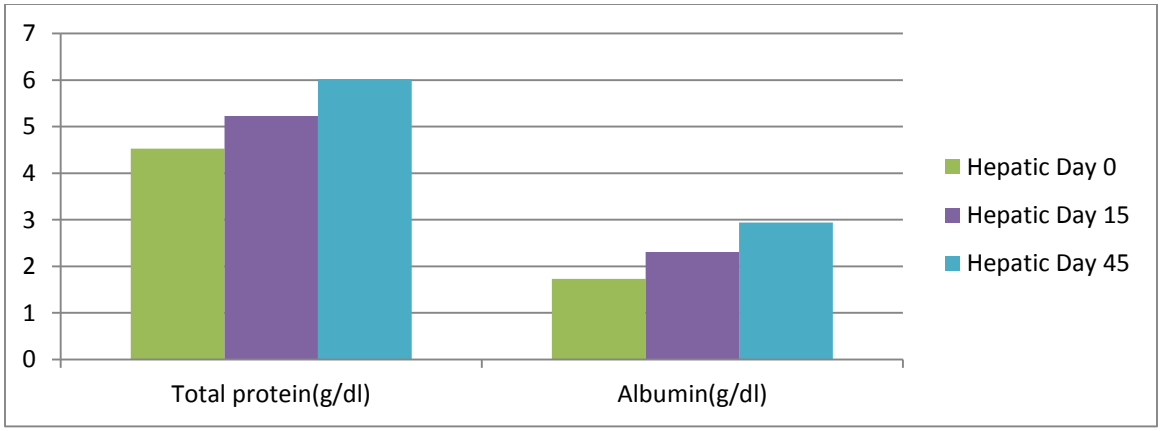


Fig. 19: Effect of treatment on Biochemical profile (Protein Parameters) in canine ascites of Hepatic origin

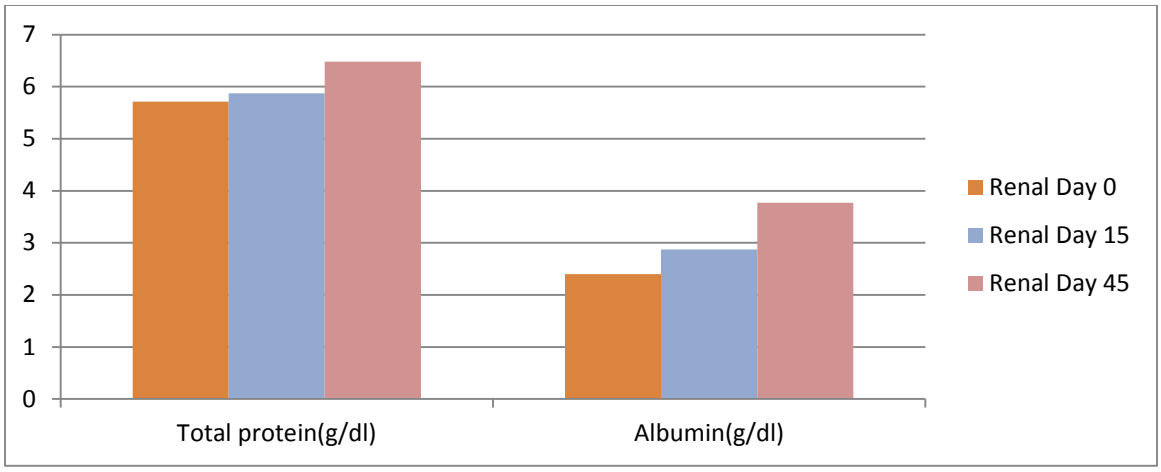


Fig. 20: Effect of treatment on Biochemical profile (Protein Parameters) in canine ascites of renal origin

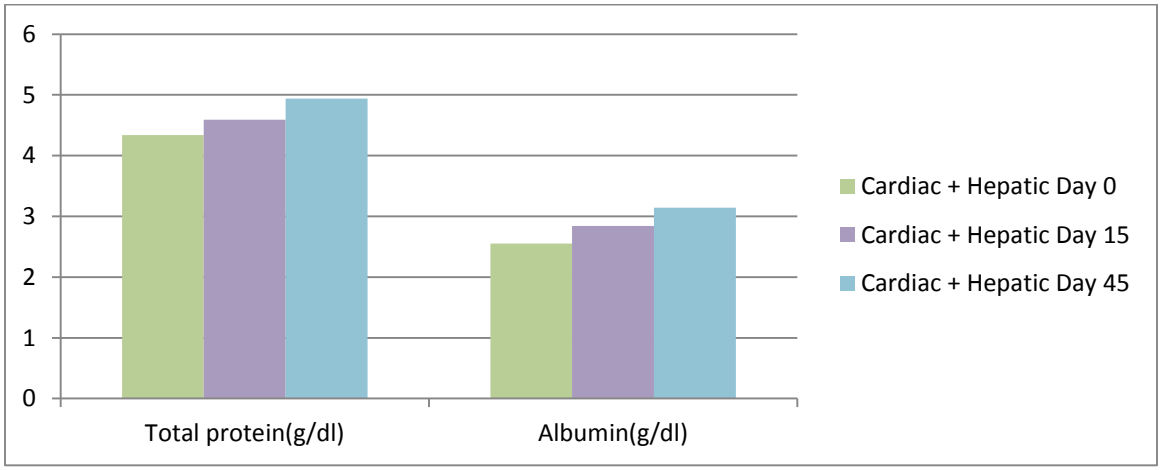


Fig. 21: Effect of treatment on Biochemical profile (Protein Parameters) in canine ascites of both Cardiac and Hepatic origin

4.5.2.3 ALT

The mean ALT values of hepatic group ranged from 160.14±13.86 IU/L at 0 day which showed significant ($p<0.05$) decrease after treatment to 54.57±4.24 IU/L at 45th day. In renal group the mean ALT value was 135±31.84 IU/L at 0 day which significantly ($p<0.05$) decreased to 61.71±8.20 IU/L on 45th day. The mean ALT value of cardiac and hepatic group on day 0 was 125±10.33 IU/L which significantly ($p<0.05$) decreased to 43.85±2.63 IU/L on 45th day (Table 13a, Fig 22, 23 and 24).

4.5.2.4 AST

The mean AST of hepatic group ranged from 185.14±57.97 IU/L on 0 day significantly ($p<0.05$) decreased to 49.28±4.41 IU/L on 45th day. In renal group, the mean AST value significantly ($p<0.05$) decreased from 147±26.23 IU/L on 0 day to 45.85±5.62 IU/L on 45th day (Table 13a, Fig 22, 23 and 24).

4.5.2.5 ALP

The mean ALP value in hepatic group significantly ($p<0.01$) decreased from 562.14±176.05 IU/L on 0 day to 160.42±59.02 IU/L on 45th day. In renal group, the mean ALP value was 272.14±52.98 IU/L on day 0 which showed significant ($p<0.01$) decrease to 90.28±11.93 IU/L on 45th day (Table 13b, Fig 22, 23 and 24).

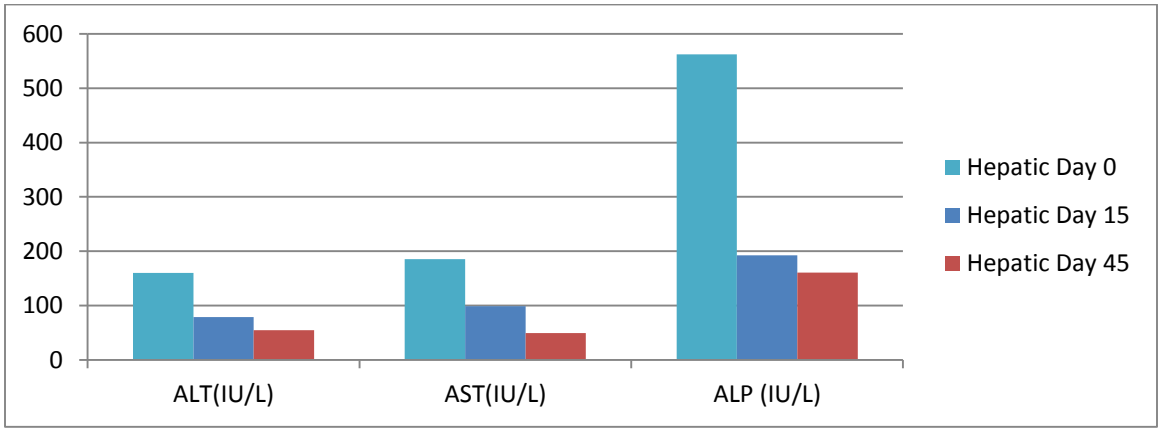


Fig. 22: Effect of treatment on Biochemical profile (Enzymatic Parameters) in canine ascites of Hepatic origin

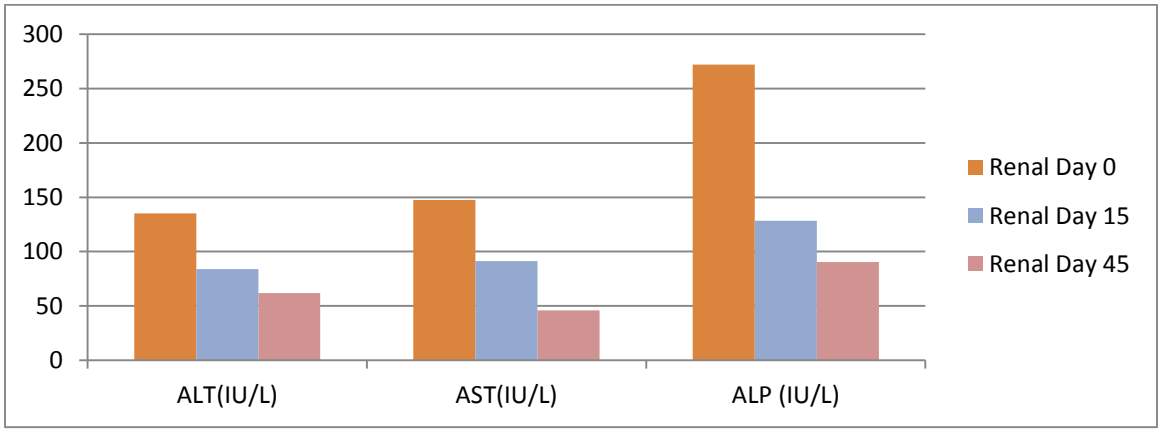


Fig. 23: Effect of treatment on Biochemical profile (Enzymatic Parameters) in canine ascites of renal origin

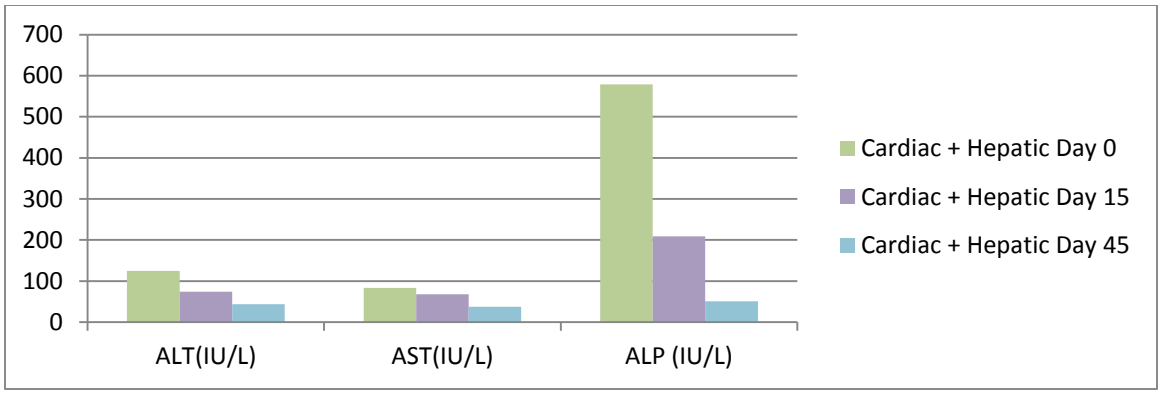


Fig. 24: Effect of treatment on Biochemical profile (Enzymatic Parameters) in canine ascites of both cardiac and hepatic origin

4.5.2.6 BUN

The mean BUN value in renal group showed significant ($p<0.05$) decrease from 63.14±27.83 IU/L on 0 day to 18.14±2.60 IU/L on 45th day. Non- significant difference were observed in the BUN value of hepatic group and both cardiac and hepatic group on day 0 and 45th day (Table 13, Fig 25, 26 and 27).

4.5.2.7 Creatinine

The mean Creatinine value in renal group showed significant ($p<0.05$) decrease from 4.16±2.10 IU/L on 0 day to 1.17±0.21 IU/L on 45th day. Non- significant difference were observed in the creatinine value of hepatic group and both cardiac and hepatic group on day 0 and 45th day (Table 13b, Fig 25, 26 and 27).

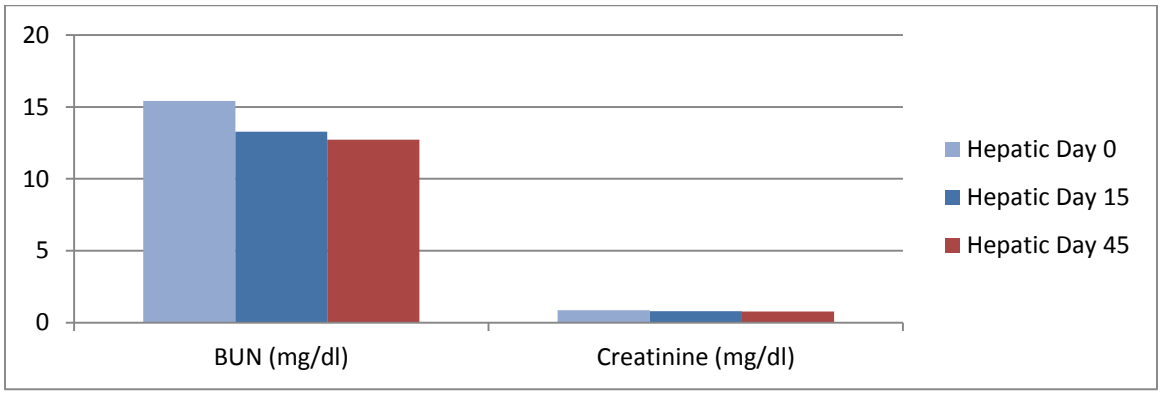


Fig. 25: Effect of treatment on Biochemical profile of (KFT Parameters) in canine ascites of Hepatic origin

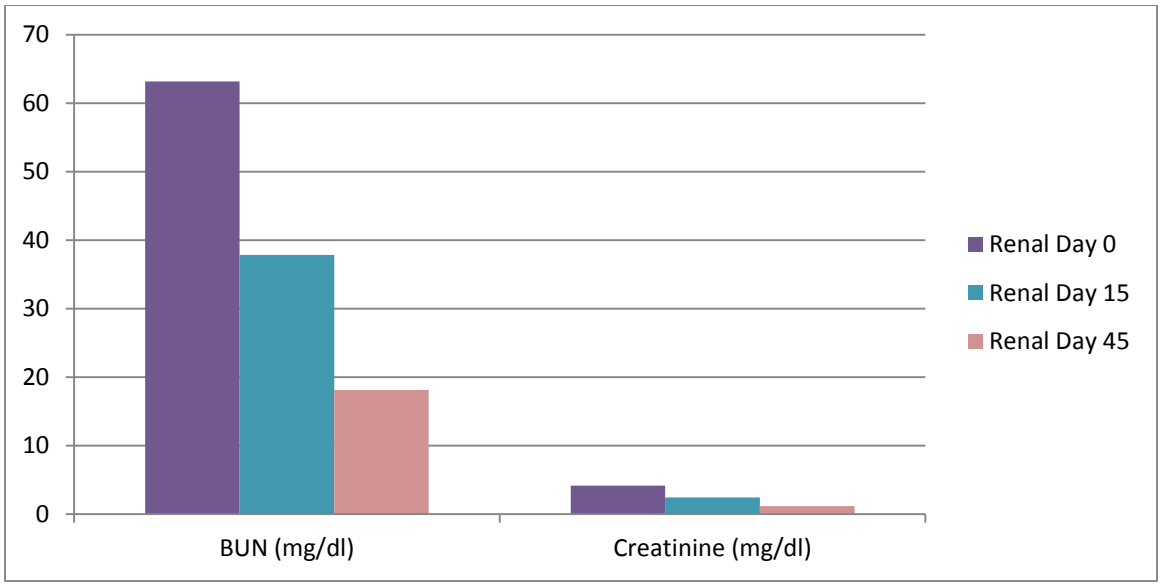


Fig. 26: Effect of treatment on Biochemical profile of (KFT Parameters) in canine ascites of renal origin

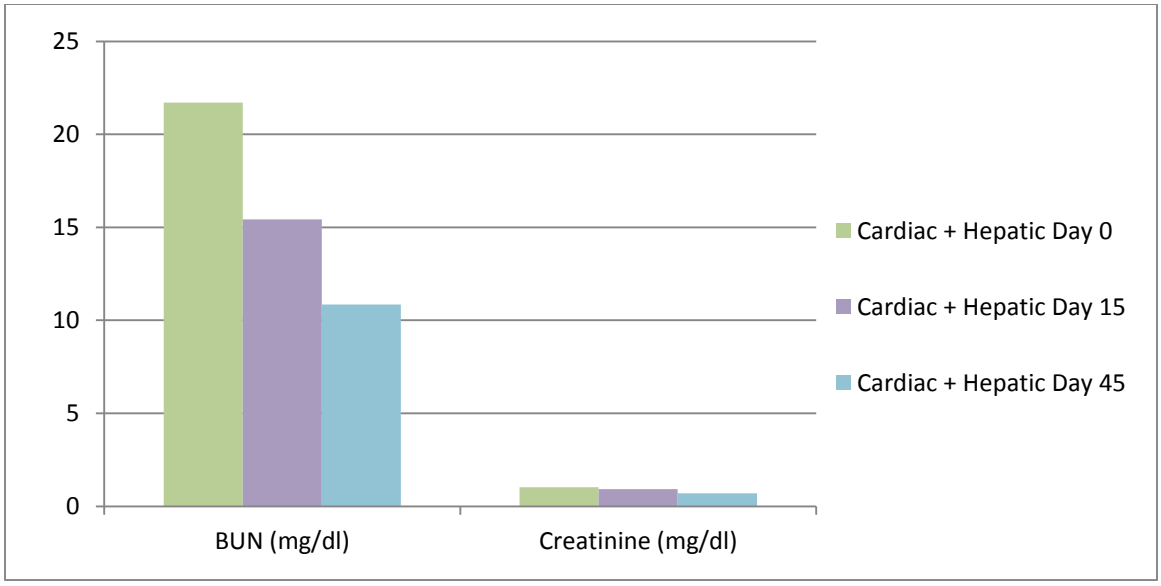


Fig. 27: Effect of treatment on Biochemical profile of (KFT Parameters) in canine ascites of both cardiac and hepatic origin

Table 13(a): Effect of treatment on biochemical parameters in canine ascites (Mean±SE)

Groups	TP (g/dl) *			Alb (g/dl) *			ALT (IU/L) *			AST (IU/L) *		
	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45
Hepatic	4.53 ± 0.24^{aA}	5.24 ± 0.18^{abAB}	6.03 ± 0.28^{B}	1.74 ± 0.13^{aA}	2.32 ± 0.28^{a}	3.03 ± 0.30^{bA}	160.14 ± 13.86^{bB}	78.71 ± 12.60^{a}	54.57 ± 4.25^{a}	185.14 ± 57.97^{bB}	98.71 ± 26.36^{aB}	49.29 ± 4.41^{a}
Renal	5.71 ± 0.83^{B}	5.87 ± 0.45^{BC}	6.49 ± 0.27^{B}	2.41 ± 0.44^{aB}	2.87 ± 0.15^{a}	3.78 ± 0.31^{bB}	135.14 ± 31.84^{bB}	83.86 ± 15.11^{a}	61.71 ± 8.20^{a}	147.57 ± 2.62^{bB}	91.28 ± 1.88^{abAB}	45.86 ± 5.63^{a}
Cardiac+ Hepatic	4.34 ± 0.14^{A}	4.60 ± 0.11^{A}	4.95 ± 0.09^{A}	2.55 ± 0.15^{B}	2.84 ± 0.13	3.14 ± 0.12^{AB}	125.14 ± 1.03^{bB}	74.42 ± 5.20^{a}	43.86 ± 2.63^{a}	83.14 ± 5.23^{A}	68.29 ± 3.59^{AB}	37.29 ± 3.83

*Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.05$ and different superscript $^{a, b, c}$ indicate significant difference between rows at $p<0.05$

**Different superscript $^{A, B, C}$ indicate significant difference between columns at $p<0.01$ and different superscript $^{a, b, c}$ indicate significant difference between rows at $p<0.01$

Table 13(b): Effect of treatment on biochemical parameters in canine ascites (Mean±SE)

Groups	ALP (IU/L) **			BUN (mg/dl) *			Creatinine (mg/dl) *		
	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45	Day 0	Day 15	Day 45
Hepatic	562.14 ± 176.05^{bC}	192.28 ± 38.94^{a}	160.42 ± 59.02^{a}	15.43 ± 3.30^{A}	13.29 ± 1.44	12.71 ± 1.64	0.86 ± 0.26^{A}	0.81 ± 0.13	0.78 ± 0.05
Renal	272.14 ± 5.30^{bB}	129.43 ± 1.89^{ab}	90.29 ± 1.19^{a}	63.14 ± 27.83^{bB}	37.86 ± 16.72^{ab}	18.14 ± 2.60^{a}	4.16 ± 2.10^{bB}	2.46 ± 1.25^{ab}	1.17 ± 0.21^{a}
Cardiac+ Hepatic	578.71 ± 59.40^{bC}	209.14 ± 36.26^{a}	50.71 ± 5.73^{a}	21.71 ± 0.68^{A}	15.42 ± 0.78	10.86 ± 1.22	1.03 ± 0.05^{A}	0.93 ± 0.03	0.71 ± 0.04

*Different superscript A, B, C indicate significant difference between columns at $p<0.05$ and different superscript a, b, c indicate significant difference between rows at $p<0.05$

**Different superscript A, B, C indicate significant difference between columns at $p<0.01$ and different superscript a, b, c indicate significant difference between rows at $p<0.01$

4.6 PERITONEAL FLUID ANALYSIS

Peritoneal fluid was collected aseptically in the sterile tubes and examined for color, consistency and pH. Majority of the samples collected were clear however, a few samples were turbid in nature. Other samples were yellow/straw coloured and some sample were serosanguinous. The amount of fluid varied from of 0.5 to 3 liters. The mean value of pH varied non-significantly ($p>0.05$) among the different etiologies of ascites in dogs. Cytological smear prepared out of samples showed presence of different types of cells such as mesothelial cells, neutrophils and few erythrocytes (Plate 3).

The mean value of total protein present in the ascetic fluid of the dogs with ascites due to hepatic origin (2.49±0.12 g/dl) was significantly higher ($p<0.05$) than that of dogs with ascites of renal origin (1.95±0.29 g/dl) or both cardiac and hepatic origin (1.58±0.27 g/dl).

The value of total cell count of the peritoneal fluid was significantly ($p<0.05$) higher in dogs with ascites of hepatic origin (262.86±61.91 /µl) than ascites of cardiac and hepatic origin (194.43±34.51 /µl) and renal origin (101.86±10.91/µl) (Table 14, Fig 28).

The mean value of SAAG was significantly ($p<0.05$) higher in ascites of both cardiac and hepatic group (1.54±0.18) than the ascites of hepatic group (1.08±0.14) and renal group (1.36±0.14) (Table 14, Fig 28).

Table 14: **Characteristics of ascitic fluid obtained from the ascitic dogs classified according to their specific etiologies**

Groups	pH	Total Cell Count (/µl)	Total Protein (g/dl)	Albumin (g/dl)	SAAG
Hepatic	7.28± 0.18	262.86±61.91^{b}	2.49±0.12^{b}	0.63±0.11	1.08±0.14^{a}
Renal	7.43± 0.20	101.86±10.91^{a}	1.95±0.29^{ab}	1.05±0.32	1.36±0.14^{a}
Cardiac + Hepatic	7.43± 0.20	194.43±34.51^{a}	1.58±0.27^{a}	1.01±0.12	1.54±0.18^{b}

Different superscript a, b, c indicate significant difference between rows at $p<0.05$

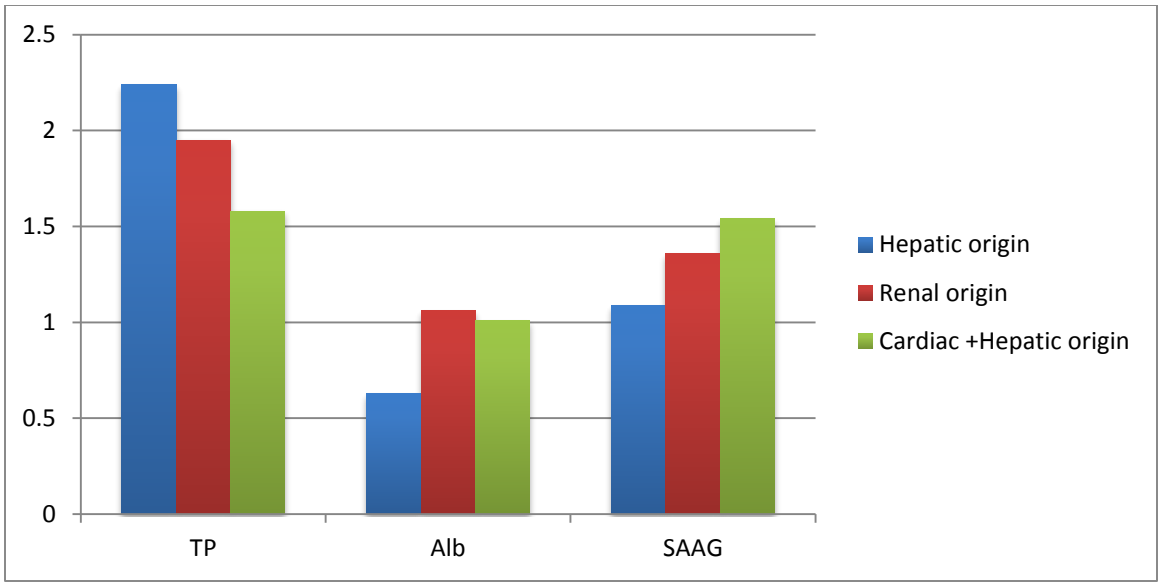


Fig. 28: Ascitic fluid characteristics in ascitic dogs of different causative types

4.7 RADIOGRAPHIC FINDINGS

All ascitic dogs were subjected to radiographic examination to find out involvement of different visceral organs. Standard radiographic factors were used (mAs) to avoid errors. Ventro-dorsal and lateral radiographs showed distended radiopaque abdomen, glass ground appearance along with the loss of details of soft tissue in abdominal cavity. There was pressure on diaphragm exhibited by arc of contour. The thoracic radiographic findings involved mild congestion of lungs and enlargement of heart whereas the abdominal radiographic findings were intestinal loops floating in the ascitic fluid, loss of liver density, ground glass appearance, free fluid in abdomen (Plate 3).

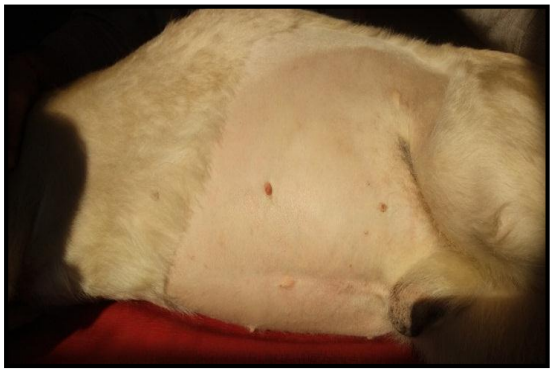
4.8 ULTRASONOGRAPHIC FINDINGS

Standard B-mode ultrasonographic examination of abdomen was adopted in all the cases. The abdominal cavity was examined in both transverse planes. All the ascitic animals of hepatic, renal and both cardiac and hepatic origin showed mild to extensive anechoic area in abdominal cavity suggestive of fluid accumulation (Plate 4). This technique was found to be useful in differentiating the conditions related to pyometra, mummified fetus and bladder rupture. Abdominal ultrasonography was performed in 15

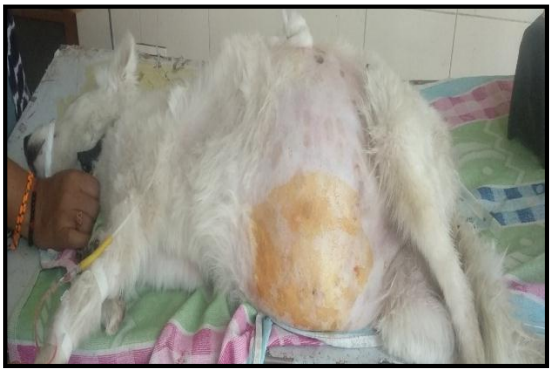
dogs where liver size was subjectively assessed to be small (n=5), normal (n=8) and enlarged (n=2) in 5, 8 and 2 dogs. Increased echogenicity of liver in comparison to the spleen was identified in 5 dogs. Kidney size was subjectively assessed to be small (n=5), normal (n=7) and enlarged (n=3). Increased cortical echogenicity of kidney was observed in 3 dogs.

4.9 ELECTROCARDIOGRAPHIC FINDINGS

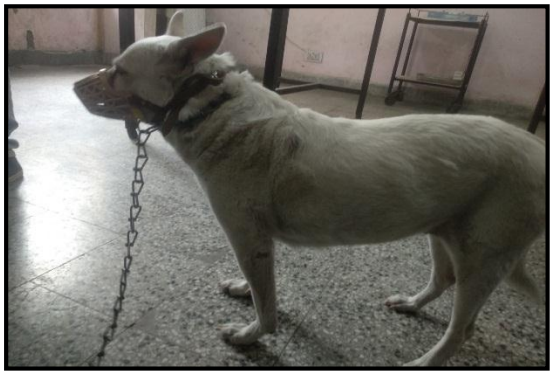
Electrocardiographic findings were recorded in the present study to reveal involvement and functioning of heart. Ascitic cases which were not of hepatic and renal origin were subjected to the ECG after radiographic and ultrasonographic examination to rule out the cardiac involvement for ascites formation in dogs. The notable changes observed in ECG were: absence of P wave representing atria fibrillation, ST elevation and inverted T wave indicating myocardial infarction, only two cases showed no major alterations except sinus arrhythmias which were not alarming (Plate 5).



A: Abdominal swelling

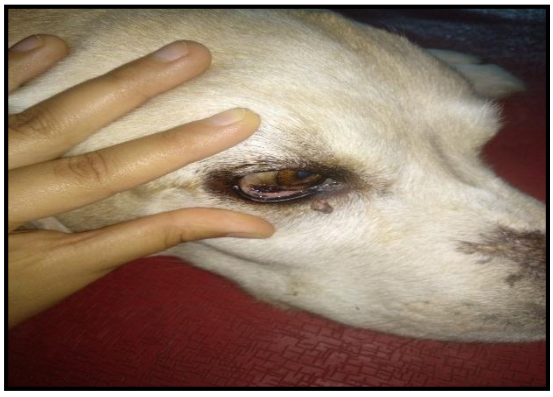


B: Swollen abdomen

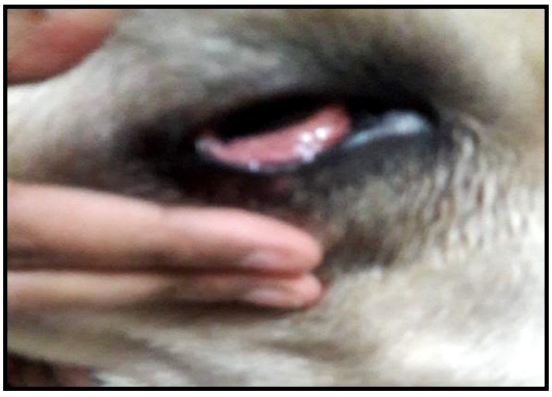


C: Swollen abdominal region with rough body coat

D: Pale mucus membrane



E: Yellow mucus membrane

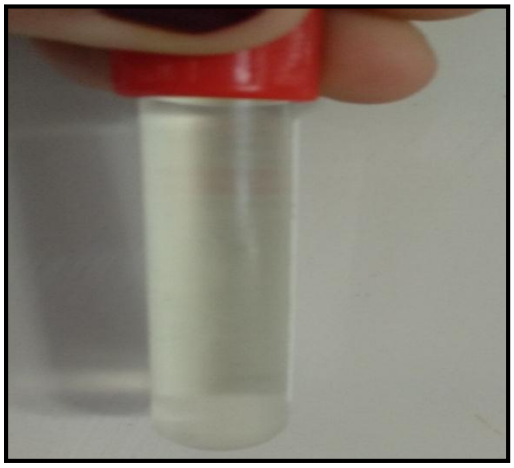


F: Pink mucus membrane

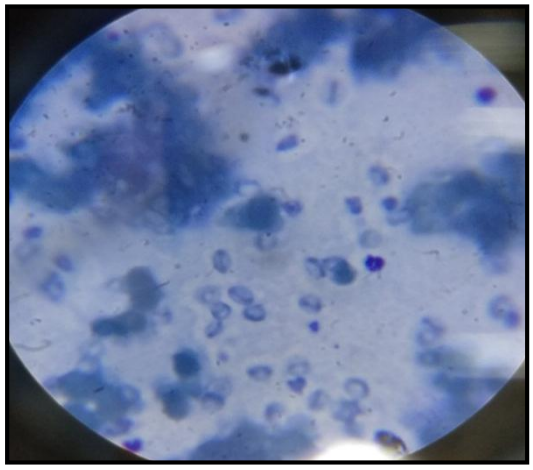
Plate 1: Different clinical signs in canine ascitic cases

A: Straw coloured Ascitic Fluid

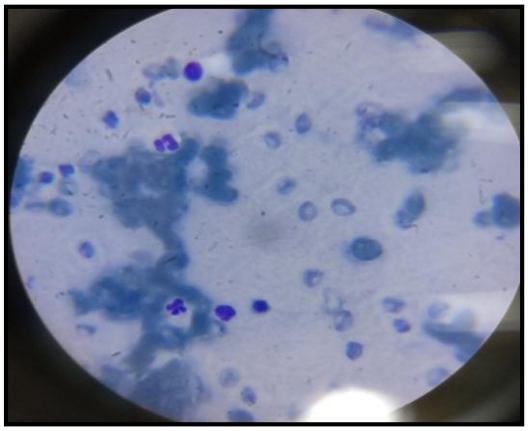
B: Reddish tinged color Ascitic Fluid



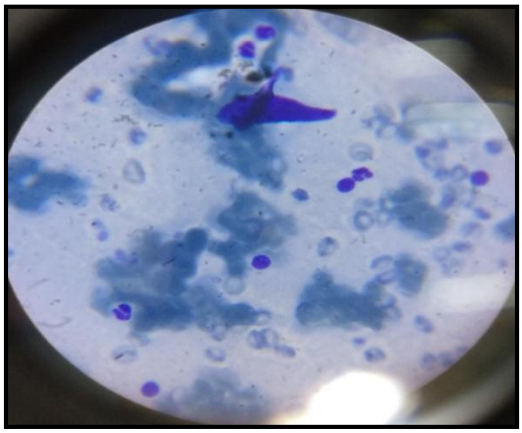
C: Clear watery Ascitic Fluid



D: Cytological smear of ascitic fluid with mesothelial cells

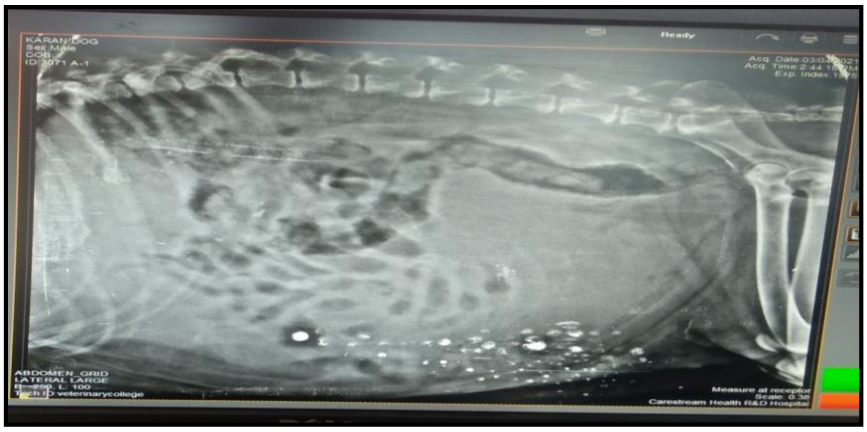


E: Cytological smear of ascitic fluid with neutrophils

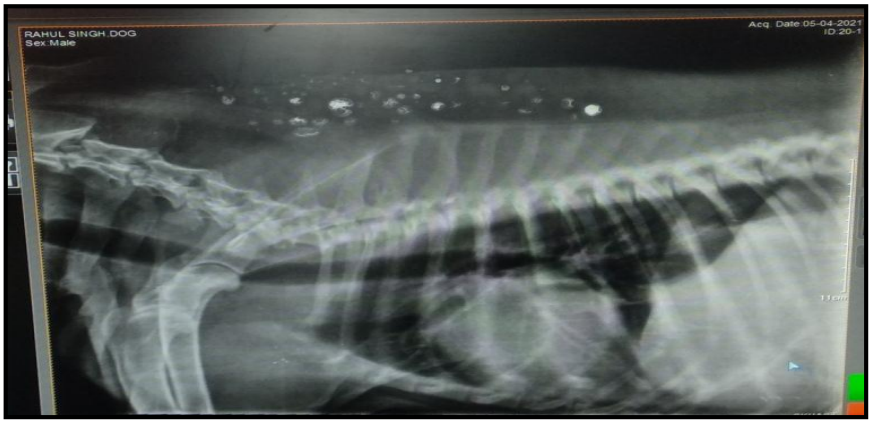


F: Cytological smears of ascitic fluid with WBCs

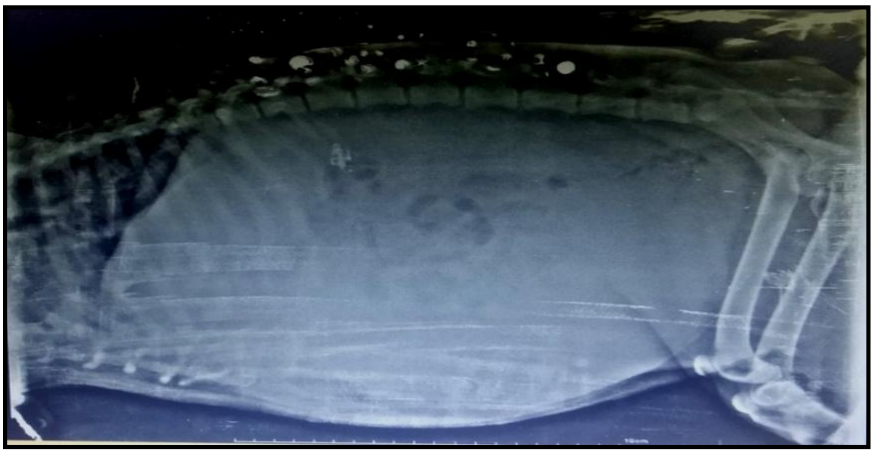
Plate 2: Peritoneal fluid analysis of canine ascitic cases



A: Radiographic view of abdominal cavity showing ground glass appearance

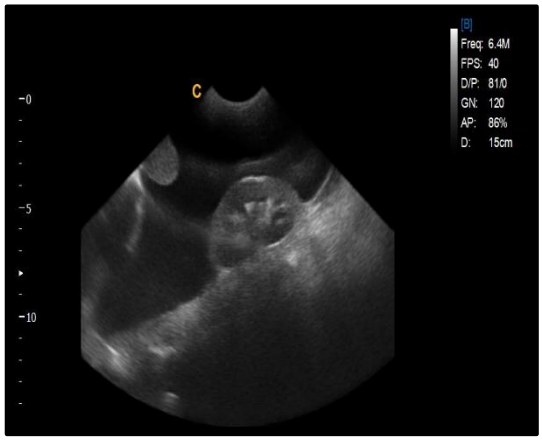


B: Radiographic view of thoracic cavity to detect abnormality of heart

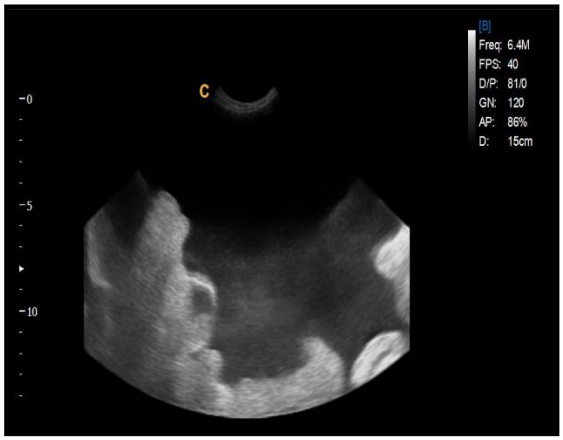


C: Radiograph view of abdominal cavity

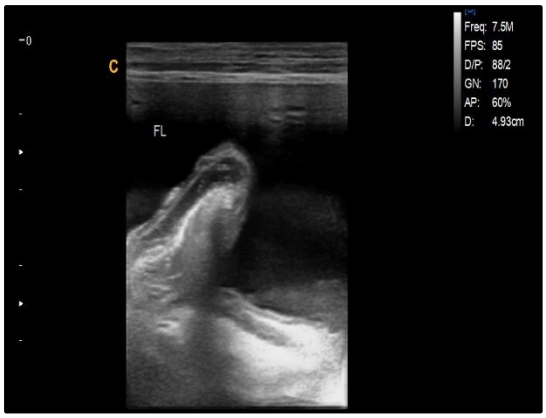
Plate 3: Radiographic findings of canine ascitic cases



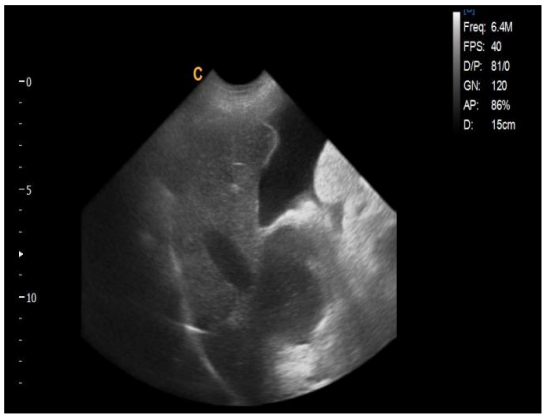
A: Ultrasonographic view of internal organ



B: Ultrasonographic view of ascitic fluid

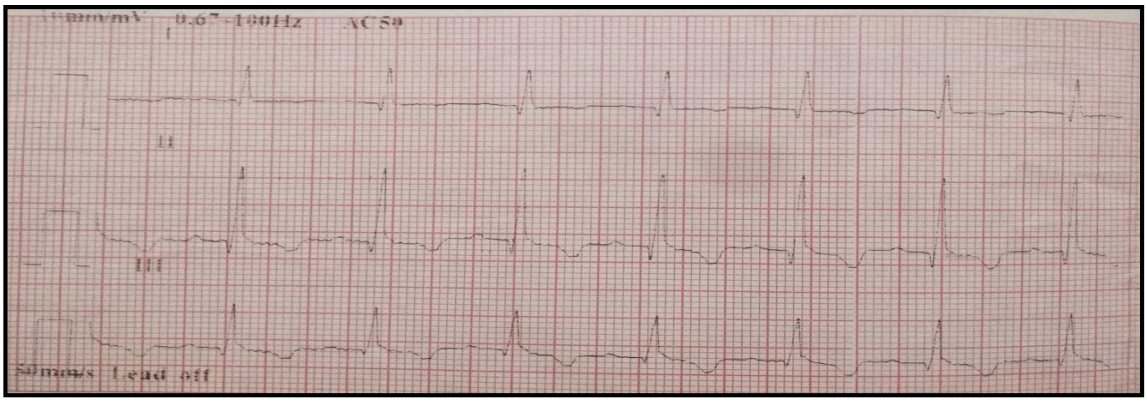


C: Ultrasonographic view of ascitic fluid

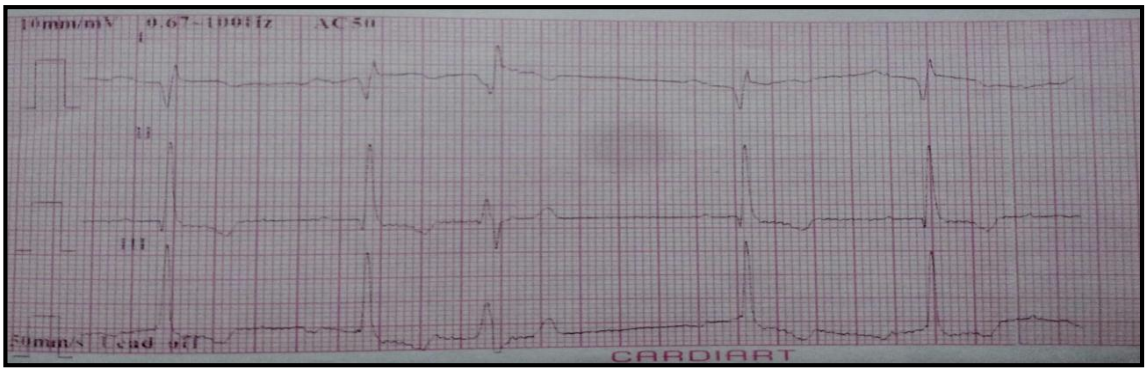


D: Ultrasonographic view of internal organ

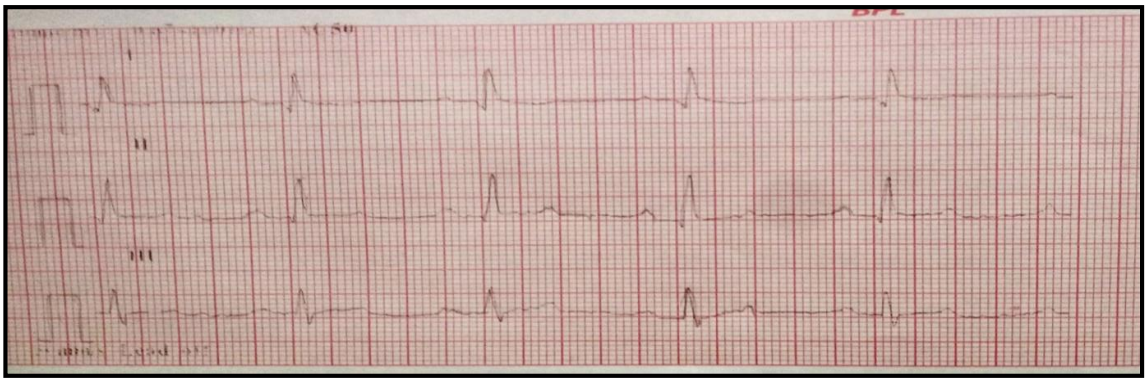
Plate 4: Ultrasonographic findings in canine ascitic cases



A: ECG showing ST elevation and inverted T wave



B: ECG showing absence of P wave



C: ECG showing sinus bradycardia

Plate 5: Electrocardiographic findings in canine ascitic cases

Chapter-V

Discussion

CHAPTER-V

DISCUSSION

Canine ascites is a notable clinical ailment in dogs and described as the excessive fluid accumulation in the abdominal cavity (Moore *et al.,* 2003) and occurs in response to numerous pathological processes. Among the numerous pathological processes, hepatic, renal and cardiac origins are the most common cause of ascites. Ascites of cardiac origin may be due to number of factors like, heartworms, congestive cardiomyopathy, right heart failure and congenital pulmonary stenosis (Peden *et al.,* 1982). Some of the common hepatic diseases which lead to ascites are liver insufficiency, chronic active hepatitis, cirrhosis and cholangitis (Peden *et al.,* 1982). Most of the hepatic diseases are associated with portal hypertension. Other causes of ascites development are hypoproteinemia due to renal loss of protein in disease like glomerulonephritis, amyloidosis, neoplasm, ruptured urinary bladder and hemorrhage from trauma (Peden *et al.,* 1982). The present study was undertaken to analyze hematological and biochemical parameters along with peritoneal fluid in dogs presented with ascites before and after the treatment.

5.1 PREVALENCE

During the study period of August 2020 to June 2021 a total of 2550 dogs were examined and only 21 dogs were diagnosed for ascites making overall prevalence of 0.82 percent. A similar pattern was observed by Saravanan *et al.* (2012) who reported 0.36 % prevalence of canine ascites at Izatnagar, (Uttar Pradesh). Prevalence of canine ascites in Enugu State, Nigeria ranged from 0.6-1% with a mean of 0.78% as reported by Ihedioha *et al.* (2013). Behera *et al.* (2017) reported 0.59 % prevalence of ascites in dogs from Odisha.

5.1.1 Age wise Prevalence

Age wise prevalence of canine ascites was found to be higher in the age group of 2-5 years (42.85%) followed by 5-10 years (28.57%), whereas minimum prevalence of

14.28% was observed in the age group of 0-2 year and 10-15 years. Thus, the age wise prevalence was found to be higher among older dogs when compared with younger one. Similar findings were reported by Saravanan *et al.* (2012) where ascites was highest in the age group of 4-5 years followed by 2-4 years and >5 years aged dogs. Upadhyay *et al.* (2014) also reported higher prevalence of ascites in dogs in the age group of above 5 years (12.35%). The higher prevalence in old aged dogs than the young animals might be due to the fact that vital organs like heart, liver, kidney are damaged with progressing age, except where the organ damage is congenital (Behera *et al.,* 2017). The present study was also supported by earlier findings of Tyagi *et al*. (2004), Routray *et al.* (2010) and Pathak *et al*. (2012).

5.1.2 Sex wise Prevalence

On the basis of sex, more prevalence of ascites was recorded in females (54%) than males (45%) in the present study. Similar reports were submitted by Upadhyay *et al.* (2014) where sex wise prevalence was higher in female (60.71%) than male (39.28%). Behera *et al.* (2017) reported higher prevalence of ascites in females (58.63%) compared to males (41.37%). Whereas Ihedioha *et al.* (2013) reported no significant association between the prevalence and sex of the animal.

5.1.3 Breed wise Prevalence

In the present study, breed wise prevalence of ascites in dogs was more in Labrador (47.61%), followed by Spitz (28.57%), German shepherd (14.28%) and least in Golden retrievers (9.52%). This was supported by the report of Padhi (2016) where the prevalence of ascites was more in Labrador (33.33%), followed by spitz (25%) and German shepherd (16.67%) dogs. Similarly Behera *et al.* (2017) reported breed wise prevalence to be higher in Labrador retriever (41.37%) followed by German spitz (18.9%), German shepherd (17.24%), Golden retriever, Pomeranian (1.72%). The breed wise prevalence was found to be higher in Labrador, which may be an indication of higher breed distribution of Labradors. However Saravanan *et al.* (2012) observed that prevalence of ascites in dogs was more in spitz breed followed by Labrador and non-descript dogs.

5.1.4 Etiology wise Prevalence

In the present study, the common etiological causes responsible for ascites in dogs were found to be of hepatic, renal and combined cardiac and hepatic origin and shared equal prevalence of 33.33%. Similarly, studies of Turkar *et al.* (2009), Dabas *et al.* (2011) and Ihedioha *et al.* (2013), Padhi (2016) and Sreehari (2017) reported hepatic, renal and cardiac systems as the common etiological cause for the ascites.

5.2 CLINICAL SIGNS

Abdominal distension and fluid thrill were recorded in all the 21 dogs examined in the present study. Other clinical signs like anorexia (38.09%, n=8), inappetence (47.61%, n=10), pale mucous membrane (61.90%, n=13), yellow mucus membrane (19.04%, n=4), respiratory distress (47.61%, n=10), tachycardia (42.85%, n=9) and sub cutaneous edema of limbs (28.57%, n=6) were recorded along with above clinical signs. Similar clinical signs were observed by Bhagat *et al.* (2011), Pathak *et al.* (2012), Saravanan *et al. (2012)*, and Singh *et al.* (2019) in their studies.

5.2.1 Hematological Studies

The hemoglobin concentration significantly ($p<0.05$) decreased in ascitic dogs of hepatic (7.54±0.86 gm %), renal (7.38±0.18 gm %) and both cardiac and hepatic origin (9.97±0.22 gm %) on day 0 when compared to the hemoglobin level of healthy dogs (11.95±0.22 gm %). Significant ($p<0.01$) decrease was observed in the packed cell volume on day 0 in the dogs with hepatic (28.24±2.34 percent), renal (24.25±1.46 percent), and both cardiac and hepatic origin (30.08±0.25 percent) ascites when compared to healthy dogs (34.75±0.18 percent). The total erythrocytes count value was significantly ($p<0.05$) decreased in ascitic dogs of hepatic (4.35±0.47 $x10^6$/µl, and), renal (3.84±0.38 $x10^6$/µl) and both cardiac and hepatic (3.57±0.25$x10^6$/µl) origin when compared to total erythrocytes count values of healthy dogs (5.92±0.14$x10^6$/µl) on day 0.

The lowered values of hemoglobin concentration, PCV and TEC count in all the cases of ascites of different origin were in accordance with the studies of earlier authors like Kumar (2002), Ihedioha *et al.* (2012), Jain *et al.* (2013), Saravanan *et al.* (2014),

Tantary *et al.* (2014) and Sharma *et al.* (2017). Reduced portal blood flow increases transit time of erythrocytes in spleen which leads to its degeneration. Increased fragility of erythrocytes and decreased nutrient uptake due to inappetence or anorexia and reduced availability of micronutrients from liver may be the cause for lowered haemoglobin concentration (Bush, 2002). Anaemia in hepatic disease might be associated with inefficient utilization of systemic iron stores (Cynthia, 2010) or due to the hepatic insufficiency which depletes the store of erythrocytic maturation factor (Chakrabarti, 1997; Sharma *et al.*, 2001). Also, fibrosis of large amount of liver mass after chronic hepatic disorder may lead to relatively lower PCV, RBC count and hemoglobin concentration in the dogs suffering from ascites (Stockham and Scott, 2008). The decreased TEC count in ascites due to renal origin may be due to reduced level of erythropoietin hormone because to renal insufficiency.

The TLC value of ascitic dogs of hepatic (17.17±2.69 $x10^3$/ µl) origin were significantly ($p<0.05$) higher when compared to the TLC values of apparently healthy dogs (11.68±0.21$x10^3$/ µl). The neutrophilic count showed significant ($p<0.05$) increase in dogs with ascites of renal (78.71±4.13 per cent) and both cardiac and hepatic origin (86.14±2.69 per cent) on day 0 when compared to the neutrophilic count of apparently healthy dogs (66.85±4.10 percent). Significant ($p<0.01$) decrease was observed in the lymphocytic count of dogs with ascites of renal (18.57±3.63 percent) and both cardiac and hepatic (10.71±2.10 percent) origin on day 0 when compared to apparently healthy dogs (24.14±1.60 percent). The monocytes count significantly ($p<0.05$) decreased in the ascitic dogs of hepatic (3.14±0.34 percent), renal (3.42±0.64 percent) and both cardiac and hepatic origin (3.57±0.52 per cent) when compared to healthy dogs (5.42±0.36 percent).

The higher total leukocyte count and differential leukocyte count, especially absolute neutrophil and lymphocyte counts recorded among ascitic dogs with multietiologies is probably indicative of the infection and inflammation of the condition. Tantary *et al.* (2013) and Pradhan *et al.* (2008) also reported higher neutrophil count in ascitic dogs. Slight decrease in hemoglobin concentration and leukocytosis with increase in Neutrophils was reported in ascitic cases by Kumar (2002), Pradhan *et al.* (2008) and Das (2012).

5.2.2 Biochemical studies

Significant ($p<0.05$) decrease was observed in the total plasma protein values of dogs with ascites of hepatic (4.53±0.24 g/dl) and both cardiac and hepatic (4.34±0.14 g/dl) origin whereas non-significantly ($p>0.05$) low total protein values were recorded in the dogs with ascites of renal origin (5.71±.83 g/dl) when compared to healthy dogs (6.34±0.18 g/dl). Significant ($p<0.05$) decrease was observed in the plasma albumin level of dogs with ascites of hepatic (1.73±0.13 g/dl) origin whereas non-significantly ($p>0.05$) lower values were observed in the dogs with renal (2.40±0.43 g/dl) and both cardiac and hepatic (2.55±0.15 g/dl) origin ascites, compared to healthy dogs (2.98±0.16 g/dl).

Damaged hepatocytes and the fibrous tissue reduces the protein synthesizing ability of the liver cells and thus leads to the lowed serum total protein for dogs with hepatic disorders. The protein loss in urine due to the damaged kidney is the cause for lowered values of total protein in renal diseases (Stockham and Scott, 2008). Decreased albumin synthesis by the liver cells, loss through the kidney in urine or/and haemodilution interlinked with the cardiac failure are some of the common cause for the significant lowered value of serum albumin recorded in all the cases of ascites. Decrease total serum protein level and hypoalbuminemia may be due to lack of production occurring with hepatic disease as albumin is synthesized exclusively by the liver. Protein-calorie malnutrition from an increased volume of distribution resulting in a dilution effect on serum albumin concentration that inhibits albumin release from hepatocytes in ascitic dogs (Cynthia, 2010). The similar findings were observed by Reynolds (2000), Ihedioha *et al. (*2012) and Tantary *et al.* (2013).

Significant ($p<0.05$) increase was observed in the ALT level of dogs with ascites due to hepatic (160.14±13.86 IU/L), renal (135.14±31.84 IU/L) and both cardiac and hepatic origin (125±10.33 IU/L) compared to healthy dogs (47.85±4.39 IU/L). Significant ($p<0.05$) increase was observed in the AST level of dogs with ascites of hepatic (185.14±57.97 IU/L) and renal (147.57±26.23 IU/L) origin compared to healthy dogs (32.57±2.83 IU/L). Significant ($p<0.01$) increase was observed in the ALP level in dogs with ascites of hepatic (562.14±176.05 IU/L), renal (272.14±52.98 IU/L) and both the cardiac and hepatic (578.71±59.39 IU/L) origin compared to healthy dogs

(63.20±1.77 IU/L). The reports from the studies by Vijayakumar (2002), Pradhan *et al.* (2008), Jain *et al.* (2013), Tantary *et al.* (2013) and Saravanan *et al.* (2014) were in agreement to the findings of the present study.

Increase serum AST and ALT may indicate damage and necrosis of membranes of hepatocytes. The activity of these liver enzymes increases on the damage to hepatocytes due to the increased permeability which depends upon the severity and the number of damaged hepatocytes (Susan and Sherding, 2006). Venous congestion of liver along with the weakened cardiac action results in elevated ALT activity for dogs with ascites due to the both cardiac and hepatic involvement. This is supported by the findings of Ihedioha *et al.* (2012) and Tantary *et al.* (2013) in their studies. Cardiac muscle damage in dogs suffering from ascites of both cardiac and hepatic origin results in elevated levels of AST enzyme while higher levels of serum AST activity recorded for dogs with renal disease may occur as a result of hypoxia linked to anemia (Stockham and Scott, 2008). Cholestatic disorders along with the hepatic disorder are reported to the cause of significant increase in the levels of ALP enzyme (Stockham and Scott, 2008; Ihedioha *et al.*, 2012; Tantary *et al.*, 2013).

Significant ($p<0.05$) increase was observed in the BUN levels of dogs with ascites of renal (63.14±27.83 mg/dl) origin when compared to healthy dogs (20.42±1.06 mg/dl). Significant ($p<0.05$) increase was observed in the creatinine levels of dogs with ascites of renal (4.16±2.10 mg/dl) origin when compared to healthy dogs (1.29±0.06 mg/dl). The results in present study were supported by earlier studies of Kumar (2002), Ihedioha *et al.* (2012), Padhi (2016) and Sood and Gupta (2018). Damaged kidneys have lowered glomerular filtration rate which lowers the clearance of the creatinine and urea levels from the blood and hence the increased levels of these parameters are recorded during the renal disease. Damaged hepatocytes are unable to regulate the urea synthesis which leads to significant lower serum urea levels (Stockham and Scott, 2008).

5.3 THERAPEUTIC STUDIES

5.3.1 Clinical observations

After following the treatment (as per the etiology of ascites) the animal's health began to show improvement which was analyzed based on the reduction in severity of the

symptoms and signs and the swollen abdomen in all the cases. In dogs with ascites of hepatic origin were treated with antibiotics, antiemetic, diuretics and supportive therapy along with hepato protective drugs whereas ascitic cases of renal origin were treated with salt free diet, antibiotics, antiemetic, diuretics and supportive therapy. Ascitic dogs with cardiac and hepatic involvement were given ACE inhibitors, pimobendan at the dose rate of 0.5 mg/kg orally along with diuretics based on radiographic and ECG findings. Nutritional supplements like multivitamin injections were given. Salt free diet along with high quality protein was advised to be included in feed. Fluid therapy was carried out using dextrose 10% which helped in checking dehydration and depleting liver glycogen Abdominocentesis was also performed in animals duly following aseptic procedures as well as monitoring vital parameters.

5.3.2 Hematological observations

Significant ($p<0.05$) increase was observed in hemoglobin concentration in hepatic (11.44±0.64 gm %) and renal (10.82±0.42 gm %) origin on day 45 when compared to day 0. Mean ± SE value of PCV in dogs with ascites of hepatic origin was 28.24±2.34 percent on day 0 which showed significant ($p<0.01$) increase on day 45 (34.57±0.92 percent). The mean values of PCV in ascites due to renal origin on day 0 was 24.25±1.46 percent which showed significant ($p<0.01$) increase on day 45 (37.58±1.00 percent) whereas, in dogs with ascites of cardiac and hepatic origin showed non-significant difference in the values of PCV on day 0 (30.08±0.25 percent) and 33.03±0.46 percent on day 45. Significant ($p<0.05$) increase was observed in the TEC count in the dogs with ascites due to hepatic (6.23±0.72 $\times 10^6$/µl), renal (6.37±0.31$\times 10^6$/µl) and both cardiac and hepatic (5.09±0.21 $\times 10^6$/µl) origin on 45th day in comparison to day 0. There was significant increase in hemoglobin and PCV value in dogs with ascetic of hepatic origin and renal disorder on day 15th and 45th post-treatment. The TLC value significantly ($p<0.05$) decreased in hepatic (11.64±0.88 x10^3/ µl) group on day 15th whereas non-significant ($p>0.05$) difference was observed on day 45th (12.57±2.85 x10^3/ µl). The neutrophilic count significantly ($p<0.05$) decreased in dogs with ascites of renal origin on 45th day (64.71±3.01 percent). Similarly, dogs with ascites of cardiac and hepatic origin showed significant ($p<0.05$) decrease in values of neutrophil

count on day 45th (68.86±1.87 percent). The lymphocytic count significantly ($p<0.01$) decreased in dogs with ascites of hepatic (15.29±1.98 percent) origin and significantly ($p<0.01$) increased in dogs with ascites of both cardiac and hepatic (23.14±1.18 percent) origin on 45th day. Significant ($p<0.05$) increase was observed in monocyte count in ascitic dogs of both cardiac and hepatic origin on day 45th (5.14±0.34 percent).

The values of TLC were comparable with healthy animals after 15th days of treatment which further improved by day 45th. Neutrophil percentage decreased significantly in dogs with ascetic of renal and both cardiac and hepatic origin after 15th days post treatment, which is in support of findings by Tantary *et al.* (2013). The results of present study are in agreement with the finding of Kumar (2002), Tantary *et al.* (2013), Padhi (2016) and Sreehari (2017).

5.3.3 Plasma Biochemical Observation

Significant ($p<0.05$) increase was observed in TPP level of dogs with ascites of hepatic origin from day 0 (4.53±0.24 g/dl) to 45th day (6.02±0.28 g/dl) whereas non-significant difference ($p<0.05$) was observed in the other two groups. Albumin levels in ascitic dogs due to hepatic (3.03±0.30 g/dl) and renal (3.78±0.31 g/dl) origin showed significant ($p<0.05$) increase on day 45th. Non-Significant increase ($p<0.05$) in total protein on day 15th and 45th post treatment was observed as compared to day '0' in ascitic dogs of renal and both cardiac and hepatic origin, whereas significant ($p<0.05$) increase in total protein level was observed in the dogs with ascites due to hepatic origin. Significant increase ($p<0.05$) was observed in the albumin values on day 45th when compared to day 0 in hepatic and renal origin ascitic dogs. Ascites causes high level of albumin distribution and lowering the blood albumin concentration leading to decrease in plasma osmotic pressure and aggravates the formation of ascitic fluid (Richter, 2003; Tennant and Center, 2008).

Significant ($p<0.05$) decrease in the ALT values were recorded in the dogs with ascites due to hepatic (54.57±4.24 IU/L), renal (61.71±8.20 IU/L) and both cardiac and hepatic (43.85±2.63 IU/L) origin on day 45th. Similarly, AST values significantly ($p<0.05$) decreased in ascitic dogs of hepatic (49.28±4.41 IU/L) and renal (45.85±5.62

IU/L) origin on 45th day. The ALP values significantly decreased ($p<0.05$) in the ascitic dogs of hepatic (160.42±59.02 IU/L) and renal (90.28±11.93 IU/L) origin on day 45th. After therapeutic management, significant decrease ($p<0.05$) in AST and ALT value was observed on day 15 and day 45 as compared to day 0 in the ascitic dogs of all groups. This was also observed by Tantary *et al.* (2013) and Padhi (2016). There was significant decrease in ALP value in all the group of ascitic dogs on 15th and 45th day post-treatment, supported by Ihedioha *et al.* (2012) and Tantary *et al.* (2013).

Significant ($p<0.05$) decrease in BUN levels (18.14±2.60 IU/L) and creatinine levels (1.17±0.21 IU/L) were observed in ascitic dogs of renal origin on day 45th when compared to day 0. Non-significant ($p>0.05$) differences were observed in the other two groups of ascites in dogs. There was significant decrease ($p<0.05$) in serum levels of BUN and creatinine on day 45th post treatment among the ascitic dogs of renal origin, is similar to the results reported by Ihedioha *et al.* (2012).

Fluid therapy especially dextrose 10% has been well established in checking dehydration, depleting liver glycogen and preventing tissue catabolism. Egg albumin was given as a protein supplement that has high biological value (Mosier and Bradley 1979). Other high quality, low protein diet in numerous small feedings is desirable to diminish bacterial conversion of excess protein to ammonia in the colon. Adequate energy should be included to minimize catabolism of proteins (Kumar *et al.,* 2016). The effected dogs were given sodium free diet to check sodium retention or secondary hyperaldosteronism as reported by Wyllie *et al.* (1980).

5.4 PERITONEAL FLUID ANALYSIS

In the present study, ascitic fluid was analyzed for color, turbidity, pH and nature after its aseptical collection. Majority of the samples collected were clear however, a few samples were turbid in nature. Other samples were yellow/straw coloured and some sample were serosanguinous. The amount of fluid varied from of 0.5 to 3 liters (Sreehari, 2017). The mean value of pH varied non-significantly ($p>0.05$) among the different etiologies of ascites in dogs. Cytological smear prepared out of samples showed presence of different types of cells such as mesothelial cells, neutrophils and few erythrocytes. Presence of mesothelial cells are helpful in differentiating from carcinoma cells.

Significantly ($p<0.05$) higher values of total protein in ascitic fluid were observed in the dogs with ascites due to hepatic (2.49±0.12 g/dl) origin than the dogs with ascites due to renal (1.95±0.29 g/dl) or both cardiac and hepatic origin (1.58±0.27 g/dl). The value of total cell count of the peritoneal fluid was significantly ($p<0.05$) higher in dogs with ascites of hepatic origin (262.86±61.91 /µl) than ascites of cardiac and hepatic origin (194.43±34.51 /µl) and renal origin (101.86±10.91/µl). Ascitic fluids which are clearer and colorless in appearance having total protein levels <2.5 g/dl and total cell count <2500 cells/ml are transudate (Burgess, 2004). Mononuclear cells like lymphocytes and mesothelial cells with fewer neutrophils are present in such fluids (Alleman, 2003).

The mean value of SAAG was significantly ($p<0.05$) higher in ascites of both cardiac and hepatic group (1.54±0.18) than the ascites of hepatic group (1.08±0.14) and renal group (1.36±0.14). Serum ascitic albumin gradient (SAAG) is used to differentiate ascitic fluid into two main categories i.e. ascites related to portal hypertension >1.1 g/dl and ascites associated with normal portal pressure have a SAAG <1.1 g/dl is suggestive of presence of portal hypertension (Saravanan *et al.,* 2012). SAAG ≥1.1 g/dl is suggestive of ascites due to ailments like cirrhosis, hepatitis, CHF, massive liver metastasis, portal vein thrombosis etc which lead to portal hypertension. SAAG <1.1 g/dl is suggestive of ascites due to ailments like peritoneal carcinomatosis, TB, pancreatic ascites, biliary ascites, nephrotic syndrome, bowel obstruction or infarction etc which are observed with normal portal pressure (Hoefs, 1983; Beg *et al.,* 2001). Color of ascitic fluids are generally found clear to pale yellow or straw and in few cases it was found bloody. The peritoneal fluid obtained from all the cases were either transudate or modified transudate, which is in support of findings by Stockham and Scott, (2008). Similar observations were reported by Saravanan *et al.* (2012), Vijaykumar *et al.* (2013), and Padhi, (2016).

5.5 RADIOLOGY

To obtain the clearer clinical picture standard radiographic procedures and techniques were followed in this study. In all the cases typical ground glass appearance of abdominal cavity was noticed. Cardiac causes of ascites were ruled out by taking standard thoracic radiographs and cardiomegaly supported by the clinical findings (Peden and Zenoble 1982). Increased fluid within the peritoneal cavity resulted in loss of

differential opacity between the adjacent structures and differentiation among different structures of abdomen. In majority of the cases, the fluid was restricted to the peritoneal space. In the present study all the radiographs showed homogenous radio opacity evenly distributed in the abdominal cavity. The findings correlate with clinical symptoms. The radiological findings obtained in the study correlate with findings of Kumar *et al.* (2011), Bhadesiya *et al.* (2015) and Kumar *et al.* (2016).

5.6 ULTRASONOGRAPHY

Use of abdominal ultrasonography is considered more sensitive than radiography for detection of peritoneal fluid accumulation in abdomen (Mc Grothy and Doust, 2004). The presence of ascitic fluid in the abdominal cavity greatly enhanced the image of various abdominal organs, viz. liver, spleen, intestine, urinary bladder and right and left kidney. Animals were placed in dorsal recumbency and sternal recumbency in few cases to carry out the abdominal ultrasonography for diagnosis of free abdominal fluid. Etiological factors which lead to ascites can be easily detected with ultrasonographic imaging and also it gives clue about the lesions like diffuse or focal (Nyland and Mattoon, 1995). In our study animals with hepatitis, cirrhosis, hepatomegaly, degenerative changes in kidneys, splenomegaly, multiple involvement of heart and liver were diagnosed. Ultrasonographic appearances are correlated with findings of Vijayakumar (2002), Kashiide *et al.* (2014), Saravanan *et al.* (2014) and Bhadesiya *et al.* (2015).

5.7 ELECTROCARDIOGRAPHY

Electrocardiographic findings recorded in the present study to reveal involvement and functioning of heart were: absence of P wave representing atria fibrillation, ST elevation and inverted T wave indicating myocardial infarction, few animals in the present study showed no major alterations except sinus arrhythmias which were not alarming. The changes were correlative with radiological and ultrasonographic findings and useful in defining the probable etiological agents responsible for ascites. Similar findings had also been reported by Guneri *et al.* (2000) and Kumar *et al.* (2011), Kumar and Srikala (2014), and Bhadesiya *et al*. (2015).

Chapter-VI

Summary and Conclusions

CHAPTER-VI

SUMMARY AND CONCLUSIONS

Canine ascites is one of the notable clinical ailments and is mainly associated with vital organs like liver, kidney and heart which require quick diagnosis along with immediate treatment, to enhance the chances of recovery.

The present study was conducted on a total of 2550 dogs presented to small animal OPD of Teaching Veterinary Clinical Complex, R. S. Pura as well as from the other sources in and around the Jammu district for health examination and only twenty one dogs had ascites (0.82%). Although ascites is very uncommon disorder, it attracts the attention of owners because of the unappealing appearance of the animal.

The breed wise prevalence of ascites was more in Labrador, followed by Spitz, German shepherd and Golden Retriever dogs. Out of 21 ascitic dogs, ten were Labrador (47.61%), three were German shepherd (14.28%), six were Spitz (28.57%) and two were Golden retriever (9.52%). On the basis of sex, more incidences of ascites were recorded in females (57.14 per cent) than males (42.85 per cent) in the present study.

Age wise prevalence of ascites in dogs was found to be higher in the age group of 2-5 years (42.85%) followed by 5-10 years (28.57%), whereas minimum prevalence of 14.28% was observed in each age group of 0-2 years and 10-15 years. The prevalence of ascites was significantly ($p<0.05$) higher in older dog than in younger ones.

The 21 ascitic cases were classified into three causative types- hepatic disorders, renal diseases and disease with involvement of both cardiac and hepatic system on the basis of clinical symptoms, physical examination, haematobiochemical and ascitic fluid analysis, radiology, electrocardiography and ultrasonography.

All the 21 dogs were showing abdominal distension. Out of 21 affected dogs, 8 (38.09%) were anorectic, 10 (47.61%) were showing inappetence, while 3 (14.28%) were having normal appetite. 13 (61.90%) dogs had pale mucous membrane, 4 (19.04%) dogs had slight yellow to yellow mucous membrane and 4 (19.04%) had pale pink mucous

membrane. Pyrexia was observed in 6 (28.57%) dogs. Other clinical signs were reduction in body weight, weakness and dullness. The dogs exhibiting the above clinical signs were subjected to abdominal palpation for confirmatory diagnosis of ascites. The abdominal palpation was performed with assisted abdominal pressure on one side and presence of fluid .thrill on opposite side. All the dogs are found positive for fluid thrill test. Respiratory distress was present in 10 (47.61 %) dogs and 9 (42.85%) dogs were showing signs of tachycardia. Sub cutaneous edema of limbs was shown by 6 (28.57 %) dogs.

The mean PCV, TEC counts and hemoglobin concentration of the ascitic dogs was significantly ($p<0.05$) lower than that the healthy dogs. The total WBC of the dogs with hepatic disorder was significantly ($p<0.05$) higher than that of dogs with ascites due to other causative types and control. However, the mean absolute monocytes count of the dogs was significantly ($p<0.05$) lower that of the control.

The mean value of ALT activity of the dogs was significantly ($p<0.05$) higher than that of the control whereas, no significant difference was observed between the groups. The mean serum AST activity of the dogs with hepatic disorders and renal disease was significantly ($p<0.05$) higher than that of dogs with ascites of both cardiac and hepatic disorders and the control. Significantly ($p<0.05$) higher serum ALP activity were observed in dogs of all the causative type and when compared between the groups with hepatic disorders and ascites due to the involvement of both cardiac and hepatic disorders. The mean serum total protein of the dogs with liver disorders and cardiac and hepatic disease were significantly ($p<0.05$) lower than that of the control, but that of dogs with renal diseases were non-significantly ($p>0.05$) different from the control. The mean serum albumin concentration of dogs with hepatic diseases was significantly ($p<0.05$) lower than that of the control, and the dogs with renal disease and both cardiac and hepatic diseases were non-significantly ($p>0.05$) lower than the control. Dogs with renal disease had a mean serum creatinine concentration significantly ($p<0.05$) higher than that of the dogs with hepatic diseases and both the cardiac and hepatic disorders, and the control. The mean serum urea concentration of the dogs with renal disease was significantly higher than that of dogs with ascites due to other etiologies and about three times higher than that of the control.

The mean of total protein value of the ascitic fluid of dogs with ascites due to hepatic origin was significantly higher than the dogs with ascites due to renal or both cardiac and hepatic origin. The total cell count value of ascitic fluid was significantly ($p<0.05$) higher in dogs with ascites of hepatic origin compared to dogs with ascites of renal or both cardiac and hepatic origin. Significantly ($p<0.05$) higher values of SAAG was observed in dogs with ascites of both cardiac and hepatic involvement compared to dogs with ascites of hepatic or renal origin.

CONCLUSIONS

Following conclusions were drawn based on the findings of present study:

i. The overall prevalence of ascites among dogs was observed to be **0.82%.**

ii. Breed wise prevalence of ascites was more in Labrador (47.61%), followed by Spitz (28.57%), German shepherd (14.28%) and Golden Retriever (9.52%).

iii. On the basis of sex canine ascites was recorded more in females (57.14 per cent) and (42.85 per cent) in males .

iv. Age wise prevalence of ascites in dogs was found to be higher in the age group of 2-5 years (42.85%) followed by 5-10 years (28.57%), whereas minimum prevalence of 14.28% was observed in the age group of 0-2 year and 10-15 years. Significantly ($p<0.05$) higher prevalence of ascites was recorded among older dog than younger ones.

v. The classification of the ascites with respect to the etiologies showed that ascites due to hepatic, renal and both cardiac and hepatic origin were the common cause in the present study and share equal rate of 33.33 percent prevalence.

vi. Among the different causative types, serum chemistry abnormalities and haematological abnormalities recorded were:

    a. Dogs with hepatic disorders had a significantly ($p<0.05$) very high serum activity of ALT, AST and ALP, very low serum levels of total protein,

albumin and urea, and significantly lower PCV, RBC count, haemoglobin, and significantly higher TLC, absolute neutrophil and lymphocyte counts.

b. Dogs with renal disease had a significantly ($p<0.05$) high serum activity of ALT, AST, ALP, low serum total protein, albumin, very high serum levels of creatinine and urea, very low PCV, RBC count and haemoglobin concentration.

c. Dogs with both cardiac and hepatic involvement had significantly ($p<0.05$) very high serum ALT and ALP activity, and low total protein levels with moderately lower levels of haemoglobin, PCV and TEC levels were observed.

vii. Analysis of ascitic fluids revealed the nature of fluid to be transudate and modified transudate. The total protein values of transudate was <2.5 g/dl whereas in modified transudate it ranged between 2.5-3 g/dl. The SAAG values of the ascitic fluid helped in diagnosing the causative type of ascites, SAAG >1.1 g/dl was related to portal hypertension suggestive of ascites due to ailments like cirrhosis, hepatitis, CHF, massive liver metastasis, fulminate liver failure, portal vein thrombosis etc. Whereas, SAAG <1.1 g/dl was related to normal portal pressure suggestive of ascites due to ailments like peritoneal carcinomatosis, TB, pancreatic ascites, biliary ascites, nephrotic syndrome, bowel obstruction or infarction.

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CERTIFICATE-IV

Certified that all the necessary corrections as suggested by the external examiner and the advisory committee have been duly incorporated in the thesis entitled **"ClinicoTherapeutic studies on Canine Ascites"** submitted by **Ms. Apurva,** Registration No: **J-19 MV-589.**

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