INVESTIGATIONS ON ASCITES IN DOGS

THESIS

BY

ABHINEET KAUR BHATTI

(V-2018-30-011)

Submitted to

CHAUDHARY SARWAN KUMAR

HIMCHAL PRADESH KRISHI VISHVAVIDYALAYA

PALAMPUR-176062, INDIA

in

Partial fulfilment of the requirements for the degree

of

MASTER OF VETERINARY SCIENCE

(DEPARTMENT OF VETERINARY MEDICINE)

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Dedication

Through good and bad times, you have always supported and motivated me for everything to let my future be bright and better

Dedicated to my lovely Grandmother and Parents with love and happiness

Dr. Des Raj Wadhwa
(Major Advisor)
Professor and Head

Department of Veterinary Medicine
CSK Himachal Pradesh Krishi Vishvavidyala
Palampur-176061 (H.P.) India

CERTIFICATE-I

This is to certify that the thesis entitled, "**Investigations on ascites in dogs**" submitted in partial fulfilment of the requirements for the award of the degree of **Master of Veterinary Science** in the discipline of **Veterinary Medicine** of CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur is a bonafide research work carried out by **Abhineet Kaur Bhatti (V-2018-30-011)** daughter of **Mrs. Manjit Kaur Bhatti** and **S. Jagdish Singh Bhatti** under my supervision and that no part of this thesis has been submitted for any other degree or diploma.

The assistance and help received during the course of this investigation have been fully acknowledged.

29/10/2020
Dr. Des Raj Wadhwa
(Major Advisor)

Place: Palampur

Date: 29th October 2020

CERTIFICATE-II

This is to certify that the thesis entitled, "**Investigations on ascites in dogs**" submitted by **Abhineet Kaur Bhatti (V-2018-30-011)** daughter of **S. Jagdish Singh Bhatti** to the CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur in partial fulfilment of the requirements for the degree of **Master of Veterinary Science** in the discipline of **Veterinary Medicine** has been approved by the Advisory Committee after an oral examination of the student in collaboration with an External Examiner.

01/12/2020

(Dr. Des Raj Wadhwa)
Chairperson

1/12/2020

External Examiner

1/12/2020

(Dr. R.K. Asrani)
Member and Dean PG's Nominee

(Dr. Ajay Katoch)
Member

1/12/2020

(Dr. Adarsh Kumar)
Member

Professor & Head
Deptt. of Veterinary Medicine
DGCN COVAS, CSKHPKV, Palampur

01/12/2020

Head of the Department

Dean, Postgraduate Studies

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A great teacher is not the one trying to be the best teacher; instead, they simply try to bring out the best in their students. And I am way blessed by the almighty to have wonderful mentors in my life.

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Abhineet Kaur

ABHINEET KAUR BHATTI

Place: Palampur

Dated: 29th October 2020

(Signature and Name)

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LIST OF ABBREVATIONS

Abbreviations	Meaning
%	Per cent
mg/dl	Milligram per decilitre
g/dl	Gram per decilitre
pg	Pico gram
fl	Femtolitre
U/L	Units per litre
µg/dl	Microgram per decilitre
mEq/L	Milliequivalent per litre
RBCs/µl	Red Blood Cells per Microlitre
WBCs/µl	White Blood Cells per Microlitre
<	Less than
>	More than
^{0}F	Degree Fahrenheit
^{0}C	Degree Celsius
Hb	Haemoglobin
PCV	Packed Cell Volume
TLC	Total Leucocyte Count
TEC	Total Erythrocyte Count
DLC	Differential Leucocyte Count
RBCs	Red Blood Corpuscles
WBCs	White Blood Corpuscles
MCV	Mean Corpuscular Volume
MCH	Mean Corpuscular Haemoglobin
MCHC	Mean Corpuscular Haemoglobin Concentration
ALT	Alanine Amino Transaminase
AST	Aspartate Amino Transaminase
ALP	Alkaline Phosphatase
BUN	Blood Urea Nitrogen
GGT	Gamma Glutamyl Transferase

A/G	Albumin Globulin ratio
CSKHPKV	Chaudhary Sarwan Kumar Himachal Pradesh Krishi Vishvavidyalaya
pCO_2	Partial pressure of carbon dioxide
tCO_2	Total carbon dioxide
BE	Base excess
HCO_3	Bicarbonate
AnGap	Anion Gap
BEact	Base excess actual
BEecf	Base excess extracellular fluid
BB	Base Buffer
$stHCO_3$	Standard bicarbonate
st pH	Standard pH
cH+	Total H+
o.i.d.	Once a day
b.i.d.	Twice a day
p.o.	Per os (Orally)
et al.	*et alia* (and others)
Inj.	Injection
Inf.	Infusion
Syp.	Syrup
IM	Intramuscular
IV	Intravenously
SC	Sub cutaneous
kg	Kilogram
mg	Milligram
mmHg	Millimetre mercury
mmol/L	Milli mole per litre
USG	Ultrasonography
ECG	Electrocardiogram
sec	Second
mV	Millivolt
HRS	Hepato-Renal Syndrome

LR	Labrador Retriever
GS	German Shepherd
ND	Non-Descript
GR	Golden Retriever
VHS	Vertebral Heart Size
CMM	Conjunctival Mucous Membrane
MILN	Medial Iliac Lymph Nodes
SAAG	Serum Ascitic Albumin Gradient
RAAS	Renin Angiotensin Aldosterone System
ADH	Anti-Diuretic Hormone
No.	Number
ACE	Angiotensin Converting Enzyme
FNAC	Fine Needle Aspiration Cytology

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Department of Veterinary Medicine
DGCN College of Veterinary and Animal Sciences
CSK Himachal Pradesh Krishi Vishvavidyalaya, Palampur-176062 (H.P.)

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ABSTRACT

The present study was aimed to study the clinical, haemato-biochemical and therapeutic aspects of ascites in dogs. The study was conducted on 2063 dogs presented to TVCC, from October 2018 to March 2020. Based upon the history, clinical signs and laboratory findings and imaging techniques, 47 dogs were found to be positive for ascites, thus representing overall incidence of 2.27%. Out of the 47 dogs, 35 dogs suffered ascites due to liver disorders (74.46%), 6 dogs due to cardiac disorders (12.76%) and 6 dogs due to renal disorders (12.76%). The incidence of ascites was higher in younger age group (1-4 years of age, 48.93%), in male dogs (76.59%) and Labrador retriever (29.78%) was the most commonly affected breed. Abdominal distension, exercise intolerance, melena, vomiting, anorexia, pale mucous membrane, dullness and depression were the common clinical signs observed. Haematology revealed normocytic hypochromic anaemia and leucocytosis. Biochemically, hypoproteinemia, hypogylcemia, increased levels of ALT, AST and ALP was observed in all the ascitic dogs. An increased level of BUN and creatinine was observed in ascitic dogs with renal disorders. The ascitic fluid analysis revealed increased levels of total protein in all the dogs and Serum Ascitic Albumin Gradient was >1.1 g/dl in dogs with liver disorders. Radiographically, ground glass appearance was observed in majority of the cases and pleural effusion and increased vertebral heart size was observed in dogs with cardiac disorders. Increased echogenicity and size of liver, kidneys and spleen, presence of free fluid, round margins of the liver lobes were some of the common ultrasonographic findings. Echocardiography revealed pericardial effusion, pleural effusion, regurgitation of the blood, ventricular hypertrophy in ascitic dogs with cardiac disorders. Electrocardiography of ascitic dogs with cardiac disorders revealed ventricular arrthymia, ventricular hypertrophy and first degree heart block. The ascitic dogs with liver disorders were treated with diuretic (Frusemide + Spironolactone), Silymarine, liver safe antibiotic and liver supportive and 13 (37.14%) dogs showed signs of recovery. Diuretic (Frusemide+Spironolactone), digoxin, ACE inhibitor (Enalapril), antibiotic and liver supportive was used for the treatment of ascitic dogs with cardiac disorders and one dog showed signs of recovery. The ascitic dogs with renal disorders were treated with 8.4% sodium bicarbonate, fluid therapy, diuretic (Frusemide+ Spironolactone), antibiotic, liver supportive and one dog showed signs of recovery. It was concluded that silymarine along with liver safe antibiotic, diuretic and liver supplements was quite useful for the treatment of ascitic dogs with liver disorders.

Abhineet Kaur Bhatti
Date: 29/10/2020

Dr. Des Raj Wadhwa
(Major Advisor)
Date: 29/10/2020

29/10/2020
Professor & Head
Deptt. of Veterinary Medicine
DGCN COVAS, CSKHPKV, Palampur

Head of the Department

INTRODUCTION

CHAPTER 1 INTRODUCTION

Ascites is defined as the accumulation of serous and sero-sanguinous fluid in the peritoneal space. The word ascites is derived from Greek words, where *"askos"* means "bag" and *"ites"* means "like a". It is also known as "hydroperitoneum drops".

It is likely to occur when the disease or some pathological condition occurs with liver, heart, kidneys, spleen, peritoneum and intestine. Ascites is usually associated with hypo-albuminemia, portal hypertension and retention of sodium and water (Johnson 1987). The major hepatic dysfunction causing ascites are chronic active hepatitis, hepatic cirrhosis and hepatic neoplasms (Leins and Monroe 1997). Ascites is also observed in congestive heart failure, protein losing nephropathy, due to inflammatory bowel disease, intestinal lymphoma, and intestinal lymphangiectasia (Dossin and Lavouè 2011), kinking of caudal vena cava (Pelosi et al. 2011), peritonitis, haemorrhage, neoplasia and even at time of rupture of urinary bladder or gall bladder (Rutgers and Biourge 2007). Some of the renal diseases like glomerulo nephritis, nephrotic syndrome also contributes to the onset of ascites (Ettinger and Feldman 2000). Ascites develops due to disturbance in the starling forces, which includes the increased pressure of the venous or lymphatic hydrostatic pressure, vascular permeability, increased intra peritoneal oncotic pressure and decrease in the oncotic pressure of capillary (Richter 2003). The development of ascites is secondary to portal hypertension and low albumin concentration (Center 2015). Portal hypertension leads to splanchnic pooling of blood with subsequent reduction in systemic arterial pressure and activation of the Renin Angiotensin Aldosterone system (RAAS). RAAS activation results in further retention of fluid and thus leading to development of ascites (Bexfield and Watson 2009).

Ascitic fluid can be classified as transudate, modified transudate and exudate depending on protein, cellular content and specific gravity (Hall 2005). The analysis of the ascitic fluid is useful in differential diagnosis. The gross examination and its analysis are useful in diagnosing the pathological condition. The analysis include abdomenocentesis, examination of the fluid grossly including specific gravity, total protein, albumin, nucleated cell count and serum ascitic albumin gradient SAAG (Kuiper et al. 2007). Canines suffering from chronic liver disease due to increased hydrostatic pressure along with portal hypertension, the fluid is clear and colourless pure transudate that contain very few nucleated cells (<2500 cells/ml), low protein content (<2.5 g/dl) and specific gravity of

1.016. Modified transudate fluid is slightly cloudy having protein content from 2.5 g/dl to 5.0 g/dl with nucleated cells <7000/ml and specific gravity in the range of 1.017-1.025, is due to post sinusoidal hypertension arising because of right side heart failure. The exudative ascites is formed secondary due to increased permeability of the peritoneal surface affected by inflammatory processes or neoplastic, the cause can be pancreatitis, bile peritonitis, malignant disease in peritoneum and septic peritonitis, inflammatory diseases. Haemorrhagic ascites is secondary to pathological accumulation of blood in peritoneal cavity that is secondary to trauma, spontaneous ruptures of blood vessels and coagulopathy (Zoia et al. 2017).

The condition can also be classified on the basis of Serum Ascitic Albumin Gradient (SAAG) as “high gradient” and “low gradient”. The normal value of SAAG is 1.1 g/dl, if SAAG is more than 1.1 g/dl the ascites is developed due to portal hypertension (transudate ascites) whereas less than 1.1 g/dl is unrelated to portal hypertension (Burgess 2004). SAAG is considered as a marker of portal hypertension and the use of this index replaces the exudates-transudate concept in ascitic fluid (Tarn and Lapworth 2010).

The serum makers such as serum bilirubin, serum alkaline phosphate, Gamma glutamyl transferase, glucose, total protein and albumin levels, alanine aminotransferase and aspartate aminotransferase are helpful in diagnosing a liver impairment (Tiwari et al. 2011; Baneerji 2003). Cardiac and renal involvement along with liver can also be diagnosed by complete blood count, serum biochemistry, urine analysis, radiography or ultrasonography (Varshney and Hoque 2002), electrocardiography and echocardiography (Bonagura and Schober 2009; Kumar et al. 2011)

There is wide range of causes of ascites and is very challenging to determine the cause, as there is no specific and sensitive test for the diagnosis of the condition. Ascites should be evaluated quickly and rapidly for a good prognosis as it is potentially a serious clinical sign. Hence, together with a complete history, physical examination, radiographic examination, ultrasonography, electrocardiography and laboratory test should be done to identify the main cause and then treated accordingly.

For treating ascites and to eliminate the root cause, an effective therapy is required that directs the interventions with an aim to eliminate the causative factors, minimizing the fibrosis and control the complication that arise. Low sodium diet and diuretics (Frusemide, spironolactone) are most commonly used to reduce and control the abdominal distension

due to presence of the fluid in peritoneal space. But in few cases mainly in cirrhosis, it is observed that animals fail to respond to the diuretics especially frusemide because of high levels of serum aldosterone (Center 2006). There is dysfunction of hemodynamics in cirrhosis that results in refractory ascites and hepato-renal syndrome (HRS). The refractory ascites and HRS is independent predictor of short survival time of animal (Salerno et al. 2010).

The perusal of records in clinics reveals that ascites in dogs is frequently encountered and is observed that by performing routine clinical examination it is not possible to determine the cause of ascites. No systematic work has been carried out on the clinically affected dogs. Thus, the present study on clinico-therapeutic aspects is being undertaken for better management of the condition with the following objectives:

- To study the detailed clinical appraisal of dogs suffering from ascites.
- To study the haemato-biochemical changes in the affected dogs and to formulate suitable therapeutic measures based upon the above investigation.

Review of Literature

CHAPTER 2 REVIEW OF LITERATURE

2.1 ASCITES

Ascites refers to the accumulation of fluid within the peritoneal cavity that is secondary to the hypoproteinemia, hepatic insufficiency, congestive heart failure, renal diseases and abdominal carcinomatosis (Ettinger 1989). Ascites due to liver disease are usually associated with hypoalbuminenia, portal hypertension and retention of sodium and water (Johnson 1987).

2.1.1 Prevalence studies and risk factors

James et al. (2008) studied 17 dogs suffering of ascites due to pre-sinusoidal portal hypertension and found that 12 dogs (70.5%) were 4 years of age or younger at time of presentation.

Raffan et al. (2009) conducted study in 34 ascitic dogs suffering from chronic hepatitis and maximum cases were observed in Labrador retriever (7) followed by Springer Spaniels (3) and Great Dane (3). The ages ranged between 1 to 13 years (mean 5 years) and 18 were female and 16 were males.

Ihedioha et al. (2011) conducted study in 14 dogs suffering from ascites where males (57.10%) were more affected than females (42.90%), 3 years and above age were among the majority with 71.40 per cent followed by 1-2.9 years with 21.40 per cent and least by <1 year of age with 7.10 per cent. Breed disposition revealed that Alsatian-Rottweiler Cross (50%) was highest in affected group and Alsatian-Mongrel Cross and Rottweiler was among the least with 14.30 per cent of incidence.

Saravanan et al. (2012) reported that higher incidence of ascites was found in Spitz dog (7 dogs) followed by Labrador Retrievers (2 dogs) and 1 non-descriptive dog. Male dogs had higher incidence (6/10) than female dogs (4/10) and incidence was high in 4-5 years of age group (5/10), followed by 2-4 years (3/10) and least in >5 years (2/10) of age group.

Sarvanan et al. (2013) conducted a study on 15 dogs and found that the occurrence of ascites was breed, sex and age dependent with higher incidence in the age group of 5 to 7 years and more among males (60%) than in females (40%). Pomeranian (33.33%) was at

higher risk followed by Labrador retriever (20%), Boxer (16.66%), Doberman pinscher (13.33%), Mongrels (10%) and least in Alsatian (6.66%).

Saravanan et al. (2014) studied 72 dogs having ascites and found that incidence in males (88.9%) was higher as compared to females (66.7%). The Spitz dogs (54.2%) had higher incidence followed by Mongrel (16.4%), Labrador retriever (12.5%) and least incidence was observed in Great Dane (1.4%) and Golden Retriever (1.4%).

Kocatürk et al. (2016) studied 8 ascitic dogs having cardiomyopathy out of which 5 were female dogs and 3 were male dogs. Mixed breed (4/8) were more in number and least were the Pointer (1/8) and Anatolian Sheepdog (1/8). The average age of dogs was 54.8 ± 30.8 months (range: 10-96 months).

Behera et al. (2017) screened 58 dogs positive for ascites, among them higher incidence was seen in >6 years of age (36.21%) following 0-3 years age group (34.48%) and 3-6 years of age group with 29.31 per cent and also that females suffered (58.63%) more than males (41.37%). Incidence was most common in Labrador breed (41.37%), then Spitz (18.9%), followed by German shepherd (17.24%) and least by Pomeranian and Cocker spaniel breed (1.72%). Majority of the cases were in the month of April (24.14%) and least in July.

Zoia et al. (2017) studied 70 dogs having ascites, 49 were male dogs (44 sexually intact and 5 neutered) and 21 were female dogs (13 sexually intact and 8 spayed). The mean age of the dogs was 8.6 ± 3.4 years. 18 dogs were crossbred and 52 were purebred with German shepherd dogs being the highest affected breed followed by Labrador retrievers and least being the Great Dane, Beagle and Rottweiler.

2.2 AETIO-PATHOGENESIS

2.2.1 Hepatic origin

Sherlock and Shaldon (1963) gave the underfilling theory; during ascites the effective blood volume decreases that induced the hormonal change and resulted in renal sodium retention. The changes in starling forces occurred due to cirrhosis of liver (production of fewer albumins) and the hepatic venous outflow obstruction.

Lieberman et al. (1969) observed that in cirrhosis and ascites the total blood volume remains normal or increased as seen in spontaneous diuresis, proposing the overflow hypothesis for the formation of ascites.

Wadhwa et al. (1995) studied 5 dogs between the age of 1 and 10 years, suffering ascites due to liver involvement.

Center (1999) stated that portal hypertension that results from sinusoidal hypertension occurs due to accumulation of peri-sinusoidal collagen along with activation of hepatic stellate cells (Ito cells) that was the main inciting event in the development of ascites.

Vijayakumar (2002) asserted that ascites developed due to venous stasis and venous hypertension consequently, two pathophysiologic events that occur include high protein hepatic lymph via hepatic or post hepatic obstruction or low protein splanchnic lymph that results from pre hepatic obstruction. The increased salt and fluid accumulation was due to hyperaldosteronism, increased renin & angiotensin levels, increased ADH and other hormonal dysfunction.

Kumar et al. (2003) studied 20 dogs, among which 17 dogs (85%) exhibited ascites of liver origin that was confirmed by serum biochemical profile.

Kruth et al. (2005) reported that transudate ascites was caused due to increased venous hydrostatic pressure with portal hypertension (like, congestive heart failure, liver diseases) or decreased colloid osmotic pressure (eg. Protein losing enteropathy, protein losing nephropathy or severe hepatic failure) and haemorrhagic ascites was secondary to accumulation of blood in peritoneal cavity due to pathological reasons.

James et al. (2008) did a retrospective study in 17 dogs suffering from ascites due to pre-sinusoidal portal hypertension.

Ihedioha et al. (2011) investigated the 14 dogs suffering with grade 3 ascites, and found that 14.3 per cent had ascites due to cirrhotic liver disease and 21.4 per cent due to chronic liver disease.

Elhiblu et al. (2015) studied 140 dogs among which 6 ascitic dogs were suffering from liver cirrhosis and the prevalence was 4.3 per cent. The study also indicated that hypoproteinemia was the most common finding in chronic disorders like cirrhosis and

portosystemic vascular abnormalities as liver is main site for synthesis and degradation of proteins. Low serum albumin concentrations due to liver disease indicate diffuse and chronic hepatopathies.

Kumar and Dhana Lakshmi (2015) diagnosed a 5 year old Pomeranian cross suffering with ascites of hepatic origin.

Kumar et al. (2016) stated that increased level of AST indicates hepatic insufficiency along with extensive damage that results in the leakage of enzymes from hepatic cell into blood stream.

Behera et al. (2017) screened 58 cases that were positive for ascites and out of which 21 dogs (36.2%) suffered due to hepatic involvement.

Zoia et al. (2017) studied 70 ascitic dogs; hypertension and malignancies were the main causative factor among 8 dogs (4 each). Bile peritonitis, concurrent protein losing enteropathy and protein losing nephropathy and hepatic failure affected 6 dogs (2 each). Benign tumour, necrotizing hepatitis, iatrogenic, traumatic and protein losing nephropathy affected least dogs (1 each).

Neelam et al. (2019) presented a case of ascites of hepatic origin in a Bully female pup of 4 months.

2.2.2 Cardiac origin

Epstein et al. (1985) proposed the vasodilation theory, stating that in peripheral arterial vasodilation, the cardiac output increases due to reduced cardiac preload. Increase in plasma concentration of renin, aldosterone, norepinephrine and vasopressin is observed as compensatory reaction that results in moderate vasoconstriction with sodium and water retention. This expansion of plasma reduce the activity of sodium retaining hormonal system and the plasma concentration of renin, aldosterone, norepinephrine and vasopressin lies within the normal range in some without ascites.

Laflamme (1997) stated that with right-sided heart failure, hepatic congestion and portal hypertension may occur that lead to decrease hepatic and gastrointestinal function. Pulmonary congestion occurs in left sided heart failure and interferes with normal gas exchange leading to building of carbon dioxide and respiratory acidosis.

Baumwart et al. (2005) studied 48 Boxer breed dogs that suffered from cardiomyopathy and left ventricular systolic dysfunction.

Martin et al. (2009) examined 369 dogs that were pure bred except four dogs and all suffered with cardiomyopathy.

Ihedioha et al. (2011) investigated the dogs suffering with grade 3 ascites and found that 50 per cent of the dogs were having ascites due to congestive heart failure.

Borgarelli and Buchanan (2012) stated that in increased right atrial pressure, the most common manifestation was respiratory distress, ascites and hepatomegaly. Pleural, pericardial effusion and splenomegaly may also develop but were rare.

Pelosi et al. (2012) observed cardiomegaly and peritoneal effusion in 9 year old male Rat Terrier.

Jan (2013) stated that ascites in dogs was due to combination of portal hypertension and moderate hypoalbuminemia in dogs with liver dysfunction.

Srivastava and Syed (2013) diagnosed ascites of hepatic origin in 6 year male Boxer dog, and stated that ascites was only caused when the albumin concentration was critically low along with reduced osmotic pressure and also portal hypertension was present in ascites that gave rise to different liver dysfunction.

Kumar and Srikala (2014) reported that in animals with long history of mitral valve insufficiency, pulmonary hypertension and right side heart failure might develop. Right side heart failure may also develop as secondary due to persistent elevation of left atrial and pulmonary venous pressures. Ascites, pleural, pericardial effusion, hepatomegaly and splenomegaly may develop as a consequence of increased right atrial pressure.

Zoia et al. (2017) did cross-sectional haemostatic study of ascitic fluid in 70 dogs, 14 dogs suffered due to right sided heart failure, 6 dogs had myocardial, valvular and arrhythmogenic diseases.

Alsaad et al. (2018) asserted that in congestive heart failure, the decrease in blood pressure and installing of cardiogenic attach were the main outcomes and myocardium restores this by tachycardia, tachypnea, vasoconstriction and increasing the volume and the

poor irrigation of internal organs lead to pulmonary oedema, liver failure and passive venal congestion.

2.2.3 Renal Origin

Kumar et al. (2003) studied 20 dogs, and 15 per cent suffered ascites of renal origin. They stated that increased sinusoidal pressure resulted in imbalance in starling forces and serum protein was driven into interstitial space. Increased renal retention of sodium and fluid aggravates the formation of ascites with increased aldosterone release and creates a vicious cycle.

Ihedioha et al. (2011) investigated the dogs suffering with grade 3 ascites and found 14.3 per cent due to kidney disease.

Behera et al. (2017) screened 58 cases positive for ascites and 9 dogs (15.51%) suffered due to renal diseases.

2.2.4 Miscellaneous

Dabas et al. (2011) reported a case in 8 year old mongrel female dog having ascites of splenic origin.

Gonde et al. (2014) found peritoneal effusion in a 5 year old male Labrador that had *Babesia gibsoni*.

Behera et al. (2017) screened 58 cases positive for ascites, 6 dogs (10.34%) were of genital origin and other 6 dogs (10.34%) due to parasitic origin.

Zoia et al. (2017) did a cross sectional haemostatic study of ascitic fluid in 70 dogs, with majority (29 dogs) suffered due to hemangiosarcoma, non-septic peritonitis, septic peritonitis and idiopathic peritoneal effusion caused ascites in 3 dogs each.

2.3 CLINICAL SIGNS

Wadhwa et al. (1995) studied 5 dogs with signs of inappetance, gradual loss of condition, bilateral abdominal distension and oliguria. The mucous membranes were pale and fluid thrills were evident on tactile percussion of abdomen. The two dogs on palpation exhibited pain on palpation of hepatic area while two showed symptoms of gastro-enteritis additionally. Average body temperature was 38.86 ± 0.9 ^{0}C and all showed tachycardia (118.5 ± 5.25/min) and tachypnea (38.0 ± 5.38/min).

Vijayakumar (2002) stated that abdominal distension, respiratory distress, abdominal discomfort, groaning while lying down, scrotal or penile oedema, weight gain, vomiting, cough, anorexia, lethargy, episodic weakness and fever are some of the signs that were commonly seen in dogs suffering ascites.

Shaw and Rush (2007) reported that dogs with chronic pericardial effusion typically had signs secondary to right sided heart failure, including lethargy, exercise intolerance, respiratory difficulty, weight loss and abdominal distension. Physical examination findings included tachycardia, hepatomegaly, ascites and tachypnea or dyspnoea, combination of muffled heart sounds, jugular venous distension and poor pulse quality.

James et al. (2008) studied 17 dogs with clinical signs of lethargy (6/17), inappetance (9/17), vomiting (7/17), diarrhoea (4/17), melena (5/17), weight loss (4/17), polydipsia/polyuria (3/17), neurological signs (1/17), mild jaundice (1/17) and cyclic pyrexia associated with neutropenia (1/17), with abdominal distension in all from day 1 to 3 months.

Rautray et al. (2010) studied two cases with signs of abdominal distension, inappetance, weakness, pale mucous membrane and presence of fluid thrill. The temperatures of the dogs were 102.6 ^{0}C and 103 ^{0}C, respectively.

Dabas et al. (2011) reported a case with signs of abdominal distension, inactiveness and weakness and fever. The mucous membrane was pale, presence of fluid thrill and on palpation revealed hard mass posterior to the left costal arch.

Pelosi et al. (2012) studied a case of cardiomegaly and peritoneal effusion in 9 year old male Rat Terrier dog that had signs of mild tachycardia, increased respiratory rate, weak regular pulse and abdominal distension.

Sarvanan (2013) stated that ascites interfere with respiration causing general discomfort and disturb fluid and electrolyte metabolism. Anorexia, lethargy, weakness, dyspnoea, discomfort, pale mucous membrane, rectal temperature 103.2 ^{0}F and distended abdomen with fluid thrill in palpation were the main clinical signs recorded in ascites affected dogs.

Srivastava and Syed (2013) observed a dog with signs of lethargy, difficulty in breathing, severe distension of abdomen, frequent vomiting, and hollow flanks with prominent spines. Conjunctival mucous membrane was pale and rectal temperature was within normal range, and the tactile percussion revealed fluid thrill.

Gonde et al. (2014) diagnosed a case of abdominal distension due to *Babesia gibsoni* affecting liver with signs of pyrexia (104 ^{0}F), pale mucous membrane, and heart rate 148 beats per minute and pulse rate 58 per minute.

Saravanan et al. (2014) studied 72 dogs with abdominal distension that had additional signs of inappetance (69.4%), lethargy (63.9%), and pale mucous membrane (45.8%), respiratory dyspnoea (15.3%), pedal oedema (12.5%), diarrhoea (8.3%), vomiting (6.9%), melena (6.9%) and polyuria/polydipsia (4.2%).

Bhadesiya et al. (2015) found enlarged abdomen and tactile percussion revealed fluid accumulation on physical examination. Clinical examination revealed normal rectal temperature 102.4 ^{0}F, capillary refill time <2 seconds, dyspnoea with pulse rate, respiratory rate and heart rate as 84 pulse per minute, 36 breaths per minute and 98 beats per minute, respectively, in a female German shepherd dog.

Kashyap et al. (2015) observed dyspnoea and tachycardia, normal rectal temperature, laboured respiration, inappetance, pale mucous membrane and undulating movements (thrills) of the fluid on taping abdomen in 5 year old Saint Bernard dog.

Chutia et al. (2016) examined a 3 years old crossbred spayed female dog with no defecation since spaying. Clinical examination revealed tedious and depressed voluminous abdomen, pale mucous membrane, normal rectal temperature (101.6 ^{0}F), and heart rate was 130 beats per minute and respiration rate was 28 breaths per minute. Fluid thrill was present on percussion.

Kocatürk et al. (2016) recorded signs of abdominal distension, dyspnoea (6/8), lethargy (4/8), exercise intolerance (8/8) and anorexia (5/8) in 8 dogs. Clinical examination revealed weak femoral pulse, distension of jugular vein, increased cardiac auscultation area and mitral and/or tricuspid murmurs.

Kumar et al. (2016) treated a German shepherd female dog with history of inappetance and enlargement of abdomen. Physical examination revealed dyspnoea,

dehydration, slight pale mucous membrane, rough, lustreless body coat and distension along with hollowness at flank region which on taping had undulating movement (fluid thrills).

Kozat and Sepehrizadeh (2017) stated that depression, weakness, CNS signs, icterus, anorexia, vomiting, change in spleen size, diarrhoea, weight loss, dark brown urine, fever, polydipsia, polyuria, abdominal pain, ascites, coma, change in liver size, dark or light coloured stools, haemorrhage and pruritus were the clinical signs associated with liver disease.

Singh and Kumar (2017) managed a pup of three and half months with signs of anorexia, lethargy, distended abdomen and difficulty in breathing. Respiratory distress, dehydration, fluid thrill, rough body coat and pale mucous membrane were the clinical signs. The respiratory rate and heart rate recorded were 30 breaths/min and 87 beats/min, respectively.

Mukherjee et al. (2017) delineated a case of a 4 year male Labrador retriever that had symptoms of anorexia from 45 days, dyspnoea, abdominal enlargement. Physical examination revealed normal body temperature (101 ^{0}F) and pale mucous membrane. Fluid wave in abdomen was evident on percussion.

Regmi and Shah (2017) studied a dog with signs of abdominal distension, discomfort, dyspnoea and anorexia. The mucous membrane was pale and tachycardia was evident, on taping the abdomen undulating fluid movements were observed.

Alsaad et al. (2018) reported a case of a male Mullinoise breed a police (K9) dog of 5.7 years weighing 23 kg with signs of acute illness 1-4 days, dyspnoea with open mouth breathing, lethargy, reduced food intake, distended abdomen, reduced exercise tolerance, coughing and weight loss. Clinical examination revealed pale mucous membrane, harsh lung sounds with pulmonary crackles, loud murmur on auscultation of heart area were audible, slight hepatomegaly, distended jugular vein and a weak femoral pulse was detected.

Neelam et al. (2019) presented a case in a Bully female pup of 4 months with signs of progressive abdominal distension, inappetance, lethargy, weakness. The clinical signs were of pyrexia, palpable fluid thrill, dyspnoea, dehydration and pale mucous membrane.

Samad (2019) found distended abdomen, normal rectal temperature, pale mucous membrane, anorexia and signs of dyspnoea, tachycardia with fluid thrill on tactile percussion in Spitz dog.

2.4 HEMATO-BIOCHEMICAL PROFILE

2.4.1 Hepatic

Wadhwa et al. (1995) studied 5 dogs with bilateral distension of the abdomen. The haematology revealed low haemoglobin (7.8 ± 0.22 g/dl) and packed cell volume (24.50 ±2.95 %), whereas the total and differential count was almost unaffected. The serum samples were positive for Vandan Bergh test showing liver involvement along with hypoproteinemia (5.38 ± 0.7 mg/dl).

Kumar et al. (2003) studied 20 dogs and 85% developed ascites as hepatic origin. The mean values of haemoglobin (Hb), packed cell volume (PCV), total leucocyte count (TLC) and total erythrocyte count (TEC) as 8.07±0.22 g/dl, 26.58 ± 0.95%, 9.80 ± 0.23 × 10^3 / cumm, 4.96 ± 0.16 × 10^6/cumm. The mean serum biochemical analysis revealed alanine amino transferase (ALT) (56.11 ± 2.38 U/L), aspartate aminotransferase (AST) (66.76 ± 2.22 U/L), alkaline phosphatase (ALP) (185.6 ± 6.25 U/L) were increased. The blood urea nitrogen (BUN) (22.32 ± 0.75 mg/dl), serum creatinine (0.87 ± 0.01 mg/dl) were within the normal range and total protein (4.31 ± 0.13 g/dl), albumin (1.7 ± 0.06 g/dl) and A/G ratio (0.65 ± 0.01) were at lower side.

Gupta et al. (2004) did haematological and biochemical study of the blood sample of a German shepherd female dog having ascites of hepatic origin, showing normal levels of haemoglobin, total leukocyte count, blood urea nitrogen (BUN), and creatinine. Hypoproteinemia, hypoalbuminemia and increased levels of AST and ALT were recorded.

James et al. (2008) performed retrospective study on 17 dogs suffering from ascites due to pre-sinusoidal portal hypertension, the haemato-biochemical changes noticed were anaemia and microcytosis with increase in ALT in 6/17 dogs, ALP in 9/17 dogs, bilirubin in 6/17 dogs, cholesterol was decreased 9/17, urea in 13/17 dogs, total protein in 14/17 dogs and serum albumin in 11/17 dogs.

Raffan et al. (2009) conducted study in 42 dogs confirmed with chronic hepatitis through histopathology and 14 dogs had ascites along with.

Sarma et al. (2009) studied 45 dogs and 33 per cent (15 dogs) suffered from ascites. The haemto-biochemical profile of the affected dogs revealed low haemoglobin (9.48±0.30 g/dl), PCV (34.50±1.26 %) and total serum protein level (4.31±0.20 g/dl), increased Alanine Aminotransferase (228.53±22.17 IU/L) while Blood urea Nitrogen (14.27±1.31 mg/dl) and creatinine (0.70±0.09 mg/dl) were within normal range.

Rautray et al. (2010) observed 2 cases of ascites due to hepatitis with haemato-biochemical parameters as Hb 7.6 g/dl and 8 g/dl, TEC 2.4×10^6/µl and 2.8×10^6/µl, TLC 12,100/µl and 13,500/µl, ALT 72 U/L and 69 U/L, AST 60 U/L and 66 U/L, BUN 29 U/L and 26 U/L and creatinine 0.6 mg/dl and 0.8 mg/dl, respectively.

Ihedioha et al. (2011) reported 14 cases among which 5 suffered from ascites due to liver impairment and divided in group 1 (2 dogs suffering cirrhotic liver disease) and group 2 (3 dogs suffering chronic active hepatitis). ALT, ALP and AST was decreased in group 1 but increased in group 2. Increase in creatinine, hypoproteinemia and hypoalbuminemia was observed in both the groups. PCV, Hb, MCV were on the lower side but TLC was within the normal limits.

Srivastava and Syed (2013) studied a 6 year old Boxer dog suffering from ascites, the haematology revealed decrease in red cells while biochemical profile showed elevation in ALT, AST and ALP enzymes, decrease in BUN, albumin, cholesterol and blood glucose indicated decreased liver function capability.

Saravanan et al. (2013) studied 15 ascitic dogs in which the haematology revealed low haemoglobin (9.49±0.28 g/dl) and PCV (34.39±1.36 %) whereas the TLC and DLC showed non-significant results. The serum biochemistry revealed low serum total protein concentration (4.40±0.20 g/dl) but increased levels of ALT (Alanine Aminotransferase) (228.53±22.17 IU/L).

Chaturvedi et al. (2013) carried out a study on 10 ascitic dogs and haemato-biochemistry analysis revealed mild anaemia (haemoglobin 10.31±0.23 g/dl), hypoproteinemia (4.39±0.07 g/dl) and hypoalbuminemia (1.05±0.09 g/dl), decreased albumin: globulin ratio (0.66±0.05), increased activity of liver enzymes, AST (104.5±5.82 IU/L) and ALT (89.5±0.01 IU/L). The other parameters (TLC, Glucose, Creatinine, BUN) were within the normal range.

Gonde et al. (2014) reported 5 year old male Labrador with peritoneal effusion due to *Babesia gibsoni*. The haemato-biochemical alterations that were noticed were elevation in the levels of bilirubin (1.8 mg/dl), alanine amino transferase (ALT; 313 U/L), alkaline phosphatase (ALP; 563 U/L), blood urea nitrogen (BUN; 30 mg/dl), and neutrophils (72%). Haemoglobin (Hb; 6 g/dl), total erythrocyte count (TEC; 3.1×10^6/ μL), packed cell volume (PCV; 18.4 %), platelets (1.58×10^5/ μL), total protein (3.2 g/dl), albumin (1.0 g/dl) being at low levels.

Sarvanan et al. (2014) studied 72 ascitic dogs, the haematology of the dogs revealed decreased haemoglobin (8.90±0.25 g/dl), PCV (27.37±0.68 %), RBC ($2.73±0.10×10^6$/μl) and biochemistry analysis revealed increased activity of BUN (31.29±3.26 mg/dl), creatinine (1.17±0.15mg/dl), ALT (173.47±12.29 IU/L), AST (161.18±11.15 IU/L), ALP (120.74±7.89 IU/L), total bilirubin (0.99±0.07 mg/dl) and decrease activity of total protein (5.15±0.10 g/dl) and albumin (2.02±0.03 g/dl).

Elhiblu et al. (2015) did haemato-biochemical study in 140 dogs with liver cirrhosis. The levels of haemoglobin (Hb), lymphocytes, packed cell volume, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and platelet count were significantly lower while the total leukocyte count (TLC), neutrophils and mean corpuscular haemoglobin concentration (MCHC) were significantly higher. The levels of glucose, total protein, albumin, A/G ratio and fibrinogen were lower and creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), prothrombin time and APTT were higher than the control group.

Manoj and Lakshmi (2015) performed haemato-biochemical examination in 5 year old Pomeranian cross dog with ventral abdomen distension that revealed Hb 9.2 g/dl, total erythrocyte count 2.2 × 106/μl, total leucocyte count 12,100/μl, neutrophil 79%, lymphocytes 14%, monocytes 2%, eosinophil 3%, basophil 0%. Biochemical estimation revealed ALT 70 U/L, AST 62 U/L, BUN 27 mg/dl and creatinine 0.6 mg/dl.

Kumar et al. (2016) did the haemato-biochemical in 9 year old German shepherd female dog having ascites of hepatic origin. Neutrophils with decreased erythrocytes and haemoglobin and increased SGPT value with decreased total protein were the main findings.

Kozat and Sepehrizadeh (2017) stated that ALT serum rising were specific remarked in dogs with highest sensitivity (more than 80%) for hepatic disorders but have

less sensitivity (under 60%) in cases of hepatic congestion, neoplasia and portosystemic vascular anomalies. ALP activity has a high sensitivity (80%) for hepatobiliary disease, but its specificity is low (50%). Elevation in ALP along with increased GGT activity increases the specificity up to 90% in liver diseases. ALT and AST increase in hepatocellular leakage whereas ALP and GGT increase in cholestasis.

Regmi and Shah (2017) conducted a case study in 11 year old male German shepherd with haemato-biochemical analysis revealing decrease in haemoglobin (9.6 g/dl), total protein (4.8 g/dl), albumin (2.2 g/dl) and blood glucose (55 mg/dl) and increased neutrophils (85%), total bilirubin (0.8 mg/dl), aspartate aminotransferase (62 U/L), alanine aminotransferase (128 U/L) and alkaline phosphatase (810 U/L). The blood urea nitrogen and creatinine were within normal range.

Lakshmi et al. (2017) studied 140 dogs, among them 32 dogs suffered from ascites due to diffuse liver parenchymal disorders. A significant decrease was observed in the platelet count ($1.87 \pm 0.07 \times 10^3/\mu l$), and increase in the value of prothrombin time (10.60 ± 0.21 sec.) and activated partial thromboplastin time (34.41 ± 1.44 sec).

Lakshmi et al. (2018) studied 32 dogs suffering from diffuse liver parenchymal disorder with ascites that had haematological findings as decreased haemoglobin (9.06 ±0.24 g/dl), TEC ($4.64 \pm 0.13 \times 10^6/\mu l$), PCV (38.81 ± 3.69 %) and increased TLC ($23.73 \pm 0.92 \times 10^3/\mu l$). The biochemical findings revealed increase in ALT (203.19 ±19.16 U/L), AST (184.84 ±22.32 U/L), ALP (284.82 ±18.39 U/L), GGT (6.64 ±0.51 U/L), total bilirubin (1.17 ±0.04 mg/dl), BUN (20.08±0.69 mg/dl) and creatinine (1.21±3.03 mg/dl) and decrease in total protein (4.77±0.10 g/dl), albumin 2.01±0.03 g/dl), glucose (90.86±1.39 mg/dl) and cholesterol (118.72±17.25 mg/dl).

Neelam et al. (2019) reported a case of Bully female pup having ascites of hepatic origin, with Haemoglobin 7.7 g %, TLC 13410/cmm and DLC- Neutrophils 86%, Lymphocytes 12%, eosinophil 1% and monocytes 1%. The biochemical analysis showed higher value of SGPT (251 U/L) and SGOT (113.2 U/L). Hypoproteinemia was also observed (4.5g/dl).

2.4.2 Cardiac

Shaw and Rush (2007) stated that in canine pericardial effusion, mild anaemia, increased leucocytes, nucleated erythrocytes, schistocytes or acanthocytes can be found in laboratory examination. Serum biochemistry analyses were generally in normal range

except increase in blood urea nitrogen and creatinine due to pre-renal azotaemia developed by decrease cardiac output.

Ihedioha et al. (2011) investigated the reported ascites cases for haematology and serum chemistry test in dogs suffering from chronic heart failure. The haematology revealed that the PCV and MCV (mean corpuscular volume) were decreased with RBCs, Hb and TLC within normal limits. The serum chemistry levels of the liver enzymes (ALT, AST), creatinine, urea, and globulin were increased with low levels of total protein and albumin.

Kumar and Srikala (2014) presented a case report on a female Labrador suffering with right side heart failure, with haemoglobin 5.8 g/dl and total erythrocyte count 7.8 × 10^6/mL revealing anaemia with normal level of total leukocyte count (TLC; 10.4 × 10^3/mL), and differential leukocyte count (DLC; neutrophil- 68, lymphocyte-29, monocyte-01 and eosinophil-02). Moderate decrease in total protein (4.12 g/dl), albumin (3.04 g/dl) and increased alanine aminotransferase (ALT; 78.60 U/L) and alkaline phosphatase (ALP; 128 U/L) were recorded.

Mukherjee et al. (2017) did a case of 4 year old male Labrador retriever ascitic dog that revealed Haemoglobin 10.2 gm %, Total Leukocyte Count 12,000/cu mm, Neutrophils 86%, Lymphocytes 12%, Eosinophil 1% and Monocyte 1% on haematological analysis and serum biochemistry revealed Blood Glucose 65 mg/dl, Total Protein 5.9 g/dl, Albumin 3.8 g/dl, Globulin 2.1 g/dl, Total bilirubin 1.23 mg/dl, Direct Bilirubin 0.50 mg/dl, Indirect Bilirubin 0.73 mg/dl, Alkaline Aminotransferase (ALT) 65.8 IU/L, Aspartate aminotransferase (AST) 87.9 IU/L, Alanine amino phosphatase (ALP) 471.1 IU/L, Blood Urea Nitrogen (BUN) 23.6 mg/dl and Creatinine 1.89 mg/dl.

Alsaad et al. (2018) reported a case of a male Mullinoise breed a police (K9) dog suffering from ascites due to congestive heart failure. The haemato-biochemical profile revealed anaemia (8.5 g/dl) and thrombocytopenia (110 ×10^3/L) and increased MCV (93.33 fl). The level of total cholesterol was increased (440 mg/dl) and total protein was decreased (2.9 g/dl).

2.4.3 Renal

Kumar et al. (2003) studied 20 dogs that were suffering from ascites and found that 15 per cent of the dogs developed the condition due to renal disturbances. The mean values

of haemoglobin (Hb), packed cell volume (PCV), total leucocyte count (TLC) and total erythrocyte count (TEC) as 8.66 ± 0.98 g/dl, 28.66 ± 1.76%, 18.65 ± 0.72 × 10^3 / cumm, 4.73 ± 0.29 × 10^6/cumm. The mean serum biochemical analysis as alanine amino transferase (ALT) (22.0 ± 2.30 U/L), aspartate aminotransferase (AST) (29.0 ± 2.30 U/L), alkaline phosphatase (ALP) (57.96 ± 2.97 U/L) were in normal range, blood urea nitrogen (BUN) (39.0 ± 2.97 mg/dl), serum creatinine (1.26 ± 0.06 mg/dl) were increased and total protein (TP) (3.8 ± 0.20 g/dl), albumin (1.43 ± 0.08 g/dl) and A:G ratio (0.60 ± 0.007) were decreased.

Ihedioha et al. (2011) investigated the reported ascites cases for haematology and serum chemistry test in dogs suffering from kidney disease. The haematology revealed that the PCV, RBCs, Hb, MCV and TLC were decreased as compared with the healthy group. ALT, AST, creatinine and urea were increased but Hypoproteinemia and hypoalbuminemia with increased level of globulin was observed.

2.4.4 Hepato-Renal

Chutia et al. (2016) studied 3 years old female crossbred dog having ascites due hepato-renal dysfunction (HRS), the haematology revealed increased WBC (29.09 m/mm^3), platelets distribution width 910.5), RBC distribution width (12.7), Lymphocytes (44.4%) and Monocytes (15.9%) and decreased RBC (5.59 M/mm^3), Haematocrit (29.5%), Haemoglobin (7.8 g/dl), Mean Corpuscular Haemoglobin (16.9 Pg), Mean Corpuscular haemoglobin Concentration (26.4 g/dl) and Granulocytes (39.7%).

Phom et al. (2019) studied 6 cases of ascites due to hepato-renal problems. The haemato-biochemical analysis revealed anaemia (6.60 ± 1.80 g/dl), neutrophil (80.00 ± 0.60%) leucocytosis (14.12 ± 1.36 ×10^3/µl), hypoprotenemia (3.90 ± 0.26 g/dl), hypoalbuminemia (1.70 ± 0.20 g/dl), hyponatremia (114.66 ± 3.75 mEq/L), hypochloraemia (87.00 ± 1.52 mEq/L), hyperbilirubinemia (2.56 ± 0.13 mg/dl), increased alanine aminotransferase (ALT) (116.00 ± 2.64 U/L), alkaline phosphatase (ALP) (137.67 ± 1.45 U/L), blood urea nitrogen (BUN) (54.00 ± 1.75 mg/dl) and creatinine (2.05 ± 1.22 mg/dl) level in all dogs.

2.4.5 Hepato-Cardiac

Bhadesiya et al. (2015) did a case of a two month old German shepherd female dog with haemato-biochemical findings of anaemia (7.9 g/dl), thrombocytopenia (102 ×10^5/cmm), increased level of liver enzymes, serum glutamic pyruvic transaminase

103 U/L, serum glutamic oxaloacetic transaminase 108 U/L, Alkaline Phosphatase 101 U/L, cholesterol 308 mg/dl, troponin-1 0.12 ng/ml and decreased total protein 6.8 g/dl, albumin 2.4 g/dl and globulin 4.4 g/dl.

2.5 ELECTROLYTE ANALYSIS

Shaw and Rush (2007) stated that in canine pericardial effusion, mild hyponatremia, hypochloremia and hyperkalemia occur in some cases especially in dogs with considerable ascites due to decrease in effective circulatory volume.

Ihedioha et al. (2011) studied 14 dogs suffering ascites, classified them in group 1 (cirrhotic liver disease), group 2 (chronic active hepatitis), group 3 (congestive heart failure) and group 4 (kidney disease) and estimated sodium and chloride levels, where the levels of both were decreased; 100.00±5.66 mmol/L, 79.20±9.62 mmol/L (group 1), 106.32±7.88 mmol/L, 83.17±8.23 mmol/L (group 2), 93.87±17.76 mmol/L, 89.57±3.41 mmol/L (group 3), 104±9.06 mmol/L, 87.40±6.00 mmol/L (group 4), respectively.

Saravanan et al. (2014) did electrolyte estimation in 72 ascitic dogs that revealed slight increase in the sodium ion (139.2±1.7 mEq/L) and slight decrease in potassium ion (4.3±0.1 mEq/L).

Lakshmi et al. (2018) studied 32 dogs suffering from ascites due to diffuse liver parenchymal disorders, the electrolyte estimation revealed a significant decrease in sodium (134.77±1.23 mEq/L), potassium (3.56±0.08 mEq/L) and chloride (97.87±0.85 mEq/L).

2.6 ASCITIC FLUID ANALYSIS

2.6.1 Physical and Biochemical Examination

Wadhwa et al. (1995) studied ascitic fluid in 5 dogs; the collected fluid was clear watery, pale yellow, non-cloudy and odourless with mean pH, specific gravity and protein as 7.52 ± 0.03, 1.01 ± 0.80 and 1.04 ± 0.08 g/dl, respectively.

Vijayakumar (2002) classified the ascitic fluid into 3 categories on the basis of protein, specific gravity and cells along with the condition they arise. Transudate: protein 2.5 g/dl, SG <1.018, cells 1000/cmm (hypoproteinemia, liver disease). Modified transudate: Protein 2.5 to 5 g/dl, SG >1.018, cells <5000/cmm (cardiac heart failure, abdominal tumour, obstruction of hepatic vein, liver disease). Exudate: Protein >4.0 g/dl,

SG >1.018, cells 5000-10,000/cmm (bleeding condition, tumour, bacterial infection caused by bowel comprise, chylo abdomen, urine, bile leakage, peritonitis).

Kumar et al. (2003) studied ascitic fluid in hepatic (Group 1) and renal (Group 2) ascites. Fluid was transudate and clear in both the groups, the specific gravity was 1.008 to 1.015 and 1.010 to 1.015, of group 1 and group 2, respectively. The pH ranged from 7.5 to 8.0 and chemical examination revealed presence of glucose and protein in traces in both groups.

Gupta et al. (2004) examined ascitic fluid collected by paracentesis from a 10 years old female German shepherd, the fluid was clear, watery, odourless and straw coloured.

Mondal et al. (2012) stated that the transudate was colourless/clear with protein <2.5 g/dl, specific gravity of <1.017 and WBC <1,000/µl. Modified transudate was light yellow to medium yellow and was clear with protein content >2.5 g/dl, specific gravity <1.017-1.025 and WBC >1,000/µl. Exudate was medium yellow to tan and was cloudy with protein content >3.0 g/dl, specific gravity >1.025 and WBC >5,000/µl.

Sarvanan et al. (2012) analysed the ascitic fluid of 10 dogs positive for ascites. Total protein 0.770 ± 0.086 g/dl, albumin 0.296 ± 0.057 g/dl, globulin 0.473 ± 0.099 g/dl, A/ G ratio 1.024 ± 0.378, SAAG 1.793 ± 0.185 g/dl, were the findings.

Srivastava and Syed (2013) analysed the ascitic fluid, the fluid was transudate in nature as it was negative for Revalta reaction. They stated that the ascitic fluid more or less turbid and pink coloured in pre and post-hepatic cause. In intrahepatic cause a moderate portal hypertension and clear, non-haemorrhagic colourless transudate fluid is observed. Pink colour of the fluid indicates portal vein or thoracic vena cava obstruction or a tumour (cardiac failure).

Vijayakumar et al. (2013) studied ascitic fluid of 12 dogs suffering the condition due to hepatobiliary dysfunction. The fluid was colourless and clear transudate, the mean specific gravity was 1.016 to 1.015 ± 0.0, cell count being 244 to 263 cells/ cmm ± 8.3 to 7.7, total protein 1.401 to 1.798 ± 0.24 to 0.31 g/dl, albumin 0.98 to 0.96 ± 0.22 to 0.31 g/dl, SAAG 1.14 to 1.19 ± 0.20 to 0.37.

Gonde et al. (2014) analysed the ascitic fluid of 5 year old Labrador male dog revealing the fluid to be clear and transparent with total protein and albumin content as 0.4 g/dl and 0.2 g/dl, respectively, that indicated hypoproteinemia and hypoalbuminemia.

Kumar and Srikala (2014) studied a case of female Labrador suffering from ascites due to right side heart failure. The aspirated peritoneal fluid was colourless, transparent, and odourless with a mean protein content of 2.8 g/dL, specific gravity of 1.010 and cellular content of 1350/cmm, suggested that the fluid was transudate in nature.

Saravanan et al. (2014) found clear ascitic fluid in 72 dogs; the specific gravity (1.014±0.001), total nucleated cell count (388.93±66.32/cmm), total protein (1.96±0.08 g/dl), albumin (0.83±0.05 g/dl) and serum ascitic albumin gradient (1.20±0.05 g/dl) revealed the ascitic fluid to be transudate in nature. They stated that ascitic fluid analysis and serum ascitic albumin gradient (SAAG) may be a key indicator for the diagnosis of aetio-pathogenesis of ascites in dog.

Bhadesiya et al. (2015) examined the ascitic fluid collected from a female German shepherd dog and the fluid was complete transudate with pH 10.0. Microscopic examination and gram staining were negative for epithelial cells or pus cells and bacteria, respectively.

Rai and Chandrapuria (2017) examined ascitic fluid of 12 dogs that on examination revealed clear to straw colour and total protein was less than 2 g/dl.

Regmi and Shah (2017) conducted study on ascites in a 11 year old German Shepherd dog and the ascitic fluid examination revealed to be transudate (clear watery with slight reddish tinge) with serum albumin ascitic gradient (SAAG) >1.1 gm/dl that suggested ascites to be due to portal hypertension caused by liver cirrhosis. Total leukocyte count (42 cells/mm^3), neutrophils (55%), lymphocytes (45%), sugar (79 mg/dl), protein (2.1 g/dl), albumin (0.8 g/dl) and globulin (1.3 g/dl) were also estimated.

Zoia et al. (2017) did haemostatic study of ascitic fluid in 70 dogs, 28 dogs had transudate fluid, 10 due to decrease colloid osmotic pressure (group 1) and 18 due to increased hydrostatic pressure (group 2), 13 had exudate (group 3) and 29 had haemorrhagic ascites (group 4). Group1 had serum total protein <4 g/dl and group 2 had serum total protein >4 g/dl and ascitic total protein in both group 2 and group 3 was similar but group 3 had highest fluid total nucleated cell count. Group 4 had similar serum and

ascitic total protein but the peripheral haematocrit was >50% whereas in all other groups it doesn't reached 25%.

Phom et al. (2019) conducted a study on ascitic fluid in 6 dogs suffering from ascites due to hepato-renal dysfunction. The ascitic fluid analysis revealed that the colour of the ascitic fluid was exuded ascites fluid and the values of specific gravity, total leukocyte count (cells/ml), total protein(g/dl), albumin(g/dl) and SAAG(g/dl) were 1.027, 8054, 3.5 0.1 and 0.60 respectively.

2.6.2 Cytology

Wahwa et al. (1995) did cytology of ascitic fluid in 5 dogs and the smear showed no RBCs and mesothelial cells.

Mondal et al. (2012) stated that in transudate fluid predominant cell type were mesothelium and mononuclear phagocytes and in modified transudate the predominant cell type was mononuclear cells whereas in exudate neutrophils (non-septic: non-degenerate, septic: degenerate) were the predominant cell type.

Vijayakumar et al. (2013) studied ascitic fluid cytology in 12 dogs that contained mostly mononuclear cells such as lymphocytes, macrophages and mesothelial cells with low number of non-degenerate neutrophils.

Saravanan et al. (2014) did ascitic fluid (transudate) cytology that revealed few mesothelial cells, lymphocytes, monocytes and neutrophils in almost all the dogs (70 dogs) whereas two dogs showed tumour cells in the ascitic fluid.

Phom et al. (2019) conducted a study on ascitic fluid in 21 dogs, whose cytology revealed the presence of few mesothelial cells, lymphocytes, monocytes and neutrophils in most of the ascitic dogs.

2.7 URINALYSIS

Chiyoda et al. (1992) selected 11 patients suffering with ascites, few white and red blood cells (in 2 patients) per high-power field in the sediment, 3+ tests for protein, were the findings in urine analysis.

Chakrabarti et al. (1994) analysed urine in 5 dogs suffering from ascites, the analysis revealed slight to moderate proteinuria, bilirubinuria and increased urobilinogen level.

Vijayakumar (2002) stated that during ascites of renal origin, proteinuria along with inactive urine sediment (hyaline cast) and altered protein: creatinine ratio (normal =<0.5) was observed.

Kumar et al. (2003) performed urinalysis in 20 dogs, 17 dogs had liver origin ascites (group 1) and 3 dogs of kidney origin ascites (group 2). Both the groups had pale yellow to deep yellow colour of the urine and appearance slight turbid to moderate was observed. The specific gravity ranged from 1.010 to 1.015 and 1.010 to 1.020 for group 1 and group 2, respectively. Chemical examination revealed trace proteins in group 1 and mild to moderate protein in group 2 with 3-6 pus cells per HPF in microscopic examination.

Gupta et al. (2004) examined urine of 10 years old female dog suffering from ascites. Physical examination revealed pale yellow colour and was clear. On chemical examination the urine was acidic in reaction with traces of albumin and had no sugars.

Bhadesiya et al. (2015) did urinalysis in a female dog having ascites that was negative for ketones, blood and nitrates and also negative for crystals and casts. The physical examination revealed straw yellow colour, non-turbidity, pH of 6.5 and specific gravity of 1.030.

Kozat and Sepehrizadeh (2017) stated that urine specific gravity can be decreased in patients with hepatic insufficiency or portosystemic shunts. Bilirubinuria (<2+ on a dipstick) can be a normal finding and urate urolithiasis is more common in patients with PSS than those with other types of hepatic dysfunction.

2.8 ELECTROCARDIOGRAPHY

Laflamme (1997) reported that in hypokalemia, electrocardiograph abnormalities were observed that included flattening of T wave and increase in the PR interval.

Vijayakumar (2002) stated that reduced amplitude of electrocardiograph was observed in ascites and ECG can be normal during chronic Hepatic Failure. Elevation of T wave can be present in nephrotic syndrome. Right ventricular enlargement can be

evident with tall P wave in lead II and deep S wave in lead I and II indicates atrial/ ventricular arrhythmia.

Shaw and Rush (2007) observed that sinus tachycardia was a frequent finding in pericardial effusion whereas premature ventricular contraction occurs less frequent. Low voltage QRS complex (<1 mV) may occur due to increased electrical impedance and also stated pericardial effusion can't be ruled out by a normal electrocardiography.

Kumar and Srikala (2014) performed electrocardiography in 8 years old Labrador female dog that revealed low voltage QRS complex, which suggested effusion in body cavity.

Kocatürk et al. (2016) performed electrocardiography in 8 dogs suffering ascites that revealed cardiac rhythms abnormalities. Atrial fibrillation was found in 5 dogs, ventricular extra systoles in 2 dogs, atrio-ventricular block in 1 dog and sinus tachycardia in 2 dogs.

Mukherjee et al. (2017) did electrocardiography in a 4 year old male Labrador retriever ascitic dog that revealed ST segment depression indicating myocardial ischemia.

Phom et al. (2019) carried out ECG in 6 dogs having ascites due to hepato renal problem and found sinus tachycardia, atrial standstill and prolonged QRS duration.

2.9 IMAGING STUDIES

2.9.1 Radiography

Whiteley et al. (1989) performed radiography of 48 dogs, 14.6 per cent (7/48) had abnormal livers (3 lymphosarcoma, 3 metastatic carcinomas, 1 metastatic sarcoma), 25 per cent (12/48) had symmetrical hepatomegaly (5 lymphosarcoma, 4 metastatic carcinoma, 2 primary carcinomas, 1 metastatic sarcoma) and 60.4 per cent (29/48) had asymmetrical hepatomegaly (17 primary carcinomas, 5 metastatic sarcomas, 3 lymphosarcomas, 3 metastatic carcinomas, 1 primary sarcoma). Peritoneal effusion was seen in 9 dogs (5 primary carcinomas, 2 lymphosarcomas, 1 metastatic carcinoma, and 1 metastatic sarcoma).

Shaw and Rush (2007) stated that in pericardial effusion the thoracic radiographs reveal mild to severe enlargement of cardiac silhouette that increase in conjunction with

chronicity and this silhouette appears globoid or rounded with sharp edges. Abdominal radiographs may show hepatomegaly or ascites that is secondary to right sided heart failure.

Srivastava and Syed (2013) did radiography in 6 year old Boxer dog, abdominal radiograph showed significant ascites due to which the abdominal structures were obscured by nature of fluid.

Bhadesiya et al. (2015) performed radiography in a female German shepherd dog that revealed ground glass in lateral abdominal radiograph and elevated trachea and rounding of cardiac silhouette in lateral thoracic radiograph.

Kocatürk et al. (2016) found enlarged heart size, deviation of trachea, mild to severe pulmonary oedema, increased vertebral heart scale (14.2±1.3), pulmonary pattern, and caudal vena cava distension and pleural on thoracic radiograph and peritoneal effusion on abdominal radiograph in 8 dogs.

Kumar et al. (2016) studied a nine year old German shepherd female dog, ground glass appearance in abdominal cavity and thoracic cavity and slight enlargement of the heart were the findings of the radiographic examination.

Neelam et al. (2019) reported a case of 4 month Bully female dog that revealed ground glass appearance of abdominal cavity and thoracic cavity with slight pneumonic changes in lungs.

Kozat and Sepehrizadeh (2017) stated that abdominal radiographs allowed assessment of hepatic size, shape, opacity and location and may also allow identifying extra-hepatic abnormalities that affect liver. The abdominal radiograph was often normal in patients with hepatobiliary disease.

Phom et al. (2019) conducted study in 6 dogs suffering from ascites due to hepato-renal dysfunction and radiographic examination showed ground glass appearance and VHS (Vertical Heart Size) was 13.25 ± 0.35.

2.9.2 Ultrasonography

Whiteley et al. (1989) reviewed 48 ascitic cases with ultrasonography and observed that 93.3% dogs had focal abnormal liver echo-texture with hepatocellular carcinomas,

remaining had metastatic carcinoma, out of which 60 per cent had metastatic disease, 20 per cent had primary disease and 20 per cent had lymphosarcoma.

James et al. (2008) studied ultrasonography in 16 dogs suffering from ascites due to pre-sinusoidal portal hypertension, liver size was small in 7/16, normal in 5/16 and enlarged in 1/16 and increased echogenicity of spleen in 7/16. Irregular capsule (3/16), nodular architectural changes (4/16) and extra-hepatic porto-systemic shunting vessels were identified in 5/16 dogs.

Sarma et al. (2009) did ultrasonography of 15 dogs having ascites that revealed presence of anechoic abdominal fluid and hepatomegaly in almost all the affected dogs and cyst in the liver in one dog.

Kumar and Srikala (2014) did ultrasonography revealing floating viscera in anechoic abdominal fluid in 8 year Labrador female dog. In liver, presence of hyperechoic to mixed echogenicity indicated hepatic disease and was greatly engorged with dilated portal and hepatic veins with hyperechoic focal areas and rounded borders of hepatic lobes suggesting hepatomegaly associated with congestive heart failure.

Gonde et al. (2014) performed ultrasonography in 5 year old male Labrador suffering from abdominal distension due to *Babesia gibsoni* and found anechoic abdominal fluid.

Saravanan et al. (2014) performed abdominal ultrasound in 72 dogs that revealed anechoic fluid in abdominal cavity. 18 dogs (25%) had focal hyper-echoic and 33 dogs (54.8%) had diffused hyper-echoic liver parenchyma with normal size of the liver. 19 dogs (26.38%) had liver cirrhosis and 10 dogs (13.90%) suffered gall bladder disorders (cholecystitis/choleithiasis/distension).

Bhadesiya et al. (2015) did ultrasonography of ascitic dogs that revealed free fluid in abdominal cavity along with hyper-echoic foci on the caudal lobe of liver in a two months old female German shepherd dog.

Elhiblu et al. (2015) did ultrasonography of liver in dogs suffering from ascites that revealed diffuse increase in echogenicity “bright liver” with rounding of liver margins. The other findings were of microhepatica (5), distension of gall bladder with wall thickness (4),

mild hepatic congestion (2) i.e. hepatic veins were moderately dilated and visualized prominently in hepatic parenchyma.

Manoj and Lakshmi (2015) performed ultrasonography of five year old Pomeranian cross with distended ventral abdomen that revealed free fluid in the peritoneum as an anechoic area that confirmed it as ascites.

Chutia et al. (2016) performed ultrasonography in 3 year old crossbred female dog that revealed enlarged size of the kidneys, hyperechoic liver with irregular border and large anechoic pocket in abdomen.

Kozat and Sepehrizadeh (2017) stated that ultrasonography allowed the assessment of hepatic parenchyma and biliary tract. Extra hepatic disease that caused secondary hepatopathy may also be detected as hepatomegaly or micro-hepatica.

Mukherjee et al. (2017) performed ultrasonography in a 4 year old male Labrador retriever dog having ascites where the urinary bladder, kidneys, spleen were of normal size and shape and liver lobes were floating in anechoic fluid. The gall bladder thickness was 2.84 mm.

Rai and Chandrapuria (2017) performed ultrasonography in 12 ascitic dogs that revealed increase in hepatic echogenicity and irregular surface of liver. The colour Doppler showed dilated portal vein and portal hypertension along with massive amount of anechoic fluid in abdomen with floating viscera.

Alsaad at al. (2018) found slight hepatomegaly and ventricular hypertrophy along with cardiac effusion and floating abdominal viscera in anechoic abdominal fluid in a male Mullinoise breed K9 dog.

Phom et al. (2019) did ultrasonographic examination on 6 dogs suffering from ascites due hepato-renal problem. The examination revealed isoechoic kidney, renal pyramids as anechoic echotexture, focal hyper-echoic and loss of echogenicity of hepatic parenchyma along with ascites.

2.9.3 Echocardiography

Shaw and Rush (2007) stated that echocardiography was a gold standard for diagnosis of pericardial effusion. Echo-free space was evident between pericardial sac and

epicardium from right parasternal view. Left parasternal view allowed better visualization of right side heart which aid in identifying right atrial masses. A fluid volume of as low as 10 to 15cm can be detected.

Kumar and Srikala (2014) performed 2-D echocardiography that revealed enlarged and globose heart with dilated right ventricle on right parasternal short axis view on B-mode whereas M-mode showed relatively increased right ventricle lumen in both diastole and systole condition. Doppler showed jet like turbulent flow and mosaic pattern of colour of mitral and tricuspid valves respectively.

Kocatürk et al. (2016) did 2-D echocardiography in 8 dogs suffering from ascites, the findings of echocardiography indicated Dilated Cardiomyopathy through increased chamber size, increased E-point to septal separation (EPSS) (1.1±0.3 cm) and poor Fractional shortening (FS) (15.8±4.8 %), pulmonary artery flow velocity (0.6 ±0.09 m/s) was higher but aortic flow velocity (0.7±0.14 m/s) was lower as compared to the healthy dogs.

Phom et al. (2019) found dilated cardiomyopathy, pleural effusion, increased E-point to septal separation and increased LA: Ao ratio in 6 dogs suffering from ascites due to hepato renal problem.

2.10 THERAPEUTIC STUDIES

2.10.1 Hepatic

Chakrabarti et al. (1994) managed 5 dogs suffering ascites with Inj. Pepsid @1-2.5 ml, Syrup Neodox forte @10-20 mg/kg, Diuretic (Tab. Lasilactone @ 1 tab). They indicated the use of Lipotrophic agents and liver extract as they help to minimize the fat accumulation and also to add neomycin to prevent liver damage by bacterial infections.

Wadhwa et al. (1995) treated 5 dogs with a history of bilateral abdominal distension due to liver involvement with Inj. Liver extract with B complex, tab Liv 52, tab Lasix 20 mg and supportive treatment included osteocalcium syrup, protein and hematinic oral supplements and fluid therapy and antibiotic during abdominocentesis (300ml to 1 litre).

Gupta et al. (2004) treated a female dog suffering from ascites of hepatic origin with liver safe antibiotic (Amoxycillin 250 mg + Cloxacillin 250 mg), liver extract with

B-complex, liver syrups (Liv-52 vet), Diuretics (Frusemide @2mg/kg), liver protective tablet (Silymarin 70 mg). Abdominocentesis was also performed.

Pradhan et al. (2008) stated that ascites can be more effectively treated by low sodium diet and potassium sparing diuretic.

Raffan et al. (2009) treated 28 dogs (14 ascitic and 14 non-ascitic) with prednisolone, antibiotics, lactulose, ursodeoxycholic acid, S-adnesylmethionine, colchicine and gastric protective drugs. Spironolactone was commonly used in ascitic group.

Rautray et al. (2010) treated 2 dogs suffering from ascites due to hepatitis with antibiotic (Ceftriaxone tazobactum 562.5 mg), tab fruselac DS (frusemide + spironolactone), liver protective drugs (Inj. Neohepatex, Syrup Liv-52), proteinex powder with dietary restrictions of salt.

Dabas et al. (2011) reported case of ascites of splenic origin in mongrel female dog and was treated with antibiotic (ceftriaxone @500mg), diuretic (frusemide @ 2mg/kg), and Inj. Pheniramine maleate @ 1ml and Inj. Ketoprofen @2ml along with supportive therapy of hematinic mixture with protein diet.

Saravanan et al. (2013) treated ascitic dogs with diuretics (Frusemide @ 2mg/kg) with supportive treatment of B complex and Vitamin C and antibiotic.

Srivastava and Syed (2013) did abdominocentesis along with fluid therapy recovering 1.0 litre of fluid aseptically, diuretics (frusemide @ 4 mg/kg), antibiotic (ampicillin 500 mg), tab. Betnelan 1mg, liver tonics were also prescribed.

Kashyap et al. (2015) treated a 5 year old Saint Bernard dog having ascites with abdominal paracentesis, Spironolactone @1 mg/kg/day, with supportive therapy of Inj. B-complex (polybion @ 3ml/day IM), Liver tonics (Liv 52), vitamin E and vitamin K, fluid therapy, protein rich diet with low copper and salt.

Manoj and Lakshmi (2015) did treatment of a 5 year old ascitic Pomeranian cross with Ceftriaxone sodium @20 mg/kg body weight, Inj. Lasix 400mg, Inj. B complex; tab Aldactone 100mg and Liv 52 syrup.

Kumar et al. (2016) treated 9 year old German shepherd female dog suffering from ascites due to liver dysfunction. The treatment included laparotomy, fluid therapy, tab lacilactone, liver tonics (Livotas pet), inj. Ataxin and Inj. Neohepatex. Protein rich diet with low salts was advised.

Kumar and Singh (2017) managed a dog of 3 and half months old suffering from ascites with abdominocentesis (200 ml), fluid therapy, diuretic (Frusemide @ 4mg/kg body weight), liver tonics (Livotas), protein diet with low salts.

Regmi and Shah (2017) managed 11 year old German shepherd having ascites by abdominocentesis followed by fluid therapy (normal saline and albumin), diuretics (frusemide @ 4mg/kg), anti-biotic (tab ampicillin 250 mg + cloxacillin 250 mg), tab Ursodeoxycholic acid 300mg, Syp. Livoferol. The diet included cooked buffalo or goat liver with restriction to salt.

Samad (2019) performed abdomenocentesis in a Spitz ascitic dog and further treated with amoxicillin suspension @ 250 mg 8 hourly, frusemide @2mg/kg, supportive treatment of B-complex and Vitamin C.

2.10.2 Cardiac

Henik (1997) suggested use of diuretics (chlorothiazide @20-40 mg/kg, frusemide @2-4 mg/kg, hydrochlorothiazide @2-4 mg/kg, spironolactone @ 1-2 mg/kg triamterene @1-2 mg/kg), alpha-blockers (prazosin @0.5-2 mg/dog, phenoxybenzamide @0.25 mg/kg), beta-blockers (atenolol @6.25-12.5 mg/dog, metoprolol @5-50 mg/dog, propranolol @0.2-1 mg/kg), calcium channel blockers (amlodipine @0.05-0.10 mg/kg) and vasodilators (benazepril @0.25 mg/kg, captopril @ 0.5-2.0 mg/kg, enalapril @0.5 mg/kg, lisinopril @0.25-0.5 mg/kg, hydralazine @ 0.5-2.0 mg/kg, sodium nitroprusside @2.5-15.0 μg/kg/min IV CRI) for systemic hypertension.

Dove (2001) reported that coenzyme Q 10 (CoQ10), Vitamin E (alpha-tocopherol), L-carnitine, taurine and fish oil (omega-3-fatty acids) were helpful in preventing and treating the heart diseases in canines. These dietary supplements also increase the life span, improve life quality and decrease the mortality rate in canines suffering from heart diseases.

Kumar and Srikala (2014) treated a dog suffering from ascites with right heart failure with angiotensin receptor blockers (losartas @25mg), aldosterone blockers (spironolactone @ 5 mg/kg), syp. Sorbiline (tricholine citrate and sorbital) and nutriceutacle (co-enzyme Q-10, 1 tab).

Mukherjee et al. (2017) suggested that ascites of cardiac origin can be treated with standard protocol that includes diuretics (Frusemide @2-4 mf/kg BID PO) and ACE inhibitors (Benazepril/ Enalapril @ 0.5 mg/kg SID to BID PO). In dogs suffering from arrhythmia, beta adrenergic blockers or calcium channel blockers should be used to improve oxygenation of heart and slow down the heart rate. Pimobendan @ 0.25 mg/kg BID PO was used to improve the survival.

Alsaad et al. (2018) managed a dog with Beta–blocker and blood pressure regulator (Nebivolol 1 mg orally for 5 days), fluid therapy (Ringer's solution) and a diuretic (Frusemide 4 mg IM for 5 days) and abdominocentesis in a male police dog weighing 23 kgs.

2.10.3 Hepato-Renal

Chutia et al. (2016) treated 3 year old female dog suffering from ascites of hepato-renal origin with diuretics (Frusemide @2 mg, tab Spironolactone @12.5 mg), cortico-steroid (Dexamethasone @2.2 mg), fluid therapy (Dextrose 10% @ 150 ml, Amino acid (Hermin @ 50 ml) and anti-biotic (ceftriaxone @10 mg/kg).

Materials and Methods

CHAPTER 3 MATERIALS AND METHODS

3.1 PLACE OF STUDY

The present investigation work was conducted on dogs presented in the Department of Veterinary Medicine, College of Veterinary and Animal Sciences, CSKHPKV, Palampur (H.P.) during the period of October 2018 to March 2020. During this period, 2063 dogs were presented in department of veterinary medicine.

3.2 SELECTION OF THE ANIMALS

On the basis of history, clinical manifestations and physical examination, cases that were suspected of ascites were studied. A total of 47 dogs were studied, among which most of the cases had appreciable abdominal distension. 10 healthy dogs that were presented for vaccination and/ or general check-up, without any clinical manifestation of illness were taken as control group.

3.3 SCREENING OF THE ANIMALS

Biochemical studies (Plasma biochemical and ascitic fluid analysis) and/ or Diagnostic studies (Radiography and Ultrasonography), wherever possible and needed were employed for confirmatory diagnosis of animals suffering from ascites.

Based on the elementary screening and final diagnosis, 47 dogs that were affected with this condition were broadly grouped into three major categories that were hepatic origin (35), cardiac origin (6) and renal origin (6).

The dogs with signs of haemoprotozoan were screened for the same and the final diagnosis revealed six ascitic dogs had haemoprotozoan infection.

3.4 CLINICAL EXAMINATION

3.4.1 History and signalment

A comprehensive study including the data of the animal was recorded with respect to breed, age, body weight and gender. The detailed history that comprised of feeding, duration of distension, appetite status, water intake, urination, defecation, vaccination, deworming, any previous illness/ treatment and response thereof. Clinical signs observed

by the owner like vomiting, coughing, exercise intolerance, weight loss, weakness and seizures, etc. were also recorded.

3.5 PHYSICAL EXAMINATION

A complete physical examination of the patient was carried; general status, rectal temperature, colour of visible mucous membrane, bodily condition, examination of the skin and dehydration status were recorded. Palpation and percussion was carried out at the abdominal region for pain and/or mass and/or organomegaly. Auscultation of the thoracic area including lungs and heart was done. In dogs with abnormal auscultation, radiography and electrocardiogram was performed whenever possible. On the basis of all the observations/ examination, a tentative diagnosis was concluded.

3.6 LABORATORY EXAMINATION

3.6.1 Sample collection

3.6.1.1 Blood sample collection and storage

After proper restraining of the animal in lateral recumbency, the blood sample was collected aseptically before and after treatment. Approximately 2ml of the blood was collected from cephalic vein or recurrent tarsal vein using sterile syringe, with needle in a sterile plastic vial containing tri-potassium salt of ethylenediamine-tetra acetic acid (3.6mg/2ml) for complete blood count. In another sterile syringe about 5ml of blood was collected in heparinized syringe for the collection of plasma for biochemical parameters. These plasma samples were preserved at -20 ^{0}C in deep freezer for further biochemical and electrolyte analysis. The blood samples were collected before treatment (Day 0) and after treatment (Day 30 and Day 90) from the animals of different groups.

For acid base estimation, 2ml of blood was collected aseptically and anaerobically in sterile heparinized syringe from cephalic or recurrent tarsal vein taking all the necessary precautions like no contact of blood with air was allowed. The blood collected was analysed for blood gases and acid base status within 5-10 minutes of collection.

3.6.1.2 Urine sample collection

Urine samples for analysis were collected by performing catheterization in cases where some observable changes were noticed by the owner. About 5ml of the midstream

urine were collected in a clean sterile syringe for urinalysis and centrifuged urine sediment smear examination. The urine samples were collected before treatment (Day 0) in affected dogs.

3.6.2 Haematological examination

The haematological examination was done on BC-2800 Vet Auto Haematology Analyser (Marketed by Fresenius Medical Care Private Limited, New Delhi) (Plate 3.1). The parameters that were determined are listed below along with the units in which they were expressed:

S. No.	Parameters	Units
1.	Haemoglobin (Hb)	Gram per decilitre (g/dl)
2.	Packed Cell Volume (PCV)	Per cent (%)
3.	Total Leucocyte Count (TLC)	($x10^9$/L) of blood
4.	Total Erythrocyte Cou0nt (TEC)	($x\ 10^{12}$/L) of blood
5.	Differential Leucocyte Count (DLC)	Per cent (%)
6.	Mean Corpuscular Volume (MCV)	Femtolitre (fl)
7.	Mean Corpuscular Haemoglobin (MCH)	Pico gram (pg)
8.	Mean Corpuscular Haemoglobin Concentration (MCHC)	Per cent (%)
9.	Platelet Count (PLT)	($x10^9$/L) of blood

Plate 3.1: BC-2800 Vet Auto Haematology Analyser

3.6.3 Plasma biochemical, mineral and electrolyte analysis

The biochemical analysis and mineral estimation and electrolyte estimation were carried on semi-automatic biochemical analyser Microlab 300 Clinical Chemistry Analyser (by Merck Limited, Mumbai) (Plate 3.2) and CB- LYTE Automatic Electrolyte Analyser (by Chariot's Biotechnology, Navi Mumbai) (Plate 3.3), respectively. These biochemical parameters and mineral estimations were done using kits that are expressed below in Table 3.1 and Table 3.2, respectively and electrolyte estimation by using standard calibration pack as listed in Table 3.2.

Table 3.1: Methods used for estimation of biochemical parameters

S. No.	Parameters	Method of Estimation	Units
1.	Alanine Aminotransferase (ALT)*	IFCC recommended methodology	U/L
2.	Aspartate Aminotransferase (AST)*	IFCC recommended methodology	U/L
3.	Alkaline phosphatase (ALP)*	DGKC-SCE recommended procedure	U/L
4.	GGT *	Szasz methodology	U/L
5.	Glucose *	GOD-PAP methodology	mg/dl
6.	Total Protein (TP)*	Direct Biuret method	g/dl
7.	Albumin *	Bromocresol green method	g/dl
8.	Globulin	Subtracting albumin from Total protein	g/dl
9.	Albumin Globulin ratio (A/G ratio)	Dividing values of albumin by values of globulin	(Numeric Value)
10.	Total and Direct Bilirubin *	Modified TAB method	mg/dl
11.	Indirect Bilirubin	Subtracting Direct Bilirubin from Total bilirubin	mg/dl
12.	Blood Urea Nitrogen (BUN)*	Urease/GLDH methodology	mg/dl
13.	Creatinine *	Modified Jaffe's method	mg/dl
14.	Cholesterol*	CHOD-PAP method	mg/dl

Table 3.2: Methods used for mineral and electrolyte estimation

S. No.	Parameters	Method of estimation	Expression of results
1.	Calcium *	Modified Arsenazo III method	mg/dl
2.	Phosphorus *	Phosphomolybdate methodology	mg/dl
3.	Sodium **	Ionic selective electrode	mmol/L
4.	Potassium **	Ionic selective electrode	mmol/L
5.	Chloride **	Ionic selective electrode	mmol/L

Plate 3.2: Semi-automatic biochemical analyser Microlab 300 Clinical Chemistry Analyser

Plate 3.3: CB- LYTE Automatic Electrolyte Analyser

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*ALT, AST, ALP, GGT, Glucose, Blood Urea Nitrogen, Creatinine, Total and Direct bilirubin, Total Protein, Albumin, Cholesterol, Calcium and Phosphorus kits: Manufactured by- Agappe Diagnostic Ltd. 'Agappe Hills', Dist. Ernakulam, Kerala- 683 562.

** Sodium, Potassium and Chloride calibration pack containing Standard A and Standard B: Manufactured by Chariot's Biotechnology, 104A, ARM Enclave, Kharghar, Navi Mumbai, Maharashtra

3.6.4 Parasitological examination

Microscopic examination of thin blood smear prepared from cephalic or recurrent tarsal vein in K_3- EDTA vials was done by staining the smears with Giemsa stain.

3.6.5 Serological detection of *Ehrlichia* by Quick Vet rapid test kit

The *Ehrlichia* Antibody detection kit is qualitative immunochromatographic lateral flow assay. The test device has testing window that has invisible T (test) zone and C (control) zone. When sample (whole blood, serum or plasma fresh or stored at 2-8^{0}C to be used in 3 days) is loaded into sample well followed by addition of canine *Ehrlichia* assay diluent on kit, sample will flow on surface of test strip. If enough *Ehrlichia* antibody in sample, visible T band will appear and the C band should always appear after loading of sample, indicating valid result.

3.6.6 Blood gas and acid base analysis

The blood samples were analysed in Idexx Vetstat acid base and electrolyte analyser (Plate 3.4a and 3.4b) (Idexx Co., USA) for determination of blood gases and acid base status comprising of partial pressure of carbon dioxide (pCO_2 mm Hg), total carbon dioxide (tCO_2 mmol/l), blood pH, plasma concentration of bicarbonate (HCO_3 mmol/l), Anion Gap (AnGap), base excess (BE mmol/l), base excess actual (BE_{act}), base excess extracellular fluid (BE_{ecf}), base buffer (BB), standard bicarbonate ($stHCO_3$), standard pH (st pH), and total H+ (cH+).

Plate 3.4a: Idexx Vetstat Acid base and electrolyte analyser

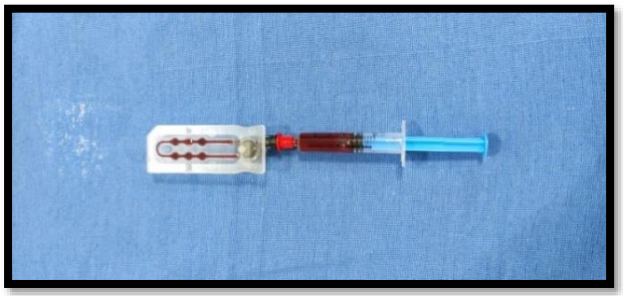

Plate 3.4b: Idexx Vetstat Cassette

3.6.7 Ascitic fluid analysis

The collected fluid was processed for physical, microscopic and biochemical analysis. The physical examination included colour, turbidity and specific gravity.

3.6.7.1 Biochemical analysis

The different biochemical parameters that were estimated were: Albumin, Total protein and SAAG. The method of estimation and its units are given in Table 3.3

Table 3.3: Method for estimation of ascitic fluid parameters

S. No.	Parameters	Method of estimation	Units
1.	Albumin *	Bromocresol green methodology	g/dl
2.	Total Protein (TP)*	Direct Biuret method	g/dl
3.	Serum Ascitic Albumin Gradient (SAAG)	Subtracting ascitic albumin from plasma albumin	g/dl

The Total Leucocyte Count (TLC) of ascitic fluid was determined by using WBC diluting fluid in the neubauer's haemocytometer (Jain 1986) and was expressed in thousands per micro-litre.

3.6.7.2 Fine Needle Aspiration Cytology (FNAC) of ascitic fluid

The ascitic fluid collected by adomenocentesis from linea alba in a clean syringe was centrifuged and smear of the sediment was examined after staining it with Giemsa stain.

Procedure:

1. The hairs over the linea alba were clipped and prepared aseptically for FNAC.
2. The needle along with the syringe was injected into peritoneal space and the fluid was aspirated.
3. Later, according to the distension and/ or the amount of fluid accumulated it was drained while the fluid therapy was administered.
4. Then, the aspirated fluid was centrifuged at 3000rpm and smear of the sediment was prepared and stained with Giemsa stain using conventional method.

3.6.8 Urine examination

The physical characterization of urine like colour, turbidity and odour of the urine were recorded.

3.6.8.1 Dip-stick testing/ Urinalysis

After collection, quantitative examination of the urine for gross changes that is colour and clarity; Chemical alterations like ketone bodies, nitrite, urobilnogen, bilirubin, protein, glucose, urinary specific gravity and pH and Microscopic evaluation for RBC's and WBC's was done by using Urine Reagent Strips in Semi-automatic analyser (Uriscan Optima-YD Diagnostic Corp. Korea) (Plate 3.5).

Plate 3.5: Semiautomatic urine analyser with urine analyser strips

3.6.8.2 Urine smear examination

Microscopic examination of the smear of the sediment of the collected urine by catherization was done after staining them with Giemsa stain.

3.7 ELECTROCARDIOGRAM (ECG)

The animals suspected of cardiac abnormality based on history, physical and clinical examination (auscultation: tachycardia, arrhythmia, adventitious sounds) electrocardiography was carried out. The animal was positioned in right lateral recumbency with the limbs kept perpendicular to the long axis of the body, on a non-

conductive surface to avoid electrical interference. The base apex bipolar leads with RA and LA electrodes were attached on the elbow joint of both the forelimbs and slightly above stifle joint on the hind limbs. The electrocardiogram was obtained using multi parameter monitor (L & T Star 55, India) (3.6a and 3.6b). The electrocardiography was recorded at paper speed of 50mm/sec and amplitude of 10mV.

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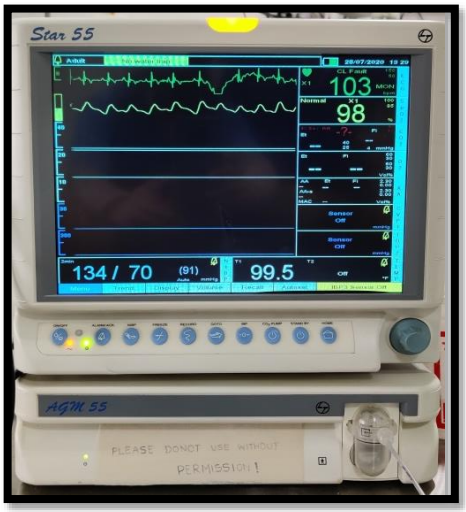
<td> 
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<tr>
<td>Plate 3.6a: L & T Star 55 Multi parameter monitor</td>
<td>Plate 3.6b: E.C.G. leads</td>
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3.8 IMAGING TECHNIQUE

3.8.1 Radiography

Abdominal radiography was performed to characterize the abnormality relating to gastrointestinal and reticuloendothelial system. The abnormalities in the size of liver, kidneys and spleen and presence of fluid in the peritoneal space were examined as per Thrall (2002). Radiographs were obtained using Siemens 80 mA mobile X-ray machine (Siemens Ltd, Goa) (Plate 3.7).

3.8.2 Ultrasonographic examination

Abdominal ultrasonography was performed to identify abnormality in the stomach, liver, spleen, intestine and kidneys and their textural change in cases where level of fluid was moderate. The ultrasonography was performed under anaesthesia or sedation. The abdomen and 5-7th intercostal space was clipped; cleaned and acoustic coupling gel was used on the skin surface for examination of the abdominal organs and echocardiography,

respectively. The ultrasonography was carried out by using Siemens acuson X 300 machine using multifrequency (5-7.5 MHz) micro convex probe (Plate 3.8).

	
Plate 3.7: Siemens 80 mA mobile X-ray machine	**Plate 3.8: Siemens acuson X 300 machine**

3.9 THERAPEUTIC MANAGEMENT

3.9.1 Treatment protocol for liver affected ascitic dogs

Dogs that were diagnosed with liver affections (hepatitis, cholecystitis, cirrhosis) were treated with Silymarine in combination with L-glutathione, N-acteyl cystenine, L-carnitine L-tartrate, L-ornithine, Choline bitartrate, and Coenzyme Q10 (Tab Hepa 20) orally at dose rate of 20-30mg/kg/day for 2 weeks. Tab fruselac DS (Frusemide +Spironolactone) was used as diuretic agent. Liver safe antibiotic Ampicillin was included with dose of 20mg/kg twice for 5-10 days. Vitamin B complex was prescribed (Tribivet) at dose of 1-2 ml I/V per day once for 5 days along with supportive therapy and diet therapy.

3.9.2 Treatment protocol for cardiac affected ascitic dogs

Dogs that suffered with cardiomyopathy and pericardial effusion were treated with diuretics, Tab Fruselac DS (Frusemide+ spironolactone @2-4mg/kg) twice daily, antibiotic (Ampicillin). Digoxin @0.025mg/kg orally twice daily and angiotensin converting enzyme

inhibitor (Enalapril) @0.5 mg/kg once daily. The treatment also included some other supportive therapy and diet therapy.

3.9.3 Treatment protocol for renal affected ascitic dogs

Dogs suffering from renal condition along with ascites were treated with diuretics (Frusemide @2-4mg/kg), 8.4% sodium bicarbonate, antibiotic (Ceftriaxone + Tazobactum) and supportive therapy.

The detail of different therapeutic agents employed for treatment of ascites (liver, cardiac and renal origin), their dose rate and schedule is enlisted in Table 3.4

Table 3.4: Drugs used for the treatment of ascitic dogs with liver, cardiac and renal disorders

Sr. No.	Generic Name	Dose rate	Trade Name	Composition
1.	Frusemide + Spironolactone	2-4 mg/kg b.wt.	Tab. Fruselac DS[1]	Frusemide 40 mg + Spironolactone 50 mg
2.	Silymarine, N-acteyl cystenine, L-carnitine tartrate, L-ornithine, Choline bitartrate, and Coenzyme Q10	1 tab/ 20 kg b.wt.	Tab. Hepa 20[2]	Silymarine 140mg, N-acteyl cystenine 50mg, L-carnitine tartrate 50mg, L-ornithine 50mg, Choline bitartrate 50mg, and Coenzyme Q10 2.5mg, Vitamin D3 400IU
3.	Digoxin	0.025 mg/kg b.wt.	Inj. Dixin[3]	Digoxin (0.5mg/2ml)
4.	Sodium bicarbonate	Based on acid base analysis of blood	Inj. Sodac 8.4%[4]	Na^+ & HCO^-_3 1000 milimoles per liter
5.	Enalapril	0.5 mg/kg b.wt.	Envas 5[5]	Enalapril Maleate 5 mg
6.	Pimobendan	0.25 mg/kg b.wt.	Safeheart 5[6]	Pimobendan 5mg
7.	Vitamin B complex	1-2 ml I/V	Inj. Tribivet[7]	Thiamine HCl 50 mg Pyridoxine HCl 50 mg Cyanocobalamin50 mcg

3.9.4 Supportive therapy

1. Fluid therapy was done with Inj. DNS[8] or Inj. Denilyte-P[9], Inj. Haemaccel[10] or Inj. Hermin[11] intravenously in dogs that were anorectic, according to the dehydration status for stabilisation.

2. Blood transfusion was carried out in severely anaemic dogs in addition to the conventional treatment.
3. Ondansetron (Inj. Neomit[12]) was used as an anti-emetic @0.2-0.5 mg/kg intravenously and an antacid like pantoprazole (Inj. Petzole[13]) @1-2 mg/kg along with fluid therapy in animals that had vomiting.
4. Haemostats (Ethamsylate (Tab K-stat[14], 500mg) were used in cases of severe bleeding.
5. Imidocarb (Inj. Imizet[15] @6.6 mg/kg) intramuscularly and Doxycycline[16] (Tab. Doxypet @ 10mg/kg) was prescribed in patients suffering from hemoprotozoan infection.
6. Ursodeoxycholic (Tab. Ursolid[17], 300mg) @15mg/kg/day in dogs with higher bilirubin.
7. Oral supplements of liver supportive and hepato-protectants (Syp. Livotas pet[18]), haematinics (Syp. aRBC pet[19]), Platelet enhancers (Syp. Immulat[21]), and protein supplements (Pow. Proteins X[21]) (200 g) were advised wherever needed.
8. The diet advised to the owner was salt free and protein rich diet that included egg whites, boneless chicken or soyabean orally and complete rest to animal was also advised alongside.

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[1] Pinnacle CVN, Lupin Ltd., Aurangabad
[2] Venus remedies Limited, Baddi, H.P.
[3] Wellona Pharma Private Ltd., Surat, Gujarat
[4] NEON Laboratories Limited, Mumbai
[5] Cadila pharmaceuticals, Maharashtra
[6] Sava Healthcare Ltd., Gujarat
[7] Intas Pharmaceuticals Ltd, Ahmedabad
[8] Inf -DNS (Dextrose 5% and Normal Saline 0.9%) (500 ml), Denis Chem Lab Ltd., Gandhinagar, Gujrat
[9] Inf.-Denilyte-P (Paediatric maintenance solution with 5% w/v Dextrose) (500 ml), Denis Chem Lab Ltd., Gandhinagar, Gujarat
[10] Inf.- Haemaccel (3.5% colloidal infusion solution of polygeline with electrolytes) (500 ml), Abbot, Piramal Healthcare Ltd., Mumbai
[11] Inf.- Hermin(Essential amino acids) (200 ml), Alembic, Krishna Drug Specialities, Delhi
[12] Inj. Neomit (Ondansetron, 2mg/ml), M/s Neon Laboratories Ltd.
13Inj-Petzole (Pantoprazole, 40mg), Zee Laboratories Ltd., Paonta Sahib
[14] Tab-K-stat (Ethamsylate 500 mg), M/s Mercury Healthcare Pvt. Ltd.
[15] Inj.-Imizet(Imidocarb Dipropionate, 120 mg/ml), M/s Intas Pharmaceuticals Ltd., Ahmedabad
16Tab Doxypet- Doxycycline (200 mg/Tab), M/s Sava Healthcare Ltd., Surendranagar, Gujarat
17Tab-Ursolid (Ursodeoxycholic Acid, 300mg/Tab), Leeford, Nagpur, Maharashtra
[18] Syp.-Livotas pet M/s Intas Pharmaceuticals Ltd., Ahmedabad
[19] Syp.- aRBC pet M/s Vetoquinol India Animal Health Ltd., Ahmedabad
20Syp.-Imulat. M/s Neo Kumfurt, New Mumbai
[21] Powder Proteinex (200 g)- M/s Apollo Pharmacy Ltd.

3.10 EVALUATION OF THE TREATMENT

On the basis of alleviation of the clinical signs, improvement in haemato-biochemical parameters after treatment and survival rate, therapeutic efficacy of different therapeutic regime was evaluated.

3.11 CONTROL GROUP

A total of 10 clinically healthy dogs formed the control group. Dogs presented for regular vaccination and elective surgery were included in this group. Haematology, biochemical analysis, minerals and electrolytes estimation, blood gas acid base analysis, urine analysis, electrocardiography and imaging techniques (radiography and ultrasonography) was carried out for comparison with the diseased dogs.

3.12 STATISTICAL ANALYSIS

The data obtained was subjected to statistical analysis by using computer software Instat from Grahpad software, 2008. The mean values of different parameters between control and diseased group; control, pre and post treatment were compared at 1% and 5% level of significance using “t” test and “ANNOVA”.

Results and Discussion

CHAPTER 4 RESULTS AND DISCUSSION

Canine ascites can be due to hepatic disease, renal or cardiac disease and evaluating them is not a simple process, as a single diagnostic test is not sufficient enough with specificity and sensitivity. A number of diagnostic tests and procedures are carried to know the basic cause of abdominal distension.

4.1 OBSERVATION ON HEALTHY DOGS

Ten healthy dogs irrespective of age, sex and breed that were presented in Department of Veterinary Medicine for general check-up or vaccination were included in the present study for comparing the results of various aspects of clinical cases of ascites. The dogs were subjected to detailed clinical and laboratory examination.

4.1.1 General clinical observation

All the healthy dogs formed the control group and were inspected clinically that showed activeness and alertness with normal appetite, defecation and urination. The mean values of rectal temperature, heart rate and respiration rate were 101.15 ± 0.29 ^{0}F, 88.7 ± 2.08 beats per minute and 17.9 ± 0.55 breaths per minute, respectively. The values of the clinical parameters are given in Table 4.1 and were within normal range.

Table 4.1: Clinical observation in healthy dogs (Mean ± S.E.)

Sr. No.	Parameters	Values (n=10)
1.	Rectal Temperature (^{0}F)	101.15 ± 0.29
2.	Heart Rate (beats per minute)	88.7 ± 2.08
3.	Respiration Rate (breaths per minute)	17.9 ± 0.55
4.	Conjunctival Mucous Membrane	Pinkish
5.	Hydration Status	Normal
6.	Behaviour	Active and Alert
7.	Urination and Defecation	Normal
8.	Faecal Examination	NAD upon direct smear examination

The physical examination revealed pinkish conjunctival mucous membrane. The dogs were screened for endoparasitic infestation by direct faecal examination and ectoparasitic infestation and the result for both the examination was negative.

4.1.2 Haematological examination

The haematological values are listed in Table 4.2. The mean values of haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC) and total leucocyte count (TLC) were 12.87 ± 0.42 g/dl, 39.05 ± 0.96 %, 6.81 ± 0.25 × 10^{12} /L and 10.69 ± 0.56 × 10^{9} /L, respectively. These values were comparable to the findings of Elhiblu et al. (2015) and Lakshmi et al. (2018) of healthy animals.

Table 4.2: Haematological profile of healthy dogs (Mean ± S.E.)

Sr. No.	Parameters	Values (n=10)
1.	Hb (g/dl)	12.87 ± 0.42
2.	PCV (%)	39.05 ± 0.96
3.	TEC (× 10^{12} /L)	6.81 ± 0.25
4.	TLC (× 10^{9} /L)	10.69 ± 0.56
5.	Lymphocytes (%)	17.08 ± 2.11
6.	Monocytes (%)	3.94 ± 0.29
7.	Granulocytes (%)	75.34 ± 2.60
8.	MCV (fl)	58.38 ± 0.87
9.	MCH (pg)	19.02 ± 0.49
10.	MCHC (g/dl)	31.63 ± 0.39
11.	Platelet count (× 10^{9} /L)	253.00 ± 20.57

Differential leucocyte count revealed 75.34 ± 2.60 % granulocytes, 17.08 ± 2.11 % lymphocytes and 3.94 ± 0.29 % monocytes, respectively and all these values were within the normal range as given by Jain (2000). However, Phom et al. (2019) reported high levels of lymphocytes. The values of mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC) and platelet count were 58.38 ± 0.87 fl, 19.02 ± 0.49 pg, 31.63 ± 0.39 g/dl and 253.00 ± 20.57 × 10^{9} /L, respectively and were comparable to the study of Elhiblu et al. (2015) and were within normal range (Klaassen 1999).

4.1.3 Blood biochemical profile

The plasma biochemical profile of healthy dogs is given in Table 4.3. The mean values of total protein, albumin, globulin, A:G ratio, cholesterol, alkaline phosphatase (ALP), aspartate amino transferase (AST), total bilirubin, direct bilirubin, indirect bilirubin, blood urea nitrogen (BUN) and creatinine were 6.68 ± .12 g/dl, 3.71 ± 0.17 g/dl, 2.97 ± 0.18 g/dl, 1.30 ± 0.12, 163.38 ± 4.93 mg/dl, 57.26 ± 8.23 U/L, 31.27 ± 4.32 U/L, 0.25 ± 0.04 mg/dl, 0.07 ± 0.01 mg/dl, 0.18 ± 0.04 mg/dl, 14.47 ± 1.22 mg/dl and 1.03 ± 0.15 mg/dl, respectively and were comparable with the findings of Elhiblu et al. (2015). The mean activities of alanine transferase (ALT) and glucose were 36.12 ± 4.29 U/L and 108.15 ± 2.82 mg/dl and the values were similar with the values recorded by Lakshmi et al. (2018) in normal healthy dogs.

Table 4.3: Plasma biochemical profile of healthy dogs (Mean ± S.E.)

Sr. No.	Parameters	Values (n=10)
1.	Total protein (g/dl)	6.68 ± .12
2.	Albumin (g/dl)	3.71 ± 0.17
3.	Globulin (g/dl)	2.97 ± 0.18
4.	A:G ratio	1.30 ± 0.12
5.	Cholesterol (mg/dl)	163.38 ± 4.93
6.	Blood Urea Nitrogen (mg/dl)	14.47 ± 1.22
7.	Creatinine (mg/dl)	1.03 ± 0.15
8.	ALT (U/L)	36.12 ± 4.29
9.	AST (U/L)	31.27 ± 4.32
10.	ALP (U/L)	57.26 ± 8.23
11.	Total bilirubin (mg/dl)	0.25 ± 0.04
12.	Direct Bilirubin (mg/dl)	0.07 ± 0.01
13.	Indirect Bilirubin (mg/dl)	0.18 ± 0.04
14.	GGT (U/L)	2.27 ± 0.18
15.	Glucose (mg/dl)	108.15 ± 2.82

4.1.4 Plasma minerals and electrolytes profile

The mean values of plasma mineral and electrolyte of healthy dogs are given in Table 4.4. The mean values of plasma calcium and phosphorus were 10.41 ± 0.89 mg/dl and 4.76 ± 0.49 mg/dl, respectively. The mean electrolyte values of plasma sodium, potassium and chloride were 141.7 ± 1.45 mEq/L, 4.42 ± 0.21 mEq/L and 104.2 ± 1.18 mEq/L respectively. The mean values of calcium, phosphorus, sodium, potassium and chloride were within normal range (Lakshmi et al. 2018).

Table 4.4: Plasma minerals and electrolytes profile of healthy dogs (Mean ± S.E.)

Sr. No.	Parameters	Values (n=10)
1.	Plasma Calcium (mg/dl)	10.41 ± 0.89
2.	Plasma Phosphorus (mg/dl)	4.76 ± 0.49
3.	Plasma Sodium (mEq/L)	141.7 ± 1.45
4.	Plasma Potassium (mEq/L)	4.42 ± 0.21
5.	Plasma Chloride(mEq/L)	104.2 ± 1.18

4.1.5 Urine Analysis

The mean values of urine analysis are given in Table 4.5. The mean values of urine pH and urine specific gravity were 6.21 ± 0.16 and 1.026 ± 0.001, respectively. The urine was negative for blood, bilirubin, urobilinogen, ketones, proteins, nitrite and glucose. These values and findings were in accordance with the findings and values reported by Ford and Mazzaferro (2009).

4.1.6 Blood gas and acid base status

The level of blood gas and acid base status is given in Table 4.6. The values of blood pH, partial pressure of carbon dioxide (pCO2), Bicarbonate (HCO3), Anion Gap (AnGap), total carbon dioxide (tCO2), Base excess (BE) were 8.42 ± 0.05 mmHg, 34.12 ± 0.49 mmol/L, 20.27 ± 0.33 mmol/L, 20.68 ± 0.71 mmol/L, -3.19 ± 0.15 mmol/L, respectively. The other parameters like Buffer base (BB), standard Bicarbonate ($stHCO_3$), and standard pH. were within the normal range as per Wadell (2012).

Table 4.5: Urine analysis of healthy dogs (Mean ± S.E.)

Sr. No.	Parameters	Values (n=10)
1.	Blood (RBC/µl)	Nil
2.	Bilirubin (mg/dl)	Nil
3.	Urobilinogen (mg/dl)	Normal
4.	Ketones (mg/dl)	Nil
5.	Proteins (mg/dl)	Nil
6.	Nitrite	Negative
7.	Glucose (mg/dl)	Nil
8.	pH	6.21 ± 0.16
9.	Specific gravity	1.026 ± 0.001
10.	Leucocytes (WBCs/µl)	Nil
11.	Clarity	Clear

Table 4.6: Blood gas and acid base analysis of healthy dogs (Mean ± SE)

Sr. No.	Parameters	Values (n=6)
1.	pH	7.12 ± 0.05
2.	pCO_2 (mmHg)	34.12 ± 0.49
3.	HCO_3 (mmol/L)	20.27 ± 0.33
4.	AnGap (mmol/L)	20.65 ± 0.78
5.	tCO_2 (mmol/L)	20.68 ± 0.71
6.	BE (mmol/L)	-3.19 ± 0.15
7.	BEact (mmol/L)	-4.35 ± 0.27
8.	BEecf (mmol/L)	-4.56 ± 0.28
9.	BB (mmol/L)	43.95 ± 0.49
10.	$stHCO_3$ (mmol/L)	21.22 ± 0.25
11.	st. pH	7.05 ± 0.05
12.	cH+ (nmol/L)	37.71 ± 0.97

4.1.7 Electrocardiography

The electrocardiography in 5 healthy animals was conducted with normal heart rate and the indices of the electrocardiograph were measured. The mean of the indices is presented in Table 4.7

Table 4.7: Electrocardiographic indices of healthy dogs (Mean ± S.E.)

Sr. No.	ECG parameter	Values (n=5)
1.	HR (beats per minute)	88.7 ± 2.08
2.	P wave interval (sec)	0.06 ± 0.0030
3.	PR interval (sec)	0.3 ± 0.004
4.	QRS interval	0.04 ± 0.002
5.	QT interval (sec)	0.15 ± 0.005
6.	T wave interval (sec)	0.65 ± 0.007
7.	PR segment	0.56 ± 0.004
8.	ST segment	0.6 ± 0.007
9.	P wave amplitude (mV)	0.20 ± 0.05
10.	R wave amplitude (mV)	1.51 ± 0.21
11.	T wave amplitude (mV)	0.26 ± 0.06

4.1.8 Imaging studies

The liver of apparently healthy dogs (n=4) were identified and were examined by ultrasonography performed through subcostal and last intercostal space in both dorsal and lateral recumbency. Multiple scans were carried out, as in a single scan the entire liver could not have been examined satisfactorily as to study the echotexture of the liver and its parenchyma.

Anatomically, the cranial border of the liver was aligning with the hyperechoic diaphragm and the caudal borders were in contact with the spleen on the left side and on the right side with right kidney at the level of renal fossa (Barr and Gaschen 2011). The kidneys showed normal echotexture with distinguishable renal cortex and renal medulla (Plate 4.29). The different lobes and the processes of the liver were not distinguishable (Plate 4.28). Hudson and Hamilton (1993) and Barr and Gaschen (2011) stated that in

absence of peritoneal effusion the liver processes cannot be easily distinguished in ultrasonography.

The hepatic blood vessels were visible as anechoic like structures in both transverse and saggital scans. These anechoic structures were traced to a considerable distance during the displacement of the liver and diaphragm during respiration along with the movement of scan head.

The wall of some of the vasculature network was relatively hyperechoic and these were assigned to be portal veins on the basis of description of echotexture by Penninck and Anjou (2008) and the hepatic veins were established as an anechoic vasculature that lacked the boundary. However, Penninck and Anjou (2008) also reported that sometimes when the ultrasonographic beam is directed perpendicular to the wall of hepatic veins they can appear hyperechoic. These findings were concurrent with the findings of Vijayanand et al. (2006).

On the right of mid-line close to the diaphragm an anechoic circle was seen that was caudal vena cava. Sometimes, the caudal vena cava can also be located by following the hepatic veins that can be seen entering it (Barr and Gaschen 2011). At the right of mid-line, a round to oval structure was also visible that was gall bladder which had normal thickening of the wall (Plate 4.30). Barr and Gaschen (2011) described it as pear shaped anechoic structure that was located between the quadrate and right medial liver lobe and its size can vary (enlarge) during anorexia or in fasted animal, the acoustic enhancement and presence of sludge is commonly found. The other structures of liver like bile duct, lobar border, hepatic arteries and small peripheral hepatic veins were not distinguishable. These observations were in correlation with those of Bhadwal et al. (1999).

4.2 OBSERVATION ON DISEASED DOGS

4.2.1 Incidence

From October 2018 to March 2020, 2063 dogs were presented at Department of Veterinary Medicine for the treatment of various conditions. A total of 47 dogs were presented with abdominal distension along with other clinical signs, thus presenting the overall incidence of 2.27 %.

4.2.2 Aetiology

A total of 35 dogs out of 47 ascitic dogs had ascites due to liver disease (74.46%), 6 dogs due to cardiac (12.76%) and 6 dogs suffered due to nephritic diseases (12.76%) (Figure 4.1). Among 47 dogs, six dogs suffered from haemoprotozoan disease, 5 ascitic dogs were of liver disorders (4 Babesiosis and 1 Ehrlichiosis) and one dog was of renal disorder (Erhlichiosis).

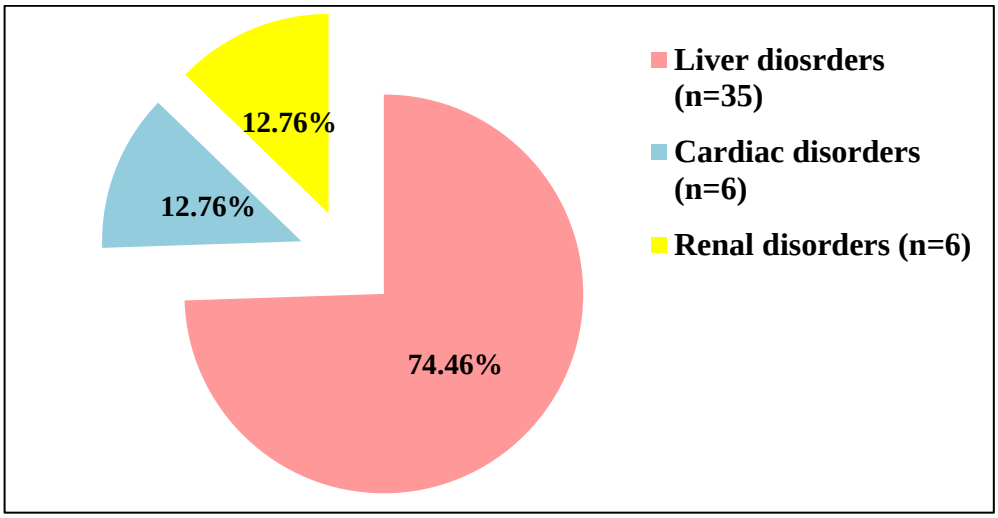


Figure 4.1: Incidence of ascites according to aetiology

Out of 35 dogs of ascites due to hepatic dysfunction, twenty one dogs (60.00%) were diagnosed with chronic hepatitis and thus forming the largest group, followed by acute hepatitis in six dogs (6/35, 17.14%). Cirrhosis and cholecystitis was diagnosed in four dogs in each group (4/35, 11.42%) (Figure 4.2).

In the study by Moore et al. (2003) and Behera et al. (2017) similar findings were found where liver was affected in majority of the cases of ascites. Raffan et al. (2009) also reported that ascites was the main complication of chronic hepatitis. Cardiomyopathy was diagnosed in five dogs (5/6, 83.33%) among the cases of cardiac disorders. Tilley and Liu (1975) also observed that in cases of cardiomyopathy, ascites was a common finding. Pericardial effusion was confirmed in two dogs (2/6, 33.33%). The above findings were in partial accordance with Ihedioha et al. (2013) who recorded 50 per cent cases of ascites due to congestive failure, 35.70 per cent cases of ascites due to hepatic disorder and 14.30 per cent cases of renal disease. The chronic renal failure was observed in two dogs (2/6, 33.33%) and four dogs had acute renal failure (4/6, 66.66%).

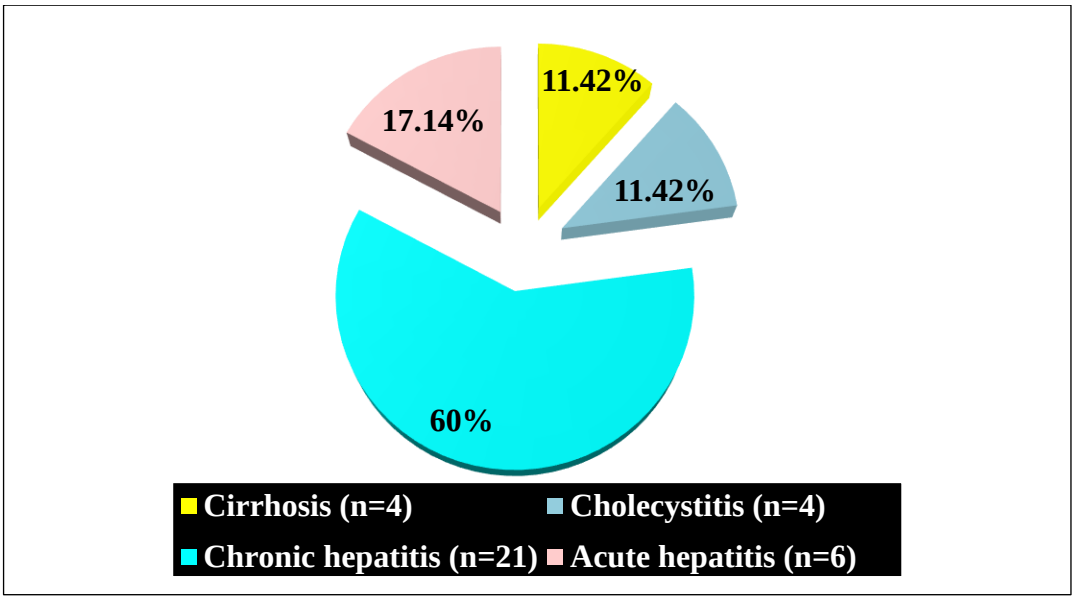


Figure 4.2: Incidence of ascites due to various liver disorders

4.2.3 Signalment

4.2.3.1 Age

Among 47 dogs that were presented with ascites, twenty three dogs (23/47, 48.93%) were in the age group of 1 to 4 years, ten dogs (10/47, 21.27%) 4-7 years of age, nine (9/47, 19.14%) above seven years and six dogs (5/47, 10.63%) were below 1 year of age group (Table 4.8, Figure 4.3). Higher incidence of ascites in the younger age group was also reported by Nottidge et al. (2003), James et al. (2008) and Turker et al. (2009). However, Sarvanan et al. (2013) reported higher incidence in age group of 5-7 years.

Ascites caused by liver damage was majority observed in the age group of 1-4 years (17/35, 48.57 %), followed by eight dogs (8/35, 22.85%) in the age group of 4-7 years, five (5/35, 14.28%) in the age groups of <1 year, and five dogs (5/35, 14.28%) in the age group of >7 years. Hepatitis was commonly observed in age group of 1-4 years (Crawford et al. 1985). Fifty per cent (3/6) of the dogs that suffered from cardiac disorders were in the young age group (1-4 years) and 33.33% (2/6) were in geriatric age group (>7 years) and 16.66 per cent (1/6) in middle age group (4-7 years). The ascites due to renal disorders was commonly observed in 1-4 years of age group (3/6, 50%) followed by >7 years (2/6, 33.33%) and least in 4-7 years of age group (1/6, 16.66%).

Table 4.8: Cause of ascites in relation to age in dogs

Sr. No.	Age group (Years)	Liver disorders	Cardiac disorders	Renal disorders	Total (N=47)
1.	< 1 year	5	-	-	5
2.	1-4 years	17	3	3	23
3.	4-7 years	8	1	1	10
4.	>7 years	5	2	2	9

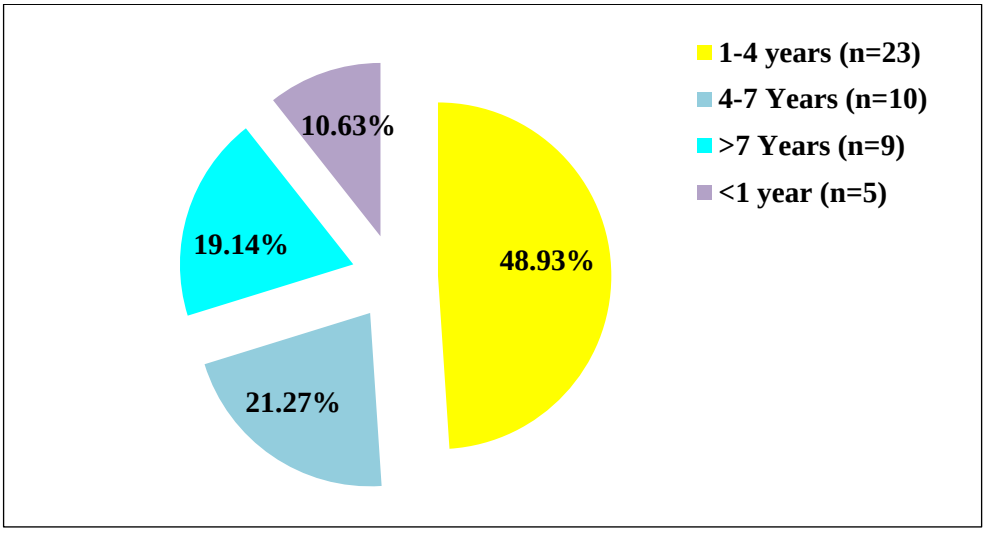


Figure 4.3: Age wise incidence of ascites due to liver, cardiac and renal disorders

Out of 35 dogs that suffered from ascites due to liver disorder, majority of the animals had chronic hepatitis (21/35, 60.00%) and was observed that most of animals were in age group of 1-4 years (12/21, 57.14 %), acute hepatitis was commonly observed in 4-7 years of age group, cholecystitis was found majorly in 1-4 years of age group (Table 4.9). Cirrhosis was observed in four dogs and each age group had 1 dog that suffered with cirrhosis (Figure 4.4).

Table 4.9: Age wise incidence of ascitic dogs with liver disorders (n=35)

Sr. No.	Age group (Years)	Cirrhosis	Chronic Hepatitis	Acute Hepatitis	Cholecystitis
1.	>1 year	1	3	1	-
2.	1-4 years	1	12	2	2
3.	4-7 years	1	3	3	1
4.	>7 years	1	3	-	1

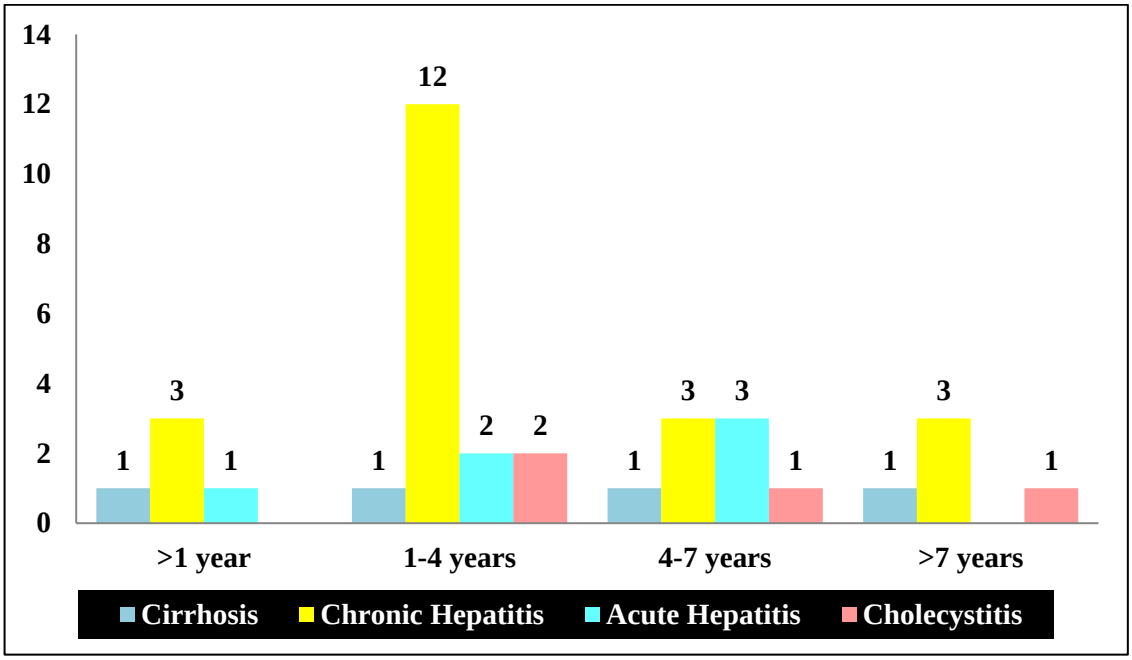


Figure 4.4: Age wise incidence of ascites due to liver disorders

4.2.3.2 Breed

The Labrador retriever (14/47, 29.78%) was most common breed that was affected with ascites, followed by non-descript mongrel (7/47, 14.89%), German shepherd (7/47, 14.89%), Pomeranian (6/47, 12.76%), Pug (3/47, 6.38%), Golden Retriever (3/47, 6.38%). Although, higher risk of ascites was observed in Pomeranian (33.33%) followed by Labrador retriever (20%), Boxer (16.66%), Doberman pinscher (13.33%), Mongrels (10%) and least in Alsatian (6.66%) (Sarvanan et al. 2013).

Table 4.10 depicts the breed wise incidence of dogs suffering from ascites due to liver, cardiac and renal disorders.

Table 4.10: Breed wise incidence of ascites in dogs (n=47)

Breed	Liver disorders	Cardiac disorders	Renal disorders	Total	Incidence (%)
Labrador retriever (LR)	9	3	2	14	29.78
German shepherd (GSD)	4	1	2	7	14.89
Non-descript mongrel (ND)	6	1		7	14.89
Pomeranian	5		1	6	12.76
Golden retriever (GR)	3			3	6.38
Pug	2	1		3	6.38
Rottweiler	1			1	2.12
Pointer	1			1	2.12
Dalmatian	1			1	2.12
Gaddi	1			1	2.12
Pit bull	2			2	4.25
Great Dane			1	1	2.12

It was observed that most of the Labrador retriever dogs suffered from liver disorders (9/14, 64.28%), three Labrador dogs suffered from cardiac disorder (3/14, 21.42%) and two from renal disorders (2/14, 14.28%). German shepherd and non-descript mongrel dogs were among second commonly affected dogs. Four German shepherd dogs (4/35, 11.42%) and six non-descript mongrel dogs (6/35, 17.14%) were suffering from ascites due to liver disorders. Behera et al. (2017) reported that Labrador retriever breed was commonly affected with ascites followed by Spitz, German shepherd and least was Boxer, and Cocker spaniel. The five Pomeranians, all the three Golden retrievers and two Pit bulls, two Pugs and Rottweiler, Pointer, Dalmatian and Gaddi were having ascites due to hepatic insufficiency. The only one Great Dane had ascites due to renal disorders (Figure 4.5).

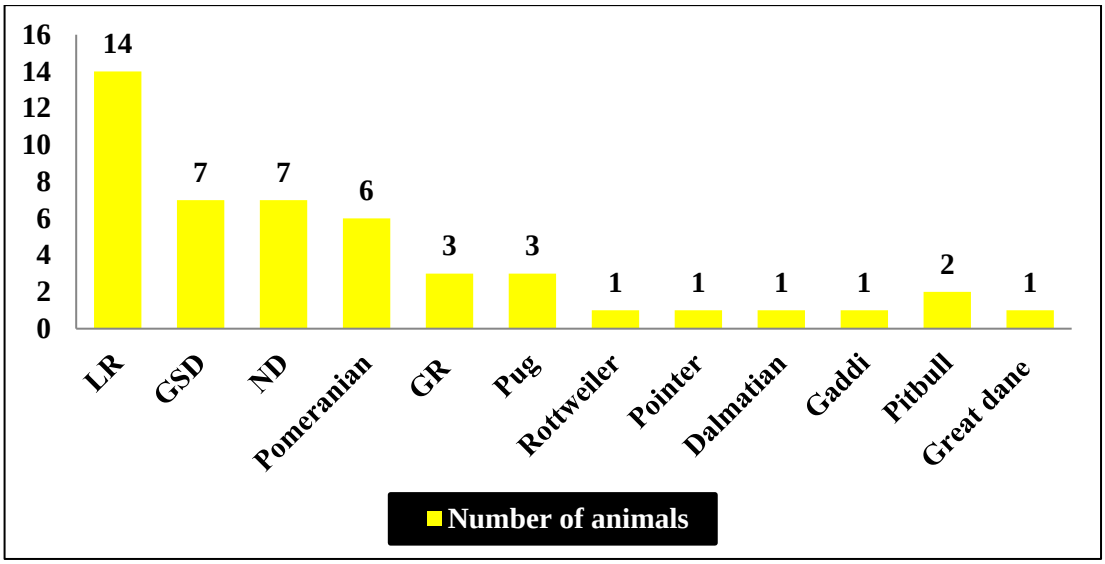


Figure 4.5: Breed wise incidence of ascites

4.2.3.3 Sex

Males were commonly affected with the condition of ascites as compared to females. The sex wise predisposition is given in Table 4.11 in relation to aetiology. Out of 47 dogs affected with ascites, thirty six (36/47, 76.59%) dogs were male and eleven dogs (11/47, 23.40%) were females (Figure 4.6).

Sex predisposition revealed that 74.28 per cent male dogs (26/35) and 25.71 per cent female dogs (9/35) suffered from ascites due to liver disorders. Similarly, male dogs suffered more from cardiac disorders (5/6, 83.33%) than the females (1/6, 16.66%) and renal disorders (5/6, 83.33%) than female dogs (1/6, 16.66%). Of the twenty one dogs that suffered chronic hepatitis fourteen (14/21, 66.66%) were males and seven (7/21, 33.33%) were females. Sarvanan et al. (2013) and Ihedioha et al. (2013) had observed similar findings in their individual study where the males were more affected with ascites than the females. However, Raffan et al. (2009) reported higher incidence of ascites due to chronic hepatitis in female dogs (18/42, 42.85%) than male dogs (16/42, 38.09%). Ware (2009) also observed that males suffered more with cardiomyopathy as compared to females, but Kocatürk et al. (2016) observed female dogs suffered more from cardiomyopathy than male dogs in his study. Katoch et al. (2017) also reported higher prevalence of renal disorder in males as compared to females as observed in the present study.

Table 4.11: Sex wise incidence of ascites in dogs

Sex	Liver disorders	Cardiac disorders	Renal disorders	Total (N=47)
Male	26	5	5	36
Female	9	1	1	11

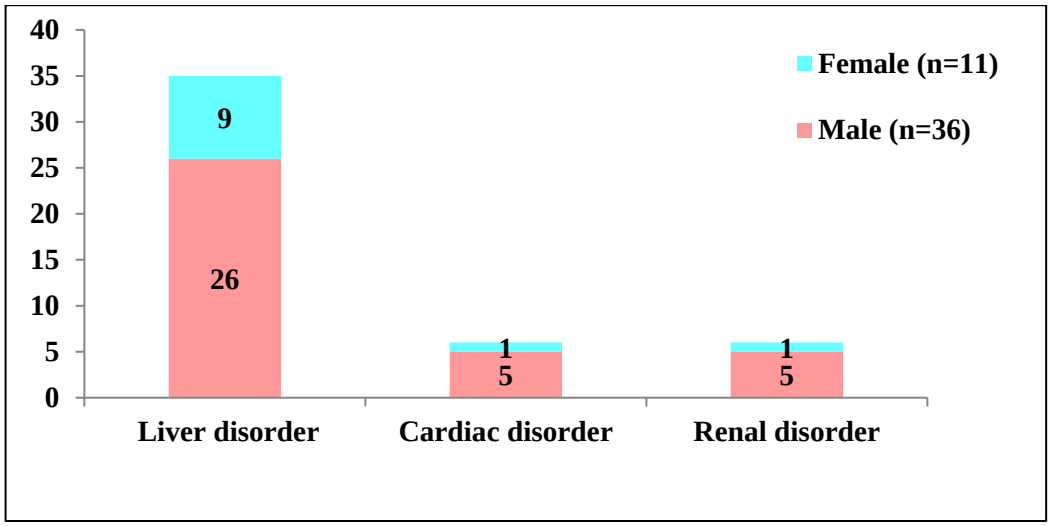


Figure 4.6: Sex wise incidence of ascites

4.2.4 Clinical Signs

4.2.4.1 Liver diseases

Based on history, clinical signs, duration of abdominal distension and physical parameters, the development of ascites was classified as rapid onset (7-10 days) and gradual onset (more than 10 days). Rapid onset of ascites was seen in eighteen dogs (18/35, 51.42%) and gradual onset in twelve dogs (12/35, 34.28%).

Out of 35 dogs that suffered ascites due to liver impairment, anorexia and inappetance was reported in 7 dogs (20%) and 19 dogs (57.14%), respectively. Appetite was normal in nine dogs (25.71%). Dullness and depression was observed in 12 dogs (34.28%) and 23 dogs (65.71%) were active and alert. Vomiting was reported in eight dogs (38.09%), diarrhoea in three dogs (8.57%) and melena in 13 dogs (37.14%). Icteric conjunctival mucous membrane was observed in six dogs (17.14%) and seven dogs (20%)

had pale conjunctival mucous membrane. Exercise intolerance was also reported in 15 dogs (42.85%). Three dogs (8.57%) had pyrexia and weight loss was complained in five dogs (14.28%), enlarged lymph nodes were found in nine dogs (25.71%) and ecchymosis was observed in five dogs (14.28%). (Table 4.12, Figure 4.7). Sarvanan et al. (2014) and Vijayakumar et al. (2013) also observed similar clinical signs in dogs suffering from ascites due to hepatobiliary disorders.

Dogs suffering from ascites due to chronic hepatitis had partial anorexia (12/21, 57.14%) and complete anorexia (7/21, 33.33%). Out of the four dogs suffering from cirrhosis, jaundice was observed in one dog, anorexia in three and depression in two dogs. Sevelius (1995) also found decreased appetite and lethargy as most common complaints in dogs suffering from cirrhosis and chronic hepatitis in his study. The signs of vomiting and diarrhoea were not among the common signs in dogs suffering from chronic hepatitis in the present study. Batt and Twedt (1994) stated that in liver disorders vomiting can occur because of the direct stimulation of chemotactic trigger zone of the fourth ventricle due to the non-clearance of endotoxins by liver. Sevelius (1995) also observed in his study that jaundice was not a common finding in chronic hepatitis and stated that melena observed in liver disorder could be due to the gastric ulceration or because of coagulopathy. In dogs that suffered from cholecystitis, pyrexia, anorexia, dullness and depression, exercise intolerance and jaundice were the main findings. Center (2015) reported that in cases of cholecystitis, fever, vomiting and mild to moderate jaundice were the main signs. Loss of weight was observed in five dogs in various hepatic disorders (5/21, 23.80%) in the present study. In cases of liver disorders weight loss was observed because of the lack of nutrition and increase in the tissue catabolism (Hess and Bunch 2000).

Table 4.12: Clinical signs observed in ascitic dogs with liver disorders (n=35)

Sr. No.	Clinical Signs		Number of affected dogs	Percentage
1	Abdominal distension		30	85.71
2	Anorexia		7	20.00
3	Inappetance		19	54.28
4	Dullness and depression		12	34.28
5	Vomiting		8	22.85
6	Diarrhoea		3	8.57
7	Melena		13	37.14
8	CMM	Icteric	6	17.14
		Pale	7	20.00
		Pink	22	62.85
9	Exercise intolerance		15	42.85
10	Pyrexia		3	8.57
11	Weight loss		5	14.28
12	Enlarged lymph nodes		9	25.71
13	Ecchymosis		5	14.28
14	Engorged abdominal blood vessels		3	8.57

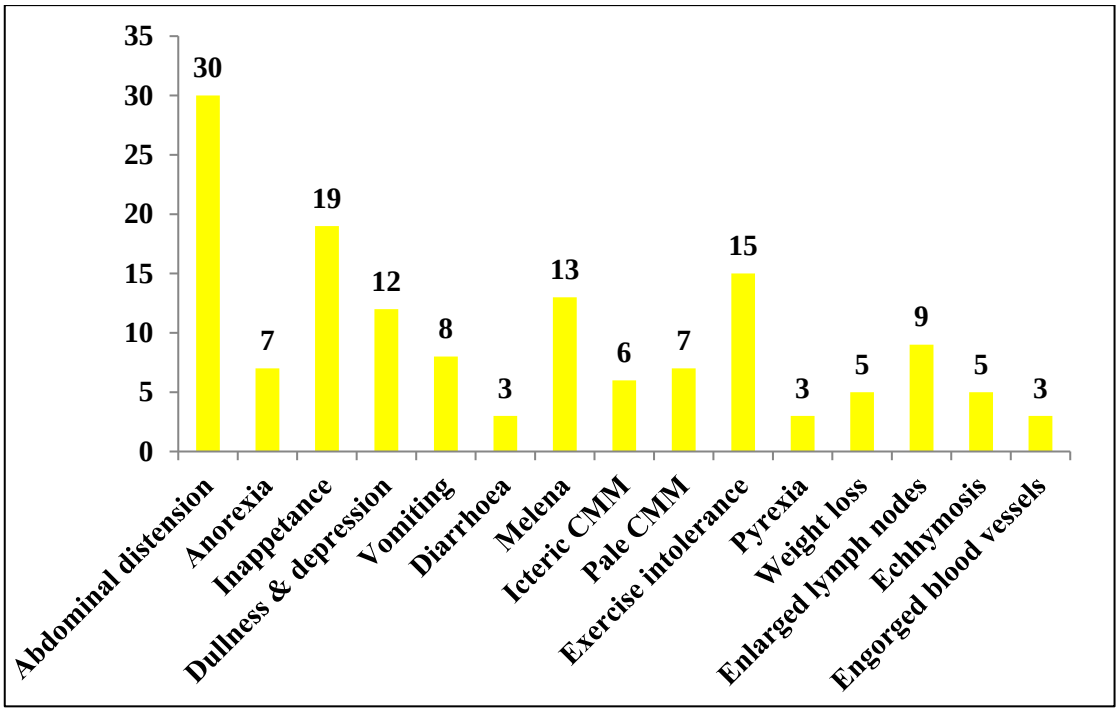


Figure 4.7: Clinical signs observed in ascitic dogs with liver disorders

4.2.4.2 Cardiac Diseases

Six dogs (6/47, 12.76%) were diagnosed with ascites due to cardiac disorders where abdominal distension was observed in five dogs (5/6, 83.33%), inappetance was reported in four dogs (4/6, 66.66%) and anorexia in one dog (1/6, 16.66%) and one dog (1/6, 16.66%) had normal appetite. On the basis of history and physical examination, two dogs (33.33%) developed ascites rapidly (7-10 days) and three dogs (50%) developed gradually (more than 10 days). Two dogs had history of vomiting (2/6, 33.33%), diarrhoea in one dog (1/6, 16.66%) and melena in two dogs (2/6, 33.33%). The other signs observed were coughing (3/6, 50%), laboured breathing/ exercise intolerance (5/6, 83.33%), pyrexia (2/6, 33.33%). The physical examination revealed pale conjunctival mucous membrane (3/6, 50%), muffled heart sounds (3/6, 50%) and tachycardia (2/6, 33.33%) (Table 4.13, Figure 4.8). Shaw and Rush (2007) reported that muffled heart sounds, tachycardia and ascites were the main signs observed in cardiac disorders. During exercise or strenuous work or metabolic changes, there is impairment of the skeletal muscles that results in the reduction of the exercise tolerance and the coughing occurs due to the cardiogenic pulmonary oedema that blocks the airway (Gibbs et al. 1982; Bjorling and Keene 1989).

Table 4.13: Clinical signs observed in ascitic dogs with cardiac disorders (n=6)

Sr. No.	Clinical signs		Number of affected dogs	Percentage
1	Abdominal distension		5	83.33
2	Anorexia		1	16.66
3	Inappetance		4	66.66
4	Vomiting		2	33.33
5	Diarrhoea		1	16.66
6	Melena		2	33.33
7	Coughing		3	50.00
8	Exercise intolerance		5	83.33
9	Pyrexia		2	33.33
10	CMM	Pale	3	50.00
		Pink	3	50.00
11	Muffled heart sounds		3	50.00
12	Tachycardia		2	33.33

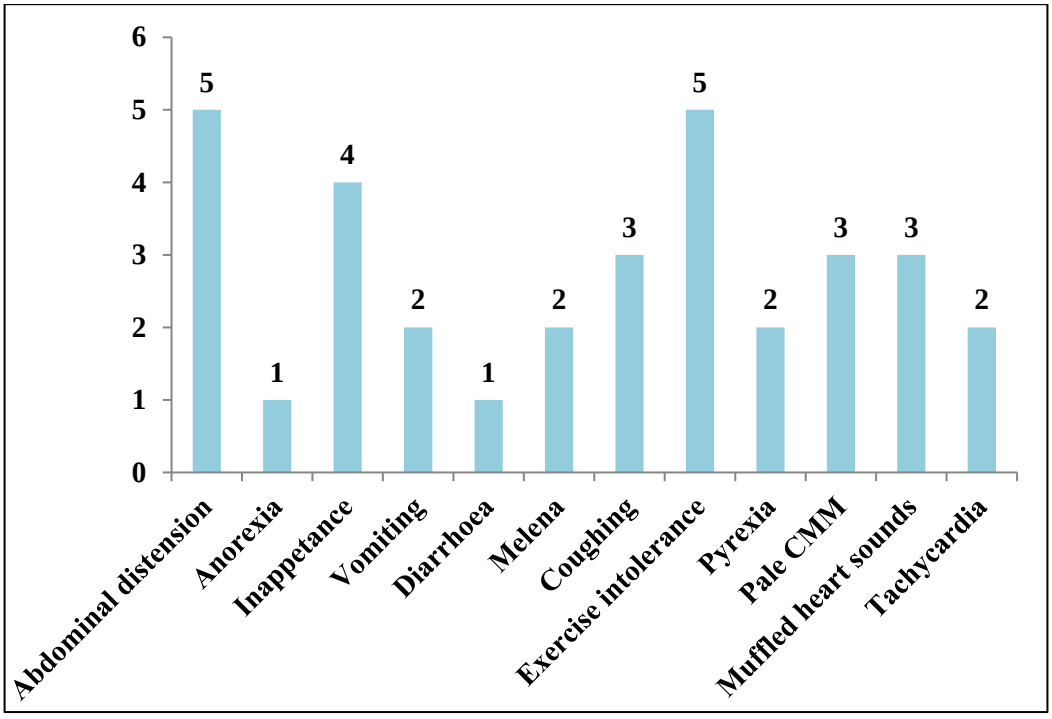


Figure 4.8: Clinical signs observed in ascitic dogs with cardiac disorders

4.2.4.3 Renal disorders

History and physical examination revealed that abdominal distension was observed in four dogs. In three dogs (50%), it was rapid onset and in one dog (16.66%) it was gradual onset. Anorexia was reported in one dog (1/6, 16.66%) and inappetance in five dogs (5/6, 83.33%). Exercise intolerance (3/6, 50%), vomiting (2/6, 33.33%), melena (1/6, 16.66%), pale mucous membrane (2/6, 33.33%) and halitosis (2/6, 33.33%) were other signs that were recorded (Table 4.14, Figure 4.9). Incorporative to the present study, Tufani et al. (2015) reported the signs of anorexia (73.81%), vomiting (80.95%), halitosis (61.09%) and ascites (30.95%) in cases of renal disorder. Polzin (2010) in his study explained that the signs of anorexia and vomiting occur due the development of uraemia. Grauer (2005) stated that because of the urea degradation into ammonia by bacteria, uremic breath was observed.

Table 4.14: Clinical signs observed in dogs with ascites due to renal disorders (n=6)

Sr. No.	Clinical signs		Number of affected dogs	Percentage
1	Abdominal distension		4	66.66
2	Complete anorexia		1	16.66
3	Partial anorexia		5	83.33
4	Vomiting		2	33.33
5	Halitosis		2	33.33
6	Melena		1	16.66
7	Exercise intolerance		3	50.00
8	CMM	Pale	2	33.33
		Pink	4	33.33

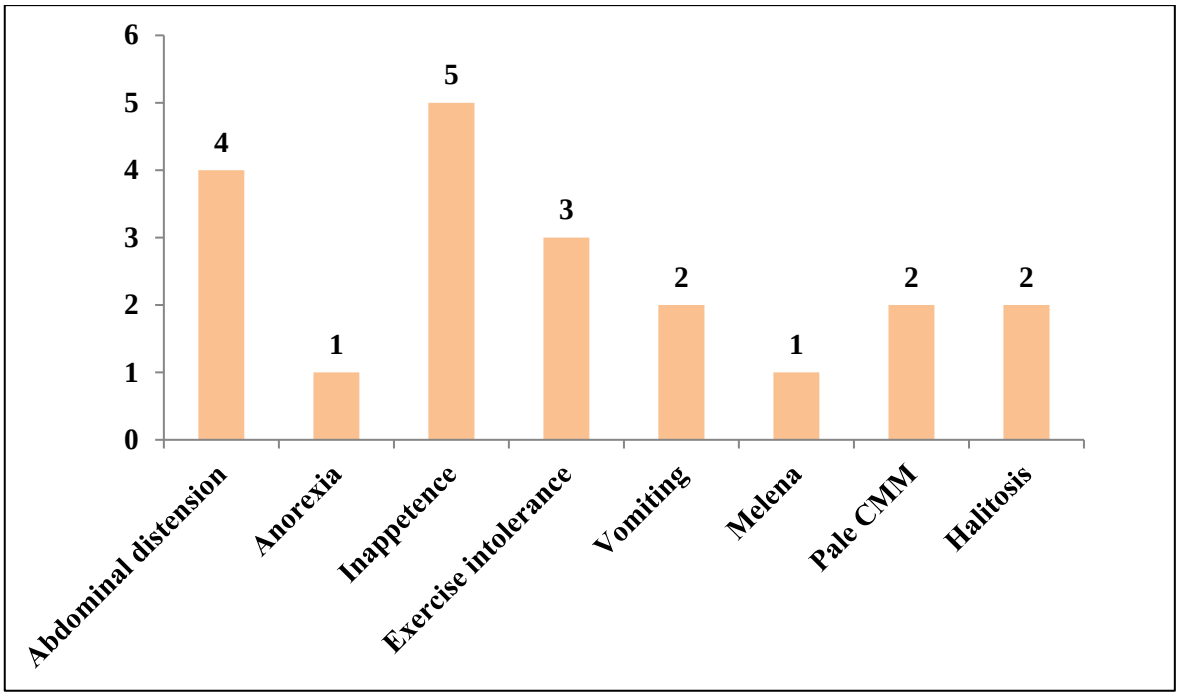


Figure 4.9: Clinical signs observed in ascitic dogs with renal disorders

The different clinical signs observed in ascitic affected dogs with liver, cardiac and renal disorders are depicted in Plate 4.1 to 4.10.

Clinical signs observed in ascitic dogs with liver, cardiac and renal disorders	

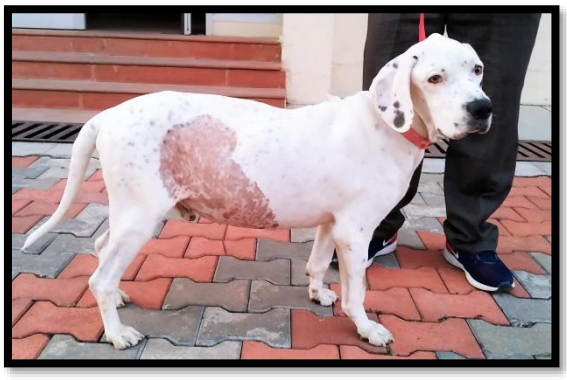
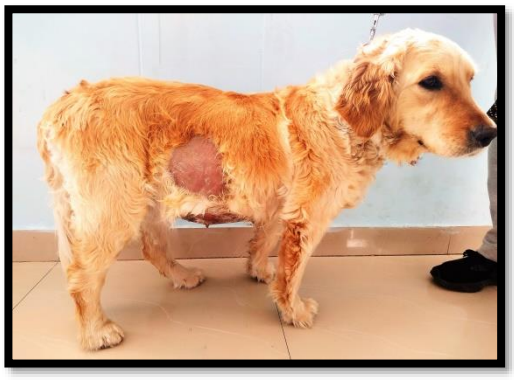

	

Plate 4.1: Severe abdominal distension in ascites affected dog	**Plate 4.2: Moderate abdominal distension in ascites affected dog**

Plate 4.3: Mild abdominal distension in ascites affected dog

Plate 4.4: Dullness and Depression in ascites affected dog

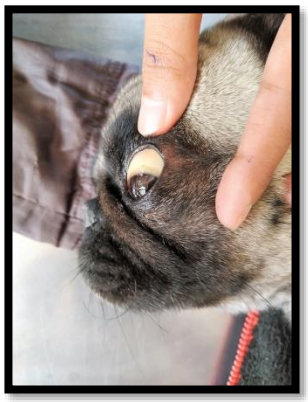


Plate 4.5: Icteric Conjunctival mucous membrane of ascites affected dog

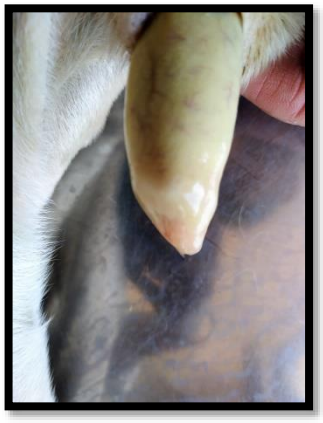


Plate 4.6: Icteric mucous membrane of ascites affected dog

Plate 4.7: Pale conjunctival mucous membrane of ascites affected dog

Plate 4.8: Pale mucous membrane of ascites affected dog

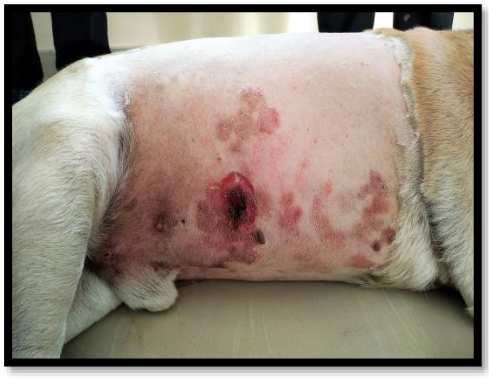


Plate 4.9: Echhymosis on andominal skin in ascites affected dog

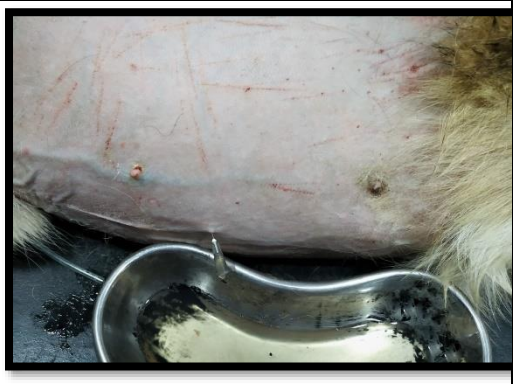


Plate 4.10: Engorged abdominal blood vessels in ascites affected dog

4.2.5 Physical examination

4.2.5.1 Condition of the body

Physical examination revealed that five dogs (5/47, 10.63%) had marked loss of weight among which three dogs (3/21, 14.28%) suffered from chronic hepatitis and two dogs (2/4, 50%) suffered with cirrhosis. Loss of appetite, lack of nutrition and tissue catabolism can be the reasons for weight loss. Weight loss was seen in one dog that suffered with cardiac disorder that could be due to cachexia because of the increase in TNF-alpha, IL-1β, norepinephrine, cortisol and insulin resistance due to activation of the neuroendocrine (de Morais and Schwartz 2004).

4.2.5.2 Abdominal distension

Distension of abdomen because of ascites was classified into 3 grades based on the severity, Grade 1 (mild), Grade 2 (moderate) and Grade 3 (Severe). Out of 35 dogs that had ascites of hepatic origin, 22 dogs (22/35, 62.85%) had grade 2 ascites, 10 suffered (10/35, 28.57 %) with grade 1 and three dogs (3/35, 8.57%) had grade 3 ascites. All the four dogs having cholecystitis had moderate ascites. Out of 21 dogs having chronic hepatitis, 14 dogs had grade 2 ascites, two had grade 1 and one dog had grade 3 ascites. Out of six dogs having acute hepatitis, three dogs had moderate ascites and three had mild ascites. In case of cirrhosis, two dogs had moderate ascites and two dogs had severe ascites. Moderate ascites was seen in most cases of cardiac disorder (5/6) and renal disorder (4/6).

4.2.5.3 Rectal temperature

The mean rectal temperature of the dogs sufferings from ascites due to liver disorder, cardiac and renal disorders were, 101.50 ± 0.18 ^{0}F, 100.68 ± 0.21 ^{0}F, 101.43 ± 0.28 ^{0}F, respectively (Table 4.15). The mean rectal temperature of the dogs with liver and renal disorders was slightly higher than the rectal temperature of the dogs with cardiac disorder that was comparable to rectal temperature of healthy dogs.

4.2.5.4 Heart rate and Respiration rate

The mean heart rate of liver, cardiac and renal disorders affected ascitic dogs was 103.65 ± 2.91 beats per minute, 105.00 ± 4.23 beats per minute and 104.16 ± 3.30 beats per minute, respectively (Table 4.15, Figure 4.10) which were significantly ($P<0.05$) higher than healthy dogs. The mean heart rate of the dogs affected with cardiac disease was slightly higher than the dogs of the other two groups. Ware (2009) reported that due to the activation of sympathetic mechanism, there is decrease in the cardiac output that results in increase in the heart rate. The mean respiration rate of the liver, cardiac and renal disorders affected dogs were 29.91 ± 0.70 breaths per min, 30.5 ± 1.33 breaths per min and 31.00 ± 1.23 breaths per min, respectively (Table 4.15, Figure 4.10). The respiration rate of all the affected dogs was significantly ($P<0.01$) higher than the healthy dogs. The increase in the respiration rate could be due to the decrease in haemoglobin and hypoxia.

Table 4.15: Clinical parameters of ascitic dogs with liver, cardiac and renal disorders

Group	Rectal temperature (^{0}F)	Heart rate (beats per minute)	Respiration rate (breaths per minute)
Healthy dogs (n=10)	101.15 ± 0.29	88.70 ± 2.08	17.9 ± 0.55
Liver disorders (n=35)	101.50 ± 0.18	103.65 ± 2.91 *	29.91 ± 0.70 **
Cardiac disorders (n=6)	100.68 ± 0.21	115.83 ± 3.00**	30.50 ± 1.33**
Renal disorders (n=6)	101.43 ± 0.28	104.16 ± 3.30**	31.00 ± 1.23**

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

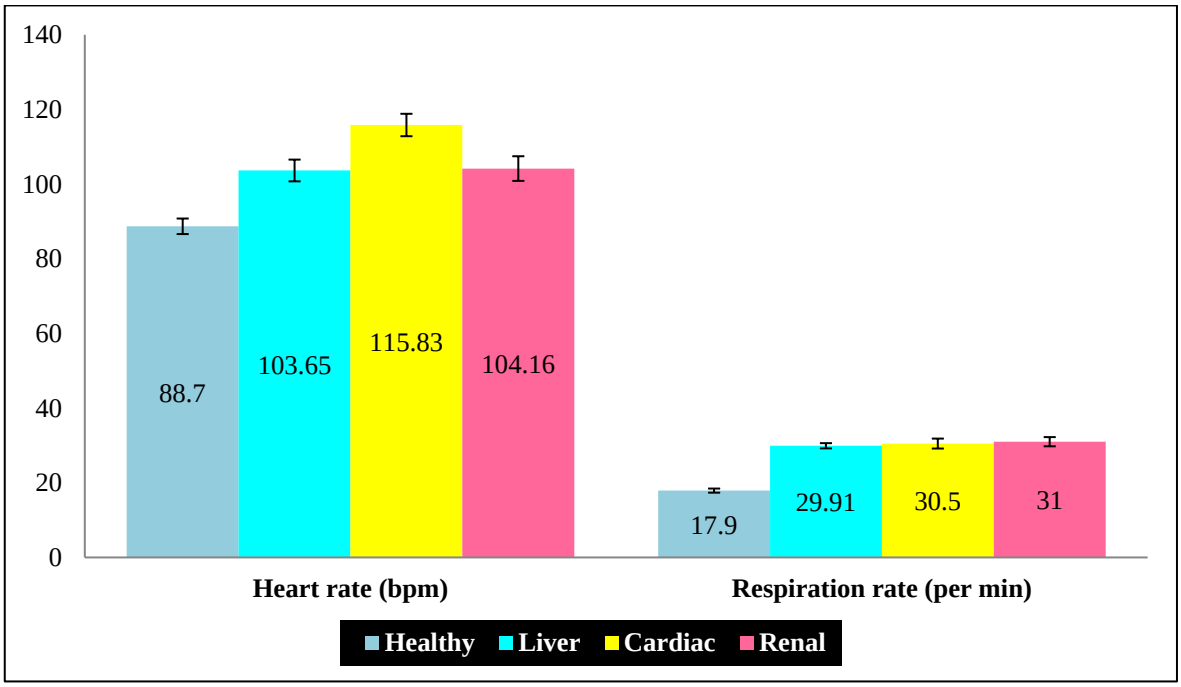


Figure 4.10: Heart rate and respiration rate of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

4.2.5.5 Thoracic auscultation

The thorax was auscultated on regular basis and was found that dogs suffering from renal disorder had no abnormality on auscultation but the dogs suffering from cardiac disorder had some abnormalities. Muffled heart sounds were heard in four dogs and arrhythmia in one dog. Out of four dogs with muffled heart sounds two had harsh lung sounds with rales which could have been due to pleural effusion. The two dogs with liver disorders had muffled heart sounds that could have been because of the displacement of the base of the heart due to abdominal fluid pressure.

4.2.6 Haemato-Biochemical changes

4.2.6.1 Haematology

The haematological profile of ascitic dogs suffering from liver, cardiac and renal disorders is presented in Table 4.16 and Figure 4.11. The mean haemoglobin level of the ascitic dogs was statistically comparable to healthy dogs. However, it was non-significantly lower in liver and renal affected dogs.

The grading of the haemoglobin was done where it was graded as mild anaemia (8-10 g/dl), moderate anaemia (6-8 g/dl) and severe anaemia (<6 g/dl). It was observed that one dog with cardiac disorder (1/6, 16.66%), one with renal disorder (1/6, 16.66%) and six dogs with liver disorder (6/35, 17.14%) had mild anaemia. Moderate anaemia was seen in two dogs (2/6, 33.33%) with renal disorders and two dogs (2/35, 5.71%) with liver disorders. Seven dogs with liver disorder (7/35, 20%) had severe anaemia. Due to the lack of efficient use of the stored iron (Watson and Bunch 2009) and mild suppression of the bone marrow in hepatobiliary disease (Dial 1995) anaemia was observed. Anaemia could also be the result of anorexia (partial/ complete) as there is lack of uptake of nutrients.

The total erythrocyte count in dogs having liver and renal disorders was 5.24 ± 0.35 and 5.71 ± 0.86 $\times 10^{12}$/L, respectively that was significantly ($P<0.05$) lower than healthy dogs. The packed cell volume remained statistically non-significant. Kumar et al. (2003) reported low levels of total erythrocyte count in ascitic dogs due to liver and renal disorders. Low levels of erythrocytes was due to non-storage of erythrocyte maturation factor in liver in hepatic insufficiency condition and decrease in erythrocyte synthesis due to reduced levels of erythropoietin hormone in renal insufficiency (Chakrabarti 1997).

The mean total leukocyte count and granulocytes were 30.84 ± 4.00 × 10^9/L and 81.55 ± 2.43% and 18.23 ± 3.27 × 10^9/L and 82.05 ± 5.14%, respectively, in liver and renal disorders that were significantly high (P<0.05) as compared to healthy dogs, that could be due to inflammation. The findings were comparable with the study conducted by Ihedioha et al. (2011). Though, Gupta et al. (2004) observed normal values of total leukocyte and haemoglobin in ascitic dogs with hepatic disorder. Lymphocytes and monocytes showed no significant change. Wadhwa et al. (1995) and Sarvanan et al. (2013) also observed no significant change in differential leukocyte count in dogs suffering from ascites. However, Kumar et al. (2003) reported low levels of total leukocyte count and packed cell volume in ascitic dogs with liver and renal disorders.

The haematological profile of the dogs suffering from ascites due to different liver disorders is presented in Table 4.17 and Figure 4.12. The mean haemoglobin level was 8.42 ± 1.49 g/dl and 8.50 ± 1.51 g/dl in cirrhosis and cholecystitis affected dogs, respectively that were significantly (P<0.05) lower as compared to the level of haemoglobin of healthy dogs (12.87 ± 0.42 g/dl) along with normal MCV and MCHC indicating normocytic normochromic anaemia. Wadhwa et al. (1995) and Randhawa et al. (1998) reported low levels of haemoglobin in dogs suffering from ascites. Willard and Tvedten (1999) and Benjamin (2010) stated that in chronic inflammatory disease and/ or secondary to multiple factors, the DNA synthesis is impaired that results in arrest of maturation at pro-rubricyte to rubricyte stage and therefore, normocytic normochromic non-regenerative anaemia is observed.

The dogs that had chronic hepatitis, four among the 21 (4/21, 19.04%) had severe anaemia, one had moderate anaemia (1/21, 4.76%) and one had mild anaemia (1/21, 4.76%). One among the six dogs (1/6, 16.66%) that suffered from acute hepatitis had moderate anaemia, one had severe anaemia (1/6, 16.66%) and two had mild anaemia (2/6, 33.33%) and two dogs (2/4, 50%) that suffered with cirrhosis had mild anaemia and one had severe anaemia (1/4, 25%). Similar to the present study, a significant decrease was observed in haemoglobin in dogs suffering with liver disorders in the study conducted by Ihedioha et al. (2011). Acute pancreatitis, bacterial cholangitis, cholecystitis and biliary rupture had non-regenerative anaemia and mild neutrophilia with shift to left were common findings (Johnson and Sherding 1994; Nelson and Couto 1998).

Table 4.16: Haematological profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Group	**Hb (g/dl)**	**TEC ($\times10^{12}$/L)**	**TLC ($\times10^{9}$/L)**	**Granulocytes (%)**	**Lymphocytes (%)**	**Monocytes (%)**	**PCV (%)**	**PLT ($\times10^{9}$/L)**	**MCV (fl)**	**MCH (pg)**	**MCHC (g/dl)**
Healthy dogs (n=10)	12.87 ± 0.42	6.81 ± 0.25	10.69 ± 0.56	75.34 ± 2.60	17.08 ± 2.11	3.94 ± 0.29	39.05 ± 0.96	201.90 ± 8.75	58.38 ± 0.87	19.02 ± 0.49	31.63 ± 0.39
Liver diseases (n=35)	11.16 ± 0.96	5.24 ± 0.35**	30.84 ± 4.00*	81.55 ± 2.43*	15.00 ± 2.24	3.45 ± 0.23	35.07 ± 2.02	199.05 ± 6.44	59.3 ± 1.52	23.38 ± 1.80**	32.59 ± 0.59
Cardiac diseases (n=6)	13.46 ± 0.85	6.84 ± 0.52	21.22 ± 7.06	67.03 ± 6.99	20.55 ± 5.08	4.05 ±0.86	40.8 ± 2.75	214.16 ± 9.24	32.59 ± 0.59	21.71 ± 1.49**	35.83 ± 2.10*
Renal diseases (n=6)	11.96 ± 2.11	5.71 ± 0.86*	18.23 ± 3.27*	82.05 ± 5.14*	9.15 ± 1.93	3.01 ± 0.52	33.16 ±5.45	208.83 ± 9.40	51.28 ± 6.78	20.68 ± 0.97**	35.95 ± 1.17

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

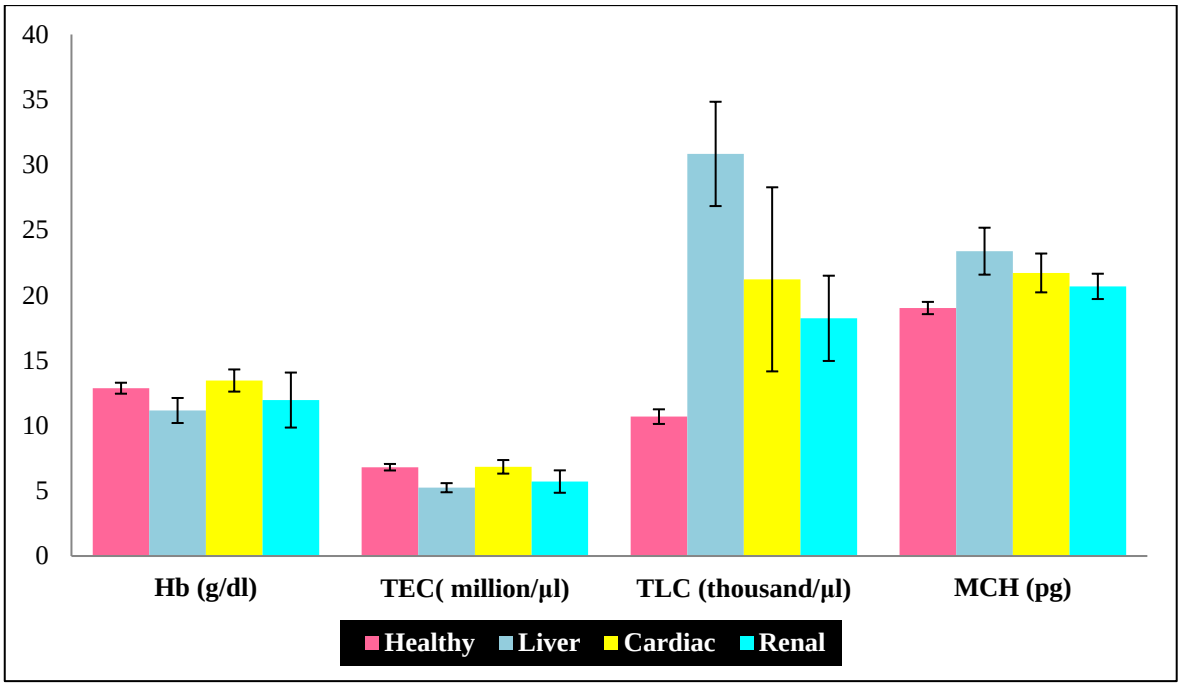


Figure 4.11: Haematological profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Total erythrocyte count decreased significantly ($P<0.05$) among all the liver groups and a significant ($P<0.01$) decrease in packed cell volume was observed in ascitic dogs with cirrhosis and acute hepatitis and chronic hepatitis, however, it was decreased non-significantly in dogs with cholecystitis as compared with healthy dogs. Anaemia in liver disorder was evident as liver apart from kidney is involved in erythropoietin production and also the other factors required for erythropoiesis (Sharma et al. 2001).

The total leukocyte count was significantly ($P<0.05$) increased among all the liver affected dogs as compared to healthy dogs. The platelet count remained non-significant and was in the normal range. Nottidge et al. (2003) recorded decreased level of haemoglobin, packed cell volume, red blood cells, lymphocytes and increased total leukocyte count and neutrophils in cases of liver cirrhosis and in dogs with ascites (Parker 2002; Pradhan et al. 2008).

Table: 4.17: Haematology profile of ascitic dogs with liver disorders (Mean ± S.E.)

Groups	Hb (g/dl)	TEC ($\times 10^{12}$/L)	TLC ($\times 10^{9}$/L)	Granulocytes (%)	Lymphocytes (%)	Monocytes (%)	PCV (%)	Platelets ($\times 10^{9}$/L)	MCV (fl)	MCH (pg)	MCHC (g/dl)
Healthy dogs (n=10)	12.87 ± 0.42	6.81 ± 0.25	10.69 ± 0.56	75.34 ± 2.60	17.08 ± 2.11	3.94 ± 0.29	39.05 ± 0.29	201.90 ± 8.75	58.38 ± 0.87	19.02 ± 0.49	31.63 ± 0.39
Cirrhosis (n=4)	8.42 ± 1.49**	3.92 ± 0.80**	28.9 ± 5.31**	84.60 ± 3.49	12.02 ± 3.12	3.30 ± 0.49	24.00 ± 3.66**	189.00 ± 7.42	63.82 ± 5.34	21.92 ± 1.36**	32.75 ± 1.25
Chronic hepatitis (n=21)	11.76 ± 0.96	5.67 ± 0.48**	29.12 ± 5.27*	78.97 ± 3.73	17.52 ± 3.46	3.55 ± 0.32	31.92 ± 2.75*	192.00 ± 8.31	59.14 ± 2.26	22.77 ± 1.83**	32.94 ± 0.45
Acute hepatitis (n=6)	12.65 ± 4.36	4.37 ± 0.52**	40.35 ± 8.93**	85.8 ± 4.24*	11.05 ± 3.76	3.10 ± 0.54	24.93 ± 2.97 **	210.00 ± 6.46	55.81 ± 1.88	27.51 ± 8.72**	31.60 ± 3.18
Cholecystitis (n=4)	8.50 ± 1.51**	5.64 ± 1.35*	20.72 ± 5.03**	85.70 ± 3.49*	10.67 ± 2.69	3.62 ± 0.84	34.15 ± 8.11	209.15 ± 8.26	60.8 ± 0.75	21.85 ± 0.99**	32.1 ± 0.70

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

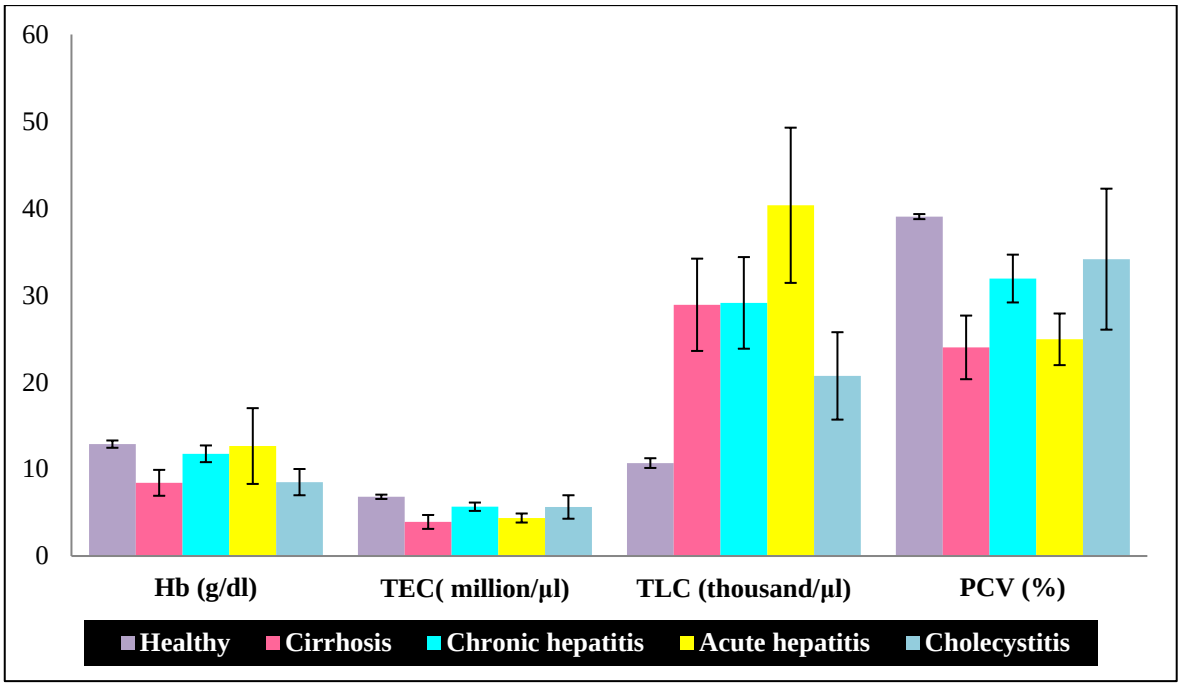


Figure 4.12: Haematological profile of ascitic dogs with liver disorders (Mean ± S.E.)

4.2.6.2 Biochemical

The mean plasma biochemical profile of ascitic dogs with liver, cardiac and renal disorders are presented in Table 4.18 and Figure 4.13 to Figure 4.16. The mean plasma value of AST was significantly ($P<0.05$) high in all the ascitic dogs with liver diseases (83.43 ± 6.53 U/L), cardiac (66.5 ± 5.58U/L) and renal (45.75 ± 4.57 U/L) disorders as compared to healthy dogs. The mean plasma levels of ALT and GGT was non-significant among liver, cardiac and renal diseases and were comparable to healthy dogs. The total bilirubin was significantly ($P<0.05$) high in liver (1.39 ± 0.21 mg/dl) and renal disorders (1.59 ± 0.86 mg/dl), the direct and indirect bilirubin were significantly increased in both liver and renal disorder group (Figure 4.14). The ALP activity increased significantly ($P<0.01$) in liver (131.57 ± 5.57 U/L), cardiac (140.33 ± 20.29 U/L) and renal diseases (125.5 ± 9.44 U/L) as compared to healthy dogs. ALP is abundantly found in tissues but is highly concentrated in liver, bone and kidney (Miller et al. 2010) therefore damage caused to them can increase the ALP levels. The ALP can cause *in vivo* dephosphorylation of bacterial endotoxins to end its toxic effects (van Veen et al. 2005; Poelstra et al. 1997). Detrano et al. (2000) reported that increase in ALP indicates inhibition of the vascular calcification that is associated with myocardial infarction and coronary death. The levels of glucose remained non-significant in cardiac (104.00 ± 13.58 mg/dl) and renal disorders dogs (97.66 ± 4.28 mg/dl) but were significantly ($P<0.05$) decreased in liver disorders

group (83.16 ± 3.70 mg/dl) (Figure 4.15). Low level of plasma glucose in ascitic dogs due to hepatic insufficiency was reported by Pradhan et al. 2008.

The mean values of BUN (Figure 4.15) and creatinine (Figure 4.16) were significantly ($P<0.01$) higher in renal disorder dogs (106.83 ± 9.71 mg/dl and 6.08 ± 1.62 mg/dl, respectively) as compared to healthy dogs. The decrease in glomerular filtration rate is associated with kidney diseases and results in increased levels of BUN and creatinine (Ihedioha et al. 2011)

The mean plasma biochemical values of ascitic dogs with liver disorders are presented in Table 4.19 and Figure 4.17 to 4.19. The mean plasma levels of ALT was significantly ($P<0.01$) high in dogs with acute hepatitis (112.03 ± 6.55 U/L) and cholecystitis (51.66 ± 7.31 U/L) (Table 4.19, Figure 4.17). The AST activity was significantly ($P<0.05$) increased in chronic hepatitis (72.52 ± 1.62 U/L), acute hepatitis (133.00 ± 5.93 U/L) and cholecystitis (105.75 ± 4.04 U/L). Increase in the activity of AST suggests hepatic insufficiency with extensive damage to the liver that leads to release of the enzymes into the blood flow (Pradhan et al. 2008; Kumar et al. 2016). The ALP activity was significantly ($P<0.01$) increased in dogs with cirrhosis (128.75 ± 8.26 U/L), chronic hepatitis (139.00 ± 8.31 U/L) and acute hepatitis (103.5 ± 6.88 U/L). ALP enzyme activity is commonly used as a marker for hepatobiliary and bone diseases (Tonelli et al. 2009). Vijayakumar et al. (2013) reported high levels of ALT, AST, ALP and GGT in dogs with hepatobiliary diseases. The increase in the levels of ALT and AST was due to active hepatic injury and necrosis of the hepatocytes that resulted in excessive release of these enzymes (Coles 1986; Stockham and Scott 2008). The levels of ALT and AST remained non-significant in cirrhosis. Similar findings were reported by Ihedioha et al. (2011). Due to the cholestatic disorder that is linked with liver damage, there was increase in ALP levels in cirrhosis and hepatitis (Coles 1986; Stockham and Scott 2008). The ALT and AST enzymes are primarily released by damaged or dying hepatocytes and in cirrhosis, majority of the hepatocytes are overtaken by fibrocytes or fibrous tissue leading to decrease in hepatocytes, therefore, the serum activity of these enzymes was decreased (Coles 1986; Stockham and Scott 2008). A significant ($P<0.05$) increase in the activity of GGT was observed in dogs with cirrhosis and cholecystitis. Tenant and Center (2008) reported that increase in the activity of ALT, AST levels and ALP, GGT levels (Nelson and Couto 1998) suggests hepatobiliary disorders.

Table 4.18: Plasma biochemical profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Group	ALT (U/L)	AST (U/L)	ALP (U/L)	GGT (U/L)	Total bilirubin (mg/dl)	Direct bilirubin (mg/dl)	Indirect bilirubin (mg/dl)	Cholesterol (mg/dl)	BUN (mg/dl)	Creatinine (mg/dl)	Glucose (mg/dl)
Healthy dogs (n=10)	36.12 ± 4.29	31.27 ± 4.32	57.26 ± 8.23	2.27 ± 0.18	0.25 ± 0.04	0.07 ± 0.01	0.18 ± 0.04	163.38 ± 4.93	31.48 ± 1.57	1.03 ± 0.15	108.15 ± 2.82
Liver disorders (n=35)	55.83 ± 5.53	83.43 ± 6.53**	131.57 ± 5.57**	3.81± 0.49	1.39 ± 0.21*	0.62 ± 0.11*	0.76 ± 0.12*	102.57 ± 7.15**	31.91± 2.86	1.29 ± 0.20	83.16 ± 3.70*
Cardiac disorders (n=6)	42.00 ± 6.26	66.5 ± 5.58*	140.33 ± 20.29**	1.83 ± 0.17	0.45 ± 0.12	0.20 ± 0.03	0.32 ± 0.11	143.00 ± 15.54	54.00 ± 9.72	1.25 ± 0.07	104.00 ± 13.58
Renal disorders (n=6)	31.23 ± 6.11	45.75 ± 4.57*	125.5 ± 9.44**	3.64 ± 1.04	1.59 ± 0.86*	0.43 ± 0.10*	0.99 ± 0.22*	119.00 ± 5.25	106.83 ± 9.71**	6.08 ± 1.62**	97.66 ± 4.28

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

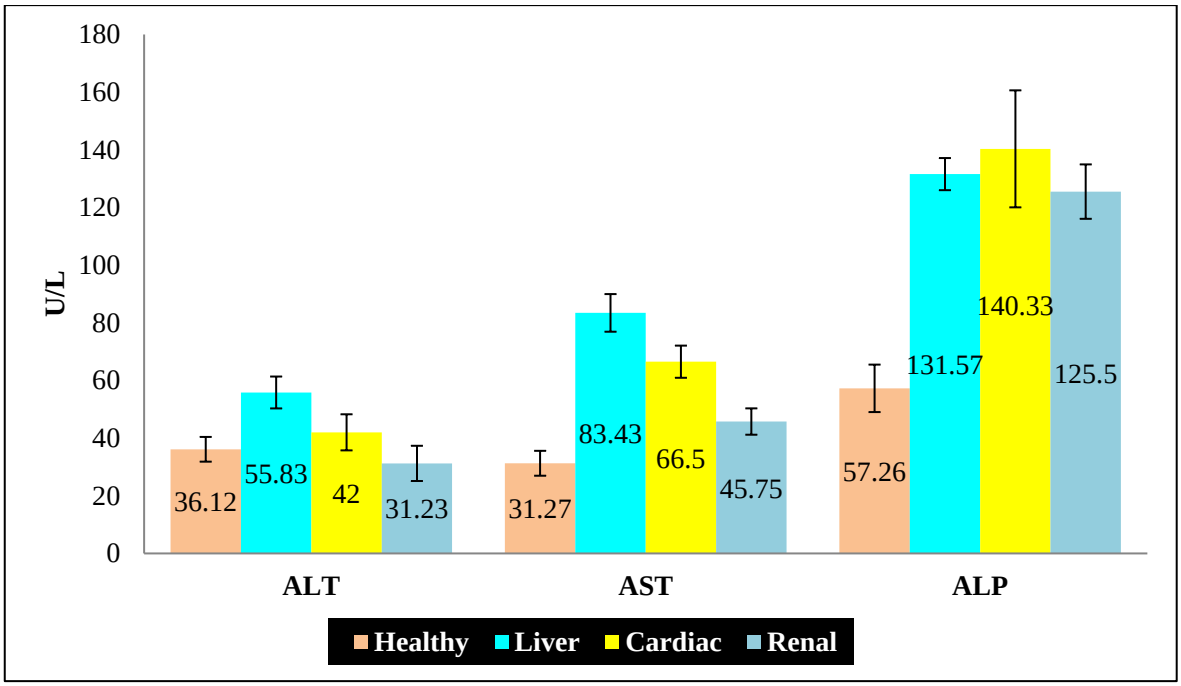


Figure 4.13: Plasma biochemical profile (ALT, AST and ALP) of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

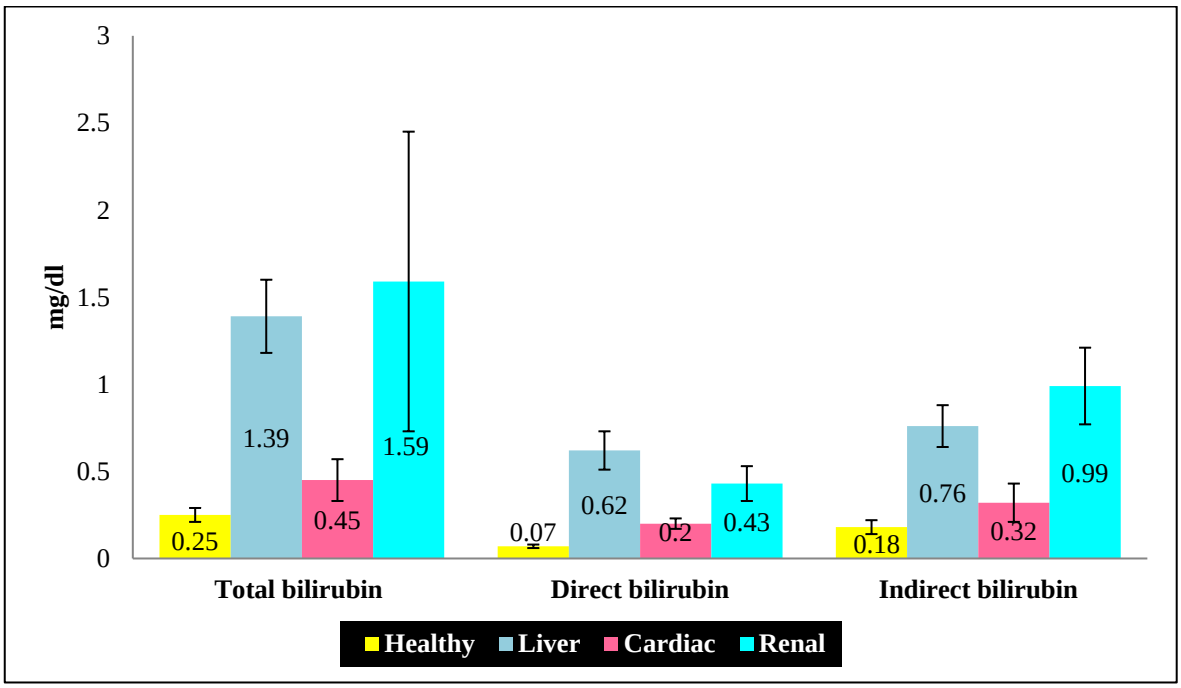


Figure 4.14: Total and differential bilirubin profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

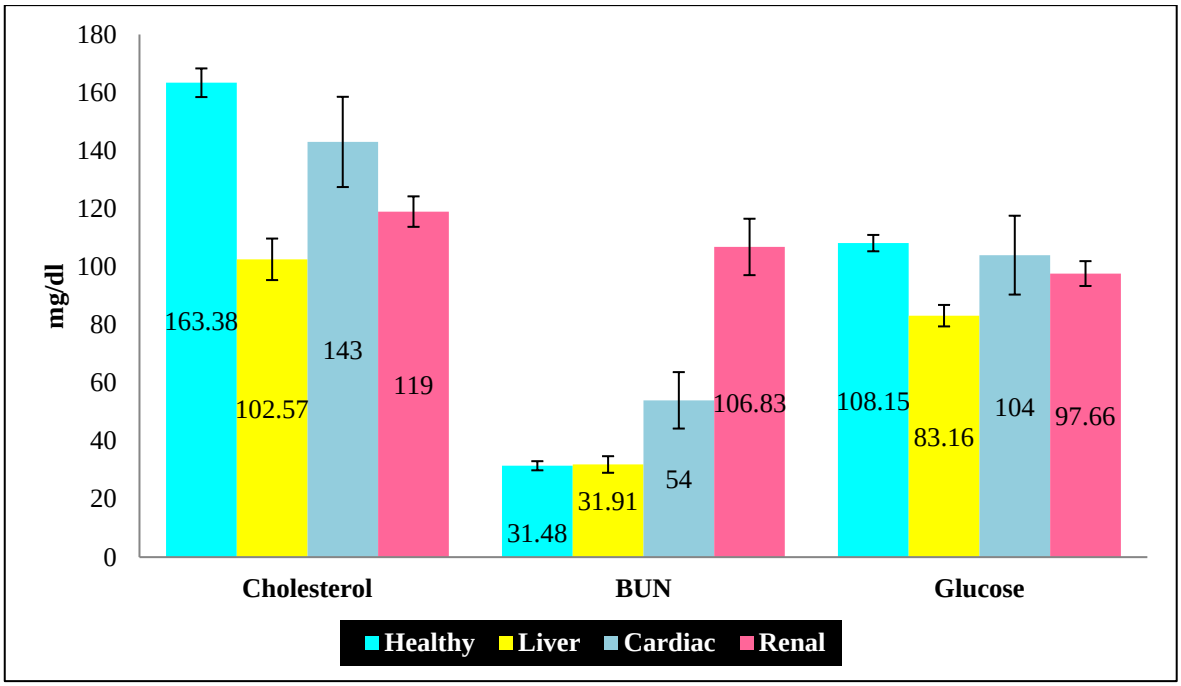


Figure 4.15: Plasma biochemical profile (Cholesterol, BUN and Glucose) of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

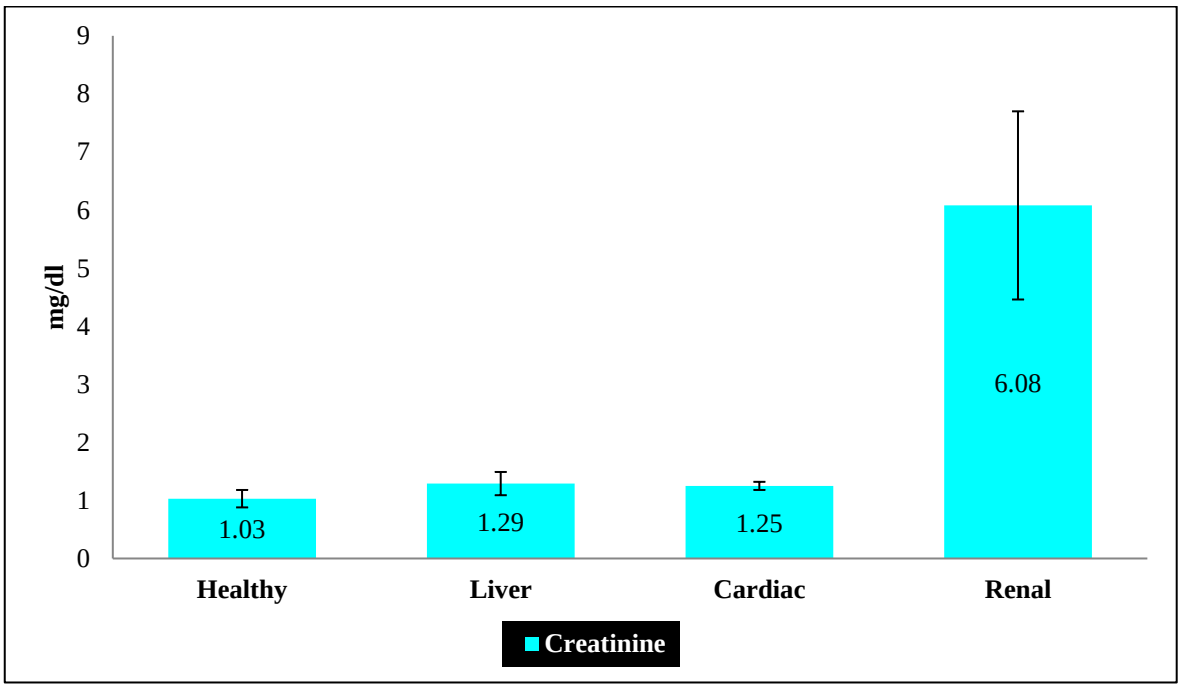


Figure 4.16: Plasma creatinine level of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

The mean total bilirubin was significantly high ($P<0.05$) in dogs with cirrhosis (1.62 ± 0.71 mg/dl), acute hepatitis (2.03 ± 0.64 mg/dl) and chronic hepatitis (1.31 ± 0.26 mg/dl) and ($P<0.01$) in dogs with cholecystitis (0.61 ± 0.08 mg/dl) as compared to healthy dogs (Figure 4.18). The level of direct bilirubin among the groups was high in acute hepatitis and the level of indirect bilirubin was high in case of chronic hepatitis. In the previous studies by Vijaykumar et al. (2001) and O' Neill et al. (2006), high levels of bilirubin was also reported in cases of cholecystitis, chlolelithiasis and cholangiohepatitis. Kozat and Sepehrizadeh (2017) stated that hyperbilirubinemia can be due to hepatobiliary or extra hepatic diseases.

The mean plasma values of BUN was significantly ($P<0.05$) decreased in chronic hepatitis as compared to healthy control animals. Willard (2010) reported that a low level of BUN in chronic hepatitis was due to hepatic insufficiency, decreased intake of proteins and / or because of excessive loss as a result of decrease synthesis.

The mean plasma glucose level was significantly ($P<0.05$) decreased in chronic hepatitis (84.90 ± 5.98 mg/dl), cirrhosis (79.25 ± 3.40 mg/dl), acute hepatitis (81.14 ± 5.41 mg/dl) and cholecystitis (81.00 ± 3.53 mg/dl). The low glucose levels indicate hepatic insufficiency (Pradhan et al. 2008; Kumar et al. 2016). The mean plasma cholesterol levels were significantly ($P<0.01$) decreased in all the liver groups indicating portosytemic shunt disruption (Figure 4.19). Webster (2019) reported decreased level of cholesterol and increased levels of ALT, AST, ALT and GGT indicative of chronic hepatitis. Goodman (1963) stated that cholesterol esterifying enzyme activity is decreased in liver disorders leading to reduction in esterified cholesterol. As the bile acid formation, bile secretion and cholesterol absorption are impaired due to liver diseases, it results in overall decrease in liver plasma cholesterol thus, hypocholesteroliemia. Hypocholesteremia can also be due to chronic liver disease leading to lower production or absorption from the intestine or conversion into bile acids (Webster 2010).

Table 4.19: Plasma biochemical profile of ascitic dogs with liver disorders (Mean ± S.E.)

Group	ALT (U/L)	AST (U/L)	ALP (U/L)	GGT (U/L)	Total bilirubin (mg/dl)	Direct bilirubin (mg/dl)	Indirect bilirubin (mg/dl)	Cholesterol (mg/dl)	BUN (mg/dl)	Creatinine (mg/dl)	Glucose (mg/dl)
Healthy dogs (n=10)	36.12 ± 4.29	31.27 ± 4.32	57.26 ± 8.23	2.27 ± 0.18	0.25 ± 0.04	0.07 ± 0.01	0.18 ± 0.04	163.38 ± 4.93	31.48 ± 1.57	1.03 ± 0.15	108.15 ± 2.82
Cirrhosis (n=4)	45.97 ± 2.16	44.07 ± 4.08	128.75 ± 8.26**	7.30 ± 0.93**	1.62 ± 0.71*	0.78 ± 0.29**	0.84 ± 0.43*	106.75 ± 4.67**	45.75 ± 9.00	0.98 ± 0.19	79.25 ± 3.40**
Chronic hepatitis (n=21)	42.45 ± 5.20	72.52 ± 1.62*	139.00 ± 8.31**	3.73 ± 0.70	1.31 ± 0.26*	0.46 ± 0.11*	0.84 ± 0.18**	93.81 ± 10.80**	24.09 ± 1.55*	1.40 ± 0.31	84.90 ± 5.98*
Acute hepatitis (n=6)	112.03 ± 6.55**	133.00 ± 5.93**	126.16 ± 7.73**	2.46 ±0.43	2.03 ± 0.64*	1.30 ± 0.41**	0.72 ± 0.29*	107.00 ± 5.85**	48.32 ± 11.13	1.21 ± 0.32	81.14 ± 5.41**
Cholecystitis (n=4)	51.66 ± 7.31**	105.75 ± 4.04**	103.5 ± 6.88*	2.73 ± 0.63*	0.61 ± 0.08**	0.28 ± 0.07**	0.33 ± 0.05*	116.47 ± 8.76**	34.48 ± 5.46	1.11 ± 0.35	81.00 ± 3.53**

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

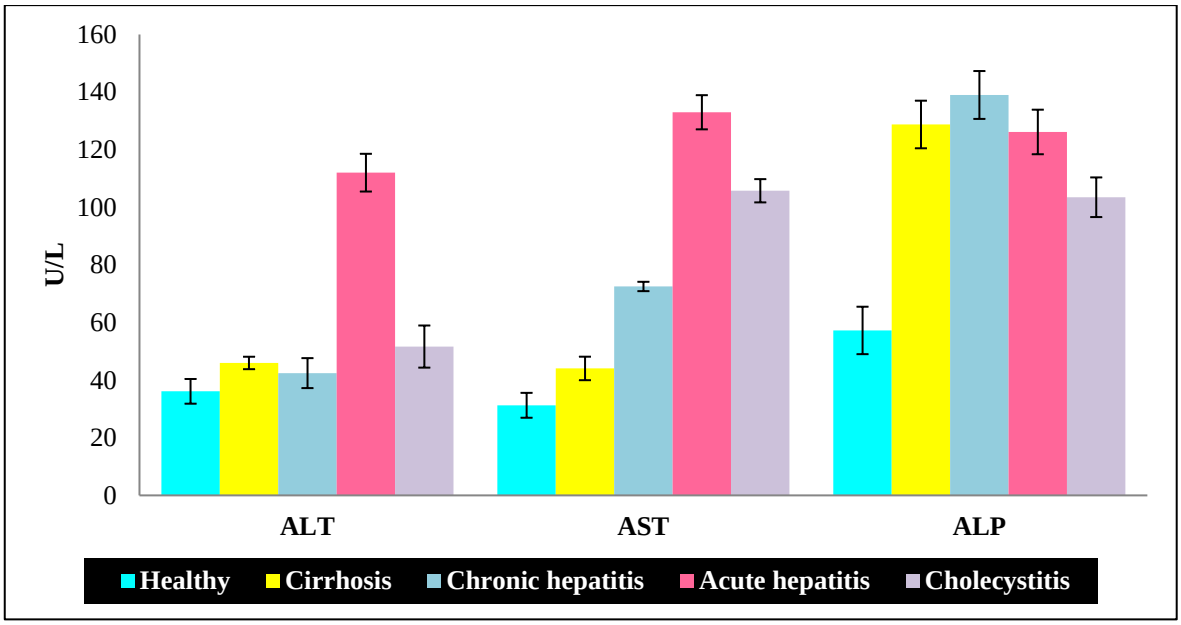


Figure 4.17: Plasma biochemical profile (ALT, AST and ALP) of ascitic dogs with liver disorders (Mean ± S.E.)

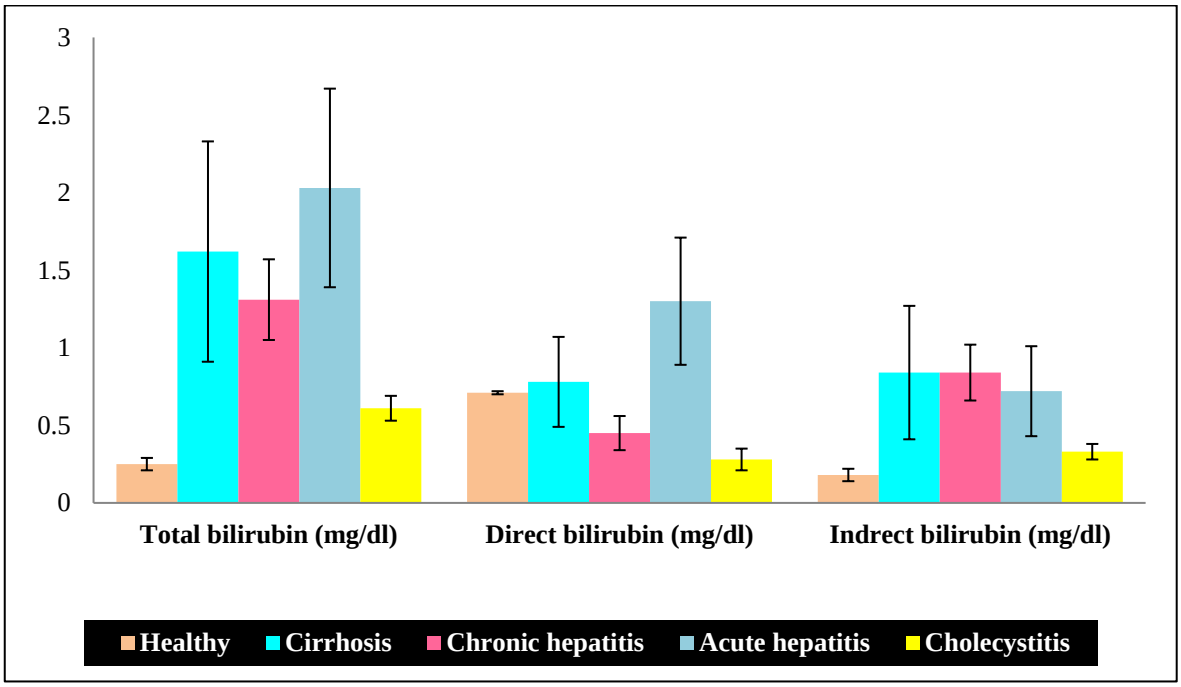


Figure 4.18: Total and differential bilirubin profile of ascitic dogs with liver disorders (Mean ± S.E.)

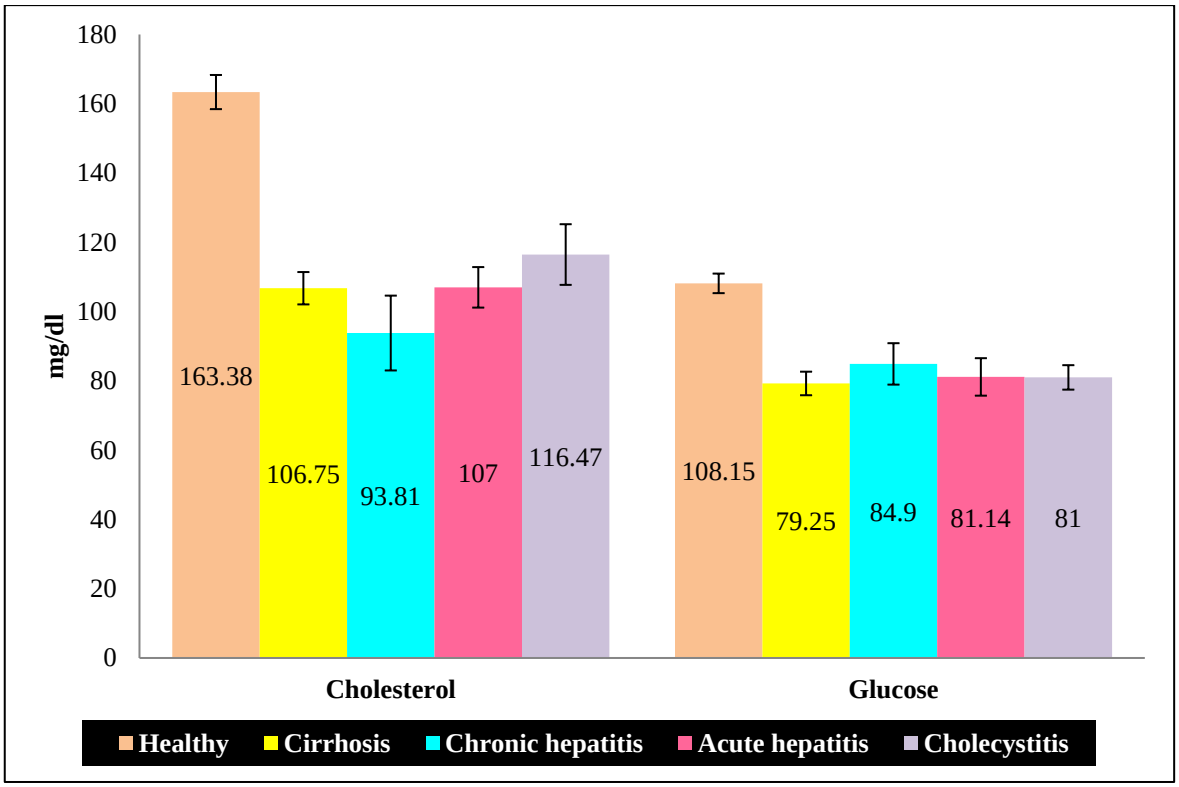


Figure 4.19: Plasma biochemical profile (Cholesterol and Glucose) of ascitic dogs with liver disorders (Mean ± S.E.)

4.2.6.3 Plasma Protein profile

The plasma protein profile of dogs with ascites due to liver, cardiac and renal disorders is presented in Table 4.20 and Figure 4.20. The mean plasma total protein was significantly ($P<0.05$) decreased in cardiac (4.98 ± 0.71 g/dl), renal affected dogs (5.31 ± 0.39 g/dl) and liver affected dogs (5.28 ± 0.29 g/dl) as compared with healthy dogs. The hypoalbuminemia was also observed among all the groups. The decreased levels of albumin was due to the accumulation of the fluid and can be attributed to decreased synthesis of albumin in liver in canine with liver disease, loss through the urine in renal disease and haemodilution associated with heart failure in dogs with cardiac disease (Reynolds 2000; Stockham and Scott 2008).

Table 4.20: Plasma protein profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Group	Total plasma protein (g/dl)	Albumin (g/dl)	Globulin (g/dl)	A/G ratio
Healthy dogs (n=10)	6.68 ± 0.12	4.33 ± 0.32	2.36 ± 0.25	2.23 ± 0.38
Liver disorders (n=35)	5.28 ± 0.29*	3.39 ± 0.18*	1.85 ± 0.24	2.93 ± 0.38
Cardiac disorders (n=6)	4.98 ± 0.71*	2.18 ± 0.25**	2.80 ± 0.62	0.67 ± 0.21*
Renal disorders (n=6)	5.31 ± 0.39*	2.67 ± 0.45*	2.63 ± 0.23	1.10 ± 0.29

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

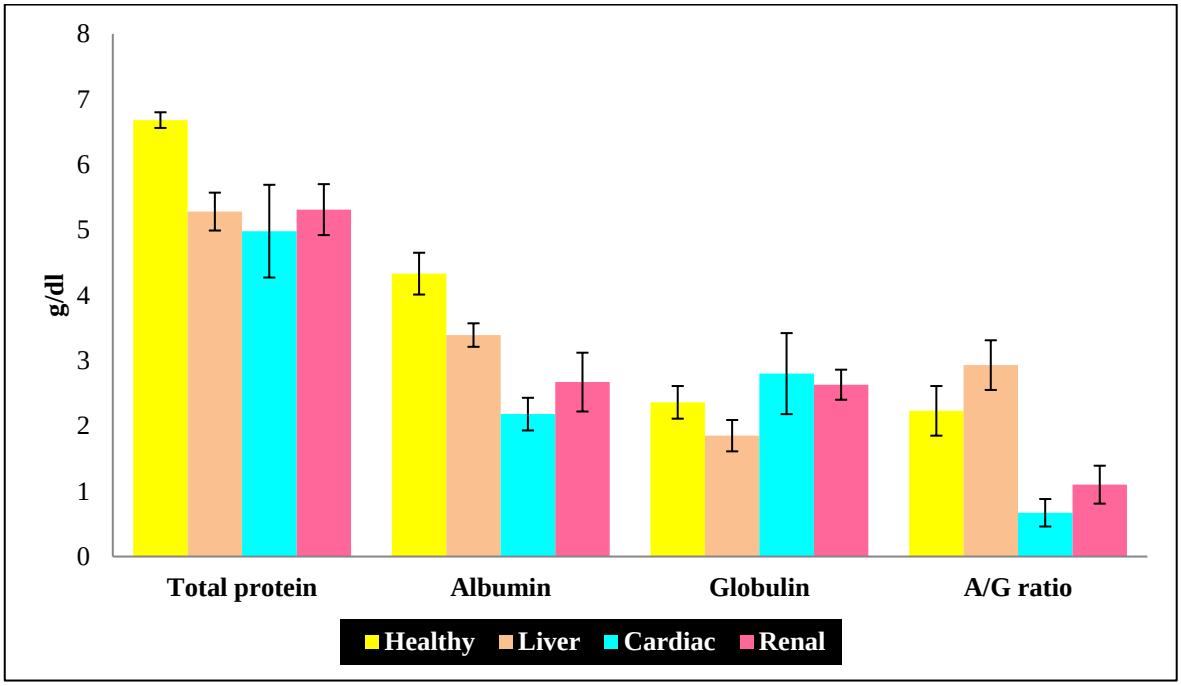


Figure 4.20: Plasma protein profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

The plasma protein profile of dogs with ascites due to different liver disorders is presented in Table 4.21 and Figure 4.21. The mean plasma values of total protein was significantly (P<0.01) low in acute hepatitis (5.17 ± 0.53 g/dl) and cholecystitis (4.82 ± 0.88 g/dl) and (P<0.05) chronic hepatitis (5.38 ± 0.41g/dl). Hypoalbuminemia was significant (P<0.05) in cirrhosis (3.27 ± 0.29 g/dl), chronic hepatitis (3.33 ± 0.27 g/dl) and cholecystitis (3.36 ± 0.53 g/dl). Webster (2010) stated that hypoalbuminemia occurs due to decreased synthesis, sequestration of albumin in abdominal fluid or in hepatic failure. The levels of plasma globulin were non-significantly lower than healthy dogs. Ihedioha et al. (2011) reported similar findings in hepatic disorder.

Table 4.21: Plasma protein profile of ascitic dogs with liver disorders (Mean ± S.E.)

Group	Total plasma protein (g/dl)	Albumin (g/dl)	Globulin (g/dl)	A/G ratio
Healthy dogs (n=10)	6.68 ± 0.12	4.33 ± 0.32	2.36 ± 0.25	2.23 ± 0.38
Cirrhosis (n=4)	5.43 ±0.96	3.27 ± 0.29**	2.16 ± 0.86	2.09 ± 0.57
Chronic hepatitis (n=21)	5.38 ± 0.41*	3.33 ± 0.27*	1.97 ± 0.35	2.72 ± 0.47
Acute hepatitis (n=6)	5.17 ± 0.53**	3.67 ± 0.43	1.49 ± 0.40	3.94 ± 1.31
Cholecystitis (n=4)	4.82 ± 0.88**	3.36 ± 0.53*	1.45 ± 0.61	3.37 ± 1.61

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

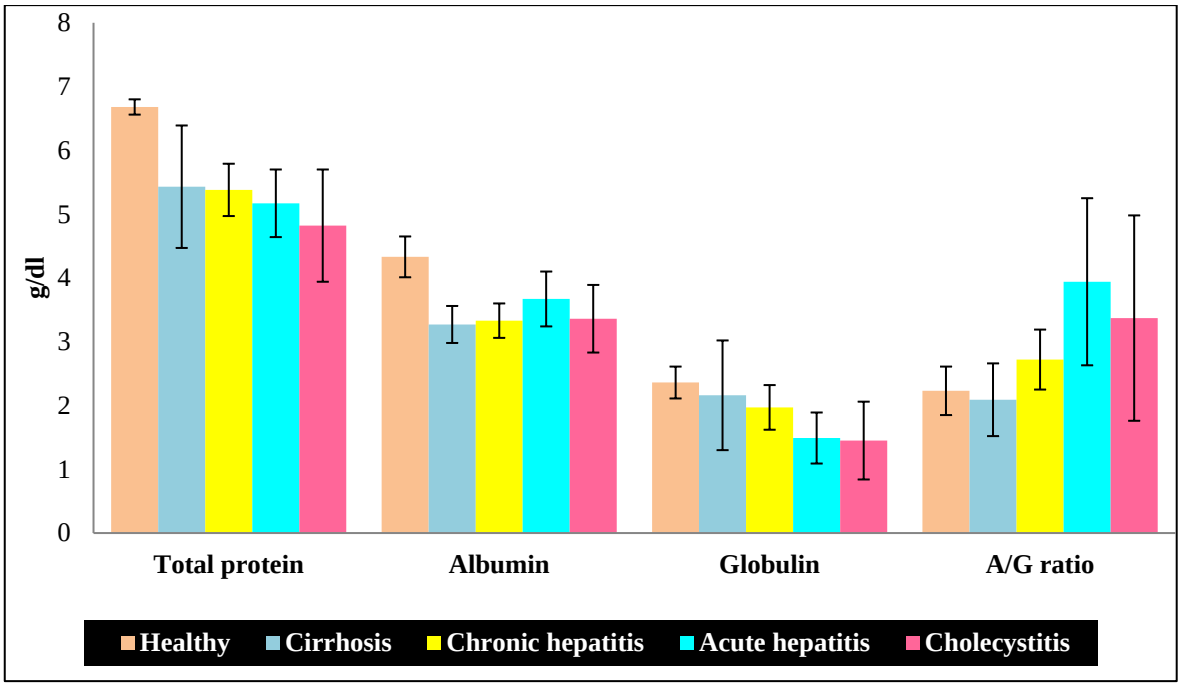


Figure 4.21: Plasma protein profile of ascitic dogs with liver disorders (Mean ± S.E.)

4.2.6.4 Mineral and electrolyte analysis

The plasma mineral and electrolytes of the dogs suffering from ascites are presented in Table 4.22. The calcium and phosphorus levels of the dogs suffering from ascites due to liver, cardiac and renal disorders were 6.07 ± 0.32 and 4.32 ± 0.48, 8.25 ± 0.96 and 3.7 ± 0.90, and 8.37 ± 1.71 and 6.25 ± 1.49 mg/dl, respectively (Figure 4.22). The level of calcium was significantly low in all the groups. The level of phosphorus was significantly ($P<0.05$) low in dogs suffering from liver and cardiac disorder but was significantly higher ($P<0.05$) in dogs suffering from renal disorders (6.25 ± 1.49) on comparison with healthy dogs.

The mean values of plasma electrolytes that are sodium, potassium and chloride in dogs with ascites due to liver, cardiac and renal disease were 138.02 ± 1.73 mmol/L, 3.92 ± 0.10 mmol/L and 100.66 ± 0.99 mmol/L, 132.74 ± 0.90 mmol/L, 4.05 ± 0.03 mmol/L and 98.95 ± 0.90 mmol/L and 136.46 ± 2.15 mmol/L, 4.05 ± 1.33 mmol/L and 100.36 ± 1.37 mmol/L, respectively (Figure 4.23 and 4.24). The mean values of sodium and chloride were significantly low in all the groups as compared with the healthy dogs, whereas the mean values of potassium was significantly low in dogs suffering from ascites due to liver (3.92 ± 0.10 mmol/L) and cardiac disorders (4.05 ± 0.03 mmol/L). However, the values were within the normal range (Klaassen 1999).

Table 4.22: Plasma minerals and electrolytes profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Parameters	Healthy dogs (N=10)	Liver disorder (n=35)	Cardiac disorder (n=6)	Renal disorder (n=6)
Calcium (mg/dl)	10.41 ± 0.89	6.07 ± 0.32**	8.25 ± 0.96**	8.37 ± 1.71*
Phosphorus (mg/dl)	4.76 ± 0.49	4.32 ± 0.48*	3.7 ± 0.90**	6.25 ± 1.49*
Ca: P	2.27 ± 0.17	2.15 ± 0.38	2.80 ± 0.60*	2.23 ± 0.97
Sodium (mmol/L)	141.7 ± 1.45	138.02 ± 1.73**	132.74 ± 0.90**	136.46 ± 2.15**
Potassium (mmol/L)	4.42 ± 0.21	3.92 ± 0.10**	4.05 ± 0.03**	4.05 ± 1.33
Chloride (mmol/L)	104.2 ± 1.81	100.66 ± 0.99**	98.95 ± 0.90**	100.36 ± 1.37**

*Significant at 5% level (P<0.05)
**Significant at 1% level (P< 0.01)

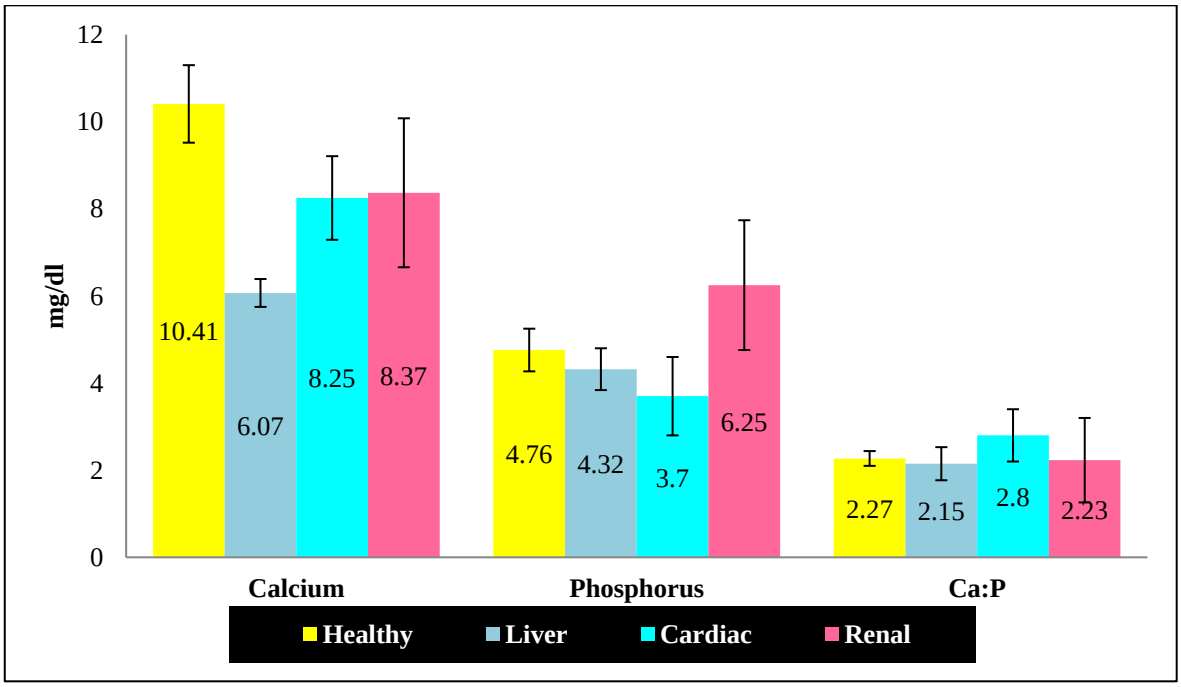


Figure 4.22: Plasma mineral profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

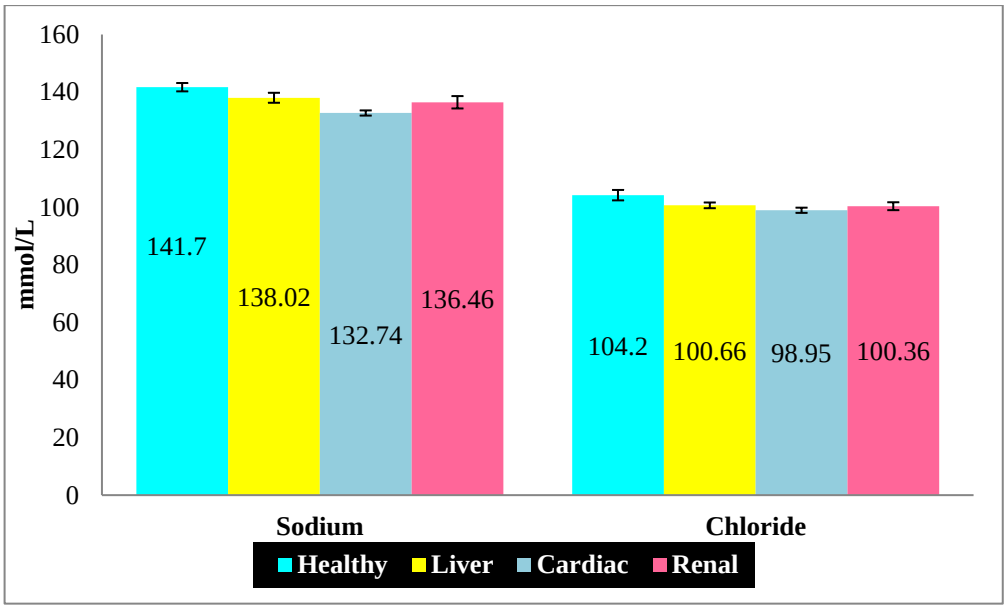


Figure 4.23: Plasma electrolyte (Sodium and Chloride) profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

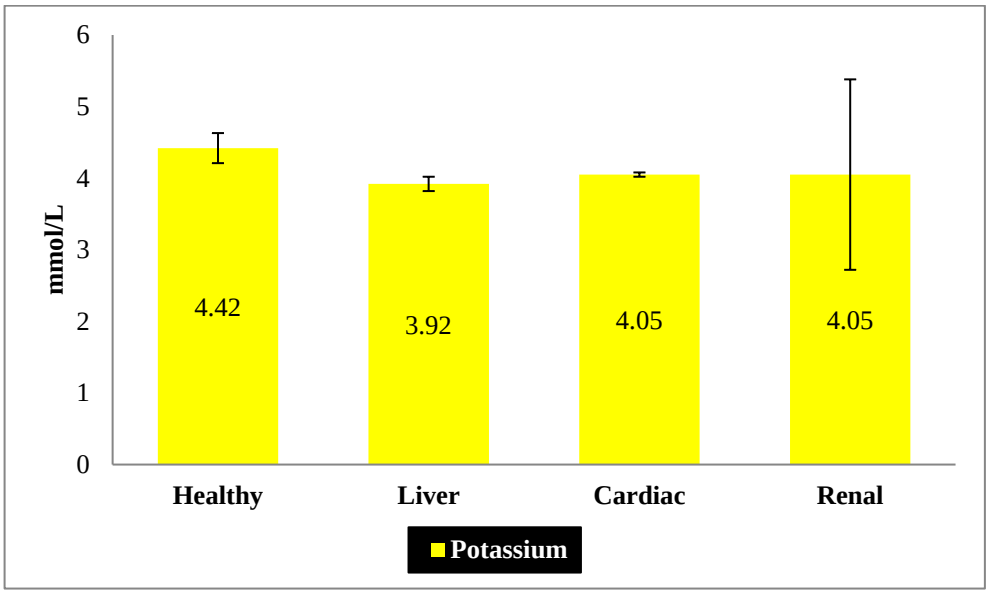


Figure 4.24: Plasma electrolyte (Potassium) profile of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

4.2.6.5 Blood gas and acid base status

The blood gas and acid base status of liver, cardiac and renal affected ascitic dogs is represented in Table 4.23. The values of pH, pCO_2, HCO_3, AnGap, tCO_2, BE, BEact, BEecf, BB, $stHCO_3$, st. pH and cH+ were non-significant among the groups and on

comparison with healthy dogs that indicated no alterations in acid base status of the ascites affected dogs.

Table 4.23: Blood gas and acid base status of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Sr. No.	Parameter	Healthy dogs (n=6)	Liver disorders (n=10)	Cardiac disorders (n=3)	Renal disorders (n=4)
1.	pH	7.12 ± 0.05	7.24 ± 0.01	7.15 ± 0.02	7.25 ± 0.12
2.	pCO_2 (mmHg)	34.12 ± 0.49	30.80 ± 3.43	32.54 ± 2.12	33.21 ± 1.65
3.	HCO_3 (mmol/L)	20.27 ± 0.33	20.06 ± 2.78	22.65 ± 1.39	21.66 ± 2.65
4.	AnGap (mmol/L)	20.65 ± 0.78	26.50 ± 1.23	24.62 ± 3.41	23.58 ± 4.21
5.	tCO_2 (mmol/L)	20.68 ± 0.71	20.28 ± 0.73	21 ± 2.90	19.65 ± 2.01
6.	BE (mmol/L)	-3.19 ± 0.15	-3.29 ± 0.18	-4.45 ± 1.22	-4.11 ± 2.31
7.	BEact (mmol/L)	-4.35 ± 0.27	-4.44 ± 0.22	-5.02 ± 0.39	-4.12 ± 0.35
8.	BEecf (mmol/L)	-4.56 ± 0.28	-4.12 ± 0.21	-6.88 ± 0.48	-5.68 ± 2.15
9.	BB (mmol/L)	43.95 ± 0.49	40.91 ± 0.47	45.22 ± 2.21	42.32 ± 1.65
10.	$stHCO_3$ (mmol/L)	21.22 ± 0.25	20.87 ± 0.25	22.92 ± 2.26	23.61 ± 2.23
11.	st. pH	7.05 ± 0.05	7.31 ± 0.01	7.28± 0.04	7.15 ± 1.2
12.	cH+ (nmol/L)	37.71 ± 0.97	35.65 ± 1.25	35.76 ± 1.85	32.96 ± 1.25

4.2.6.6 Ascitic Fluid analysis

The ascitic fluid analysis revealed significant ($P<0.05$) increase in total protein of the cardiac (3.21 ± 0.92 g/dl) and renal affected dogs (3.48 ± 0.92 g/dl) in comparison with liver affected dogs (1.68 ± 0.23 g/dl) (Table 4.24, Figure 4.25). The ascitic fluid albumin levels were significantly ($P<0.01$) low in liver disorders (0.98 ± 0.12 g/dl) as compared to cardiac (2.39 ± 0.56 g/dl) and renal disorders (2.67 ± 0.62 g/dl). The other parameters, total leukocyte count, red blood cell count, specific gravity and pH were quite close in all the affected groups (Table 4.24). The fluid was classified into 3 categories, transudate, modified transudate and exudate on the basis of total protein, specific gravity, total leukocyte count, pH and dominant cell type on cytology (Mondal et al. 2012).

Transudate fluid was observed in twelve dogs with liver disorders (12/35, 34.28 %), one in cardiac affected dogs (1/6, 16.66%). Exudate ascitic fluid was observed in nine dogs (9/35, 25.71%) with liver disorder, four dogs (4/6, 66.66%) with cardiac disorder and three in dogs with renal (3/6, 50%) disorders and modified transudate was observed in fourteen dogs (14/35, 40%) with liver disorders, one with (1/6, 16.66%) cardiac disorder and three dogs (3/6, 50%) with renal disorders (Plate 4.11 and Plate 4.12). The mean SAAG of the fluid in dogs with liver disorders (2.35 ± 0.12 g/dl) was greater than 1.1 g/dl and was significantly higher than the SAAG values observed in cardiac (0.53 ± 0.12 g/dl) and renal (0.57 ± 0.12) disorders. Similar findings were reported by Beg et al. (2001) and Sarvanan et al. (2012), who stated that SAAG >1.1 g/dl is related with portal pressure (Burgess 2004) and was noticed in cirrhotic liver, cardiac failure, Portal-vein Thrombosis and hepatic failure (Das et al. 1998).

Table 4.24: Ascitic Fluid analysis of dogs with liver, cardiac and renal disorders (Mean ± S.E.).

Group	Total protein (g/dl)	Albumin (g/dl)	SAAG (g/dl)	TLC (/µl)	RBC (/µl)	Specific gravity	pH
Liver disorders (n=30)	1.68 ± 0.23^{a}	0.98 ± 0.12^{a}	2.35 ± 0.12^{b}	37.74 ± 5.29	55.6 ± 11.64	1.021 ± 0.002	6.68 ± 0.50
Cardiac disorders (n=6)	3.21 ± 0.92^{b}	2.39 ± 0.56^{b}	0.53 ± 0.12^{a}	25.00 ± 5.77	79.16 ± 40.91	1.020 ± 0.003	6.51 ± 0.15
Renal disorders (n=5)	3.48 ± 0.92^{b}	2.67 ± 0.62^{b}	0.57 ± 0.12^{a}	25.00 ± 6.7	74.16 ± 41.84	1.020 ± 0.003	6.83 ± 0.18

Values with different superscript in a row differ significantly (P<0.05) in each groups

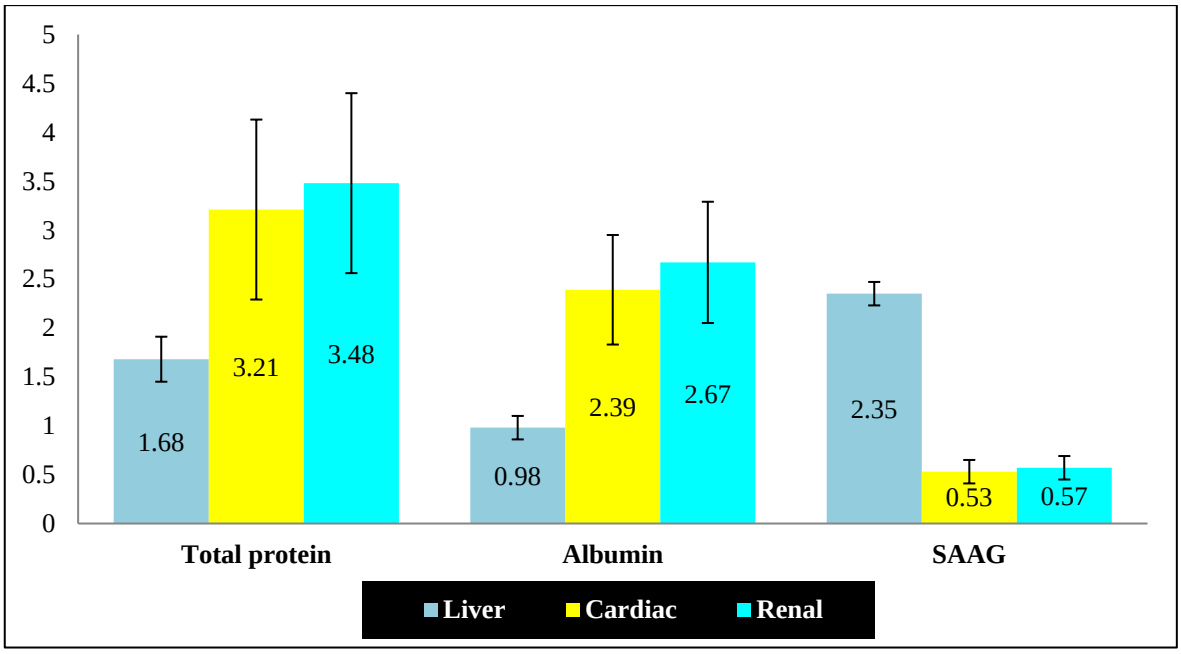
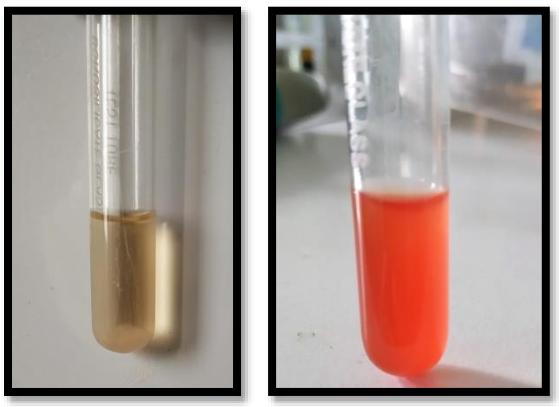


Figure 4.25: Ascitic fluid profile of dogs suffering from ascites due to liver, cardiac and renal disorders (Mean ± S.E.)

The cytology of the peritoneal fluid revealed presence of neutrophils, RBCs, inflammatory cells mainly lymphocytes and bacterial colonies (Plate 4.13). Mondal et al. (2012) reported that neutrophils, lymphocytes, plasma cells, eosinophil, mast cells, macrophages, mesothelial cells and micro-organisms can be seen under microscope in cytology of the sediment of the ascitic fluid. Saravanan et al. (2014) observed similar findings; few mesothelial cells, lymphocytes, monocytes and neutrophils in cytology of the ascitic fluid however presence of tumour cells was also reported.



(a) Pure transudate	(b) Modified transudate

Plate 4.11: Transudate and Modified transudate ascitic fluid

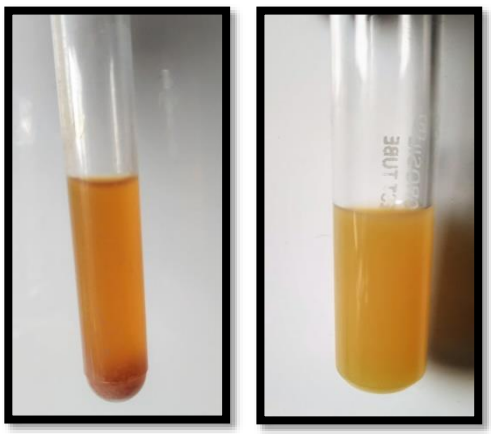
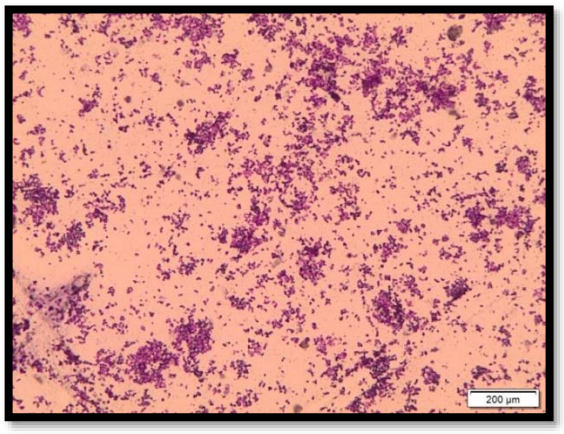


Plate 4.12: Exudate ascitic fluid

	**Plate 4.13: Ascitic fluid cytology slide showing bacterial colonies indicative of infection**

4.2.7 Urinalysis

The urinalysis of the dogs affected with ascites due to liver, cardiac and renal disorders is presented in Table 4.25. The physical examination of the urine revealed dark yellow to light colour and was clear in appearance. One dog with liver disorder had dark orange coloured urine. Bilirubin, ketones and urobilinogen were observed in cases of ascitic dogs due to liver disorder. Similar findings were reported by Gupta et al. (2004). Kozat and Sepehrozadeh (2017) stated that bilirubin in urine was due to haemolytic or hepatobiliary disease. However, Archer (2005) stated that bilirubinuria can be a normal finding when dipstick test is carried and is <2+. The urinary protein levels in ascitic dogs due to liver, cardiac and renal disorders were 55.45 ± 6.34, 16.66 ± 3.07 and 15 ± 3.41 mg/dl, respectively, with marked increase in ascitic dogs suffering from liver disorders (55.45 ± 6.34 mg/dl). Vijayakumar et al. (2002) reported proteinuria in ascitic dogs with liver disorders. Glycosuria was observed in ascitic dogs with liver (77.5 ± 7.60 mg/dl) and renal disorders (52.5 ± 1.44 mg/dl). Laura (2011b) reported that glycosuria can occur due to defective renal tubular abnormalities or damage.

The urinary pH and red blood cell count remained non-significant among all the groups. The urinary leukocyte count was 14.16 ± 1.12 and 7.5 ± 1.44 in dogs suffering ascites due to liver and renal disorders, respectively. The urinary specific gravity increased significantly among all the groups. Laura (2011a) reported that high levels of urine specific gravity were observed in marked proteinuria, glycosuria and dehydration. However, Kozat and Sepehrizadeh (2017) reported that in hepatic insufficiency or portosystemic shunts (PSSs), the urine specific gravity was decreased due to inability to fully concentrate the urine leading to polyuria.

Table 4.25: Urinalysis of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

Parameters	Healthy Control (n=10)	Liver disorder (n=35)	Cardiac disorder (n=6)	Renal disorder (n=6)
Blood (RBCs/µl)	Nil	10 ±01.19**	10 ± 1.44**	6.25 ± 1.02**
Bilirubin (mg/dl)	Nil	2.33 ± 0.19**	Nil	Nil
Ketone (mg/dl)	Nil	6.66 ± 0.48**	Nil	Nil
Urobilinogen (mg/dl)	Norm	3.75 ± 0.86**	Norm	Norm
Protein (mg/dl)	Nil	55.45 ± 6.34**	16.66 ± 3.07**	15 ± 3.41**
Glucose (mg/dl)	Nil	77.5 ± 7.60**	Nil	52.5 ± 1.44**
pH	6.21 ± 0.16	6.14 ± 0.16	6.11 ± 0.20	5.91 ± 0.30
Specific gravity	1.026 ± 0.001	1.031 ± 0.001*	1.032 ± 0.003*	1.032 ± 0.002**
Leukocyte (WBCs/µl)	Nil	14.16 ± 1.12**	Nil	7.5 ± 1.44**
Clarity	Clear	Clear	Clear	Clear

**Significant at 1% level (P<0.01)
*Significant at 5% level (P<0.05)

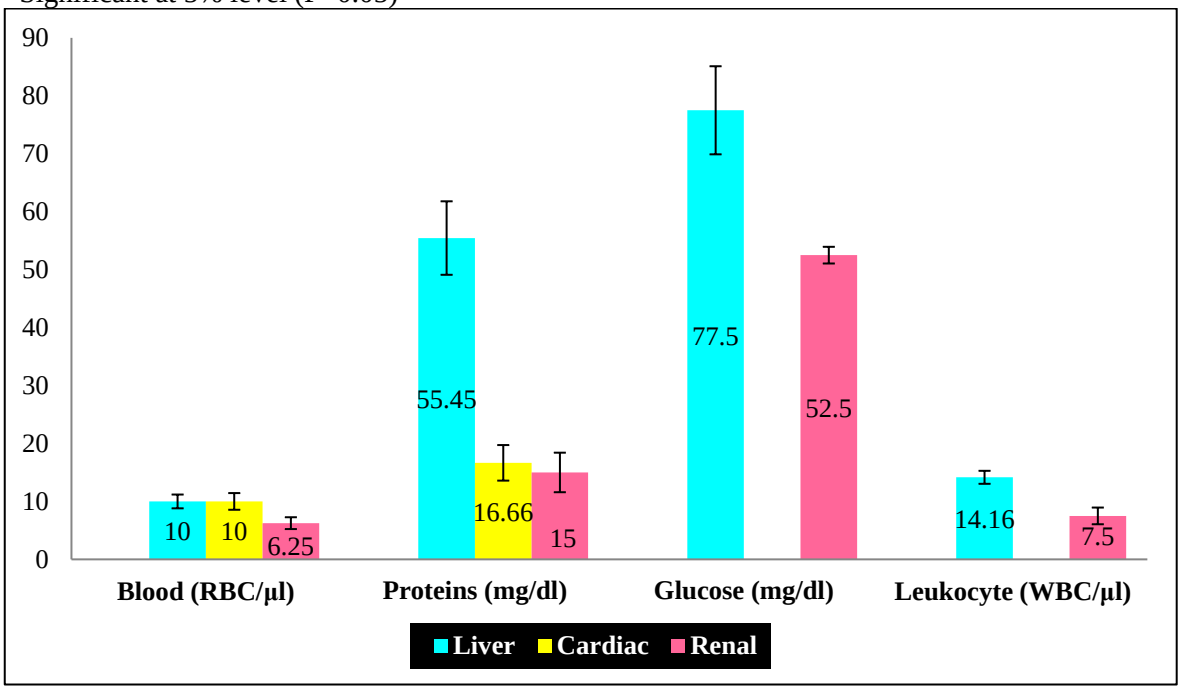


Figure 4.26: Urinalysis of ascitic dogs with liver, cardiac and renal disorders (Mean ± S.E.)

The cytology of the urine sediment revealed presence of pus cells in two dogs with renal disorder. Similar findings were reported by Kumar et al. (2003). Granular cast along

with crystals of calcium oxalate was observed in one ascitic dog with renal disorders (Plate 4.14).

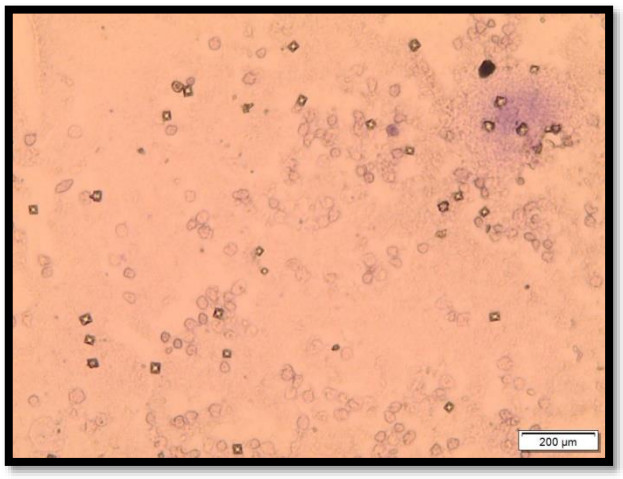


Plate 4.14: Urine cytology revealing presence of calcium oxalate indicative of crystalluria

4.2.8 Electrocardiographic changes

On comparison of the mean of electrocardiographic indices between the healthy dogs and dogs suffering from ascites due to cardiac disorders, no significant difference was observed (Table 4.26). However, tachycardia and arrhythmia was observed in 1 dog each.

Table 4.26: Electrocardiograph indices of healthy dogs and ascitic dogs with cardiac disorders (Mean ± S.E.)

ECG parameter	Healthy dogs (n=5)	Cardiac disorder (n=4)
HR (beats per minute)	88.7 ± 2.08	115.83 ± 3.00
P wave interval (sec)	0.06 ± 0.0030	0.048 ± 0.003
PR interval (sec)	0.3 ± 0.004	0.1 ± 0.008
QRS interval	0.04 ± 0.002	0.045 ± 0.003
QT interval (sec)	0.15 ± 0.005	0.16 ± 0.023
T wave interval (sec)	0.65 ± 0.007	0.076 ± 0.010
P wave amplitude (mV)	0.20 ± 0.05	0.166 ± 0.015
R wave amplitude (mV)	1.51 ± 0.21	1.266 ± 0.087
T wave amplitude (mV)	0.26 ± 0.06	0.241 ± 0.029

- Two dogs (Plate 4.15 and Plate 4.16) showed increase in PR interval that indicated slow conduction between atria and ventral due to slow conduction through the AV node depicting 1st degree heart block. Laflamme (1997) observed increased PR interval with flat T wave along with hypokalemia in ascitic dogs with cardiac disorder.
- One dog (Plate 4.17), showed increase in the QRS voltage indicating left ventricular hypertrophy. Shaw and Rush (2007) reported increased QRS interval in an ascitic dogs with cardiac disorder.
- One dog (Plate 4.18) showed QT prolongation by prolonged ventricular repolarisation usually asserted with increased risk of ventricular arrhythmia.
- Two dogs revealed normal electrocardiograph with normal indices (Plate 4.19 and Plate 4.20).

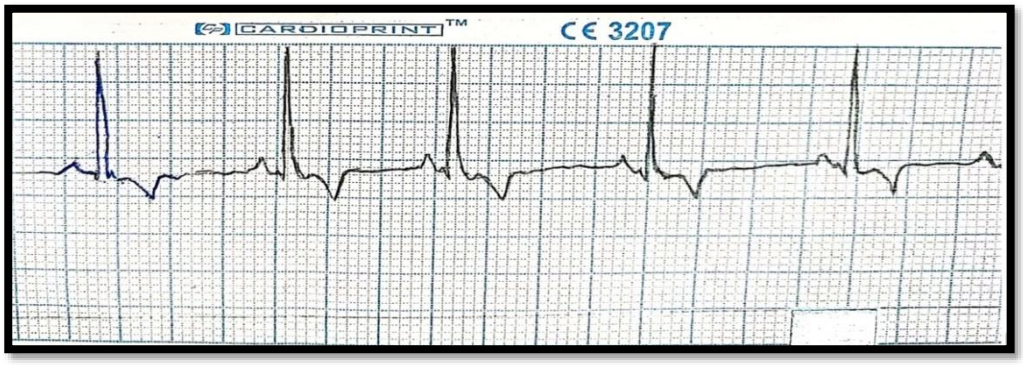


Plate 4.15: Electrocardiogram showing increased PR interval indicative of first degree heart block in dog with mild tachycardia

PR interval: 0.06 sec	QRS interval: 0.04 sec	QT interval: 0.12 sec	
T interval: 0.1 sec	P amplitude : 0.2 mV	R amplitude: 1.5 mV	T amplitude: 0.3 mV

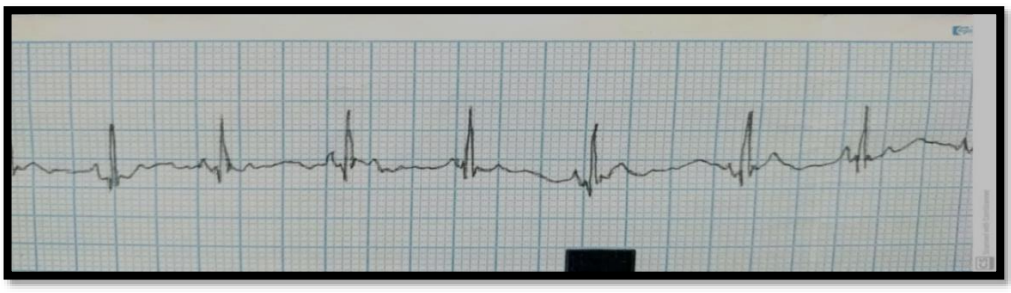


Plate 4.16: Electrocardiogram showing increased PR interval indicative of first degree heart block in dog with normal heart rate

PR interval: 0.08 sec	QRS interval: 0.05 sec	QT interval: 0.16 sec	
T interval: 0.06 sec	P amplitude: 0.2 mV	R amplitude: 1.2 mV	T amplitude: 0.3 mV

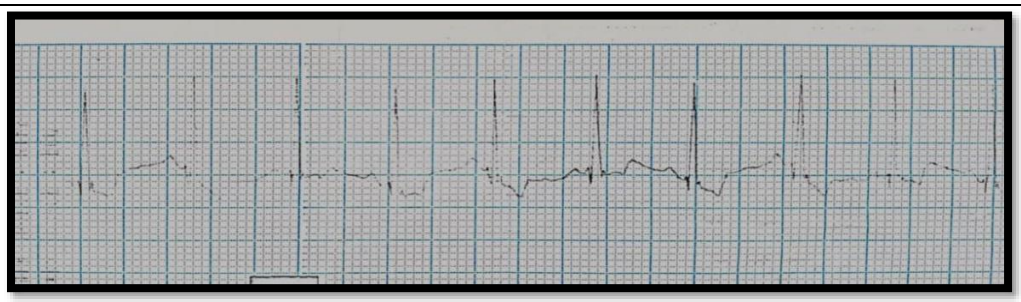


Plate 4.17: Electrocardiogram with increased QRS voltage indicative of left ventricular hypertrophy

PR interval:0.12 sec	QRS interval: 0.06 sec	QT interval: 0.12 sec	
T interval: 0.08 sec	P amplitude: 0.2 mV	R amplitude: 1.5 mV	T amplitude: 0.3 mV

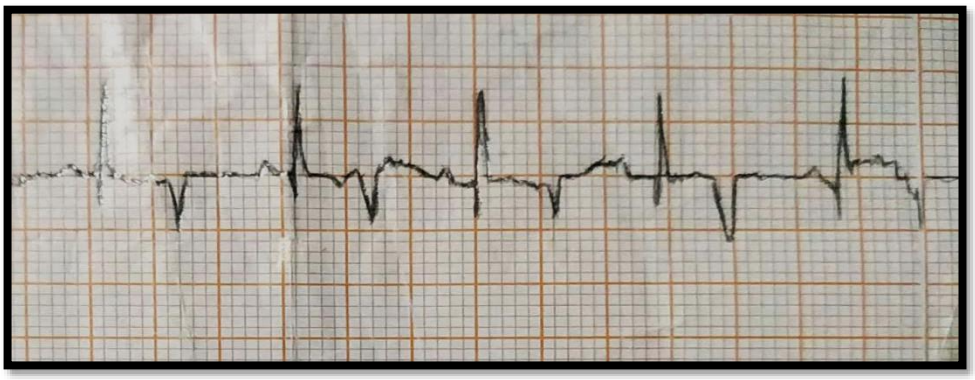


Plate 4.18: Electrocardiogram showing QT prolongation due to prolonged ventricular repolarisation in a dog with mild arrthymia

PR interval: 0.12 sec	QRS interval: 0.04 sec	QT interval: 0.28 sec	
T interval: 0.06 sec	P amplitude: 0.1mV	R amplitude: 1.4 mV	T amplitude: 0.25 mV

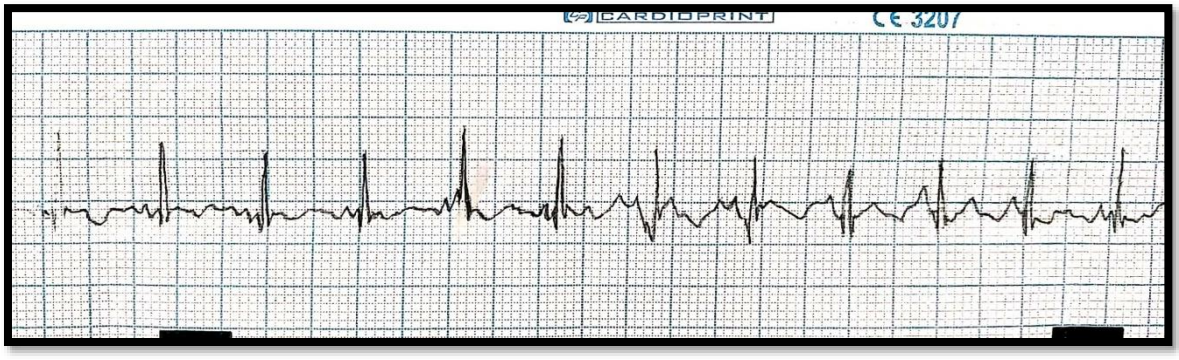


Plate 4.19: Electrocardiogram showing normal sinus rhythm in a dog with normal heart rate

PR interval: 0.08 sec	QRS interval: 0.04 sec	QT interval: 0.16 sec	
T interval: 0.12 sec	P amplitude: 0.15 mV	R amplitude: 1.0 mV	T amplitude: 0.2 mV

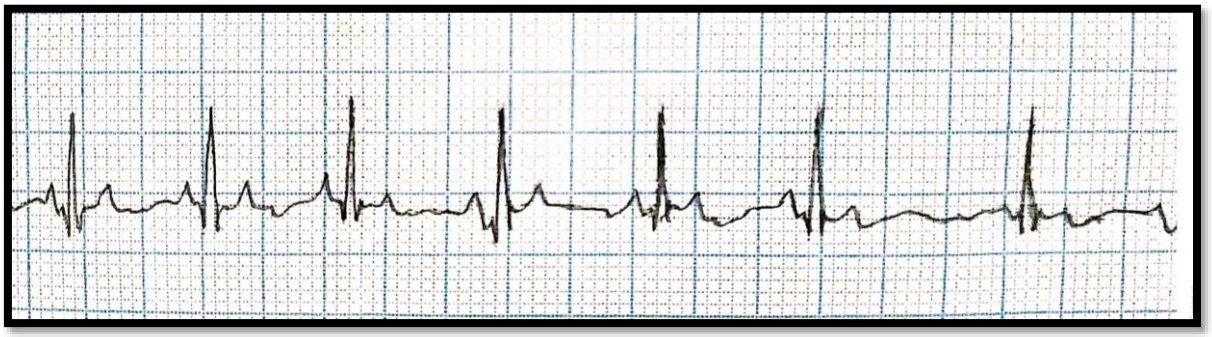


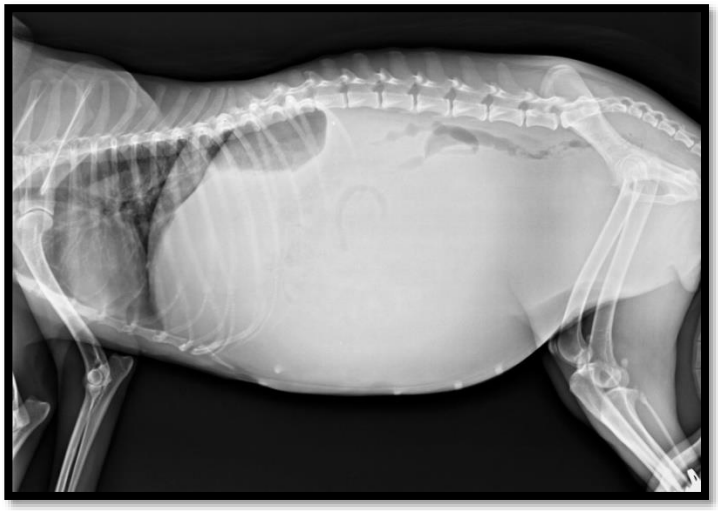
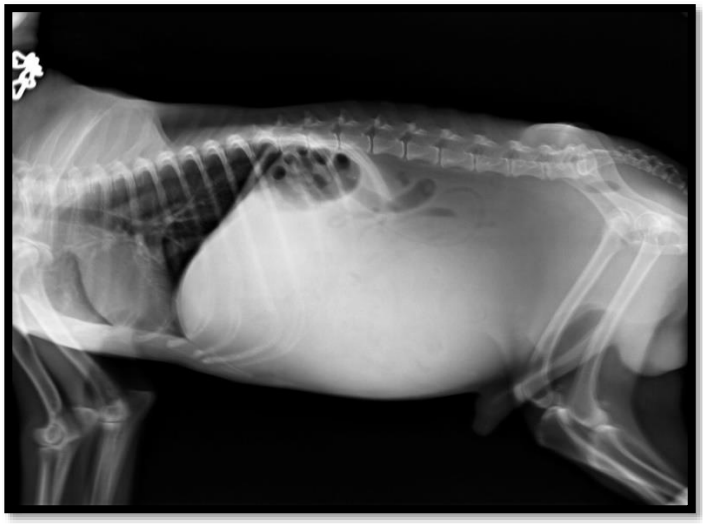
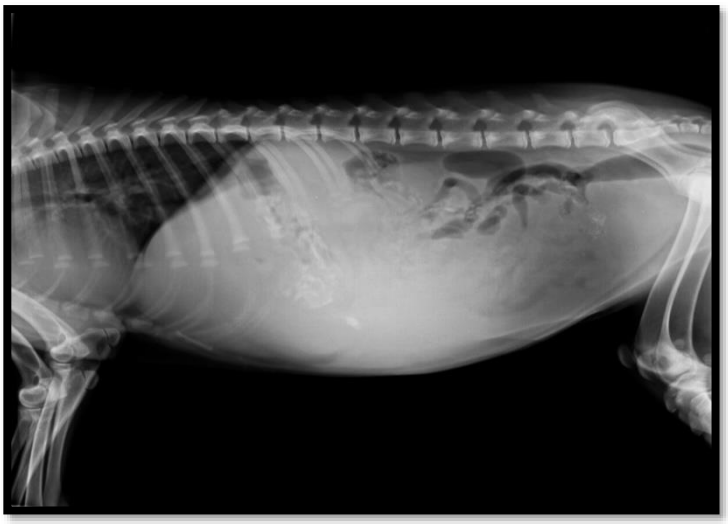
Plate 4.20: Electrocardiogram showing normal sinus rhythm in a dog with slight increased heart rate

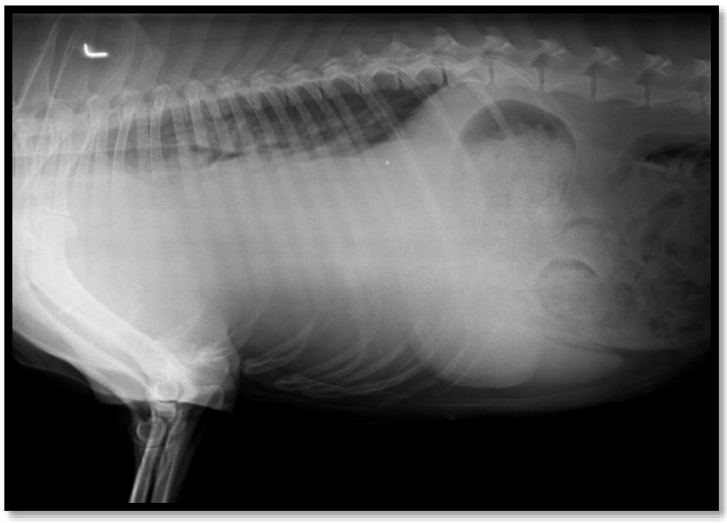
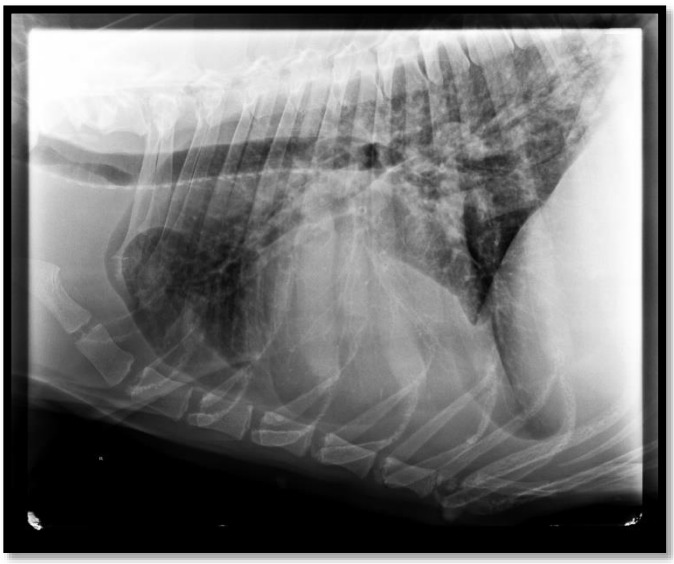
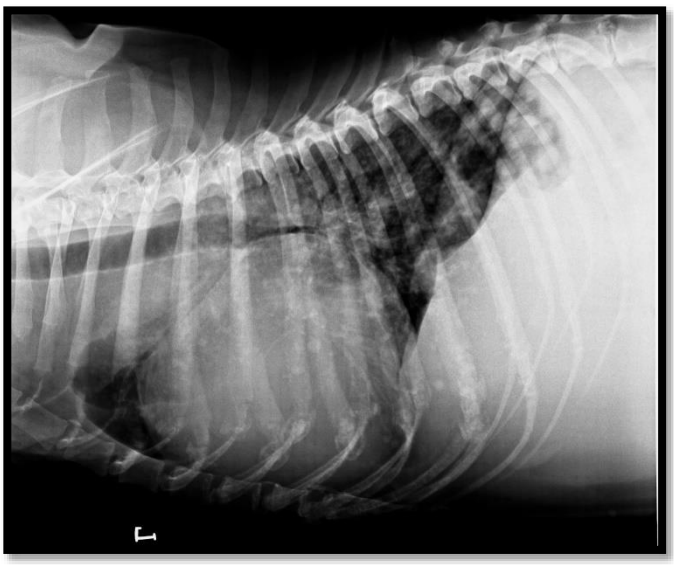
PR interval: 0.08 sec	QRS interval: 0.04 sec	QT interval: 0.12 sec	
T interval: 0.04 sec	P amplitude: 0.15 mV	R amplitude: 1.0 mV	T amplitude: 0.1 mV

4.2.9 Imaging studies

4.2.9.1 Radiography

Radiographic images showed ground glass appearance on standing abdominal view in 39 dogs (39/47, 82.97%), out of these, 30 dogs were of hepatic group, five were of cardiac group and four were of renal group and due to that the visualisation of the abdominal organs were not clear (Plate 4.21 to 4.23). The thoracic radiograph of dogs of cardiac group showed pleural effusion (Plate 4.24) in four dogs and pericardial effusion in two dogs. The dogs suffering from pericardial effusion, pulmonary oedema was the most common clinical finding (Srinivasan and Maheshkrishna 2008). Increase in the vertebral heart size (VHS) in four dogs (11, 13, 14.5, and 13) (Plate 4.26) and right ventricular enlargement was observed in one dog. The mean VHS of the dogs suffering from cardiomyopathy was 12.87 ± 0.71. Bright and Mears (1997) asserted that radiograph in dilated cardiomyopathy; generalized cardiomegaly is a representative feature. Elevation in the trachea in lateral thoracic radiographic view was observed in one dog (Plate 4.25). Similar findings were reported by Bhadesiya et al. (2015). Kocatürk et al. (2016) also observed enlargement of the heart size, deviation of the trachea, mild to severe pulmonary oedema and increased VHS.

Radiographs of the dogs suffering from ascites	
	**Plate 4.21: Abdominal radiograph: Severe ascites in a dog masking the abdominal organs**
	**Plate 4.22: Moderate ascites in a dog masking the details of abdominal organs**
	**Plate 4.23: Abdominal radiograph: Mild ascites and gas filled intestinal segments observed in a dog on abdominal radiograph**

	**Plate 4.24: Thoracic radiograph: Pleural fluid in the thoracic cavity in a dog having mild ascites**
	**Plate 4.25: Thoracic radiograph: Marginally elevated trachea with increased general opacity in a dog having ascites**
	**Plate 4.26: Thoracic radiograph: Cardiac silhouetting of cranial margins with increased soft tissue density and increased Vertebral Heart Size (13) in dog having ascites**

4.2.9.2 Echocardiography

Echocardiography was performed in ascitic dogs with cardiac disorders. Pericardial fluid was observed in two ascitic dogs (2/6, 33.33%) (Plate 4.27), poor cardiac contractibility in one dog (1/6, 16.66%), cardiac wall thickening in one dog along with left ventricular enlargement (1/6, 16.66%) and mixing of the blood (regurgitation) in one dog with dilated cardiomyopathy (1/6, 16.66%) in color Doppler.

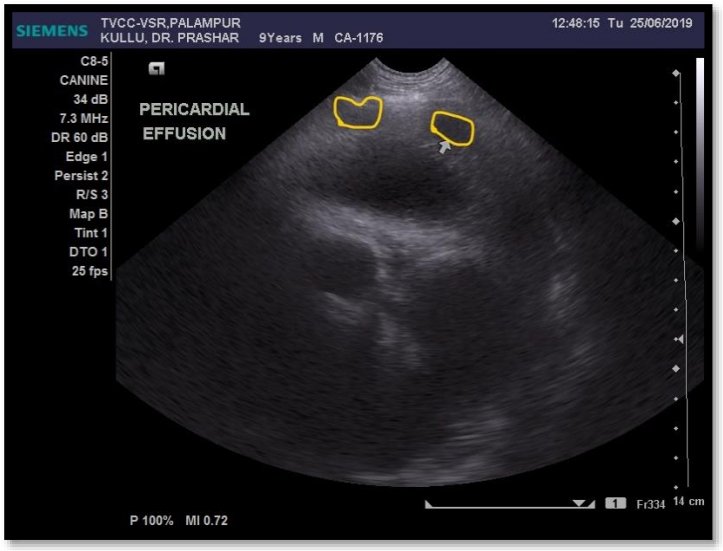


Plate 4.27: Echocardiography of ascitic dog having pericardial effusion

4.2.9.3 Ultrasonography

Ultrasonography was performed in all the dogs that were presented with abdominal distension. In 6 dogs, presence of anechoic free fluid in the abdomen was an accidental finding where the fluid was not that enough to cause distension at time of presentation of these dogs but could have in future. 5 dogs among these 6 dogs were of hepatic disorder and 1 dog was of renal disorder group. Yeh et al. (1977) stated that ultrasound is able to detect even 100ml of the ascitic fluid in the abdomen that was not being revealed on radiography.

The abdominal ultrasonography of the 47 dogs affected with ascites revealed hyperechogenicity and hypoechogenicity of the liver parenchyma in 32 (32/47, 68.08%) and two dogs (2/47, 4.25%) respectively. Hepatomegaly and splenomegaly was evident in 11 (11/47, 23.40%) and five dogs (5/47, 10.63%), respectively. Reduced size of the liver was noticed in three dogs (3/47, 6.38%). Elhiblu et al. (2015) observed diffuse increase in echogenicity “bright liver” with rounding of liver margins, microhepatica, distension of

gall bladder with wall thickness, mild hepatic congestion in ascitic dogs with liver disorders. The ultrasonographic features that were observed are presented in Table 4.27.

Table 4.27: Ultrasonographic features of ascitic dogs

Sr. No.	Ultrasonographic Feature		Number of affected dogs	Percentage
1.	Anechoic peritoneal fluid		47	100
2.	Fibrin strands in the fluid		4	8.51
3.	Inflammation of liver parenchyma		33	70.21
4.	Increase echogenicity	Liver	32	68.08
		Renal	7	14.89
5.	Decreased echogenicity	Liver	2	4.25
		Renal	0	0
6.	Increased size	Liver	11	23.40
		Kidney	2	4.25
		Heart	5	10.63
		Spleen	5	10.63
7.	Reduced liver size		3	6.38
8.	Round edges of the liver		19	40.42
9.	Wall thickness	Gall bladder	4	8.51
		Urinary bladder	4	8.51
		Heart	1	2.12
10.	Sludge	Gall bladder	5	10.63
		Urinary bladder	2	4.25
11.	Systemic hypertension		3	6.38
12.	Fibrosis of the liver		4	8.51
13.	Fatty degeneration		5	10.63
14.	Gall Bladder polyp		1	2.12
15.	Thickened liver capsule		3	6.38
16.	Decreased perfusion	Liver	3	6.38
		Renal	2	4.25

17.	Space Occupying Lesion (SOL)	Liver	5	10.63
		Kidney	1	2.12
18.	Cystic lesion	Liver	2	4.25
		Kidney	1	2.12
		Spleen	1	2.12
19.	Increased size Medial Iliac Lymph Node (MILN)		3	6.38
20.	Increased size mediastinal lymph node		1	2.12
21.	Nephroliths		2	4.25

Ultrasonography of the dogs with cirrhosis revealed presence of anechoic peritoneal free fluid, hyperechogenicity of the liver parenchyma (3/4, 75%) (Plate 4.30), round edges of the liver (3/4, 75%), hepatomegaly (2/4, 50%), reduced liver size (2/4, 50%) were the common findings. Space occupying lesion (SOL) in the liver parenchyma was observed in one dog as hyperechoic mass. Increase in the size of spleen was found in two dogs. Peri-hepatic changes like ascites, portal and splenic vein dilation, and splenomegaly occur in cirrhosis due to portal hypertension (Partington and Biller 1995). Chohan (2005) reported microhepatica, irregular liver margins, and focal lesions representing regenerative nodules, increased echogenicity of the liver parenchyma that was associated with increased fibrous tissue and ascites in dogs with liver disorder.

The common ultrasonographic changes noticed in cholecystitis were increase in the gall bladder wall thickness (3/4, 75%) (Plate 4.41), sludge in the gall bladder (4/4, 100%), increase echogenicity of the liver parenchyma (2/4, 50%), rounding of the liver margins (2/4, 50%). Lamb (1991) stated that gall bladder wall thickening could be seen in cholangiohepatitis and cholecystitis. Fluid filled cystic lesions in the liver parenchyma and near gall bladder was observed in one dog (Plate 4.42). Inflammation of the urinary bladder and hyper-echogenicity of the renal cortex was also observed in one dog. The abdominal cavity was filled with anechoic fluid in all the 4 dogs. Similar findings were reported by Smith et al. (2017) where the gall bladder wall thickness was >2mm and gall bladder oedema was defined as thickened hypo-echoic gall bladder. Thickened wall of the

gall bladder can be secondary to hypoproteinemia, right sided congestive heart failure and presence of contiguous peritonitis (Smith et al. 2017).

In case of acute hepatitis round edges of the liver (6/6, 100%) (Plate 4.40), increase in the liver size (3/6, 50%), hyper-echogenicity of the liver parenchyma (2/6, 33.33%) (Plate 4.39) and peri-portal vasculature (2/6, 33.33%), systemic hypertension (2/6, 33.33%) and thickening of the liver capsule (1/6, 16.66%), anechoic peritoneal fluid (6/6, 100%), increased size of mediastinal lymph node in one dog (1/6, 16.66%) (Plate 4.35) were the common ultrasonographic changes that were visualised.

Ultrasonography of the dogs with chronic hepatitis revealed inflammation of the liver parenchyma (20/21, 95.23%), hyper-echogenicity (20/21, 95.23%), focal hypo-echogenicity (1/21, 4.76%), focal hypechogenicity in the liver parenchyma and around hepatic vessels in two dogs (2/21, 9.52%) (Plate 4.36) indicating fat deposition, rounding of the liver margins (18/21, 85.71%), capsular thickening (3/21, 14.28%) (Plate 4.37), decreased portal vascularity (3/21, 14.28%), increase in size of liver (9/21, 42.85%) and spleen (3/21, 14.28%). Space occupying lesion and cystic lesions in the liver parenchyma was detected 2 (Plate 4.32) and 1 dog (Plate 4.35), respectively. Increase in the size of medial iliac lymph node was visualised in 3 dogs (3/21, 14.28%) (Plate 4.34) and systemic hypertension was observed in 2 dogs (2/21, 9.52%). Biller et al. (1992) found irregular liver margins, focal regenerating nodules and increased parenchymal echogenicity due to deposition of the fibrous tissue in chronic hepatitis. Peri-hepatic changes were observed by Johnson (2000).

The abdominal ultrasonography in the dogs with the cardiac disorders revealed increase in the size of liver (3/6, 50%), hyper-echogenicity of the liver parenchyma (3/6, 50%), presence of anechoic peritoneal fluid (6/6, 100%), round edges of liver lobe margins (2/6, 33.33%) (Plate 4.33) and cystic lesion in the right kidney with merging densities of the renal cortex in 1 dog (1/6, 16.66%). Partington and Biller (1995) asserted that long term passive congestion due to right sided heart diseases cause hepatomegaly, splenomegaly, liver hypo-echogenicity, dilation of hepatic vein and caudal vena cava. The thoracic ultrasonography showed pleural effusion with fibrin strands (2/6, 33.33%).

The ultrasonography of the dogs with renal disorder revealed presence of anechoic peritoneal fluid (6/6, 100%), hyper-echogenicity of the renal cortex (5/6, 83.33%), increase in the size of kidney (2/6, 33.33%) (Plate 4.43), decreased size of kidney (2/6, 33.33%)

(Plate 4.44) increased size of liver (1/6, 16.66%), inflammation of the urinary bladder (2/6, 33.33%), decreased renal perfusion (3/6, 50%), hyper-echogenicity of liver (2/6, 33.33%), increased in size of medial iliac lymph node (1/6, 16.66%), nephroliths (2/6, 33.33%), cystic lesion in the right kidney (1/6, 16.66%), end stage renal disease (2/6, 33.33%). Kumar et al. (2011) reported similar findings in dogs with renal affections, however hydronephrosis, nephritis, nephrocalcinosis were not present in the present study. Squires (2005) and Forrester and Lees (1994) stated that variety of adverse factors (toxins, drug overdose, infectious agents, neoplasia) causes renal damage that could be irreversible or reversible. Phom et al. (2019) and Vijaykumar et al. (2011) reported similar findings in dogs with ascites due to hepato-renal syndrome.

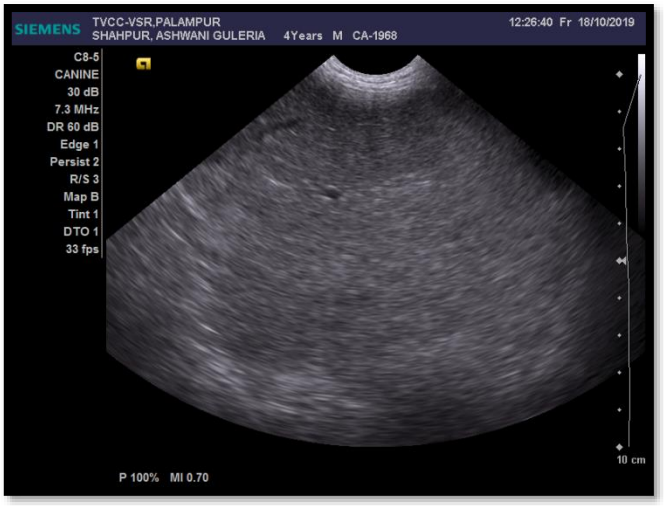
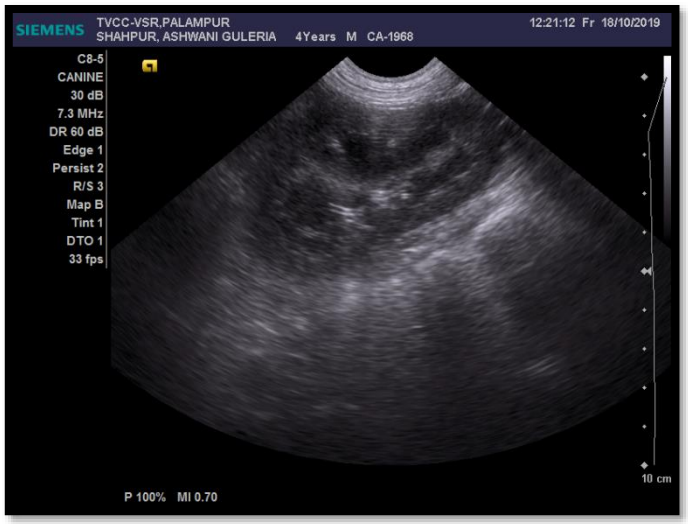
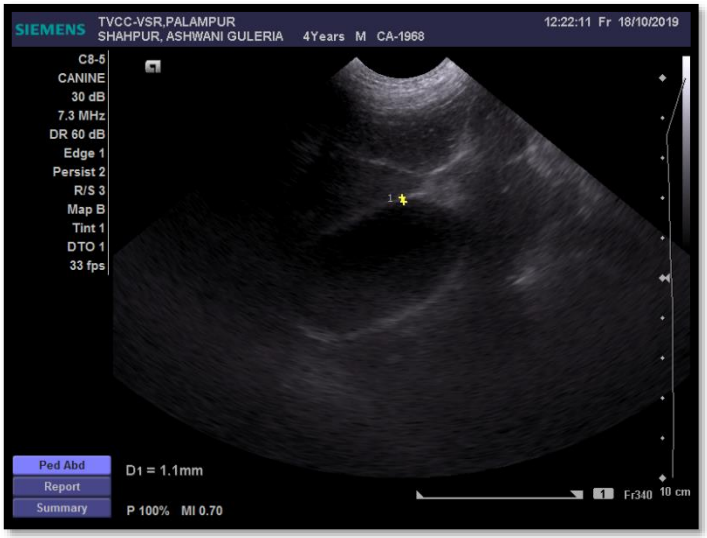
Table 4.28 describes the significant ultrasonographic changes noticed in dogs suffering from ascites due to liver, cardiac and renal disorder along with clinical registration number, clinical findings and ultrasonographic diagnosis. The ultrasonographic changes are also presented in Plates 4.30 to Plate 4.44.

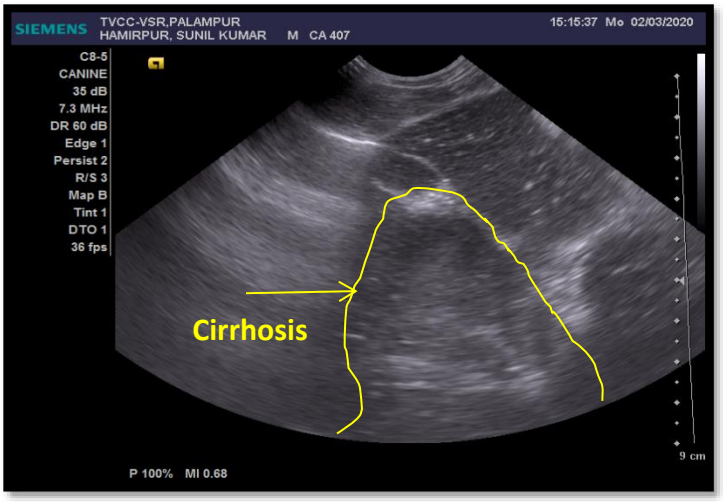
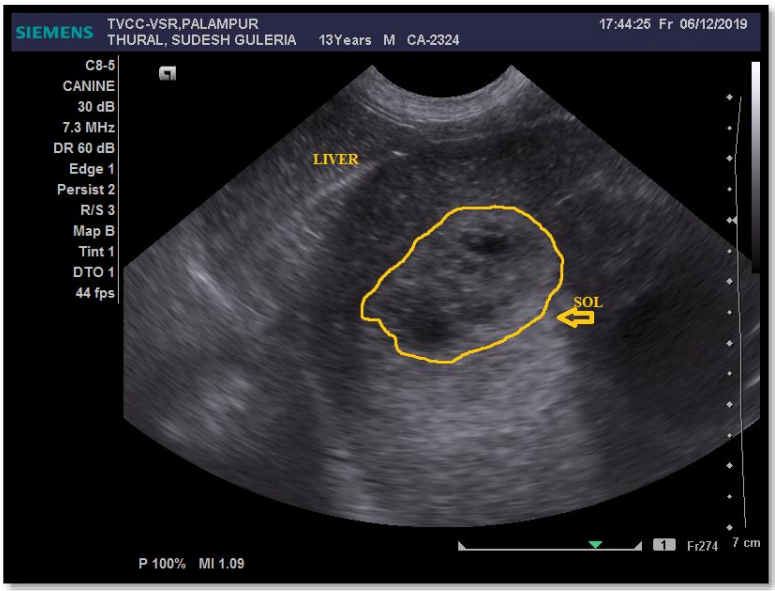
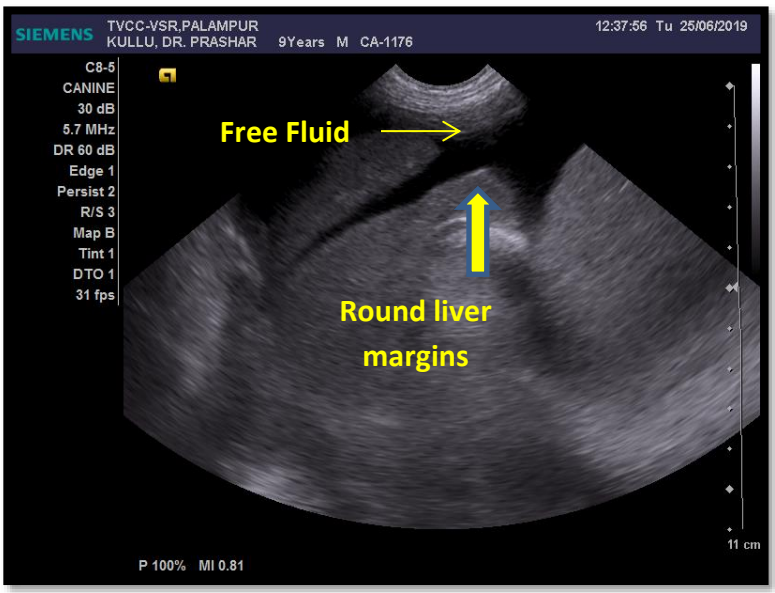
Table 4.28: Ultrasonographic changes with diagnosis of significant findings in dogs suffering from ascites

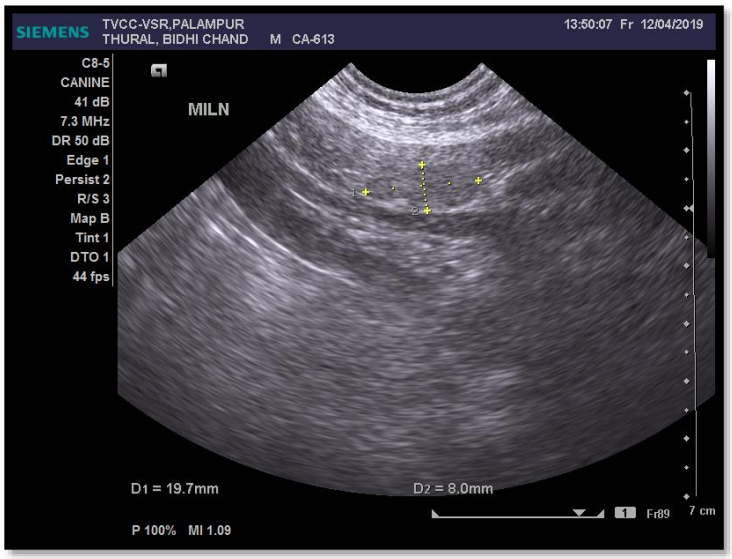
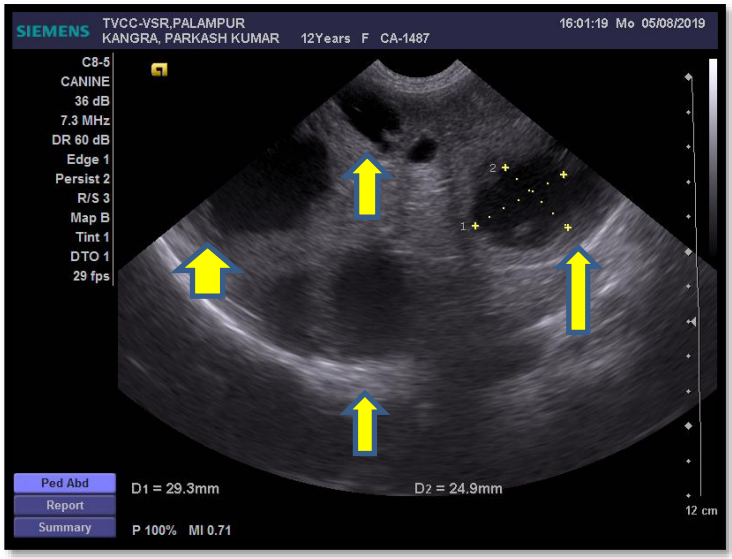
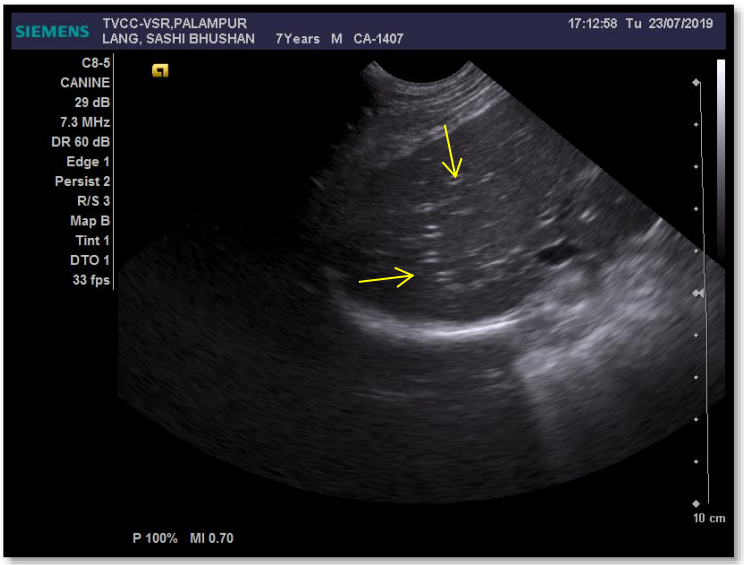
Sr. No.	Case No.	Clinical findings	USG findings	USG diagnosis
1.	CA/1443	Abdominal distension, anorexia, icteric CMM, intense yellow urine	Peritoneum filled with anechoic fluid along with fibrin deposition, Collapsed portal vessels, and enlarged size of liver	Hepatitis leading to cirrhosis
2.	CA/407 (Plate 4.31)	Abdominal distension, exercise intolerance inappetance	Peritoneum having free fluid, increased echogenicity of liver, SOL occupying liver parenchyma (hyperechoic mass)	Cirrhosis
3.	CA/613 (Plate 4.34)	Abdominal distension, inappetance, vomiting, chocolate colour faeces.	Hepatic portal veins dilated, round edges of the liver, thickened capsule of liver, enlarged MILN, free fluid peripheral to liver, enlarged size of spleen	Chronic Hepatitis
4.	CA/1449	Abdominal distension, anorexia, vomiting,	Free fluid at periphery of liver, non- homogenous echotexture of	Chronic Hepatitis

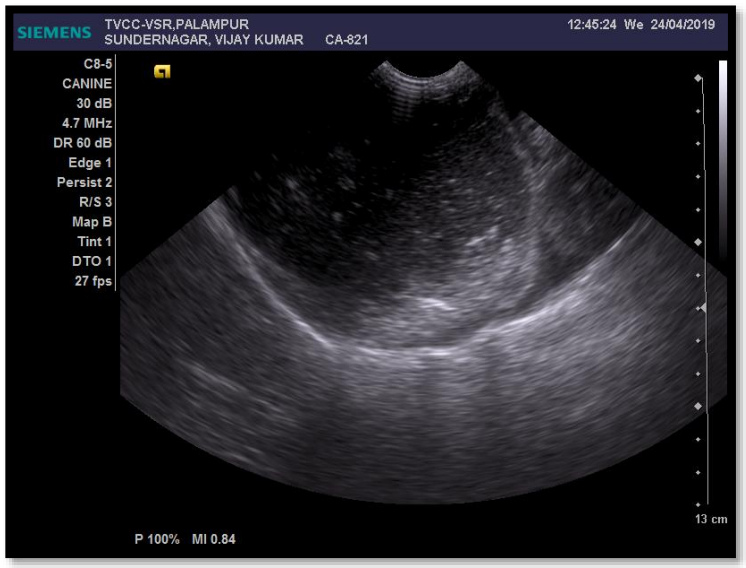
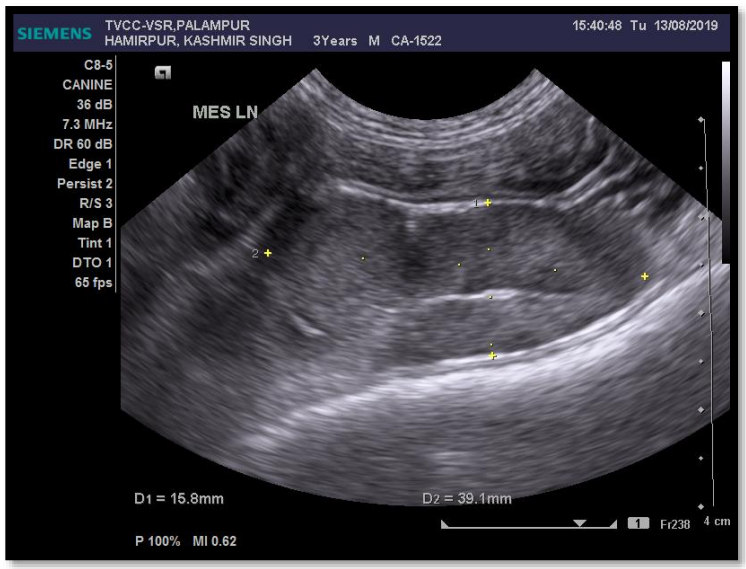
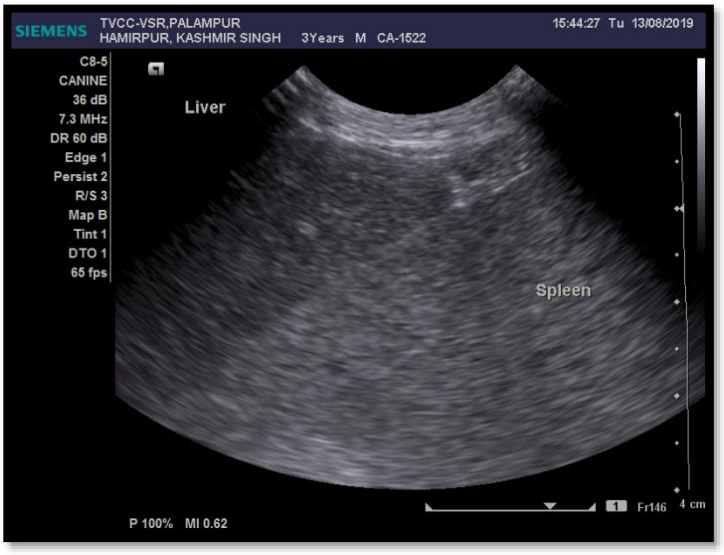
		melena	liver, portal vasculature not visible, reduced size of liver, decreased perfusion of hepatic parenchyma	
5.	CA/1487 (Plate 4.35)	Abdominal distension, exercise intolerance, inappetance, congested CMM	Anechoic cyst in spleen, non-homogenous echotexture of liver lobe, diffused masses and cyst of mixed echogenicity in hepatic parenchyma, masses have no portal and hepatic vascularity.	Chronic Hepatitis
6.	CA/1407 (Plate 4.36)	Abdominal distension, exercise intolerance, inappetance, vomiting, melena	Fatty infiltration of liver, liver parenchyma not visible, altered echotexture, hyperechoic mass on hepatic fossa on cranial part.	Chronic Hepatitis
7.	CA/2326	Vomiting, icteric CMM, diarrhoea, red tinged urine	Severe hepatomegaly, altered echogenicity, round edges, fatty degeneration of right liver lobe, thickened capsule, portal vascularity lost, free fluid around liver lobes	Chronic Hepatitis
8.	CA/821 (Plate 4.37)	Abdominal distension, inappetance, exercise intolerance	Shredded liver ends, free fluid in the peritoneum	Chronic Hepatitis
9.	CA/1522 (Plate 4.38 and 4.39)	Abdominal distension, inappetance, pale CMM	Increase echogenicity of liver parenchyma and periportal vasculature, hyperechoic. Increase echogenicity of spleen, splenomegaly, increased size of MESLN, free fluid in peritoneum	Acute Hepatitis
10.	CA/1979 (Plate 4.40)	Abdominal distension, exercise intolerance, inappetance, melena.	Hepatomegaly, liver edges are round, systemic hypertension, mild inflammatory changes in liver parenchyma, free fluid in peritoneum along with fibrin	Acute Hepatitis

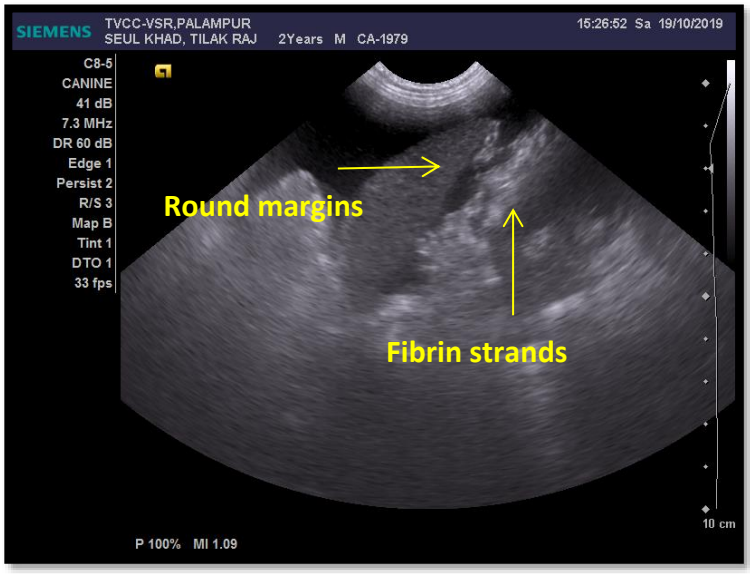
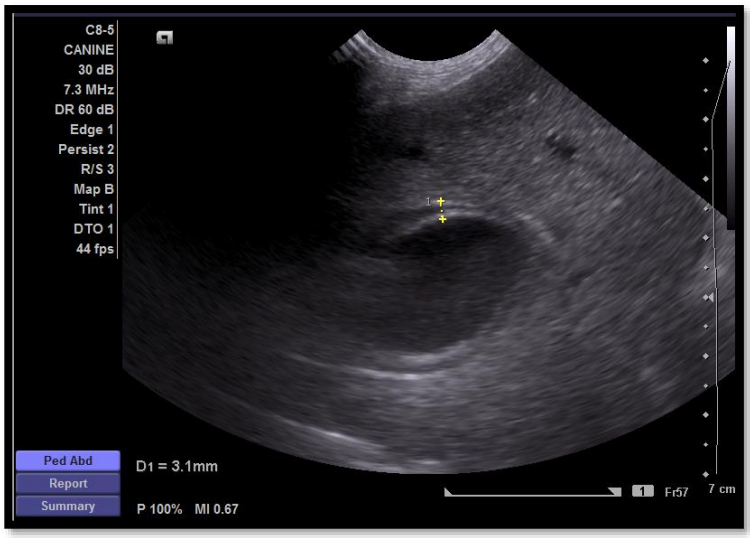
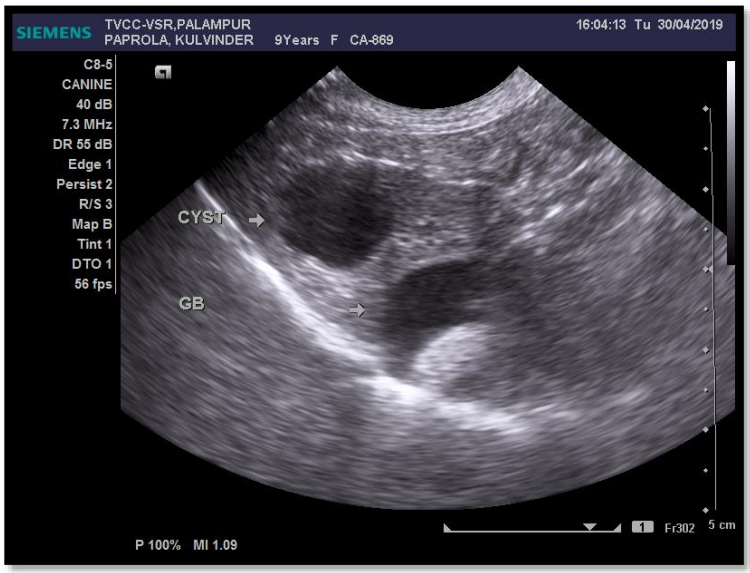
11.	CA/2278	Abdominal distension, exercise intolerance	Free fluid in peritoneum, round edges of liver, thickened capsule, liver echotexture slightly altered, distension of vascular supply in liver, severe hypertension.	Acute Hepatitis
12.	CA/483 (Plate 4.41)	Abdominal distension, inappetance melena	mild rounding of liver edges, presence of sludge in gall bladder, thickening of the gall bladder wall	Cholecystitis
13.	CA/869 (Plate 4.42)	Abdominal distension, exercise intolerance, halitosis, melena	Increased echogenicity of liver parenchyma, multiple fluid filled cystic lesion near gall bladder in liver parenchyma, thickened gall bladder wall	Cholecystitis
14.	CA/1606 (Plate 4.43)	Abdominal distension, exercise intolerance, pale CMM	Increased echogenicity of renal cortex, CMJ clear, increase in size of right kidney, increased perfusion of right kidney, renal aorta collapsed-ESRD, splenomegaly, free fluid in peritoneum	Chronic Renal Failure
15.	CA/2174 (Plate 4.44)	Inappetance, vomiting, melena	Increase in size of left kidney, hyperechoic renal cortex, CMJ non-distinct, diminished perfusion, free fluid in peritoneum	Acute Renal Failure

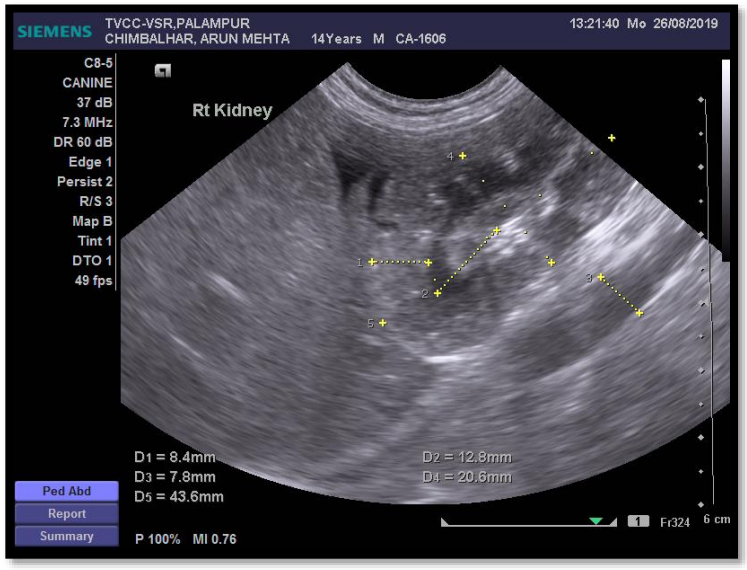
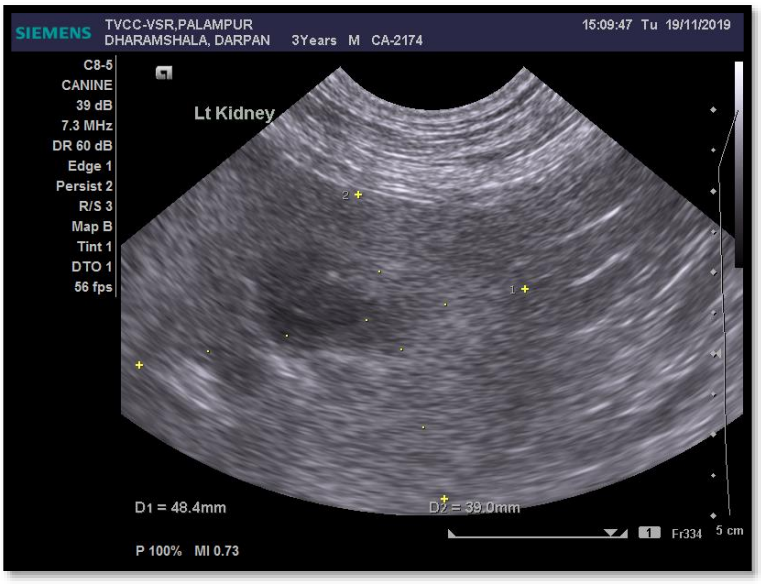
Ultrasounds of healthy dogs	
	**Plate 4.28: Normal echotexture of liver parenchyma in a healthy dog**
	**Plate 4.29: Normal echotexture of renal parenchyma in a healthy dog**
	**Plate 4.30: Normal gall bladder wall thickness in a healthy dog**

Ultrasounds of dogs suffering from ascites	
	**Plate 4.31: Hyperechoic liver lobe in an ascitic dog having liver disorder (Cirrhosis) (CA/407)**
	**Plate 4.32: Space occupying lesion in liver of an ascitic dog having liver disorder (Chronic Hepatitis) (CA/2324)**
	**Plate 4.33: Free fluid around the round margins of the liver in an ascitic dog with cardiac disorder (Dilated Cardiomyopathy) (CA/1176)**

	**Plate 4.34: Increase in the size of Medial Iliac Lymph Node in an ascitic dog having liver disorder (Chronic Hepatitis) (CA/613)**
	**Plate 4.35: Multiple cystic lesions in the liver lobe of an ascitic dogs with liver disorder (Chronic hepatitis) (CA/1487)**
	**Plate 4.36: Fat deposition (Hyperechoic) in liver of an ascitic dog having liver disorder (Chronic Hepatitis) (CA/1407)**

	**Plate 4.37: Chronic liver diseases showing irregular prominent thickening of capsule in an ascitic dog having liver disorder (Chronic Hepatitis) (CA/821)**
	**Plate 4.38: Increase in the size of Mediastina Lymph Node in an ascitic dog having liver disorder (Acute Hepatitis) (CA/1522)**
	**Plate 4.39: Increase echogenicity of the liver in an ascitic dog having liver disorder (Acute Hepatitis) (CA/1522)**

	**Plate 4.40: Rounded margins of the liver lobe and presence of free fluid along with fibrin strands in an ascitic dog having liver disorder (Acute Hepatitis) (CA/1979)**
	**Plate 4.41: Increase in the wall thickness of gall bladder (Double rim effect) (>2mm) in an ascitic dog having liver disorder (Cholecystitis) (CA/483)**
	**Plate 4.42: Cystic lesion in the liver next to gall bladder (presence of sludge) in an ascitic dog having liver disorder (Cholecystitis) (CA/869)**

	**Plate 4.43: Right kidney enlargement in a 10 kg Pomeranian ascitic dog having renal disorder (Chronic Renal Disorder) (CA/1606)**
	**Plate 4.44: Decrease in the size of left kidney in an Ascitic dogs of 35 kgs and hyperechoic renal cortex with indistinct CMJ (Acute Renal Disorder) (CA/2174)**

4.2.10 Therapeutic studies

Among 47 cases of ascites, 35 dogs suffered due to hepatic disorder, 6 suffered with cardiac disorder and 6 suffered renal disorder. All the affected dogs were treated accordingly as described in section 3.9. Recovery was observed in 15 dogs among them 13 were having liver disorders.

4.2.10.1 Therapeutics for liver disorder

Silymarine as tablet (Tab Hepa 20) with combination of other liver supportive, along with diuretics, liver safe antibiotics and vitamin B complex were prescribed. The detailed therapeutic regime is mentioned in section 3.9.1. Blood transfusion, fluid therapy,

amino acid infusion, hemaccel and treatment for haemoprotozoan were done where ever required.

4.2.10.1.1 Cirrhosis

Out of the 4 ascitic dogs that suffered from cirrhosis, only one dog showed signs of improvement whereas the abdominal distension was reduced in 2 dogs; however refractory ascites and increase in creatinine level were observed in other two dogs. This could be due to dysfunction of hemodynamics that results in refractory ascites and hepato-renal syndrome (HRS) and these are independent predictor of short survival time of the animal (Salerno et al. 2010).

The pre and post treatment values of clinical parameters, haematology and biochemical are represented in Table 4.29, 4.30 (Figure 4.27) and 4.31 (Figure 4.28, 4.29 and 4.30), respectively. Rectal temperature (101.6 ^{0}F), heart rate (95 beats per minute) and respiration rate (24 breaths per minute) were within normal range. Reduction in abdominal distension was observed at day 15 of post-treatment. The total leukocyte count decreased from 26.7 $\times 10^9$/L to 15.9 $\times 10^9$/L at day 90 of post treatment. Moderate anaemia (5.7 g/dl) was observed at the day of presentation but at day 90 of post treatment a considerable increase in haemoglobin (12.9 g/dl) was observed (Table 4.30 and Figure 4.27). The post treatment values of ALP and GGT were 60.93 U/L and 2.56 U/L, respectively, that decreased significantly ($P<0.05$) from pre-treatment values, 125 U/L and 6.9 U/L, respectively. The protein profile also improved; mild hypoproteinemia was observed on day 0 of presentation (4.71 g/dl) and on day 90 the protein level (6.78 g/dl) were within the normal range (Figure 4.29). The values of total and differential bilirubin also decreased after treatment (Table 4.31 and Figure 4.30).

4.2.10.1.2 Chronic hepatitis

Among 21 dogs that had chronic hepatitis, 9 dogs showed recovery. Reduction in the moderate abdominal distension was noticeable on day 15 of the treatment and in severe distension, the reduced abdomen was appreciable on day 30 of post treatment along with positive change towards recovery in haemato-biochemical profile.

Icteric mucous membrane and hyperbilirubinemia was reported in 4 dogs on day 0 and on day 30, reduction in the total bilirubin was observed. Ursodexoycholic acid (Tab Ursolid) was prescribed in dogs with hyperbilirubinemia along with oral liver supportive

supplements and hepato-protectants. Haematinics were prescribed in dogs with mild to moderate anaemia and protein supplements with protein rich diet and restriction to salt was advised.

Haemoprotozoan (Babesiosis) was confirmed in 4 dogs and they were treated with Imidocarb injection and doxycycline along with supportive therapy comprising platelet enhancer and haematinics as described in section 3.9.4.

The pre and post treatment values of clinical parameters, haematology and biochemical of dogs with chronic hepatitis are represented in Table 4.29, 4.30 (Figure 4.27) and 4.31 (Figure 4.28, 4.29 and 4.30), respectively. The heart rate that was significantly increased on day 0 of presentation decreased significantly after 90 days post treatment. No significant difference was observed in respiration rate in pre and post treatment; the respiration rate was significantly higher in affected dogs than the healthy dogs however, was in normal range. A significant decrease in total leukocyte count ($12.24 \pm 2.48 \times 10^9$/L) was observed on day 90 after treatment as compared to the day 0 of presentation ($29.9 \pm 9.54 \times 10^9$/L) (Table 4.30, Figure 4.27). The other haematological parameters were non-significant and were comparable with the normal healthy dogs. A significant decrease in ALT (45.27 ± 2.76 U/L to 32.31 ± 2.92 U/L) and ALP activities (71.91 ± 14.10 U/L to 53.10 ± 6.59 U/L) was noticed after 90 days of post treatment (Table 4.31, Figure 4.28). There was significant improvement in the post treatment total protein values that were increased from 5.3 ± 0.66 g/dl at day 0 of presentation to 6.58 ± 0.44 g/dl at day 90 of presentation. The indirect bilirubin also decreased significantly from 1.08 ± 0.31 mg/dl (Figure 4.31) as compared to mean pre-treatment value (0.45 ± 0.11 mg/dl) and was comparable with healthy dogs (0.18 ± 0.04 mg/dl). The post-treatment cholesterol level (158.21 ± 12.50 mg/dl) was also comparable with the healthy dogs (163.38 ± 4.93 mg/dl).

4.2.10.1.3 Acute hepatitis

Out of 6 ascitic dogs that suffered of acute hepatitis, three dogs recovered. One of the recovered dog had haemoprotozoan (*Ehrlichia* spp.), that was detected with the help of quick vet *Ehrlichia* kit. The dog was treated for haemoprotozoan along with hepatic condition. Moderate anaemia was observed in one dog and mild hyperbilirubinemia in another dog.

The mean values of pre and post treatment along with healthy dogs for clinical parameters, haematology and biochemical of dogs with acute hepatitis is presented in Table 4.29, 4.30 (Figure 4.27) and 4.31 (Figure 4.28, 4.29 and 4.30), respectively. The mean respiration rate varied significantly between the pre-treatment (27.66 ± 1.85 breaths per minute) and post- treatment (20.33 ± 2.33 breaths per minute) groups. The mean total leukocyte count decreased significantly from 28.56 ± 8.51 to 13.09 ± 2.35 $\times 10^9$/L on day 30 of post treatment. Significant difference was noticed in platelet count but was in normal range. The mean values of ALT, AST and ALP was 40.33 ± 3.17, 45.76 ± 2.47 and 59.00 ± 4.93 U/L that decreased on day 30 of post treatment from 121.00 ± 10.69, 129.00 ± 4.58 and 116.00 ± 3.05 U/L on day 0 of presentation. There was significant increase in glucose level from 81.33 ± 4.91 mg/dl to 99.26 ± 11.89 mg/dl at day 30 of post treatment. Post-treatment values were comparable to the values of the healthy dogs.

4.2.10.1.4 Cholecystitis

All the 4 ascitic dogs that suffered from cholecystitis showed no signs of improvement. Three dogs died within 15-20 days of presentation and one dog died shortly after the day of presentation. The surgery being the only permanent treatment for the condition could not be performed due to unstable condition of the dogs.

Table 4.29: Pre and Post treatment clinical parameters of ascitic dogs suffering with liver disorders (Mean ± S.E.)

Parameters	Healthy dogs (n=10)	Cirrhosis (n=1)		Chronic hepatitis (n=9)		Acute hepatitis (n=3)	
		Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
Rectal temperature (^{0}F)	101.15 ± 0.29	102	101.6	101.63 ± 0.44	101.35 ± 0.20	101.66 ± 0.71	101.35 ± 0.20
Heart rate (beats per minute)	88.7 ± 2.08^a	101	95	105.55 ± 7.13^b	96.66 ± 0.88^{ab}	94.66 ± 2.40^a	89.00 ± 2.64^a
Respiration rate (breaths per minute)	17.9 ± 0.55^a	29	24	28.22 ± 1.09^b	20.88 ± 0.99^b	27.66 ± 1.85^b	19.33 ± 2.33^b

Values with different superscript in a row differ significantly (P<0.05) in each group

Table 4.30: Pre and Post treatment haematological profile of ascitic dogs with liver disorders (Mean ± S.E.)

Parameters	Healthy dogs (n=10)	Cirrhosis (n=1)		Chronic hepatitis (n=9)		Acute hepatitis (n=3)	
		Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
TLC (× 10^9/L)	10.69 ± 0.56^{a}	26.7	15.9	29.9 ± 9.54^{b}	12.24 ± 2.48^{ab}	28.56 ± 8.51^{b}	13.09 ± 2..35^{a}
L%	17.08 ± 2.11	17.6	2.58	20.53 ± 5.31	16.84 ± 3.35	10.06 ± 5.03	18.16 ± 3.05
M%	3.94 ± 0.29	3.5	1.8	3.61 ± 0.27	3.84 ± 0.56	2.93 ± 1.11	2.76 ± 0.76
G%	75.34 ± 2.60	78.9	56.4	75.85 ± 5.47	60.15 ± 2.65	84.86 ± 8.95	88.64 ± 3.69
TEC (× 10^{12}/L)	6.81 ± 0.25^{b}	2.33	5.2	4.88 ± 0.87^{a}	6.52 ± 0.60^{b}	4.81 ± 0.53^{a}	5.13 ± 1.57^{b}
Hb (g/dl)	12.87 ± 0.42^{b}	5.7	12.9	10.54 ± 1.88^{a}	14.1 ± 1.39^{b}	9.53 ± 1.16^{a}	10.81 ± 3.17^{b}
HCT %	39.05 ± 0.96	21.5	38.2	27.96 ± 5.42	35.5 ± 1.65	26.66 ± 3.20	35.4 ± 9.35
MCV (fl)	58.38 ± 0.87	78.5	56.3	61.73 ± 4.70	61.01 ± 2.67	52.53 ± 2.26	55.86 ± 8.71
MCH (pg)	19.02 ± 0.49	24.4	21.2	25.88 ± 4.10	21.48 ± 1.13	18.63 ± 0.80	20.06 ± 3.78
MCHC (g/dl)	31.63 ± 0.39	30	33.6	32.86 ± 0.69	35.52 ± 1.32	35.83 ± 0.98	34.63 ± 2.32
PLT (× 10^9/L)	201.90 ± 8.75^{a}	185	210	189.77 ± 14.88^{a}	210.77 ± 11.32^{a}	218.66 ± 4.48^{a}	250.00 ± 5.03^{b}

Values with different superscript in a row differ significantly (P<0.05) in each group

Table 4.31: Pre and Post treatment plasma biochemical profile of ascitic dogs with liver disorders (Mean ± S.E.)

Parameters	Healthy dogs (n=10)	Cirrhosis (n=1)		Chronic hepatitis (n=9)		Acute hepatitis (n=3)	
		Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
ALT (U/L)	36.12 ± 4.29^{a}	42.36	45.2	45.27 ± 2.76 ^{ab}	32.31 ± 2.92^{a}	121 ± 10.69^{b}	40.33 ± 0.48^{a}
AST (U/L)	31.77 ± 4.32^{a}	32.2	38.6	71.91 ± 4.10^{b}	53.10 ± 6.59^{a}	129 ± 4.58^{b}	45.76 ± 2.47^{a}
ALP (U/L)	57.26 ± 8.23^{a}	125	60.93	144.66 ± 16.10^{b}	60.66 ± 3.80^{a}	116 ± 3.05^{b}	59.00 ± 4.93^{a}
GGT (U/L)	2.27 ± 0.18	6.9	2.56	3.83 ± 1.11	2.14 ± 0.57	2.55 ± 0.28	1.86 ± 0.33
Total bilirubin (mg/dl)	0.25 ± 0.04^{a}	1.59	0.76	1.65 ± 0.52^{b}	0.75 ± 0.16^{a}	0.63 ± 0.20^{a}	0.77 ± 0.12^{a}
Direct bilirubin (mg/dl)	0.07 ± 0.01	0.98	0.61	0.56 ± 0.25	0.30 ± 0.08	0.43 ± 0.16	0.45 ± 0.12
Indirect bilirubin (mg/dl)	0.18 ± 0.04^{a}	0.61	0.15	1.08 ± 0.31^{b}	0.45 ± 0.11^{a}	0.19 ± 0.09^{a}	0.65 ± 0.35^{a}
Total protein (g/dl)	6.68 ± 0.12^{b}	4.71	6.78	5.3 ± 0.66^{a}	6.58 ± 0.44^{b}	4.83 ± 0.46^{a}	6.30 ± 0.48^{b}
Albumin (g/dl)	3.71 ± 0.17	3.67	4.92	3.58 ± 0.57	4.61 ± 0.42	3.95 ± 0.13	4.33 ± 0.28
Globulin (g/dl)	2.97 ± 0.18^{b}	1.04	1.86	1.53 ± 0.40^{a}	1.96 ± 0.20^{a}	0.88 ± 0.32^{a}	1.96 ± 0.29^{b}
A/G ratio	1.30 ± 0.12^{a}	3.52	2.64	3.57 ± 0.92^{b}	2.59 ± 0.40^{a}	5.92 ± 2.04^{b}	2.28 ± 0.28^{a}
BUN (mg/dl)	14.47 ± 1.22^{a}	35	34.49	26.08 ± 1.82^{b}	16.96 ± 2.70^{a}	23.64 ± 1.69^{b}	19.46 ± 3.10^{a}
Creatinine (mg/dl)	1.03 ± 0.15	0.84	0.56	1.27 ± 0.33	0.84 ± 0.12	0.7 ± 0.05	0.65 ± 0.13
Glucose (mg/dl)	108.15 ± 2.82^{b}	86	99.21	84.16 ± 10.45^{a}	106.32 ± 12.17^{b}	81.33 ± 4.91^{a}	99.26 ± 11.89^{b}
Cholesterol (mg/dl)	163.38 ± 4.93^{a}	98	56	180.42 ± 17.13^{b}	158.21 ± 12.50^{a}	233.33 ± 10.41^{b}	165.33 ± 8.95^{a}

Values with different superscript in a row differ significantly (P<0.05) in each group

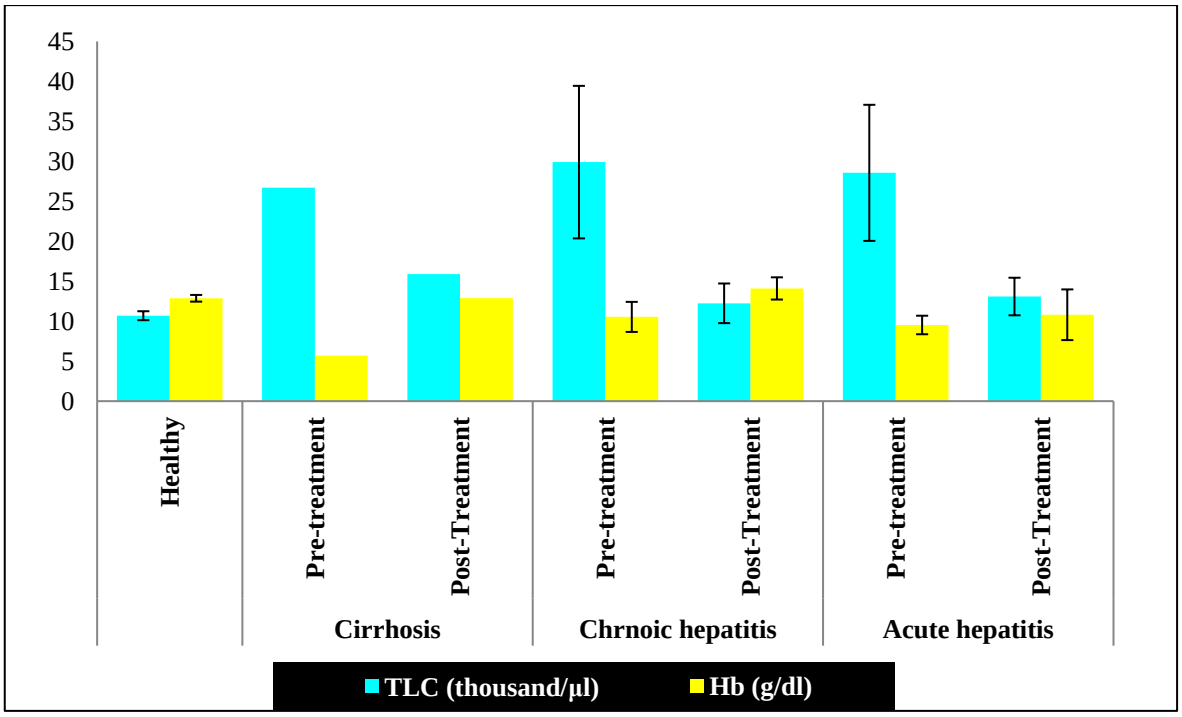


Figure 4.27: Pre and post treatment haematological profile of ascitic dogs with liver disorders (Mean ± S.E.)

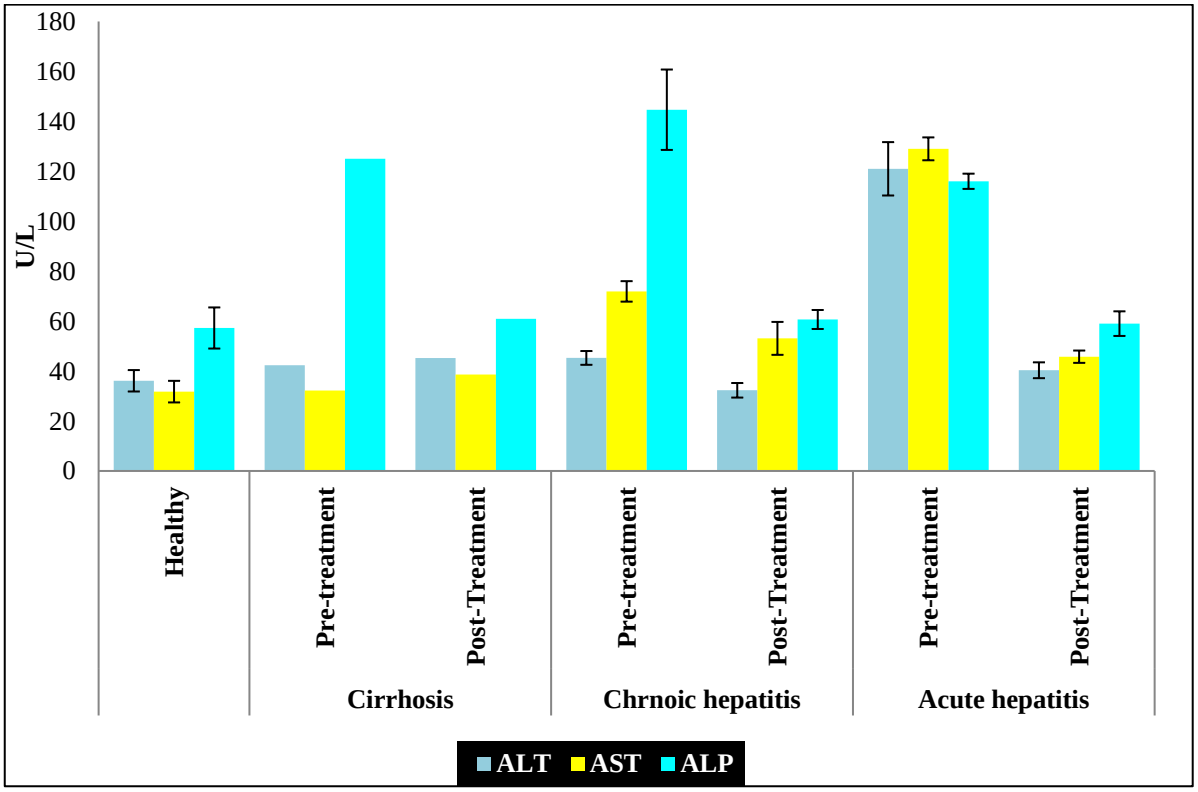


Figure 4.28: Pre and post treatment plasma biochemical profile (ALT, AST and ALP) of ascitic dogs with liver disorders (Mean ± S.E.)

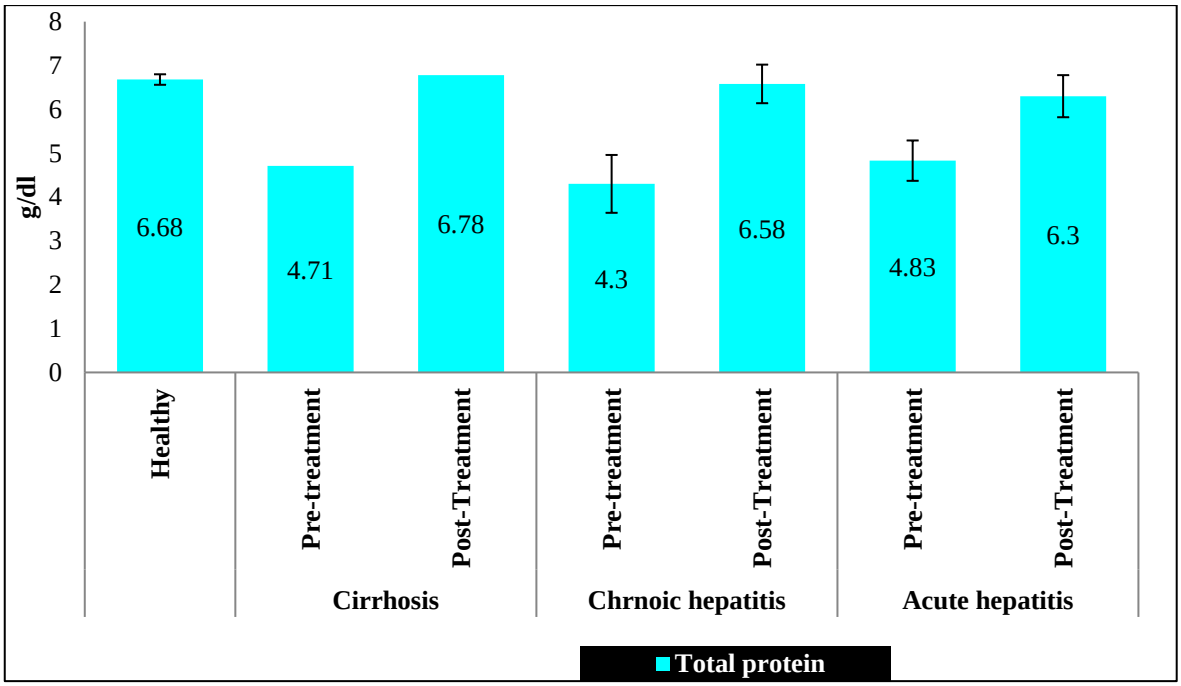


Figure 4.29: Pre and post treatment plasma protein profile of ascitic dogs with liver disorders (Mean ± S.E.)

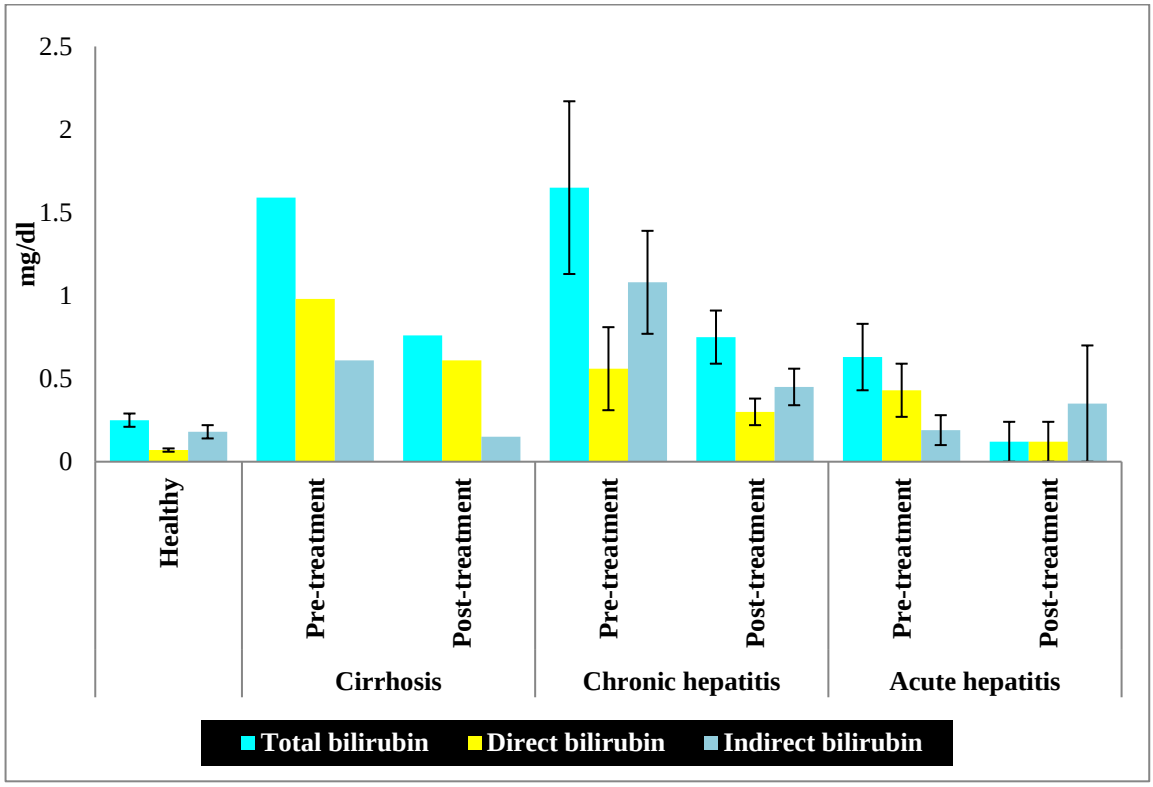


Figure 4.30: Pre and Post treatment total and differential bilirubin of ascitic dogs with liver disorders (Mean ± S.E.)

4.2.10.2 Cardiac diseases

Of the six dogs that suffered with cardiac disorders, only one dog showed improvement from ascites with reduction in the abdominal distension. The study by Monnet et al. (1995) suggested that in cardiomyopathy, ascites and pleural effusion were poor prognostic indicators. The survival rate in the present study is low that could be due to presence of ascites in all the animals and pleural effusion in 2 animals.

The dogs with abdominal distension and pleural effusion were treated with diuretics (Fruselac DS) and digoxin along with angiotensin converting enzyme inhibitor (Enalapril) and other supportive treatment. The detailed therapeutic regime is listed in section 3.9.2.

4.2.10.2.1 Cardiomyopathy

The dog was presented with complain of abdominal distension and laboured breathing and inappetance. The heart rate decreased from 120 beats per minute to 105 beats per minute and no tachycardia or arrhythmia was recorded on day 90 of post treatment. The haemato-biochemical parameters were almost in normal range, the packed cell volume decreased from 46.8% to 28.69% with normal TEC (6.26 $\times 10^{12}$/L) and haemoglobin (14.5 g/dl) at day 90 of presentation the levels of ALP also decreased from 184 U/L to 120 U/L with normal ALT (48 U/L) and AST (39 U/L). Total protein level increased from 4.65 g/dl to 6.94 g/dl at day 90 of post treatment. The dog was treated with digoxin, Enalapril, liver supplements, antibiotics and diuretics. The protein rich diet and restriction to salt was also advised. The pre and post values of clinical parameters, haematology and biochemical are presented in Tables 4.32 to 4.34.

4.2.10.3 Renal diseases

Six dogs had ascites due to renal disorder but one among the six recovered from the condition. The dogs with renal diseases were prescribed with regular fluid therapy for 5 days and depending on the condition the fluid therapy was continued for another 5 days. Diuretic was prescribed along with other supportive therapy. The detailed therapeutic regime is presented in section 3.9.3.

4.2.10.3.1 Acute renal disorder

The ascitic dog that recovered had acute renal disorder. The dog was presented with complain of inappetance, vomiting and intense yellow coloured urine and melena. The dog

was prescribed with fluid therapy, diuretic, antiemetic (Ondansetron), haemostats (Ethamsylate) and liver supplements. The abdominal distension was markedly reduced on day 15 of post treatment. The detailed pre and post treatment values of clinical parameters, haemato-biochemical analysis of the recovered dog are presented in the Table 4.32 to 4.34.

The total leukocyte count decreased from 20.1 to 12.87×10^9/L and the haemoglobin levels were increased from 8.9 g/dl to 12.68 g/dl (Table 4.33). The levels of BUN and creatinine decreased markedly from 98 mg/dl and 4.65 mg/dl to 84 mg/dl and 1.65 mg/dl, respectively at day 30 of presentation. Improvement in the values of ALP, Total protein, GGT were observed at day 30 of post treatment (Table 4.34).

Table 4.32: Pre and Post treatment clinical parameters of dogs with renal and cardiac disorders

Parameters	Cardiac (n=1)		Renal (n=1)	
	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
Rectal temperature (^{0}F)	100.4	101.9	102.4	101.9
Heart rate (beats per minute)	120	105	98	102
Respiration rate (breaths per minute)	32	28	34	25

Table 4.33: Pre and Post treatment haematological profile of ascitic dogs with cardiac and renal disorders

Parameters	Cardiac (n=1)		Renal (n=1)	
	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
TLC ($\times10^9$/L)	14.7	12.54	20.1	12.8
L%	11	5.46	16.5	4.7
M%	3.1	6.8	3.9	2.1
G%	85.9	64.1	79.6	47.1
TEC ($\times10^{12}$/L)	8.63	6.26	5.19	6.54

Hb (g/dl)	15.4	14.5	8.9	12.68
HCT %	46.8	28.69	24.3	34.4
MCV (fl)	54.3	50.21	47	46.9
MCH (pg)	17.8	23.4	17.1	16.5
MCHC (g/dl)	32.9	38.1	36.6	27.9
PLT ($\times 10^9$/L)	185	225	210	250

Table 4.34: Pre and Post treatment plasma biochemical profile of ascitic dogs with cardiac and renal disorders

Parameters	Cardiac (n=1)		Renal (n=1)	
	Pre-treatment (Day 0)	Post-treatment (Day 90)	Pre-treatment (Day 0)	Post-treatment (Day 30)
ALT (U/L)	20	48	46	45
AST (U/L)	37	39	51	50
ALP (U/L)	184	120	120	115
GGT (U/L)	1.6	1.2	4.2	1.4
Total bilirubin (mg/dl)	0.59	0.54	089	1.68
Direct bilirubin (mg/dl)	0.12	0.34	0.24	0.79
Indirect bilirubin (mg/dl)	0.47	0.2	0.65	0.89
Total protein (g/dl)	4.65	6.94	5.96	6.3
Albumin (g/dl)	2.06	4.62	2.8	3.62
Globulin (g/dl)	4.44	2.32	3.16	2.68
A/G ratio	0.46	1.99	0.88	1.38
BUN (mg/dl)	45	40	98	84
Creatinine (mg/dl)	1.2	1.54	4.65	1.65
Glucose (mg/dl)	110	120	106.5	99.86
Cholesterol (mg/dl)	185	145	140	112

4.11 THERAPEUTIC EFFICACY

The observed recovery rate of dogs suffering from ascites due to liver, cardiac and renal disorders was 31.91%. Out of 35 dogs suffering from ascites due to liver disorder 13 dogs (13/35, 37.14%) showed recovery post treatment and one dog suffering ascites due to cardiac (1/6, 16.66%) and one dog suffering ascites due to renal disorder (1/6, 16.66%) showed signs of recovery. Out of 13 recovered dogs that were affected with liver disorder; 9 recovered from chronic hepatitis (9/21, 42.85%), three recovered from acute hepatitis (3/6, 50%) and one dog recovered from cirrhosis (1/4, 25%). The low rate of recovery could have been due to the involvement of other vital organs along with the main etiological organ that caused the ascites. Six ascitic dogs with liver disorder showed cardiac (2) and renal involvement (4) with time. Ginès et al. (2003) reported that the most common clinical condition involved in hepato-renal syndrome was ascites. Two ascitic dogs with cardiac disorders and three ascitic dogs with renal disorders showed involvement of liver dysfunction. The prognosis was grave in dogs with renal disorders on involvement of liver or biliary tract disease (Nothanaget 1899). Refractory ascites was observed in ten dogs, five dogs were having liver disorder, two were having cardiac disorder and three dogs were having renal disorder. The refractory ascites and Hepato-Renal Syndrome (HRS) is independent predictor of short survival time of animal (Salerno et al. 2010).

The chronic condition of the organs involved in dogs suffering from ascites could also have been the reason of low recovery rate. The ultrasonography showed chronic condition of the liver, cardiac and kidneys in majority of the cases. Deposition of the fibrous tissue in the liver parenchyma, cardiomyopathy, mixing of the blood in the heart, end stage renal disorder that were identified on ultrasonography along with cystic and space occupying lesions that could possibly have been tumorous mass revealed that the condition was chronic form; thus, the prolonged illness resulted in deterioration of the clinical picture and dysfunction in the circulatory system.

Summary and Conclusions

CHAPTER 5 SUMMARY AND CONCLUSIONS

The present study was conducted on dogs presented in the Clinics of Dr. G. C. Negi College of Veterinary and Animal Sciences, Palampur, between October 2018 to March 2020. During this period 2063 cases of dogs were presented in the Department of Veterinary Medicine. On the basis of history and clinical examination; cases of ascites were studied. In this way, a total of 47 dogs suffering from ascites due to different aetiologies were included in this study, thus representing an incidence of ascites as 2.27 per cent. Complete haematology (TLC, DLC, TEC, Hb, PCV, MCV, MCH, MCHC and PLT), plasma biochemical analysis (ALT, AST, ALP, GGT, Total and Differential Bilirubin, Total protein, Albumin, BUN, creatinine, Cholesterol and Glucose), ascitic fluid analysis (Total protein, Albumin, SAAG), Urine analysis (Physical and chemical examination), cytology of urine and ascitic fluid was carried out along with electrocardiography, radiography and ultrasonography.

Out of 47 ascitic dogs, 35 dogs (35/47, 74.46%) had ascites due to liver disorders, 6 due to cardiac (6/47, 12.76%) disorders and 6 due to renal (6/47, 12.76%) disorders. Among the liver disorders, chronic hepatitis formed the largest group with 21 ascitic dogs (21/35, 60%) followed by acute hepatitis in 6 dogs (6/35, 17.14%), cirrhosis in 4 dogs (4/35, 11.42%) and cholecystitis with 4 ascitic dogs (4/35, 11.42%). Cardiomyopathy was diagnosed in five dogs (5/6, 83.33%), pleural effusion in 2 dogs (2/6, 33.33%) and pericardial effusion in 2 dogs (2/6, 33.33%). The chronic renal failure was observed in 2 dogs (2/6, 33.33%) and 4 dogs had acute renal failure (4/6, 66.66%).

The age distribution of the ascitic dogs revealed that majority of the dogs (23/47, 48.93%) were of young age group (1-4 years), followed by middle (4-7 years) age group (10/47, 21.72%), geriatric age (>7 years) group (9/47, 19.14%) and least by <1 year age group (5/47, 10.63%). The majority of the animals having ascites due to liver disorder were in the young age group (17/35, 48.57%) followed by middle age group (8/35, 22.85%), <1 year (5/35, 14.28%) and geriatric group (5/35, 14.28%). 50% (3/6) of the ascitic dogs with cardiac disorder were of young age group, 33.33% (2/6) were of >7 year and 16.66% (1/6) was of <1 year. Dogs of 1-4 years of age were more affected with ascites due to renal disorders (3/6, 50%) followed by >7 years (2/6, 33.33%) and least by 4-7 years (1/6, 16.66%).

It was observed that male dogs (36/47, 76.59%) suffered more than females (11/47, 23.40%) from ascites. Sex predisposition revealed that 74.28 per cent male dogs (26/35) and 25.71 per cent female dogs (9/35) suffered from ascites due to liver disorders. Similarly, male dogs suffered more from cardiac disorders (5/6, 83.33%) than the females (1/6, 16.66%) and renal disorders (5/6, 83.33%) than female dogs (1/6, 16.66%). Of the twenty one dogs that suffered chronic hepatitis fourteen (14/21, 66.66%) were males and seven (7/21, 33.33%) were females.

The breed distribution showed that Labrador retriever was the most commonly affected breed (14/47, 29.78%) followed by German shepherd (7/47, 14.89%), Non-descript (7/47, 14.89%), Pomeranian (6/47, 12.76%), Pug (3/47, 6.38), Golden retriever (3/47, 6.38%). The Labrador retriever breed had higher incidence of ascites due to liver (9/14), cardiac (3/14) and renal (2/14) disorders. German shepherd (4/7), Non-descript (6/7), Pomeranian (5/6), Golden retriever (3/3), Pug (2/3), Pitbull (2/2), Rottweiler (1), Pointer (1), Dalmatian (1), Gaddi (1) were the breed affected with ascites due to liver disorder. German shepherd (1/7), Non-descript (1/7) and Pug (1/3) were affected with ascites due to cardiac disorders. German shepherd (2/7) and Pomeranian (1/6) were affected with ascites due to renal disorders.

The common clinical signs that were noticed in ascitic dogs were abdominal distension, inappetance, and anorexia and exercise intolerance. Icteric mucous membrane, (6/35, 17.14%), pale mucous membrane (7/35, 20%), ecchymosis (5/35, 14.28%), weight loss (5/35, 14.28%), pyrexia (3/35, 8.57%), melena (13/35, 37.14%), engorged blood vessels of abdomen (3/35, 8.57%), enlarged lymph nodes (9/35, 25.71%) were the signs noticed in ascitic dogs with liver disorders. Muffled heart sounds (3/6, 50%), tachycardia (2/6, 33.33%), pale conjunctival mucous membrane (3/6, 50%) and pyrexia (2/6, 33.33%) were the signs noticed in ascitic dogs with cardiac disorders. Clinical signs noticed in ascitic dogs with renal disorders were vomiting (2/6, 33.33%), halitosis (2/6, 33.33%) and pale conjunctival mucous membrane (2/6, 33.33%).

The clinical parameters showed significant ($P<0.05$) increase in heart rate and respiration rate in ascitic dogs with liver, cardiac and renal disorders, 103.65 ± 2.91 beats per minute and 29.91 ± 0.07 breaths per minute, 115.83 ± 3.00 beats per minute and 30.50 ± 1.33 breaths per minute and 104.16 ± 3.30 beats per minute and 31.00 ± 1.23 breaths per minute, respectively. The rectal temperature of the ascitic dogs remained non-significant.

The haematological profile revealed significant decrease (P<0.05) in TEC values in ascitic dogs having liver (5.21 ± 0.35 $\times 10^{12}$/L) and renal (5.71 ± 0.86 $\times 10^{12}$/L) disorders. The TLC and granulocytes were significantly (P<0.05) increased in liver (30.84 ± 4.00 $\times 10^{9}$/L and 81.55 ± 2.43%) and renal (18.23 ± 3.27 $\times 10^{9}$/L and 82.05 ± 5.14%) disorders. A significant decrease in TEC was observed in ascitic dogs with cirrhosis (3.92 ± 0.80 $\times 10^{12}$/L), chronic hepatitis (5.67 ± 0.48 $\times 10^{12}$/L), acute hepatitis (4.37 ± 0.52 $\times 10^{12}$/L) and cholecystitis (5.64 ± 1.35 $\times 10^{12}$/L). The TLC and MCH were significantly increased (p<0.01) in all the liver disorder groups; cirrhosis (28.9 ± 5.31 $\times 10^{9}$/L and 21.92 ± 1.36 pg), chronic hepatitis (29.12 ± 5.27 $\times 10^{9}$/L and 22.77 ± 1.83 pg), acute hepatitis (40.35 ± 8.93 $\times 10^{9}$/L and 27.51 ± 8.72 pg) and cholecystitis (20.72 ± 5.03 $\times 10^{9}$/L and 21.85 ± 0.99 pg). The PCV was decreased (P<0.05) in dogs with cirrhosis (24.00 ± 3.66%), chronic hepatitis (31.92 ± 2.75%) and acute hepatitis (24.93 ± 2.97%).

The plasma biochemical analysis revealed no statistical significance in ALT levels. A significant (P<0.01) increase in levels of AST and ALP was observed in liver (83.43 ± 6.53 U/L and 131.57 ± 5.57 U/L), cardiac (66.50 ± 5.58 U/L and 140.33 ± 20.29 U/L) and renal (45.75 ± 4.57 U/L and 125.5 ± 9.44 U/L) disorders. The total and differential bilirubin was significantly (P<0.05) increased in liver and renal disorders. The levels of BUN and creatinine were significantly (P<0.01) increased in dogs with renal disorders (106.83 ± 9.71 mg/dl and 6.08 ± 1.62 mg/dl). Hypoglycaemia was evident in dogs with liver disorders (83.16 ± 3.70 mg/dl). Among the liver disorders groups, activity of ALT was significantly increased (P<0.05) in acute hepatitis (112.03 ± 6.55U/L) and cholecystitis (51.66 ± 7.31U/L). The ascitic dogs with liver cirrhosis had significantly increase (P<0.01) activity of ALP (128.75 ± 8.26 U/L) and GGT (7.30 ± 0.93 U/L). The levels of AST and ALP were significantly increased (P<0.05) in dogs with chronic hepatitis (72.52 ± 1.62 U/L and 139.00 ± 8.31 U/L), acute hepatitis (133.00 ± 5.93 and 126.16 ± 7.73 U/L) and cholecystitis (105.75 ± 4.04 U/L and 103.50 ± 6.88 U/L). The significant (P<0.05) increase in total and differential bilirubin was noticed in all the liver groups. The levels of cholesterol and glucose were significantly (P<0.01) decreased in cirrhosis (106.75 ± 4.67 mg/dl and 79.25 ± 3.40 mg/dl), chronic hepatitis (93.81 ± 10.80 mg/dl and 84.90 ± 5.98 mg/dl), acute hepatitis (107.00 ± 5.85 mg/dl and 81.14 ± 5.41 mg/dl), cholecystitis (116.47 ± 8.76 mg/dl and 81.00 ± 3.53 mg/dl).

The plasma protein profile revealed significant decrease (P<0.05) in total protein and albumin in dogs suffering from ascites due to liver (5.28 ± 0.29 g/dl and 3.39 ± 0.18

g/dl), cardiac (4.98 ± 0.71 g/dl and 2.18 ± 0.25 g/dl) and renal (5.31 ± 0.39 g/dl and 2.67 ± 0.45 g/dl) disorders. Hypo-proteinemia was evident in chronic hepatitis (5.38 ± 0.41 g/dl), acute hepatitis (5.17 ± 0.53 g/dl) and cholecystitis (4.82 ± 0.88 g/dl). A significant ($p<0.05$) decrease in albumin was observed in cirrhosis (3.27 ± 0.29 g/dl), chronic hepatitis (3.33 ± 0.27 g/dl) and cholecystitis (3.36 ± 0.53 g/dl).

The mineral and electrolyte analysis revealed hypocalcemia in dogs suffering from ascites due to liver (6.07± 0.32 mg/dl), cardiac (8.25 ± 0.96 mg/dl) and renal (8.37 ± 1.71 mg/dl) disorders. The decrease in phosphorus was observed in liver (4.32 ± 0.48 mg/dl) and cardiac (3.70 ± 0.90 mg/dl) disorders, whereas it was increased in renal (6.25 ±1.49 mg/dl) disorders. Significant decrease in sodium and chloride was recorded in liver, cardiac and renal disorders. Potassium was significantly decreased ($P<0.01$) in liver (3.92 ± 0.10 mEq/L) and cardiac (4.05 ± 0.03 mEq/L) and remained non-significant in renal disorder.

The blood gas and acid base status of liver, cardiac and renal affected ascitic dogs revealed no significant change in the values of pH, pCO_2, HCO_3, AnGap, tCO_2, BE, BEact, BEecf, BB, $stHCO_3$, st. pH and cH+ in comparison with healthy dogs that indicated no alterations in acid base status of the ascites affected dogs.

The electrocardiography of the six ascitic dogs with cardiac disorder revealed slow conduction between atria and ventricle in two dogs (2/6, 33.33%), left ventricular hypertrophy in one dog (1/6, 16.66%), ventricular arrhythmia in one dog (1/6, 16.66%) and two dogs (2/6, 33.33%) revealed normal electrocardiography with normal electrographic indices.

The ascitic fluid analysis revealed transudate fluid in 12 dogs with liver disorders (12/35, 34.28%) and in 1 dog with cardiac disorders (1/6, 16.66%). Modified transudate was observed in 14 dogs with liver disorders (14/35, 40%), 1 dog with cardiac disorders (1/6, 16.66%) and in 3 dogs with renal disorders (3/6, 50%). Exudate fluid was noticed in 9 dogs (9/35, 25.71%) with liver disorders, 4 dogs with cardiac disorders (4/6, 66.66%) and 3 dogs with renal disorders (3/6, 50%). The total protein of the ascitic fluid in liver disorders was 2.35 ± 0.12 g/dl, in cardiac was 0.53 ± 0.12 g/dl and in renal disorders was 2.67 ± 0.62 g/dl. The mean SAAG of dogs with liver disorders (2.35 ± 0.12 g/dl) was greater than 1.1 g/dl. The cytology revealed presence of neutrophils, RBCs and inflammatory cells mainly lymphocytes.

The physical examination of the urine revealed light yellow to dark yellow colour of the urine and was clear in appearance. One ascitic dog with liver disorder had dark orange colour of urine. The presence of RBCs and proteins in the urine of ascitic dogs with liver, cardiac and renal disorders was also observed however, urinary proteins were more in case of liver disorders. The presence of glucose and leukocyte was observed in the urine of ascitic dogs with liver and renal disorders. The urine pH remained non-significant and urine specific gravity was significantly ($P<0.05$) increased in all the groups.

The radiography imaging revealed ground glass appearance on standing abdominal view in 39 dogs (39/47, 82.97%) that masked the abdominal organs, out of them, 30 dogs were suffering from liver disorders, 5 had cardiac disorders and four dogs had renal disorders. The thoracic radiograph of the ascitic dogs with cardiac disorder revealed pleural effusion in 4 dogs and pericardial effusion in 2 dogs. The VHS was increased in 4 dogs and the mean VHS was 12.87 ± 0.71.

The echocardiography of the ascitic dogs with cardiac disorder dogs revealed pericardial effusion in two dogs (2/6, 33.33%), poor cardiac contractibility in one dog (1/6, 16.66%), left ventricular wall thickening in one dog (1/6, 16.66%) and mixing of the blood in 1 dog (1/6, 16.66%) with dilated cardiomyopathy.

The ultrasonography of the abdomen revealed presence of anechoic free fluid in the peritoneum in all the 47 dogs. Hyper-echogenicity and hypo-echogenicity was observed in 32 (32/47, 68.08%) and 2 (2/47, 4.25%) dogs, respectively. The fibrins in the ascitic fluid was noticed in four dogs (4/47, 8.51%), fibrosis in four dogs (4/47, 8.51%), systemic hypertension in three dogs (3/47, 6.38%), gall bladder polyp in 1 dog (1/47, 2.12%), space occupying lesion in liver and kidney was visualised in 5 dogs (5/47, 10.63%) and 1 dog (1/47, 2.12%), respectively. Cystic lesion were observed in liver (2/47, 4.25%), kidney (1/47, 2.12%) and spleen (1/47, 2.12%). Medial iliac lymph nodes (MILN) was increased in size in three dogs (3/47, 6.38%) and nephroliths were noticed in 2 dogs (2/47, 4.25%). The wall of gall bladder was thickened in four dogs (4/47, 8.51%) and sludge in gall bladder was evident in five dogs (5/47, 10.63%). The edges of the liver were round in 19 dogs (19/47, 40.42%) and inflammation of the liver parenchyma was observed in 33 dogs (33/47, 70.21%).

In case of acute hepatitis, round edges of the liver (6/6, 100%), increase in the liver size (3/6, 50%), hyper-echogenicity of the liver parenchyma (2/6, 33.33%) and peri-portal

vasculature (2/6, 33.33%), systemic hypertension (2/6, 33.33%) and thickening of the liver capsule (1/6, 16.66%) and anechoic peritoneal free fluid (6/6, 100%) were the common ultrasonographic changes that were visualised.

Ultrasonography of the dogs with chronic hepatitis revealed inflammation of the liver parenchyma (20/21, 95.23 %), hyper-echogenicity (20/21, 95.23%), focal hypo-echogenicity (1/21, 4.76 %), rounding of the liver margins (18/21, 85.71%), capsular thickening (3/21, 14.28%), decrease portal vascularity (3/21, 14.28%), increase in size of liver (9/21, 42.85%) and spleen (3/21, 14.28%). Space occupying lesion and cystic lesions in liver parenchyma was detected two and one dog, respectively. Systemic hypertension was observed in 2 dogs.

The abdominal ultrasonography in the dogs with the cardiac disorders revealed increase in the size of liver (3/6, 50%), hyper-echogenicity of the liver parenchyma (3/6, 50%), presence of anechoic free fluid (6/6, 100%) and cystic lesion in the right kidney with merging densities of the renal cortex in 1 dog.

The ultrasonography of the dogs with renal disorder revealed presence of anechoic free fluid (6/6, 100%), hyper-echogenicity of the renal cortex (5/6, 83.33%), increase in the size of kidney (2/6, 33.33%), increased size of liver (1/6, 16.66%), inflammation of the urinary bladder (2/6, 33.33%), decreased renal perfusion (3/6, 50%), hyper-echogenicity of liver (2/6, 33.33%), increased in size of medial iliac lymph node (1/6, 16.66%), nephroliths (2/6, 33.33%), cystic lesion in the right kidney (1/6, 16.66%) and end stage renal disease (2/6, 33.33%).

The combined therapy of liver safe antibiotics, diuretic, hepato-protectants, fluid therapy, liver extracts and silymarine was used for the treatment of ascitic dogs due to liver disorders along with supportive therapy and 13 dogs showed signs of recovery, among which 9 had chronic hepatitis, 3 had acute hepatitis and 1 had cirrhosis. Diuretic and digoxin, ACE inhibitor (Enalapril), anti-biotic therapy and supportive therapy were used for treating ascitic dogs with cardiac disorder and only one dog showed recovery. Fluid therapy, diuretic, anti-biotics and supportive therapy was used for treating ascitic dogs with renal disorders and only one dog showed recovery.

Conclusions

Based on the clinical, haematology, biochemical analysis of plasma and ascitic fluid, electrocardiography, urinalysis, imaging and therapeutic studies on canine ascites, following conclusions were drawn:

- Overall incidence of canine ascites was 2.27 per cent in College Veterinary Clinics.
- The major cause of ascites was found to be liver disorders with 74.46% of incidence and among the liver disorders, chronic hepatitis was the main cause of ascites, having incidence of 60 per cent.
- The young age group of 1-4 years and male dogs and breed Labrador retriever followed by German shepherd had higher incidence for ascites.
- The main clinical signs observed in ascites were abdominal distension, anorexia, and exercise intolerance. Vomiting and melena were also observed in ascitic dogs with liver and renal disorders.
- The haematology of the ascitic dogs revealed anaemia and leucocytosis.
- Activities of ALT, AST and ALP were increased and total protein was decreased in all ascites affected dogs. Hypoglycaemia and increased bilirubin was majorly observed in dogs with liver disorders and Creatinine and BUN were increased in dogs with renal disorders.
- The plasma protein levels decreased significantly in all the ascitic dogs along with decrease in plasma albumin.
- The Serum Ascitic Albumin Gradient (SAAG) was more than 1.1g/dl in ascitic dogs with liver disorders.
- The radiographic imaging revealed ground glass appearance in dogs with abdominal distension, however minute quantities of the ascitic fluid were not observed.
- Ultrasonographic imaging proved to be very useful imaging technique, as minute quantities of the ascitic fluid was also observed with true reflection of the vital organs (Liver, Heart and Kidneys).
- Combined therapy of silymarine along with supportive therapy of hepato protectants, liver supportive, liver safe anti-biotic and diuretic (Frusemide + Spironolactone) proved to be quite useful in the recovery of ascitic dogs having hepatic disorders.

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Brief Biodata of student

Name : Abhineet Kaur Bhatti

Father's Name : Jagdish Singh Bhatti

Mother's Name : Manjit Kaur Bhatti

Date of birth : 14-09-1993

Permanent Address : Guga Mari Basti, Near Bus Stand, Tapa, Dist. Barnala (Punjab) - 148108

Academic Qualifications:

Qualification	Year	School/College	Board/ University	Marks (%)	Division
10th	2009	St. Xavier's High School, Rampura (Punjab)	CBSE	70.8%	First
12th	2011	B.G.S. Public School, Barnala (Punjab)	CBSE	60.6%	Second
B.V.Sc & A.H.	2018	Khalsa College of Veterinary and Animal Sciences, Amritsar, Punjab	GADVASU, Ludhiana	63.21%	Second
M.V.Sc.	2020	DGCN College of Veterinary and Animal Sciences, Palampur, H.P.	CSKHPKV, Palampur	73.2%	First

Awards: Third prize in poster presentation at ISVM Conference held at Bikaner, 2019

Publications:

Total: Nil

Research Papers (in peered journals): Nil

Scientific Popular Articles: Nil

Others: Nil

Visits Abroad along with duration and purpose of visit: Nil