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# **CLINICAL AND ULTRASONOGRAPHIC INVESTIGATION OF ASCITES IN DOGS**

**JEGAVEERA PANDIAN. S.**

**Thesis submitted in partial fulfilment of the  
requirement for the degree of**

## **Master of Veterinary Science**

**Faculty of Veterinary and Animal Sciences  
Kerala Agricultural University, Thrissur**

**2005**

**Department of Clinical Medicine  
COLLEGE OF VETERINARY AND ANIMAL SCIENCES  
MANNUTHY, THRISSUR-680651  
KERALA, INDIA**

**DECLARATION**

I hereby declare that the thesis entitled "**CLINICAL AND ULTRASONOGRAPHIC INVESTIGATION OF ASCITES IN DOGS**" is a record of research work done by me during the course of research and this thesis has not previously formed the basis for the award of any degree, diploma, fellowship or associateship or other similar title, of any other University or Society.

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Mannuthy



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**CERTIFICATE**

Certified that the thesis entitled **“CLINICAL AND ULTRASONOGRAPHIC INVESTIGATION OF ASCITES IN DOGS”** is a record of research work done independently by **Jegaveera Pandian. S** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to him.

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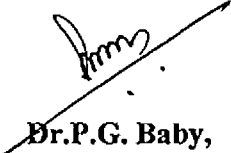
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EXTERNAL EXAMINER

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*Medicine, Madras Veterinary College for their whole- hearted help and strenuous teaching efforts.*

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# ***Introduction***

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## LIST OF ABBREVIATIONS

A: G ratio- Albumin: Globulin ratio

@- At the rate of

b.i.d- Twice daily

B.W- Body weight

cu. mm- Cubic Millimeter

EDTA- Ethylene Diamine Tetra Acetate

*et al-* et alibi

°F- Fahrenheit

Fl - Femtolitre

g- Gram

g/L- Gram per liter

g/dl- Grams per deciliter

Kg- Kilogram

MEA- Mean electrical axis

mg – Milligram

mg/dl- Milligram per deciliter

mg/lb- Milligram per pound

mmol / l- Millimol per liter

ml – Milliliter

MHz- Mega hertz

mV- Millivolts

P.O- Per Os

q.24.h- For every 24 hours

sec – second

+TV<sub>10</sub>- positive 'T' in lead V<sub>10</sub>

U/L- units per liter

# ***Introduction***

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## 1. INTRODUCTION

Ascites is a common clinical sign of many systemic disorders in dogs. But, it is not a separate clinical entity. Many factors such as nutritional, infectious, breed-associated, immune-mediated, parasitic diseases and derangements of various organ systems are incriminated as causes of ascites.

Ascites is an abnormal accumulation of fluid in the peritoneal cavity, usually a modified transudate. The pathophysiology of ascites differs depending on the organ involved. Two theories have been postulated concerning the development of ascites. They are 'Overfill theory' and 'Classic theory' (Leib, 1997).

Ascites must be differentiated from various conditions causing abdominal distension like distended bladder, ruptured bladder, pregnancy, pyometra, gastric distension, tympanitis, peritonitis, abdominal tumor and obesity. Ascites may be evident in an animal with hypoalbuminemia or portal hypertension due to major hepatic dysfunction (usually cirrhosis) or with other causes of portal hypertension. However, ascites can also occur with right-sided cardiac failure, intra-abdominal obstruction of the vena cava and hypoproteinemia due to nephrotic syndrome or protein-losing enteropathy (Maddison, 1990).

Understanding the pathophysiology of ascites aids in diagnosing, prognosticating and selecting appropriate managemental strategies. Differential diagnosis of the etiologies in ascites must include diseases of liver, heart, kidneys, endocrine glands, obstruction of blood and/ or lymph circulation, parasites, neoplasms, abscesses, peritoneal inflammations and protein-wasting diseases (Ringheim, 1975).

It is a widely accepted that abdominal ultrasonography can contribute a lot to the evaluation of abdominal disorders. It can also contribute to study the characteristics of effusion and pathologic conditions of abdominal viscera associated with ascites.

Electrocardiogram (ECG) is a good clinical diagnostic procedure for studying the functional status of heart and diagnosing congestive heart failure. It becomes one of the preliminary parameter in evaluating cardiac performance (Bolton, 1975). Echocardiography is a useful diagnostic technique in diseases of heart and proximal great vessels.

Diseases involving the liver are the most common causes of ascites. Increased portal pressure, decreased albumin production and sodium retention ultimately result in the development of ascites. Liver function tests are efficient in diagnosing liver-related ascites (Strombeck and Guilford, 1991). Elevation in the activity of alanine aminotransferase (ALT) is considered to be specific for hepatic injury in dogs and cats. Serum alkaline phosphatase (ALP) activity may be elevated in both acute and chronic liver diseases, but marked elevations are indicative of cholestasis, biliary cirrhosis or extrahepatic bile duct obstruction (Burk and Ackerman, 1996).

Post-sinusoidal hypertension usually results in high protein ascites due to the contribution of high protein hepatic lymph. But, it was necessary to determine ascitic fluid to plasma protein ratio in order to differentiate pre and post-sinusoidal hypertension (Hunt *et al.*, 1993). If the proteinuria of glomerulonephritis is severe, plasma albumin level will fall, resulting in hypoalbuminemia and eventually leading to generalized edema and ascites (Bown, 1977).

Considering these views, the present study entitled, "Clinical and Ultrasonographic Investigation of Ascites in Dogs" was taken up for,

1. Studying the pathogenesis of ascites in dogs using ultrasonography, electrocardiography and serum bio-chemical assays.
2. Assessing the efficacy of managerial procedures employed.

# *Review of Literature*

## 2. REVIEW OF LITERATURE

Two theories have been postulated to explain the pathogenesis of ascites. The first theory describing ascites formation is called 'Overflow theory'. The initial event is renal sodium retention, although the initiating cause is not known, it is postulated that hepatic diseases resulted in decreased availability of natriuretic hormone, which leads to expansion of extracellular volume and portal hypertension secondary to an increased volume of blood flow. Another theory of ascites formation is called the 'Classic theory'. It suggested that resistance to portal blood flow lead to splanchnic pooling, which resulted in decreased cardiac output, decreased renal blood flow, activation of renin-aldosterone system which resulted in sodium retention and ascites (Leib, 1997).

### 2. 1. ETIOLOGY

#### 2.1.1. Liver

Toth and Derwelis (1980) reported a case of ascites associated with hepatitis due to administration of oral antibacterial agent, trimethoprim with sulphadiazine. They concluded that it was a drug- induced reaction.

Two out of five dogs received pentobarbitone therapy for three years had developed ascites (Bunch *et al.*, 1982).

Chronic active hepatitis with increased hepatic copper concentration was diagnosed in 25 female and one male Doberman pinscher dogs. Common clinical signs were polyuria/polydipsia, weight loss, anorexia, icterus and ascites (Crawford *et al.*, 1985).

Ascites that occurred with hepatic parenchymal disease was due to combination of renal sodium retention, intrahepatic portal hypertension, formation of fluid at the surface of liver and hypoalbuminemia (Maddison, 1990).

Hunt *et al.* (1993) explained cases of ascites due to portal hypertension in three young dogs. In those cases, nodular regenerative hyperplasia or Budd-Chiari like syndrome were incriminated as causes.

Ascites was the most common clinical sign, whereas decreased appetite and lethargy were the most common owner's complaint in cirrhotic dogs (Sevelius, 1995).

Van den Ingh *et al.* (1995) observed primary hypoplasia of portal vein as a cause of abdominal distension in 42 dogs and twenty three of them showed ascites. It was concluded that portal hypertension associated with primary hypoplasia of the hepatic portal vein affected mainly young dogs and most likely to be originated congenitally.

Adamus *et al.* (1997) observed ascites in sixteen juvenile Beagle dogs associated with chronic hepatitis caused by *Leptospira*. Lesions included severe chronic hepatitis to mild diffuse hepatocellular vacuolation with bile stasis. They added that all those dogs were vaccinated against *Leptospira interrogans*.

Leib (1997) observed high incidence of chronic active hepatitis in middle-aged Doberman pinschers with the clinical signs of weight loss, vomiting, anorexia, icterus, ascites, polyuria/polydipsia, bleeding tendencies and hepatic encephalopathy. Lobular dissecting hepatitis was common in young dogs below one year of age. Besides, portal hypertension and ascites were common in those dogs.

A syndrome, resembling idiopathic or non cirrhotic portal hypertension in humans was reported in four young, male Doberman pinschers characterized by lack of arteriovenous fistula, portal vein atresia or intra-hepatic fibrosis (De Marco *et al.*, 1998).

Mucopolysaccharidosis-I (MPS-I), a genetic storage disease was observed as a cause of non cirrhotic portal hypertension and ascites in a colony of dogs and

non cirrhotic portal hypertension and nodular regenerative hyperplasia appeared to be related to the obliteration of small portal veins (Mc Entee *et al.*, 1998).

Pembleton-Corbett *et al.* (2000) concluded that portal hypertension was a predominant force in the formation of transudative abdominal effusion in dogs with hepato-biliary diseases.

Lucena *et al.* (2001) described a case of ascites in a five-month-old Spanish mastiff. On necropsy, it was found that cirrhosis was the cause of ascites. The possible involvement of canine adeno virus was postulated.

Szatmari *et al.* (2002) explained a case of ascites in a two- and - a-half-year-old German shepherd dog with circumscribed fibrosis in the wall of the portal vein, resulted in stenosis and prehepatic portal hypertension.

Yamagami *et al.* (2002) studied a case of hepatomegaly and ascites in a nine-month-old intact male, American cocker spaniel due to hepatic lymphangiomatosis.

Koide *et al.* (2004) discussed a case of ascites in a two-month-old Golden retriever with hepatic arteriovenous fistula and aortic stenosis. One of his littermate also had an intrahepatic portosystemic shunt.

Nottidge *et al.* (2003) dealt a case of ascites in a ten-month-old Alsatian dog due to liver cirrhosis and congestive heart failure. The dog was non-responsive to diuretic therapy.

Rychlik *et al.* (2005) elaborated a case of lobular dissecting hepatitis (hepatitis lobularis dissectionica) in a six-month-old, male Newfoundland dog with ascites.

### **2.1.2. Kidney**

Bown (1977) stated that all cases of glomerulonephritis were associated with proteinuria, which varied from levels of <100 mg / 100ml to 1g / 100ml. The

major protein fraction appearing in the urine was albumin due to its smaller molecular size. Proteinuria even when massive was not pathognomonic of glomerulonephritis. If the proteinuria of glomerulonephritis was severe enough, it resulted in hypoalbuminemia and generalized oedema. This syndrome was known as nephrotic syndrome. Oedema was usually subcutaneous or ascitic.

DiBartola *et al.* (1980) observed a largest amount of protein excretion through urine in dogs with amyloidosis. Besides, the authors also opined that amyloidosis might occur secondary to many disorders of chronic immunologic stimulation or chronic inflammation or even neoplasia. Sometimes, it resulted without any discernible predisposing cause.

Nephrotic syndrome manifested in any glomerular disorder wherein the urinary loss of protein was sufficient to produce hypoalbuminemia. Peripheral edema and ascites were the common consequences of hypoalbuminemia caused by nephrotic syndrome (Cowgill, 1983).

Patients with nephrotic syndrome retained sodium, which contributed to the formation of ascites, edema, hypertension, and weight gain (Fleming *et al.*, 1989). The author also stated that it was often impossible to identify the initial cause of glomerular diseases.

Forrester (1997) concluded that membranous glomerulonephritis and amyloidosis were the two most common causes of nephrotic syndrome in dogs and cats. Furthermore, the author pointed out that clinical signs of glomerular disease resulted from complications of urinary protein loss, renal failure or underlying infectious, inflammatory or neoplastic diseases.

### **2.1.3. Heart**

The two most constant clinical signs of congestive heart failure were ascites and a dry, hacky 'cardiac cough' resulting from pulmonary oedema and other clinical signs included dyspnoea, increased heart rate, rapid onset of fatigue

after physical activity, polydipsia, polyuria (especially nocturnal), hepatomegaly, and oedema of limbs (Morris *et al.*, 1976).

Edwards *et al.* (1978) reported a case of ascites and hepatomegaly in a German shepherd dog due to right atrial tumour causing portal hypertension. On surgical exploration, it was found that an unresectable right atrial tumour occluding caudal vena cava.

Van Vleet *et al.* (1981) observed congestive cardiomyopathy as a cause of ascites in large-breeds of dogs. Similarly, Calvert *et al.* (1982) reported congestive cardiomyopathy in twenty Doberman pinscher dogs. Among these dogs, only one dog had isolated right-sided heart failure and 45 % of the dogs had both left and right- sided heart failure. The authors stated that dogs with isolated right- sided heart failure had developed ascites and pleural effusion.

Calvert *et al.* (1986) studied 137 cases of canine heartworm disease. Among these, 36 had abdominal effusions characterized by modified transudate.

Heart failure was artificially induced using a surgically implanted programmable ventricular pacemaker, which stimulated the heart at a rate of 240 beats / min and a low- cardiac output state was achieved. This condition resembled a cardiomyopathic state. By these procedures, all the five dogs studied were developed ascites (Allworth *et al.*, 1995).

Davis (1995) explained the existence of an extra-adrenal sodium retaining factor in congestive heart failure which increased the responsiveness of renal tubules to aldosterone resulted in sodium retention and oedema.

Ishibashi *et al.* (2001) experimentally created tricuspid regurgitation in seven dogs and all dogs developed ascites and loss of appetite.

Hidaka *et al.* (2003) observed ascites in three dogs under two- years of age, due to right- sided heart failure associated with heartworm caval syndrome.



Moneva-Jordan (2003) stated that dogs with dilated cardiomyopathy (DCM) and chronic, severe, right-sided heart failure were prone to cardiac cachexia and ascitic distension.

Ascites, right-sided congestive heart failure, pulmonary crackles, tachypnea, dyspnoea, pulmonary thromboembolism and hepatomegaly were the common observations made in canine heartworm disease (Eslami *et al.*, 2005).

#### **2.1.4. Other Causes**

Heise (1983) presented a case of lymphangiectasia and protein-losing enteropathy in a German shepherd dog. The animal was having ascites, rapid weight loss, malodorous faeces, selective appetite and marked wasting of muscle tissue.

Barr *et al.* (1989) reported that chronic dilative myocarditis caused by *Trypanosoma cruzi* might be the reason for the resultant ascites, respiratory distress, thoracic effusion, cyanosis and weak pulse with ventricular arrhythmia in two female hunting dogs.

Malik *et al.* (1990) explained a case of ascites resulted from congenital obstruction of the caudal vena cava in a 16- week- old- female Rottweiler dog. A stenotic lesion at the junction of caudal vena cava and right atrium was noticed in selective angiography.

Fossum *et al.* (1992) diagnosed chylous ascites in three dogs associated with variable reasons namely ruptured mesenteric lymph vessel, abdominal lymphatic obstruction and complication of mesenteric lymphangiography.

A ten-year old, spayed female Beagle with two- and- a- half- year history of ascites was diagnosed as cystic peritoneal mesothelioma. Ascitic fluid of that animal was a modified transudate (Di Pinto *et al.*, 1995).

Hess and Bunch (1995) stated that increased pressure in the portal venous system resulted from impedance to blood flow at any point along its course from splanchnic circulation through the liver to the right heart was manifested as ascites and acquired portosystemic shunts.

A two- year- old Labrador retriever with persistent ascites and exercise intolerance was diagnosed as obstruction of intrathoracic caudal vena cava. This partial obstruction was probably due to automobile accident that the animal met earlier (Lisciandro *et al.*, 1995).

Lamb *et al.* (1996) explained a case of ascites associated with portal vein thrombosis in a dog positive for *Ehrlichia canis* (1:20) and *Ehrlichia equi* (1:80) serologically.

Kull *et al.* (2001) explained intestinal lymphangiectasia was the cause of ascites in 17 dogs and ascites was one of the abnormal physical examination findings in those dogs.

Harder *et al.* (2002) explained a case of ascites in a two and a half year-old, castrated male Wheaten terrier due to segmental aplasia of the caudal vena cava.

Any decrease in serum albumin concentration could cause a significant decrease in oncotic pressure resulted in fluid shift from the intravascular space into the interstitium. This lead to hypotension, edema and body cavity effusions. Albumin concentration less than 15 g/L resulted in edema and effusions (McGrotty and Knottenbelt, 2002).

Bressler *et al.* (2003) studied a case of ascites due to portal vein and aortic thrombosis associated with canine ehrlichiosis.

Caruso *et al.* (2003) opined that *Mesocestoides* infection in the peritoneal cavity led to ascites and peritonitis.

Rollois *et al.* (2003) detailed a case of ascites and hind limb oedema in a dog. Post mortem examination of the dog revealed an esophageal leiomyoma, which compressed the caudal vena cava.

Lymphadenopathy, splenomegaly, ascites, paleness of mucous membrane, kidney and liver and discrete pulmonary congestion were observed at necropsy of dogs experimentally infected with *Ehrlichia canis* (de Castro *et al.*, 2004).

## 2.2. ULTRASONOGRAPHY

### 2.2.1. Effusions

Calvert *et al.* (1986) observed modified transudate in 36 cases of canine heartworm disease.

Hunt *et al.* (1993) revealed a large amount of free fluid and freely mobile intestinal loops in three dogs with ascites associated with noncirrhotic portal hypertension.

Spaulding (1993) classified the sonographic appearance of peritoneal effusion into anechoic, homogeneously echogenic and echogenic and septated. Homogeneity and echogenic character of an effusion could be used as an estimation of cellular content which helped in categorizing the effusions. The author stated that anechoic effusions might be either a transudate, modified transudate, or less frequently, and exudates. However, echogenic /septated, and homogeneously echogenic appearances were typically associated with ascites. Apart from this, the author stated that a comparison of the peritoneal effusion with the sonographic appearance of the urine within the bladder lumen help in assessing the degree of cellularity in both areas suggesting either an exudative or transudative fluid.

Boysen *et al.* (2004) elaborated various planes of abdominal ultrasonography used to detect the abdominal fluid of post-traumatic origin. In addition to that, the authors opined abdominal ultrasonography as an efficient mean of diagnosing the presence of free abdominal fluid.

Szatmari *et al.* (2004a) quantified the peritoneal effusion subjectively using physical examination and ultrasonography. The quantity of peritoneal effusion was subjectively estimated and classified as a large (ascites detectable by physical examination), moderate (ascites not detectable by physical examination but easily seen ultrasonographically), or small (thorough ultrasonographic search needed to detect free abdominal fluid) amount.

### **2.2.2. Liver**

Hunt *et al.* (1993) observed no sonographic abnormalities in the hepatic parenchyma of three ascitic dogs, due to portal hypertension associated with non-fibrosing liver disease.

Ultrasonographic imaging of the liver in dogs could easily be done by placing the transducer caudal to xiphisternum in a longitudinal plane and angled dorsocranially between 30° - 40° to the dorsal plane in dorsally recumbent animal. The author also discussed the distinguishing features of portal and hepatic veins. They stated that the portal veins were surrounded by hyperechoic connective tissue whereas the hepatic veins, in general were not (Jian-Xin WU and Carlisle, 1995).

The liver was not always accessible to ultrasound scanning, because a portion of it was obscured by the overlying stomach. In dogs with deep thoracic conformation, the liver might be cranial to the last rib and relatively inaccessible to scanning. The liver is less echogenic than the spleen and more echogenic than the renal cortex. The architecture of the liver is composed of a uniform texture, which is interrupted by short, highly echogenic, paired parallel lines surrounding

an anechoic lumen representing the portal veins and anechoic lumen representing the hepatic veins (Burk and Ackerman, 1996).

Lamb *et al.* (1996) diagnosed ascites associated with portal vein thrombosis ultrasonographically. In that case, portal vein tributaries were dilated and an echogenic thrombus was identified in the portal vein at the porta hepatis.

An ultrasonographic study conducted in human patients with non cirrhotic portal hypertension (NCPH) revealed hyperechoic bands surrounding the portal vein branches, which were separated from adjacent liver parenchyma by a hypoechoic stripe (Gurkaynak *et al.*, 1998).

Kull *et al.* (2001) opined that imaging abnormalities were common in dogs with intestinal lymphangiectasia but were not specific enough to differentiate this disorder from other gastrointestinal disorders.

A large, hypoechoic, heterogenous mass located between the kidneys and associated with an anomalous, distended caudal vena cava was the ultrasonographic finding in a dog with segmental aplasia of caudal vena cava (Harder *et al.*, 2002).

Yamagami *et al.* (2002) characterized the ultrasonography of lymphangiomatosis in a young dog. They stated that there was increased parenchymal echogenicity with focal and more hyperechoic nodules.

Ultrasonographic examination of a dog with ascites due to *Ehrlichia canis* revealed normal liver, portal vein and aortic thrombosis (Bressler *et al.*, 2003).

Szatmari *et al.* (2004b) described the techniques of imaging the abdominal and portal vasculatures in dogs using seven different planes. With the dog in left lateral recumbency, transverse sections of the portal vein and its branches were observed via the right intercostal space and longitudinal sections were obtained via the right flank. With the dog in dorsal recumbency, longitudinal sections of the portal vein were obtained via the ventral body wall. With the dog in right

lateral recumbency, longitudinal sections were obtained via the left flank to image the right gastric-caval shunts and the left testicular or ovarian vein.

Vijayakumar *et al.* (2004a) observed an increase in the echogenicity of liver following administration of oxytetracycline, suggestive of hepatic disorder.

### **2.2.3. Kidney**

Konde *et al.* (1984) explained the normal echotexture of canine kidney by ultrasonography and they concluded that renomegaly in diuretic therapy was due to increase in the medullary size. In this study, sagittal renal cortical echo patterns were more consistent with post-mortem evaluation than transverse scans.

Douglass and Kremkau (1993) explained the phenomenon of pseudohypoechoic bladder wall lesion due to sound refraction on the curved wall in dogs with free abdominal fluid.

Nyland *et al.* (1995) stated that imaging the kidneys in sagittal, transverse and longitudinal planes was essential to arrive at a sonographic diagnosis. The authors opined that ultrasound was usually indicated in evaluating the kidney even in the presence of impaired renal function or abdominal fluid. The authors had also explained the technique of imaging kidneys ultrasonographically and they pointed out that imaging of left kidney was comparatively easier than that of right kidney.

Triolo and Miles (1995) stated that the ultrasonography was an excellent method for evaluating the kidneys and it also provided a better means for general parenchymal architecture than excretory urography.

The kidneys were usually examined in three planes; longitudinal, transverse and sagittal. These planes were referenced to the kidney and not to the patient. The renal cortex, medulla and pelvis were identified consistently. The renal cortex was brighter than medulla, with an echointensity less than that of liver and markedly less than that of spleen. The renal pelvic recesses appeared as

bright, evenly spaced, round or linear echoes. This appearance was most likely the result of the presence of the renal pelvic fat and the interlobar arteries (Burk and Ackerman, 1996).

In cases of nephrotic syndrome in dogs, ultrasound scanning did not reveal useful information to diagnose the case. Specific changes in the echogenicity of renal parenchyma could not be observed in glomerulonephritis and renal amyloidosis (Leib, 1997).

#### 2.2.4. Heart

M- mode echocardiography provided a non- invasive method of evaluating cardiac chamber size, interventricular septum, left ventricular free wall thickness and systolic and diastolic function. M- mode echocardiography was used in the diagnosis of congestive cardiomyopathy in Doberman pinschers and echocardiograms were obtained by placing the transducer in right fourth to sixth intercostal space, between the sternum and the costochondral junction. Echocardiographic findings included decreased shortening fraction, decreased septal and left ventricular free wall percent systolic thickening and increased E-point to septal separation (EPSS) (Calvert and Brown, 1986).

Barr *et al.* (1989) found that there was bilateral cardiac enlargement and septal and left ventricular free wall thinning in M- mode echocardiography of two ascitic dogs with chronic dilative myocarditis associated with *Trypanosoma cruzi* infection.

Allworth *et al.* (1995) observed a significant increase in the left ventricular internal diastolic dimension (LVIDd) ( $50.2 \pm 0.645$  mm) and left ventricular internal systolic dimension (LVIDs) ( $40.6 \pm 0.770$  mm) in dogs with pacing-induced heart failure resembling cardiomyopathy. Where as in control dogs, the values were  $37.3 \pm 0.552$  mm and  $24.6 \pm 0.760$  mm respectively.

Moise and Fox (1988) explained various parasternal windows applicable to evaluate heart using ultrasound and various measurements to study the cardiac performance in dogs and cats.

Left ventricular internal diastolic dimension (LVIDd) > 45mm in dogs that weighed  $\leq$  42 kg and left ventricular internal systolic dimension (LVIDs) > 38mm and left ventricular fractional shortening (FS) < 26% and cranial mitral valve leaflet E-point to septal separation > 8mm were considered as cardiomyopathic in a study conducted using echocardiography to diagnose DCM in Doberman pinschers (Calvert and Jacobs, 2000).

Ishibashi *et al.* (2001) concluded that despite massive tricuspid regurgitation and overt right-sided heart failure created experimentally, the intrinsic right ventricular contractile function was normal.

Left ventricular end-diastolic diameter (LVEDD) and Left ventricular end-systolic diameter (LVSED) were significantly increased in dogs with dilated cardiomyopathy (DCM). While fractional shortening (FS) was decreased (Tidholm *et al.*, 2001).

Fascetti *et al.* (2003) diagnosed dilated cardiomyopathy (DCM) in 12 dogs using echocardiography.

Hidaka *et al.* (2003) studied heartworm caval syndrome in three dogs under two years of age. In that, the authors utilized echocardiography to visualize a moderate number of heartworms in right atrium and tricuspid valve apparatus. Echocardiographic findings were worm echoes in right atrium and right ventricle and a flattening of the interventricular septum in right parasternal short-axis views of the left ventricle, suggesting high right ventricular pressure and/or volume overload.

Su *et al.* (2003) studied the two-dimensional and M-mode echocardiographic indices in the normal Taiwanese dogs. The authors stated that



ejection fraction, fractional shortening, and fractional thickness, interventricular septal thickness / left ventricular posterior wall thickness ratio, left atrial / aortic root dimension ratio, systolic time intervals, indices in mitral valvular study and stroke volume index were good indicators of evaluation of cardiac function. Measurements in end-diastole were taken at the onset of 'QRS' complex of the ECG or just before the closure of mitral valves. Those in end-systole were made at the point of maximal anterior motion of the posterior wall. The author studied echocardiographic indices in normal dogs, with out tranquilizing them. The authors preferred 2-D echocardiography to M-mode echocardiography as the entire contour of the cardiac chamber could be viewed at one time in 2-D echocardiography. They stated that, 2-D echocardiography provided more reliable estimate of chamber volume and ejection fraction than M-mode echocardiography.

Ristic (2004) stated that echocardiography provides useful information about chamber size, contractility, valve structure and motion. In dilated cardiomyopathy, there was enlargement or dilatation of ventricles and atrium and reduced contractility.

### 2.3. ELECTROCARDIOGRAPHY

Soave (1959) explained that electrocardiograms of the dog usually taken with the animal lying on its right side, strapped down lying on its back, or in position of sternal recumbency. The author stated that in nephritis and nephrotic conditions of dogs, the ECG changes were bradycardia and an increase in QT time.

Jones (1985) reported that low voltage QRS complexes in dogs with ascites might be due to increased peritoneal fluid or effusions in thoracic cavity.

Calvert *et al.* (1986) compared the radiographic and electrocardiographic abnormalities in right-sided heart failure due to canine heartworm disease. The authors stated that dogs with occult heartworm infections tend to develop earlier

and more severe right ventricular enlargement when compared to microfilaremic heartworm disease. They concluded that the ECG did not detect mild to moderate right ventricular enlargement and although the radiographic assessment of moderate right ventricular enlargement was fraught with problems, ECG was even less accurate. They stated that ECG criteria of right ventricular hypertrophy such as R/Sv4, +Tv10, MEAx, S1 were highly specific tests. While Sv2 was a highly sensitive, but not highly specific test. In addition to this, they suggested ECG as a confirmatory test for the presence of severe right ventricular hypertrophy and as an exclusion test for the absence of right-sided congestive heart failure.

Electrocardiogram of two hunting dogs having ascites due to chronic dilative myocarditis revealed first-degree heart block, chamber enlargement and ventricular-based arrhythmia (Barr *et al.*, 1989).

Electrocardiography (ECG) is one of the simplest and most widely used diagnostic tools available to evaluate heart diseases. A tall 'P' wave is called 'P' pulmonale, arising from right atrial enlargement. Although, 'P' pulmonale is caused by pulmonary disease, congenital and acquired tricuspid valve disease may also lead to right atrial enlargement (Bond, 1997). The author enlisted the ECG criteria for right ventricular enlargement. They were, 'S' wave in lead CV<sub>6</sub>LL > 0.08 sec, mean electrical axis in the frontal plane greater than +103° degree, S wave lead CV<sub>6</sub>LU > 0.7 mV, S wave in lead I > 0.05 mV, R/S ratio in CV<sub>6</sub>LU < 0.87mV, S wave in lead II > 0.35mV, S waves in leads I, II, III and aVF, positive T waves in lead V<sub>10</sub> and 'W' shaped QRS in lead V<sub>10</sub>. The author further stated that right axis deviation and deep 'S' waves in leads I, II and III were common in ECG of heartworm infected dogs with ascites. Furthermore, the author recommended echocardiography for measuring the ventricular enlargement in dirofilariasis.

Heart rate was significantly increased ( $183 \pm 49$  bpm) in dogs with dilated cardiomyopathy compared to normal dogs ( $95 \pm 16$  bpm). Rhythmic changes

observed in DCM were normal sinus rhythm, sinus tachycardia, atrial fibrillation, and ventricular premature contractions (Tidholm *et al.*, 2001).

Martin (2002) opined that deep 'S' waves in lead II are suggestive of right ventricular enlargement. The author stated that prolonged 'QRS' duration, or a shift in the mean electrical axis to the right was indicative of right ventricular enlargement and increased amplitude of 'P' wave indicated right atrial enlargement.

ECG parameters remained normal in seven refractory cases of low- protein ascites except the low- amplitude complexes (Varshney *et al.*, 2002).

Fascetti *et al.* (2003) observed ECG changes in 10 of the 12 dogs with dilated cardiomyopathy (DCM). They were left ventricular enlargement, atrial fibrillation, left bundle branch block and ventricular premature contractions.

#### 2.4. HAEMOGLOBIN AND PACKED CELL VOLUME

Kaneko *et al.* (1997) stated that the hemoglobin concentration (Hb) and packed cell volume (PCV) of normal healthy dogs were 12-18 g% and the 33-45% respectively.

Levy and Richard (1978) observed that there was plasma volume expansion preceding ascites formation. Total plasma volume increased by 13.2% when measured during ascitic phase of the cirrhosis.

DiBartola *et al.* (1980) observed normochromic normocytic anaemia (PCV < 37 %) in six of the seven dogs with proteinuria due to glomerular diseases.

Goldston *et al.* (1980) stated that prolonged occlusion of the vein and excitation of animal resulted in increased in PCV by 10 to 15%. The PCV may be low in hydremic conditions such as pregnancy, hypoproteinemia, iatrogenic overhydration...etc. These variables must be considered when the PCV is interpreted.

Hunt *et al.* (1993) observed lowered PCV (16%) in three dogs with ascites associated with non-fibrosing liver disease and portal hypertension.

A study conducted by experimental induction of cirrhosis in 10 healthy stray dogs revealed an increase in hemoglobin and erythrocyte concentration (Ertekin *et al.*, 2003).

Nottidge *et al.* (2003) observed that there was progressive reduction in hemoglobin concentration, PCV and RBC values in a dog with ascites due to liver cirrhosis and congestive heart failure. The author had also noticed normocytic, normochromic, non-regenerative anaemia in that dog.

## 2.5. SERUM BIO-CHEMISTRY

McGrotty and Knottenbelt (2002) reviewed the importance of urine protein- creatinine ratio in diagnosing proteinuria. They insisted that proteinuria can be concluded in the light of urine specific gravity.

Laboratory findings included low-protein ascites, less number of nucleated cells, target cells in the blood smear, ammonium biurate crystals in urine, negative ECG findings, hypoproteinemia, hypoalbuminemia and lower values of serum ALP, ALT and blood urea nitrogen (BUN) were found in seven refractory cases of ascites in dogs (Varshney *et al.*, 2002).

Harper *et al.* (2003) observed age- related change in haematological and serum biochemical parameters in Beagles and Labrador retrievers. Moreover, the authors suggested the use of age-specific reference ranges for the interpretation of clinical data.

Koide *et al.* (2004) evaluated the serum bio-chemical parameters of a dog with hepatic arteriovenous fistula, ascites, intrahepatic portosystemic shunt and aortic stenosis. It revealed mild microcytic, hypochromic anaemia (Mean Corpuscular Volume (MCV) 59fl, Mean Corpuscular Hemoglobin Concentration (MCHC) 31.5g/dl, and PCV 25%), prolonged clotting time, serious

panhypoproteinemia (2.5g/dl of total protein, 1.2 g/dl of albumin) and higher serum ALP concentration (1513 U/L).

Swanson *et al.* (2004) studied the age and food related changes in haemato– biochemistry of dogs. In that, the authors concluded that the red blood cells, hemoglobin, hematocrit, creatinine, total protein, albumin, sodium, chloride and alanine aminotransferase were present in greater ( $P < 0.05$ ) concentrations in old dogs. But, young dogs had greater concentrations of glucose and alkaline phosphatase.

Serum bio-chemical values were significantly elevated and their liver biopsy specimen showed foamy cytoplasm with mild to moderate hydropic changes at the end of the trial conducted by administering oxytetracycline (Vijayakumar *et al.*, 2004a).

### **2.5.1. Proteins**

The total protein, albumin and globulin concentrations in the serum of healthy dogs were 54.0-71.0 g/L, 26.0-33.0 g/L and 27.0-44.0 g/L respectively. The albumin: globulin ratio in healthy canine serum was 0.59-1.11 (Kaneko *et al.*, 1997):

Oedema develops when the plasma albumin level falls below 2 g / 100ml. The nephrotic syndrome is not common in dogs, but when it occurs as a result of glomerulonephritis, it is associated with membranous form of the disease. Uremia is not a constant feature in case of glomerulonephritis (Bown, 1977).

Sixteen dogs (76.2%) had serum albumin concentration less than 2 g/ 100 ml and 11 dogs (52.45%) had values less than 1.5 g/ 100ml on at least one determination while studying the 21 dogs with severe proteinuria (DiBartola *et al.*, 1980). Edema and ascites were evident in three dogs.

Nephrotic syndrome manifested in any glomerular disorder wherein the urinary loss of protein was sufficient to produce hypoalbuminemia, where

peripheral edema and ascites were the common consequences of hypoalbuminemia caused by nephrotic syndrome (Cowgill, 1983; Fleming *et al.*, 1989).

Heise (1983) categorized the ascitic fluid of a dog with lymphangiectasia and protein-losing enteropathy as pure transudate. Transudation occurred when the serum albumin value falls to 1.0 g/dl or less, resulting in reduced plasma oncotic pressure. The total protein, albumin and globulin were 2.8, 1.0 and 1.8 g/dl respectively in the serum of a dog with lymphangiectasia and protein-losing enteropathy.

Codner and Farris- Smith (1986) observed hyperglobulinemia (3.2- 9.3 mg/dl), thrombocytopenia, absolute lymphocytosis and normal serum albumin concentration (2.7 – 4.4 mg/dl) in dogs with sub clinical ehrlichiosis.

Hypoalbuminemia (1.6 g/dl) and proteinuria (3+) were observed in an eight- year- old male Brittany Spaniel with amyloidosis (Spyridakis *et al.*, 1986).

The protein concentration of the ascitic fluid was usually greater than 25 g/L in hepatic parenchymal disorders causing portal hypertension; but, in ascites caused by hypoproteinemia alone, the protein concentration of ascitic fluid was invariably less than 25 g/L. Ascitic fluid of low protein concentration ( <25 g/L) was found when it was formed from intestinal lymph. In contrast, post-hepatic portal hypertension occurred with obstruction from hepatic veins to the right atrium resulted in high-protein ascites (> 25g/L), mainly contributed by hepatic lymph (Maddison, 1990).

The liver synthesizes albumin, and since it is most abundant large molecule in plasma, constituting 60% of the plasma protein mass and its level determines the most (80%) of plasma colloid osmotic pressure. Hepatic function and the animal's nutritional status determine the rate of albumin synthesis. Portosystemic vascular shunts; cirrhosis and severe chronic hepatitis reduce albumin production. Volume of distribution of albumin determines the plasma

level of albumin. One-third of all albumin in the body is in the intravascular space. The remaining two-thirds is in the extravascular space, primarily the interstitial fluid, in which half of the albumin is in the skin. The plasma concentration of albumin usually increases with dehydration. The total body pool of albumin cannot be predicted accurately without measuring its volume of distribution. With hypoproteinemia and ascites, the rate of albumin synthesis may be normal and the total amount of albumin in the body may also be normal. Albumin lost directly into the peritoneal cavity may be the reason for the accumulation or persistence of ascitic fluid. Thus hypoproteinemia resulted from an increase in its space of distribution where the rate of albumin synthesis remains normal. Volumes of ascitic fluid are usually in a steady state; that is, bi-directional fluxes of the fluid into and out of the abdomen are equal. Albumin loss is accelerated with some forms of intestinal and kidney diseases. Increased permeability of the intestinal mucosa and glomerular membrane accounted for most of the losses. They concluded that most hypoalbuminemia are not due to decreased albumin synthesis but to increased loss or degradation (Strombeck and Guilford, 1991).

The protein content of the ascitic fluid was divided by the plasma protein concentration and the ratio expressed as a percentage. This parameter is referred to as "ascitic fluid to plasma protein ratio." This parameter was helpful in differentiating the pre-sinusoidal and post-sinusoidal portal hypertension (Hunt *et al.*, 1993).

Rutgers *et al.* (1993) reported that idiopathic hepatic fibrosis in fifteen young dogs as a cause of ascites. Besides, erythrocyte microcytosis, hypoproteinemia, high serum activities of alkaline phosphatase and, to a smaller extent, alanine aminotransferase were the consistent laboratory findings in all those dogs.

Hypoalbuminemia was the most consistent serum biochemical aberration in liver cirrhosis (25/26) and hepatitis (13/18) of dogs (Sevelius, 1995).

Low PCV, total serum protein, albumin and increased activities of liver enzymes in plasma and increased fasting levels of total bile acids and ammonia in 42 dogs with portal hypertension associated with primary hypoplasia of portal vein (Van den Ingh *et al.*, 1995).

Protein concentrations in ascitic fluid and serum were almost similar in microfilaremic dogs, developing ascites acutely. In contrast, dogs chronically infected with *Dirofilaria immitis* developed ascites with significantly lower ascitic fluid protein than plasma (Atwell *et al.*, 1996)

Greig *et al.* (1996) stated that hypoproteinemia and hypoalbuminemia were the serum- biochemical features in dogs affected with granulocytic ehrlichiosis.

Leib (1997) observed that common laboratory findings in chronic active hepatitis were increased ALT and ALP, hypoalbuminemia, hyperbilirubinemia, reduced BUN and anemia.

Serum albumin-effusion albumin (SA-EA) gradient had overall diagnostic accuracy of 69.4% in predicting portal hypertension in dogs with or without hepato-biliary diseases which exceeded that of hypoalbuminemia (57.1%) (Pembleton-Corbett *et al.*, 2000).

Most notable clinicopathologic findings were low serum ionized calcium concentration and hypoalbuminemia in 17 dogs with ascites due to intestinal lymphangiectasia (Kull *et al.*, 2001).

Hypoalbuminemia (24 g/L) was a feature in the serum of a dog, developed ascites due to segmental aplasia of caudal vena cava (Harder *et al.*, 2002).

Any decrease in serum albumin concentration resulted in a significant decrease in oncotic pressure with a resultant fluid shift from the intravascular space into the interstitium. This leads to hypotension, edema and body cavity



effusions. Albumin concentration less than 15 g/L commonly cause edema and effusions (Mc Grotty and Knottenbelt, 2002).

Varshney *et al.* (2002) reported low-protein ascites (0.3 – 2.0 g/dl) in peritoneal fluid and hypoalbuminemia (0.8 - 1.2 g/dl) in the serum of 7 dogs with refractory ascites. In this study, cirrhosis of liver was suspected.

Koide *et al.* (2004) evaluated the serum bio-chemical parameters of a dog with hepatic arteriovenous fistula, ascites, intrahepatic portosystemic shunt and aortic stenosis. It revealed mild microcytic hypochromic anaemia (MCV 59fl, MCHC 31.5 g/dl, and PCV 25%), prolonged clotting time, serious panhypoproteinemia (2.5 g/dl of total protein, 1.2 g/dl of albumin) and higher serum ALP concentration (1513 U/L).

Nephrotic syndrome characterized by increase in urine protein-creatinine ratio (>1.0), hypoalbuminemia (<1.5 g/dl), hypercholesterolemia (>240 mg/dl) and edema could be experimentally induced by administration of endotoxin and bovine serum albumin intravenously in dogs (Choi and Lee, 2004).

Ascites in dogs, which were experimentally infected with *Ehrlichia canis*, was resulted from vasculitis or hypoalbuminemia associated with hypergammaglobulinemia since the infected dogs had total protein levels within normal range (de Castro *et al.*, 2004).

### **2.5.2. Liver enzymes**

Kaneko *et al.* (1997) stated that normal healthy canine serum levels of ALT and ALP were 21 – 102 U/L ( $47 \pm 26$ ) and 20 – 156 U/L ( $66 \pm 36$ ) respectively.

Serum alkaline phosphatase (ALP) is an enzyme, which hydrolyses monophosphoric esters with the liberation of inorganic phosphate. It is present nearly in all tissues including red cells. The origin and the mechanism which causes the rise in serum levels of ALP in cases of hepatic dysfunction, especially

in obstructive jaundice is obscure. Raised ALP values were also found in young animals undergoing active growth of bone tissue, and in conditions in which there is interference in calcium and phosphorus metabolism. (Hoe and Harvey, 1977). Furthermore, the authors stated that 95.0 % of the dogs with ALP values exceeding 100 KA units showed liver damage, and 55.0 % with ALP values between 9 to 14 KA units showed some involvement with calcium and phosphorus metabolism. For instance an ALP value above 60 units gives a fairly definite indication of established liver damage, and even values above 20 units may be suspicious in the absence of other conditions known to cause increased ALP values.

Toth and Derwelis (1980) confirmed a case of hepatitis in a dog associated with drug hypersensitivity reaction by increased serum levels of glutamic-oxaloacetic transaminase (SGOT), glutamic-pyruvic transaminase (SGPT) and serum alkaline phosphatase (ALP).

A significant increase in ALP concentration was observed in the serum of young pups and its level was progressively decreasing as the age advanced and became static after seven months of age (Keller, 1981). The author was also opined that an increased concentration of ALT in serum of young dogs.

Bunch *et al.* (1982) observed ascites in dogs treated with anticonvulsant for two to three years. They stated that there was remarkable increase in the serum activities of ALT, ALP and GGT. Histological examination of the liver revealed macronodular or micronodular cirrhosis.

Crawford *et al.* (1985) observed an increase in liver enzyme activities and abnormal liver function tests consistently in all the cases of ascites associated with chronic active hepatitis in Doberman pinschers.

Maddison (1990) stated that the three most commonly used serum enzymes to assess hepatic disease in dogs were alanine aminotransferase (ALT), alkaline phosphatase (ALP) and  $\gamma$ - glutamyl transpeptidase (GGT). The author

had also pointed out that elevated serum concentrations of ALT indicate hepatocellular damage and the degree of elevation reflected the number of damaged hepatocytes and / or degree of damage. If the serum concentration of alanine aminotransferase (ALT) exceeded 300- 400 U/L was suggestive of moderate hepatocellular necrosis. In addition to that, the author opined that elevations could not be considered significant unless the level exceeds two or three times normal.

Rutgers *et al.* (1993) reported idiopathic hepatic fibrosis in 15 young dogs as a cause of ascites. Besides, erythrocyte microcytosis, hypoproteinemia, high serum activities of alkaline phosphatase and, to a smaller extent alanine aminotransferase were the consistent laboratory findings in all those dogs.

Sevelius (1995) stated that elevated ALT concentration was more prominent in chronic non-specific hepatitis and ALP was comparatively lower in chronic progressive hepatitis and cirrhosis. In cirrhotic dogs, serum ALT concentration of  $3.42 \pm 2.70$   $\mu\text{kat/l}$  and a serum ALP concentration of  $16.99 \pm 24.68$   $\mu\text{kat/l}$  were observed. Cirrhosis and chronic progressive hepatitis in 79 dogs showed normal to mildly increased concentrations of serum ALT and  $\gamma$ -Glutamyl transferase (GGT) and a moderate to marked increase in serum ALP concentration.

Low PCV, total serum protein, albumin and increased activities of liver enzymes in plasma and increased fasting levels of total bile acids and ammonia were observed in 42 dogs with portal hypertension associated with primary hypoplasia of portal vein (Van den Ingh *et al.*, 1995).

Greig *et al.* (1996) observed an increase in serum levels of alkaline phosphatase (ALP) in dogs infected with granulocytic ehrlichiosis.

In a study conducted in 626 randomly selected, clinically healthy Dobermans, it was found that 55 dogs had elevated levels of ALT and diagnosed as subclinical Doberman hepatitis. After the onset of clinical signs, there was a

decrease in the ALT levels and an increase in ALP concentration in serum as the disease progressed. But, the changes were not statistically significant (Speeti *et al.*, 1996).

Cornelius (1997) opined that the most common pathologic cause of increased serum ALP activity in dogs was glucocorticoid therapy. Long term use of otic, ophthalmic and probably topical glucocorticoid preparations also resulted in increased serum ALP. Osteoblastic activity in young growing animals and physiologic hyperphosphatemia were the situations in which elevated serum levels of ALP commonly seen. In-vitro levamisole inhibition test could be used to differentiate corticosteroid-induced increased ALP activity from increased ALP activity caused by cholestasis.

Leib (1997) observed that common laboratory findings in chronic active hepatitis were increased alanine aminotransferase (ALT) and alkaline phosphatase (ALP), hypoalbuminemia, hyperbilirubinemia, reduced BUN and anemia. Serum ALT concentration was often lower than the expected level with this severity owing to the lack of active inflammation in cirrhotic liver.

De Marco *et al.* (1998) reported that there was erythrocyte microcytosis, normal to mildly increased liver enzyme activities, increased concentrations of serum bile acids, reduced indocyanine green clearance and normal total bilirubin concentrations in four young, male Dobermans with idiopathic or noncirrhotic portal hypertension.

Experimentally induced cirrhosis in 10 healthy dogs by administering 0.5 ml/kg of carbon tetrachloride in olive oil (1:1) twice a week for five months by orogastric intubation resulted an increase in ALT ( $43 \pm 2.81$  to  $660.70 \pm 4.70$ ) and AST ( $40.40 \pm 1.13$  to  $369.40 \pm 3.28$  U/L) enzymes in serum (Ertekin *et al.*, 2003).

Koide *et al.* (2003) observed higher serum ALP concentration (1513 U/L) in a dog with hepatic arteriovenous fistula, ascites, intrahepatic portosystemic shunt and aortic stenosis.

Nottidge *et al.* (2003) studied a case of ascites associated with liver cirrhosis, and they arrived at conclusion that hypoalbuminemia and persistently decreasing liver enzymes were due to replacement of active hepatocytes by fibrous connective tissue. They also stated that the progressively decreasing values of these parameters were good indicators of deteriorating, non-responding condition of the liver despite therapy.

Serum bio-chemical values of dogs were significantly elevated and liver biopsy specimen showed foamy cytoplasm with mild to moderate hydropic changes at the end of the trial conducted by administering oxytetracycline (Vijayakumar *et al.*, 2004a).

### **2.5.3. Kidney Function Tests**

#### **2.5.3.1. Blood Urea Nitrogen (BUN)**

The serum samples of healthy dogs contained 10- 28 mg/dl of BUN (Kaneko *et al.*, 1997).

DiBartola *et al.* (1980) observed an increase in the level of BUN (> 80 mg/ 100ml) in 33.33% of dogs with proteinuria due to glomerular diseases.

Many non-renal factors altered the plasma urea and creatinine level. Post-prandial increase in the urea level was common irrespective of the type of food (Watson *et al.*, 1981).

Sevelius (1995) and Leib (1997) observed a reduction in BUN levels in all four types of chronic hepatitis (viz. chronic progressive hepatitis, chronic non-specific hepatitis, chronic cholangiohepatitis and liver cirrhosis) and the values had no statistically significant difference.

Harder *et al.* (2002) observed an increase in the levels of BUN (12.5 mmol/l) and creatinine (212  $\mu$ mol/ l) in a dog with ascites associated with segmental aplasia of caudal vena cava.

McGrotty and Knottenbelt (2002) reviewed the importance of urine protein: creatinine ratio in diagnosing proteinuria. They insisted that proteinuria should be concluded in the light of urine specific gravity and the authors also suggested that urine protein: creatinine ratio was a more accurate measurement of proteinuria and they stated that urine protein: creatinine ratio of more than 13 was normally associated with amyloidosis and glomerulonephritis.

Varshney *et al.* (2002) observed reduced BUN level in seven refractory cases of ascites in dogs. All these dogs were having low-protein ascites associated with portal hypertension and cirrhosis.

Nephrotic syndrome was experimentally induced by administration of endotoxin and bovine serum albumin intravenously in dogs. An increase in urine protein-creatinine ratio ( $> 1.0$ ), hypoalbuminemia ( $<1.5$  g/dl), hypercholesterolemia ( $>240$  mg/dl) and edema were the characteristics of nephrotic syndrome (Choi and Lee, 2004).

#### **2.5.3.2. Creatinine**

Normal healthy canine serum contained 0.5 – 1.5 mg/dl of creatinine (Kaneko *et al.*, 1997).

Serum biochemistry of 33.33% dogs with proteinuria revealed an increase in the concentrations of creatinine ( $> 2$  mg/ 100ml) with urine specific gravity less than 1.025 (DiBartola *et al.*, 1980).

Watson *et al.* (1981) opined that diet could alter the serum concentrations of creatinine and significant change was observed following feeding cooked meat to dogs.

#### **2.5.4. Sodium**

Kaneko *et al.* (1997) stated that healthy canine serum contained 141 – 152

mmol/l of sodium.

Unikowsky *et al.* (1983) concluded that by normalization intrahepatic pressure by providing an outflow tract for the cirrhotic liver (end-to-side anastomosis) abolished renal tubular sodium retention and formation of ascites.

Patients with nephrotic syndrome retained sodium, which contributes to ascites, edema, hypertension, and weight gain. Judicious use of loop diuretics was essential in managing edematous conditions (Fleming *et al.*, 1989).

Davis (1995) explained the existence of an extra-adrenal sodium retaining factor in congestive heart failure which increases the responsiveness of renal tubules to aldosterone so that sodium retention and edema results.

Serum sodium concentration represented the amount of sodium relative to the amount of water in extra cellular fluid and provided no direct information about the total body sodium concentration. Patients with hyponatremia or hypernatremia might have decreased normal or increased total body sodium content. Decreased serum sodium concentration (measured by flame photometry) with normal plasma osmolality was called pseudohyponatremia. This condition was due to hyperlipidemia or severe hypoproteinemia and it had no clinical significance (DiBartola, 2000). Added to that, the author opined that hyponatremia might develop due to thiazide or furosemide therapy.

Hyponatremia was primarily associated with renal sodium wasting and water retention due to an inability to excrete the ingested water. Usually, the hyponatremic patient became dehydrated if fluid intake was not compensating the urinary loss (Pak, 2000).

#### **2.5.5. Potassium**

The potassium concentration of healthy canine serum is 4.37 – 5.35 mmol/dl (Kaneko *et al.*, 1997). Signs associated with potassium imbalance could be expected when the serum concentration of potassium is below 2.5 mmol / l or

above 7.5 mmol / l. Pseudohyperkalemia might result from lysis of thrombocytes or red blood cells. Hypokalemia was sequelae of furosemide or thiazide diuretic administration (DiBartola and de Morais, 2000). Hypokalemia resulted from the shift of ion from intracellular to the extracellular compartment and decrease renal excretion of potassium (Pak, 2000).

## 2. 6. MANAGEMENTAL ASPECTS

### 2. 6. 1. Dietary Management

Dogs in mild congestive states might require only low restriction of sodium. This can be accomplished by a diet containing about 250 mg sodium / 100gm of dry diet, which results in an intake of 40 mg/kg body weight (Morris *et al.*, 1976). The authors also concluded that the sodium conservation mechanism in the dog is extremely efficient. Since salt is used ubiquitously in human foods, probably even a home-made diet would not be as deficient in sodium as to endanger a dog. They advised to restrict the dietary sodium to any level required to relieve congestion in cases of congestive heart failure.

Heise (1983) preferred strict reduction of patient's intake of long-chain triglycerides will be of use in managing lymphangiectasia and protein-losing enteropathy as even small amount of long-chain triglycerides could increase lymph flow. The author also stated that dietary management is the key to therapy in such cases.

Fleming *et al.* (1989) suggested restriction of sodium in diet (0.1 – 0.3 % of the diet), and low- protein mixed vegetable and animal protein diet for dogs with proteinuria and ascites.

Fascetti *et al.* (2003) studied the correlation between the plasma taurine concentration and DCM. All dogs with DCM had reduced plasma taurine concentrations ( $16 \pm 20$  nmol/ml) against the normal reference range of 2 – 64 nmol/ml and were treated with taurine (1000 to 3000 mg. P.O q.24.h).



Supplementation of amino acids like taurine and L-carnitine would be useful in the management of amino acid associated congestive heart failure. Supplementation of Omega-3 fatty acids will help to overcome the adverse effects of increased cytokine production (Moneva-Jordan, 2003).

### 2.6.2. General Therapy

Levy and Richard (1978) observed that a decline in central venous pressure and hemoconcentration following furosemide administration to cirrhotic dogs. But, there was lack of hemoconcentration, hyponatremia and maintenance of venous pressure after administration of mannitol.

Lumb *et al.* (1978) suggested that spironolactone, an aldosterone antagonist, was an effective drug in the treatment of certain forms of hypertension, ascites and edematous conditions in dogs. This study found no evidence to suggest that spironolactone was carcinogenic.

Jacobs (1989a) stated that furosemide is a potent drug, useful in patients with ascites due to right-sided CHF. The author also suggested that intravenous administration of furosemide @ 1-2 mg/lb followed by oral dosage of 1-2 mg/lb b.i.d was an effective mode of preload reduction and diuresis.

Spironolactone, a potassium sparing diuretic that competitively inhibits the bindings of aldosterone to mineralocorticoid receptors, was preferred for inducing diuresis in seven refractory cases of ascites in dogs (Varshney *et al.*, 2002).

Bressler *et al.* (2003) treated a case of ascites due to portal vein and aortic thrombosis with doxycycline and aspirin. They concluded that thrombi were primarily caused by canine ehrlichiosis.

Nottidge *et al.* (2003) reported that the non-responsiveness of the patient to furosemide diuresis was owing to poor renal perfusion resulting from reduced renal blood flow associated with congestive heart failure.

Uechi *et al.* (2003) compared the effects of loop diuretics, furosemide and torasemide on diuresis in dogs. In that study, they concluded that torasemide was effective in patients with edema caused by congestive heart failure and it also had high bioavailability (> 80%) and a rapid rate of absorption. Plasma aldosterone increased with torasemide, whereas no change with furosemide. Furosemide caused a dose-dependent increase in urine volume that peaked at 2 to 3 hour in dogs and cats. The diuretic effect of furosemide disappeared 6 hours after administration and the authors also suggested that the use of diuretics should depend on the degree of oedema and they should be used in combination with angiotensin converting enzyme inhibitors or angiotensin receptor blockers to avoid activation of renin- angiotensin system.

### 2.6.3. Liver

Martin *et al.* (1984) suggested that silibinin alone or used with choline conferred a good regenerative effect on hepatic tissue damaged by carbon tetra chloride by reducing the extent of lesion and the time needed for recovery.

Administration of furosemide, spironolactone, B-complex vitamins, liver extracts and amino acid infusions were considered as conventional therapy for managing ascites of hepatic origin. Addition of Liv-52 Vet tablets along with conventional therapy resulted in an early response with normalization of biochemical parameters and abated clinical symptoms more rapidly. The author also observed that, Liv-52 Vet tablets have beneficial effects in restoring liver function and in reducing ascitic distensions (Umesh, 2000).

It was concluded that silymarin @ 10 mg/kg body weight PO b.i.d along with oxytetracycline therapy was helpful in overcoming deleterious effects of oxytetracycline in the treatment of canine ehrlichiosis. It was also suggested that treatment with silymarin significantly increased the PCV and RBC count (Vijayakumar *et al.*, 2004a).

Administration of silymarin @ 10 mg/kg body weight orally resulted in reduction in the echogenicity of the liver from initial hyperechogenicity at 22<sup>nd</sup> day of trial. This was concluded that a treatment period of 21 days with above said dose was effective in hepatic diseases (Vijayakumar *et al.*, 2004b). The authors observed a pronounced increase in erythron and a significant reduction in leukogram, ALT, ALP, bile acids and bilirubin following treatment with silymarin.

A study conducted in 18 dogs with hepatic parenchymal disorders, concluded that silymarin @ 10 mg/kg b.i.d P.O for 21 days was more effective than phospholipids alone or in combination with silymarin (Vijayakumar *et al.*, 2004 c).

#### **2.6.4. Kidney**

Dimethylsulfoxide (DMSO) was given subcutaneously three times per week for one year to a dog suffered from amyloidosis and proteinuria. Despite controversy over the effectiveness and mechanism of action, the low toxicity of DMSO and the grave prognosis associated with amyloidosis necessitated the use of DMSO in the therapeutic regimen for amyloidosis (Spyridakis *et al.*, 1986).

Fleming *et al.* (1989) advocated the use of immunosuppressive agents like corticosteroids and aspirin (5-10 mg/kg/day) for dogs with ascites due to glomerular diseases and proteinuria.

Forrester (1997) preferred corticosteroids, azathioprine and cyclophosphomide as the drugs of choice for glomerular diseases.

#### **2.6.5. Heart**

McIntosh (1981) reviewed the efficacy of various vasodilators in relieving the congestive signs of CHF. The author also stated that vasodilators efficiently

reduced the left ventricular filling pressure and enhanced the cardiac output in patients with refractory CHF.

Jacobs (1989a) opined that intravenous administration of digitalis is rarely indicated in congestive heart failure. But, chronic oral inotropic support could be efficiently provided by administration of digoxin @ 0.003 to 0.004 mg/lb b.i.d. The author also elaborated toxic effects of digoxin such as secondary A-V block and other dysrhythmias.

Allworth *et al.* (1995) concluded that enalapril treatment resulted in a significant reduction in left atrial dimensions, end-systolic and end-diastolic left ventricular internal dimensions, and severity of clinical signs associated with right- sided heart failure in five dogs.

## ***Materials and methods***

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### **3. MATERIALS AND METHODS**

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy during the period from July 2004 to July 2005.

Dogs brought to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkala with abdominal distension were selected and utilized for the present study.

#### **3.1. SELECTION OF CASES**

Criteria for selection of clinical cases

1. Dogs with abdominal distension and fluid thrill on tactile percussion.
2. Associated signs like abdominal and respiratory discomfort, inappetance, melena, haematemesis, cachexia, limb edema and history of previous treatment for ascites.

Animals that were tentatively diagnosed were subjected to electrocardiography, echocardiography, abdominal ultrasound scanning and serum biochemical assay in addition to detailed clinical examination. Ten animals that were found to have abdominal effusion during ultrasound scanning formed materials for the study. All the diagnostic procedures were carried out on first, tenth and twenty first days of admission.

#### **3.2. OUTLINE OF STUDY**

##### **3.2.1. Clinical Examination**

Detailed clinical examination of ten selected dogs was done as per Houston (2000) and significant changes were recorded. The breed, sex, age of the animals was recorded.

### **3.2.2. Electrocardiography**

All the animals were subjected to electrocardiography as per the standard technique described by Bolton (1975) using BPL – CARDIART<sup>®</sup> 6108 machine. A standard six- lead electrocardiogram was recorded in BPL – CARDIART<sup>®</sup> paper strips at 25 mm/s paper speed and 1mV= 10 mm sensitivity.

#### ***3.2.2.1. Technique of electrocardiography***

Animals were restrained in right lateral recumbency and held with their humeri and femora at right angles to their body and parallel to each other. The limb leads were attached as per the method described by Bolton (1975) after applying electrode gel to establish skin- electrode contact. The recorded ECG tracings were photographed later.

Heart rate, rhythm, mean electrical axis and criteria for chamber enlargement were observed and significant changes were noted (Bolton, 1975).

### **3.2.3. Ultrasound scanning**

#### ***3.2.3.1. Equipment***

Selected animals were subjected to ultrasound scanning of liver, kidney, and heart using L&T SYMPHONY<sup>®</sup> 4.0 and HONDA HS 2000 VET<sup>®</sup> Scanners using 3.5, 5.0 and 7.5 MHz convex mechanical transducers.

#### ***3.2.3.2. Ultrasound scanning procedures***

##### **3.2.3.2.1. Peritoneal fluid**

Abdomen was scanned for the presence of anechoic fluid and the cellularity of the ascitic fluid was categorized as per the method described by Spaulding (1993) as anechoic, hypoechoic and mixed echoic/ cellular ascites.

#### 3.2.3.2.2. Liver

Animals were placed in dorsal recumbency, abdominal hairs were removed. Acoustic coupling gel was applied liberally to establish skin – transducer contact. Animal was placed in such a way that the cranial portion of the image was oriented to the viewers left on sagittal scan and right side of animal to viewers left on transverse scan. Imaging of liver was done by placing the transducer substernally and directing the beam cranially. Liver was also scanned in right dorsal and lateral inter costal approach using transverse and sagittal planes (Nyland *et al.*, 1995) (PLATE 1).

The ultrasonograms were reviewed for alterations in the echogenicity of the liver parenchyma, contour, hepatic vasculature and liver size. The echogenicity of the liver parenchyma was described as normal, hypoechoic, hyperechoic or mixed echoic (Nyland *et al.*, 1995). The recorded images were photographed.

#### 3.2.3.2.3. Kidney

The abdomen was prepared by clipping the hairs and applying acoustic coupling gel to the skin. Animals were positioned in dorsal recumbency. The left kidney was imaged with spleen as acoustic window caudal to the costal arch with firm pressure to avoid overlying bowel gas. The right kidney was imaged at the right eleventh or twelfth intercostal space. In animals with severely distended abdomen, kidneys were examined from the dorsal paralumbar region with the animal in sternal recumbency or in standing position (Nyland *et al.*, 1995). Renal ultrasonograms were evaluated for cortical and medullary echotextural changes.

#### 3.2.3.2.4. Heart

Echocardiography was done in all the patients. Both two- dimensional and M-mode echocardiography in left and right parasternal view of the heart was obtained using 7.5 MHz transducer. After shaving the hairs in both sides of the thorax from second to eighth intercostal space, acoustic coupling gel was applied to establish good transducer- skin contact. Animals were put either in left or right lateral





A



B



C

**Plate 1. Ascitic dog and scanning procedures**

- A. Dog: 7 with dilated cardiomyopathy and cardiac cachexia**
- B. Positioning for ultrasound examination of liver - dorsal recumbency**
- C. Echocardiographic examination- left parasternal approach using cut- hole made on the table.**

recumbency and the transducer was directed through the cut- out holes in the table (PLATE 1). Echocardiographic images were evaluated for dilatation or hypertrophy of cardiac chambers, kinesis of valves and walls, pericardial effusions, anatomical changes in valves and walls and contractility of the cardiac musculature.

End- diastolic diameter and end- systolic diameter were measured in M- mode echocardiography in selected cases and they were utilized for estimating end- diastolic volume and end- systolic volume, stroke volume and cardiac output (Moise and Fox, 1988).

#### **3.2.4. Abdominal Paracentesis**

After properly restraining the animal, ventral abdominal hairs were clipped and scrubbed with antiseptics. Using 20 G needle, a puncture was made at a point right lateral to umbilicus avoiding subcutaneous blood vessels. Ascitic fluid was collected in sterile screw- capped vial and subjected for biochemical analysis. In animals with severe abdominal distension and respiratory discomfort, 250- 500 ml of ascitic fluid was tapped to give ease to them (Barrett, 1975).

### **3.3. CLINICAL PATHOLOGY**

#### **3.3.1. Collection of clinical material**

Clinical materials were collected on first, tenth and twenty first days of admission. Five milliliter of whole blood was collected from saphenous or cephalic vein of the patient in dry glass vials with EDTA @ 1-2 mg per milliliter as anti- coagulant (Benjamin, 1998).

Ten milliliter of blood was collected in a clean, dry test tube for seperating serum for biochemical analysis. Sera thus separated were stored at -20° C till further analysis.

In suspected cases of nephrotic syndrome, urine was collected using urinary catheter and qualitative assessment was done using URISTICK®.

In suspected cases of canine ehrlichiosis, a peripheral blood smear and a buffy coat smear were collected and examined for morula and inclusion bodies for confirmatory diagnosis.

### 3.3.2. Hemoglobin and Packed cell volume

Haemoglobin concentration was estimated using Sahli's haemometer and packed cell volume (PCV) was measured using microhematocrit tube as described by Schalm *et al.* (1975).

### 3.3.3. Serum biochemistry

Total serum protein, albumin, globulin, A: G ratio, liver enzymes including alanine aminotransferase (ALT) and alkaline phosphatase (ALP), blood urea nitrogen (BUN), creatinine, serum sodium and potassium were estimated. All biochemical estimations were done by spectrophotometry in Merck 200 spectrophotometer using commercially available kits. Serum sodium and potassium concentrations were estimated using SYSTRONICS 128<sup>®</sup> flame photometer.

The total protein<sup>1</sup> in serum and ascitic fluid were estimated by modified Biuret method as described by Weichselbaum (1946). While albumin<sup>2</sup> in serum was estimated by bromocresol green dye binding method as described by Doumas *et al.* (1971). Alanine aminotransferase<sup>3</sup> (ALT) was estimated based on the reference method of International Federation of Clinical Chemistry (IFCC). Alkaline phosphatase<sup>4</sup> (ALP) was measured in accordance with the recommendations of Deutsche Gesellschaft fur klinische chemie (Anon, 1970).

Blood urea nitrogen<sup>5</sup> (BUN) was estimated by modified Berthelot method (Wheatherburn, 1967) and the serum creatinine<sup>6</sup> was estimated using modified Jaffe's method (Allen, 1982). The "ascitic fluid to plasma protein ratio" was calculated by dividing protein content of ascitic fluid by the plasma protein concentration and the ratio was expressed as a percentage (Hunt *et al.*, 1993).

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1 Merck Ecoline Total protein 2 Merck Ecoline Albumin 3. Merck Ecoline ALAT tris GPT 4. Merck Ecoline ALP 5. Aggape LyphoChek Urea UV 6. Aggape ChemChek creatinine.

### 3.4. COURSE OF ILLNESS

Animals were observed from the date of admission to the date of discharge / death. In the event of mortality during the course of clinical investigation and treatment, post-mortem examination was done in the Department of Veterinary Pathology, College of Veterinary and Animal Sciences, Mannuthy. Gross pathological features of the tissues were noted if significant. The correlation between ultrasonographic and post-mortem findings were studied.

### 3.5. THERAPEUTIC MANAGEMENT

On the day of admission, all the cases were treated based on the tentative diagnosis. After confirmation, necessary changes were made in the therapeutic regimen. Treatment regimen was designed according to the nature of illness and the organ/ system involved. As ascites is a hyperhydremic state, diuresis was facilitated by oral administration of furosemide (2 mg / kg b.i.d PO for 20 days) or spironolactone (2 mg/kg b.i.d PO for 20 days) or their combination (LASILACTONE<sup>®</sup>) as diuretic and silymarin (10 mg/kg b.i.d P.O), urso deoxycholic acid (URSOFALK<sup>®</sup>) @ 10 mg/kg b.i.d PO for 10 days and prednisolone (1 mg/kg PO) in hepatic disorders. In cases of congestive heart failure, administration of LASILACTONE<sup>®</sup> (2 mg/ kg b.i.d P.O for 20 days), enalapril (0.5 mg / kg P.O for 20 days), and digoxin were made routine. In nephrotic syndromes, administration of prednisolone @ 1 mg / kg P.O was adopted. Response to treatment was assessed at regular intervals till the date of discharge / death of the patient whichever was earlier.

## *Results*

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## 4. RESULTS

Dogs presented to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kakkala with clinical signs of ascites formed the material for the present study. A total of ten confirmed cases falling under various breed, sex and age were studied in detail.

All the cases were subjected to electrocardiography (ECG), ultrasonography of liver, kidney and heart, serum bio-chemical assays and abdominal paracentesis. According to the test results and findings, these cases were classified into ascites associated with liver, kidney and heart diseases.

### → 4.1 CLASSIFICATION OF CASES.

Among the ten cases subjected for study, ascites associated with liver, kidney and heart diseases were confirmed in five, three and two cases respectively. Signalments of the 10-ascitic dogs were given in Table 1.

#### 4.1.1. Age

Age-wise prevalence of ascites revealed that dogs belonging to four to seven year of age were more frequently affected followed by three years of age group.

#### 4.1.2. Breed

Breed-wise prevalence of ascites revealed that Dachshunds were the most commonly affected breed followed by Boxers, Spitz, Rottweiler, Labrador retriever, Doberman pinchers and mongrel breeds.

#### 4.1.3. Sex

Among the dogs studied, eight dogs were males and two dogs were females.

Table 1. Signalments of dogs with ascites

Dog No:	Out patient No:	Breed	Age (in years)	Sex	Organ involved
1	6921	Spitz	5	Male	Liver
2	6217	Dachshund	3	Male	Liver
3	3369	Labrador retriever	4	Male	Liver
4	2387	Rottweiler	1	Female	Liver
5	6677	Boxer	6	Male	Liver
6	9904	Dachshund	3	Female	Heart
7	7291	Boxer	7	Male	Heart
8	9272	Mongrel	15	Male	Heart
9	6877	Dachshund	6	Male	Kidney
10	1434	Doberman Pinscher	3	Male	Kidney

## 4.2. ASCITES OF HEPATIC ORIGIN

Age of the affected dogs ranged from one to six years with a mean of 3.5 years. One dog each belonged to Boxer, Dachshund, Rottweiler, Spitz and Labrador retriever breeds. Out of the five dogs found to have ascites of hepatic origin four dogs were males.

### 4.2.1. Clinical examination findings

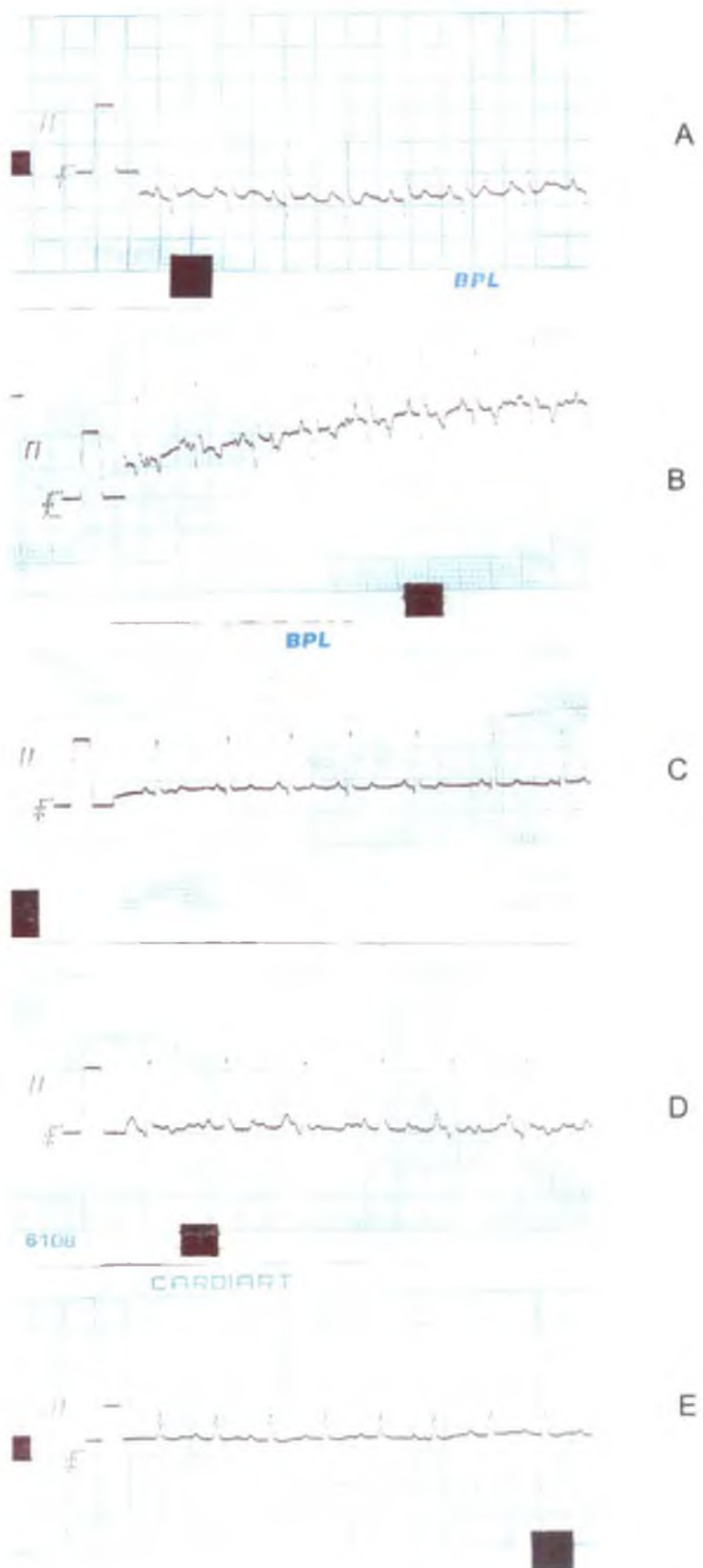
All the five dogs had moderate to marked abdominal distension. Fluid thrill was observed in all cases on tactile percussion of abdomen. Four out of five dogs were lethargic. Conjunctival mucous membrane was congested in four dogs and pale roseate in one dog. Rectal temperature ranged from 101°F to 104.4°F. All the five dogs were inappetent.

The heart rate ranged from 120 - 180 beats per minute (bpm). No abnormalities were observed in the volume, consistency, and colour of faeces in all those dogs. Four out of five dogs passed deep- yellow coloured urine. One dog was voiding normal (straw- yellow coloured) urine. Three dogs showed respiratory distress on standing position, while, all the five dogs had respiratory discomfort in lateral recumbency. In all the five dogs, liver was not palpable.

### 4.2.2. Electrocardiographic findings

Among the five cases diagnosed as ascites of hepatic origin, no marked abnormalities could be detected in the ECG parameters and mean electrical axis. All the five cases had small 'QRS' complexes (PLATE 2). Four dogs had 'P' duration of 0.04 sec and one dog had 0.08 sec on the day of admission. 'P' amplitude was within normal limits in all five cases (0.1- 0.2 mV). P-R interval ranged from 0.08 to 0.16 sec. The 'R' amplitude ranged from 0.4 – 1.1 mV. The 'QRS' duration in all the five dogs was 0.04 sec. The polarity of 'T' waves was positive in four dogs and negative in one dog. The *mean electrical axis (MEA)* of





**Plate 2. Lead II ECG tracings of dogs with ascites of hepatic origin  
 A. Dog: 1; B. Dog: 2; C. Dog: 3; D. Dog: 4; E. Dog: 5  
 Small 'QRS' complexes in all the dogs**

all the five dogs was within the normal limits, ranged from +60 ° to +90°. The ECG parameters of ascitic dogs are presented in Table 2.

### **4.2.3. Ultrasonographic findings**

#### **4.2.3.1 Ascitic fluid**

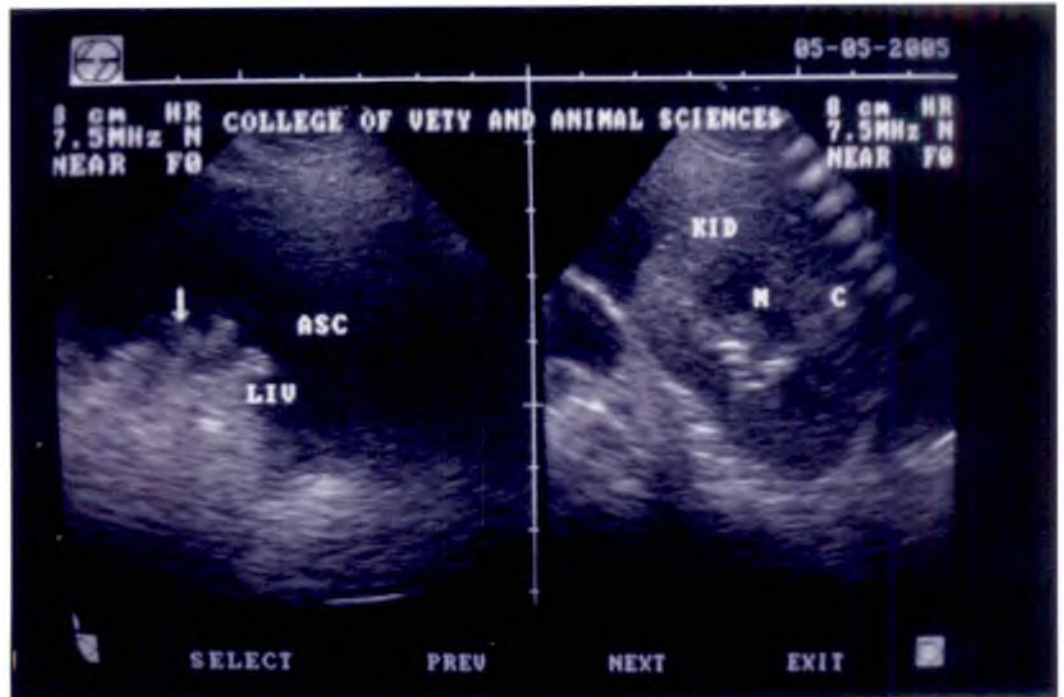
Ultrasonographic examination of abdomen revealed anechoic fluid and floating organs in four out of five cases and one case (Dog: 5) had mixed echoic fluid with echogenic, minute, floating particles (cellular ascites). Four cases had no difference between the echogenicity of ascitic fluid and urine. The ascitic fluid of dog: 5 was comparatively echogenic than urine. All the five cases revealed hypoechoic discontinuity in the urinary bladder wall. Acoustic enhancement phenomenon was observed in all the cases while examining the echogenicity of abdominal viscera.

#### **4.2.3.2 Liver**

Lobular demarcation of the liver was clear in all the five cases due to the presence of anechoic fluid. Generalized increase in the echogenicity of liver in far-field views obtained through sub-costal approach was observed in all the five cases. This pseudohyperechogenicity in liver parenchyma was markedly reduced when the probing was done through 8<sup>th</sup> to 12<sup>th</sup> intercostal space.

Four cases had diffused hyperechogenicity of liver parenchyma. Specks of hyperechoic areas were observed in the liver parenchyma of one dog. The borders of the liver lobes were even and normal in three cases and rest had uneven borders. The irregularity of the liver borders was marked in dogs: 3 and 5 (PLATE 3).

The size of the gall bladder and the echogenicity of the contents were normal in four cases. Two dogs (dogs: 3 and 5) had distended gall bladder. Mixed echoic gall bladder contents were observed in dog: 3 (PLATE 3).



A



B

**Plate 3. Cirrhotic liver**

**A. Uneven and eroded borders (Arrow) of liver in Dog: 5  
(longitudinal plane)**

**B. Hyperechoic liver parenchyma and uneven borders in Dog: 3  
(longitudinal plane)**

#### **4.2.3.3. Kidney**

Both the kidneys were scanned in transverse, longitudinal and sagittal planes. No marked change in the echogenicity of renal parenchyma was observed in four dogs. One dog (dog: 5) had slight increase in the echogenicity of the renal cortex. Scanning the kidney while the animal was in standing position reduced acoustic enhancement of the renal parenchyma.

#### **4.2.3.4. Echocardiography**

No significant changes were observed in the chamber dimension and contractility of the myocardium of all the five dogs.

#### **4.2.4. Hemoglobin and packed cell volume**

The haematological values are presented in Table 3. All the five dogs suffered from mild degree of anemia. Hemoglobin concentration ranged from 6.7 – 11.0 g % on the day of admission. In this group of dogs, PCV ranged from 25 – 36% on the day of admission. On 10<sup>th</sup> day of examination, PCV and Hb ranged from 30 - 42% and 8.5- 11 g% respectively. The corresponding values were 32-46 % and 9.0 – 13 g% on 21<sup>st</sup> day of observation. The platelet count was done in all the five dogs. Dogs: 1 and 4 had reduced thrombocyte count. The thrombocyte count of dog: 1 was 80,000/ cu. mm and that of dog: 4 was 93,000/ cu. mm on the day of admission.

#### **4.2.5. Serum Biochemistry**

The serum biochemical values are given in Table 3.

##### **4.2.5.1. Liver enzymes**

Mild to marked elevation in the serum alanine aminotransferase (ALT) concentration was observed in all the five dogs on the day of admission. The serum levels of ALT in affected dogs ranged from 126 – 393 U/L. Invariably, all the five dogs had elevated alkaline phosphatase (ALP) concentration in the serum



and values ranged from 166 to 2343 U/L on the day of admission. On 10<sup>th</sup> day of observation, concentrations of ALT and ALP ranged from 118- 173 U/L and 81 - 482 U/L respectively. On 21<sup>st</sup> day of examination, the corresponding values were 78- 136 U/L and 73- 359 U/L.

#### **4.2.5.2. Serum proteins**

Serum total protein, albumin and globulin concentrations of healthy dogs were 54.0-71.0 g/L, 26.0-33.0 g/L and 27.0-44.0 g/L respectively. The total serum protein was markedly reduced (< 40 g/L) in one case and mild reduction was observed in three cases between (40 – 60 g/L). One dog (dog: 1) had a total serum protein concentration of 65 g/L. The total protein concentration ranged from 43- 66 g/L and 51 – 65 g/L on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively.

On first day of examination, the serum albumin concentration was reduced in all the five cases and it ranged from 15 to 22 g/L. The serum albumin concentration ranged from 14 – 22 g/L on 10<sup>th</sup> day and 22 – 31 g/L on 21<sup>st</sup> day of observation.

On the day of admission, the serum globulin concentration was mildly elevated in dog: 1 (45 g/L). Other dogs had normal serum globulin level (22 – 37 g/L). The serum globulin concentration ranged from 21- 47 g/L on 10<sup>th</sup> day and it was 20 – 43 g/L on 21<sup>st</sup> day of observation. The A: G ratio ranged from 0.44- 0.68 on the day of admission. The corresponding values were 0.09 – 0.48 and 0.51- 1.55 on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively.

The protein content of ascitic fluid ranged from 6 to 26 g/L on the day of admission. The ascitic fluid protein content ranged from 2- 20 g/L and 13 – 25 g/L on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively. Ascitic fluid to plasma protein ratio ranged from 11 to 70% on the day of admission. The corresponding values were 3.23 – 41.87 % and 21.7 – 38.5 % on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively.

#### **4.2.5.3. Kidney function tests**

##### **4.2.5.3.1. Blood urea nitrogen (BUN)**

On the day of admission, BUN levels in all these dogs were within the normal reference range (10- 28 mg/dl). The level of BUN ranged from 10 -17 mg/dl on 10<sup>th</sup> day and 10 - 22 mg/dl on 21<sup>st</sup> day of examination respectively.

##### **4.2.5.3.2. Creatinine**

The serum creatinine concentration was within normal range in all the five dogs. The serum creatinine concentration ranged between 0.97 to 1.9 mg/dl on the day of admission. The corresponding values were 0.7 - 0.9 mg/dl on 10<sup>th</sup> day and 0.8 – 1.7 mg/dl on 21<sup>st</sup> day of observation.

##### **4.2.5.4. Sodium**

Two dogs (Dogs: 1 and 2) were comparatively hyponatremic and remaining dogs were normonatremic on the day of admission. The serum sodium concentration ranged from 136 - 152 mmol/l on the day of admission. The corresponding values were 135 – 142 mmol/l and 131 – 155 mmol/l on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively.

##### **4.2.5.5. Potassium**

On the day of admission, all the five dogs were normokalemic, serum potassium concentration ranged from 3.7 to 5.2 mmol/l. The serum potassium concentrations were 4.20 – 5.50 mmol/l and 4.40 – 6.20 mmol/l on 10<sup>th</sup> and 21<sup>st</sup> day of observation respectively.

Table 2. Electrocardiographic findings in dogs with ascites of hepatic origin

PARAMETERS	1 <sup>ST</sup> DAY					10 <sup>TH</sup> DAY					21 <sup>ST</sup> DAY				
	Case No:														
	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
'P' duration (sec)	0.04	0.04	0.04	0.08	0.04	0.04	0.04	0.04	0.04	-	0.04	0.04	0.04	0.04	-
'P' amplitude (mV)	0.15	0.1	0.1	0.2	0.1	0.1	0.1	0.1	0.1	-	0.1	0.1	0.1	0.1	-
P-R interval(sec)	0.12	0.10	0.08	0.12	0.16	0.08	0.12	0.06	0.08	-	0.06	0.08	0.06	0.08	-
QRS duration (sec)	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	0.04	-	0.04	0.04	0.04	0.06	-
'R' amplitude (mV)	1.1	0.9	1.0	1.1	0.4	2.0	1.4	1.0	1.8	-	2.1	1.7	1.3	1.7	-
'T' wave polarity	+ve	-ve	+ve	+ve	+ve	+ve	-ve	+ve	+ve	-	+ve	+ve	+ve	+ve	-
S-T segment	N	N	N	N	N	N	N	N	N	-	N	N	N	N	-
MEA ( degrees)	+60°	+60°	+60°	+90°	+90°	+60°	+60°	+60°	+90°	-	+60°	+60°	+60°	+60°	-
Heart rate (bpm)	150	150	180	120	150	160	128	150	120	-	160	150	150	120	-

N :Normal

+ve : Positive

-ve : Negative

- : Animal died during the course of study

### 4.3. ASCITES OF CARDIAC ORIGIN

The signalments of this group of dogs are presented in Table 1. Age of the affected dogs ranged from 3 to 15 years and they belonged to Dachshund, Boxer, and Mongrel breeds. Two dogs were males and one dog was female.

#### 4.3.1. Clinical findings

All the three dogs had visible abdominal distension and fluid waves were felt on tactile percussion of abdomen. The rectal temperatures were 102.6°F, 104°F and 101°F respectively in dogs 6, 7 and 8 on the day of admission. Vaccinations and deworming were regular in two dogs. One dog (Dog 8) was not having proper record of vaccinations and deworming.

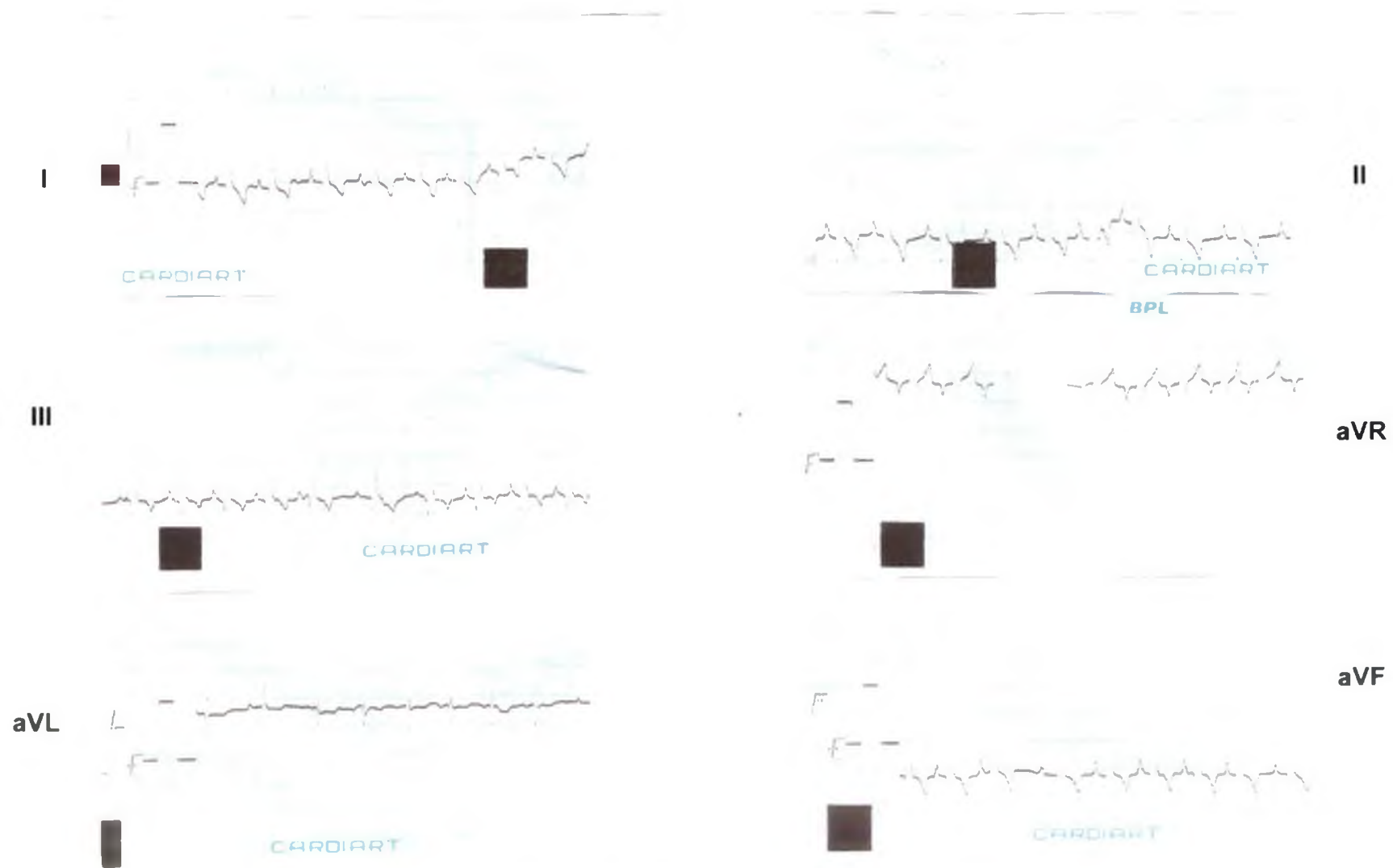
The heart rates ranged between 120 to 210 bpm. Conjunctival mucous membrane was pale-roseate in all the three dogs. Two dogs (dog: 7 and 8) were having poor appetite. The appetite was normal in one dog (dog: 6). Thoracic auscultation revealed respiratory distress and loud heart sounds in all the three cases.

Cardiac palpitation was observed in two dogs (dogs: 7 and 8). Strong pulsation of femoral artery was felt in all the three dogs. Urine was invariably deep-yellow in all those dogs. Two dogs were already treated with diuretics (furosemide) and liver extracts by local veterinarians.

#### 4.3.2. Electrocardiographic findings

Electrocardiographic findings in the three dogs are placed in Table 4. Electrocardiographic findings in the three dogs with ascites of cardiac origin were variable. The 'P' duration was within the normal limit in dog: 7 and remaining two dogs had increased 'P' duration viz 0.06 and 0.08 sec. The 'P' amplitude was at its maximum (0.4mV) in dog no: 7. Other two dogs had 'P' amplitude below 0.4 mV (i.e. 0.3 and 0.2 mV). The 'P-R' intervals of dogs 6, 7 and 8 were 0.12, 0.12





**Plate 4. ECG of Dog: 6 (Hypertrophic cardiomyopathy)**  
Deep 'Q' waves in Leads I, II and aVF  
Tall 'R' waves (3.3 mV)

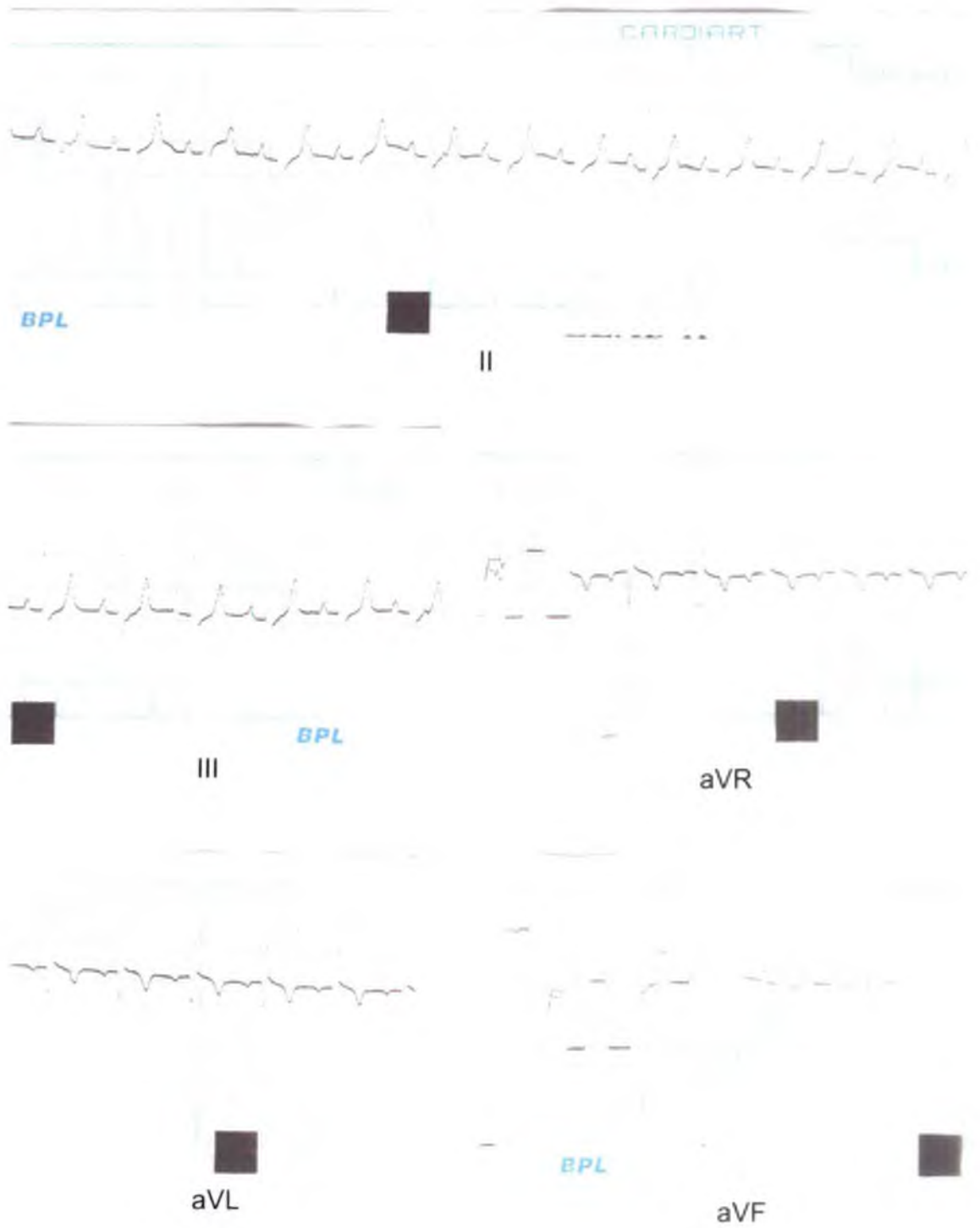


Plate 5. ECG of Dog: 7 with dilated cardiomyopathy  
- Deep 'S' waves  
Peaking of 'T' waves

and 0.13 sec respectively. Deep 'Q' waves were observed in all the three cases (> 0.5 mV) and deep 'S' waves were seen in two cases (dogs: 7 and 8). 'S-T' slurring was observed in dog: 6 (PLATE 4).

The 'R' amplitude was markedly elevated in two dogs (dogs: 6 and 8) even with the presence of peritoneal fluid. They were 3.3 mV and 2.5 mV respectively in those cases. Dog: 7 had a small 'R' wave with a height of 0.9 mV (PLATE 5). The "QRS" duration was normal in one dog (Dog: 7) and it was increased in other two dogs. The mean 'QRS' duration was 0.06 sec. The mean electrical axis was +60° in all the three cases. The polarity of 'T' wave was negative in two cases (dogs: 6 and 8) and dog: 7 had a positive, tall 'T' wave (0.6 mV). No marked change in the ECG of 10<sup>th</sup> and 21<sup>st</sup> day of observation could be observed when compared to the first day tracings. The ECG tracings of dog: 8 is presented in PLATE 6.

### **4.3.3. Ultrasonographic findings**

#### **4.3.3.1. Ascitic fluid**

Dog: 6 was found to have anechoic peritoneal fluid, which was comparatively less echoic than urine in the urinary bladder. Two cases (dogs: 7 and 8) had cellular ascites with multiple echogenic particles floating all over the anechoic fluid (PLATE 7). All the three dogs had pseudohypoechoic wall discontinuity of urinary bladder. Acoustic enhancement phenomenon was observed in abdominal viscera of all the three cases.

#### **4.3.3.2. Liver**

No marked changes observed in the echotexture of liver in all the three cases. Liver was mildly enlarged, projecting beyond the costal arch in dogs 6 and 7. Liver size was normal in dog: 8. Dilated hepatic vessels and caudal vena cava was observed in dogs 6 and 7 (PLATE 8).

#### **4.3.3.3. Kidney**

Cortico-medullary distinction was clear in all the three cases. There was slight increase in the echogenicity of renal cortex as multiple pointed foci observed in all the three cases. No abnormal focal or diffused lesions could be observed in renal cortex and medulla of all the three dogs.

#### **4.3.3.4. Heart**

On two- dimensional echocardiographic examination, bi- ventricular enlargement was noticed in two cases (dogs: 6 and 7). Dog: 8 revealed right ventricular dilatation in M- mode echocardiography and B- mode echocardiography. Dogs 7 and 8 had dilatation of ventricles and dog: 6 was having hypertrophied right and left ventricle.

Dilatation of left ventricle was marked in dog: 7 with thin interventricular septum and ventricular walls (PLATE 9). Left parasternal approach was efficient in studying the right ventricular changes. Myocardial contractility was diminished in that case. On the day of admission, the left ventricular internal dimension at end- diastole (LVIDd) was 4.2 cm and the left ventricular dimension internal at end-systole (LVIDs) was 3.6 cm. According to the formula stated by Moise and Fox (1988), the end- diastolic volume (EDV) was 527.50 ml, end systolic volume (ESV) 304.20 ml, stroke volume 223.30 ml, cardiac output 2.68 L/ min, ejection fraction 42.33%, and fractional shortening (FS) 14.28%.

Dog: 8 was having isolated right ventricular dilatation which was evident in M- mode echocardiography. 2-D echocardiography of left ventricle yielded no marked abnormality. Myocardial contractility was normal in two cases except dog: 7. Myocardial contractility was sluggish in that dog. Two- dimensional echocardiography revealed no change on 10<sup>th</sup> day from the 1<sup>st</sup> day observation.

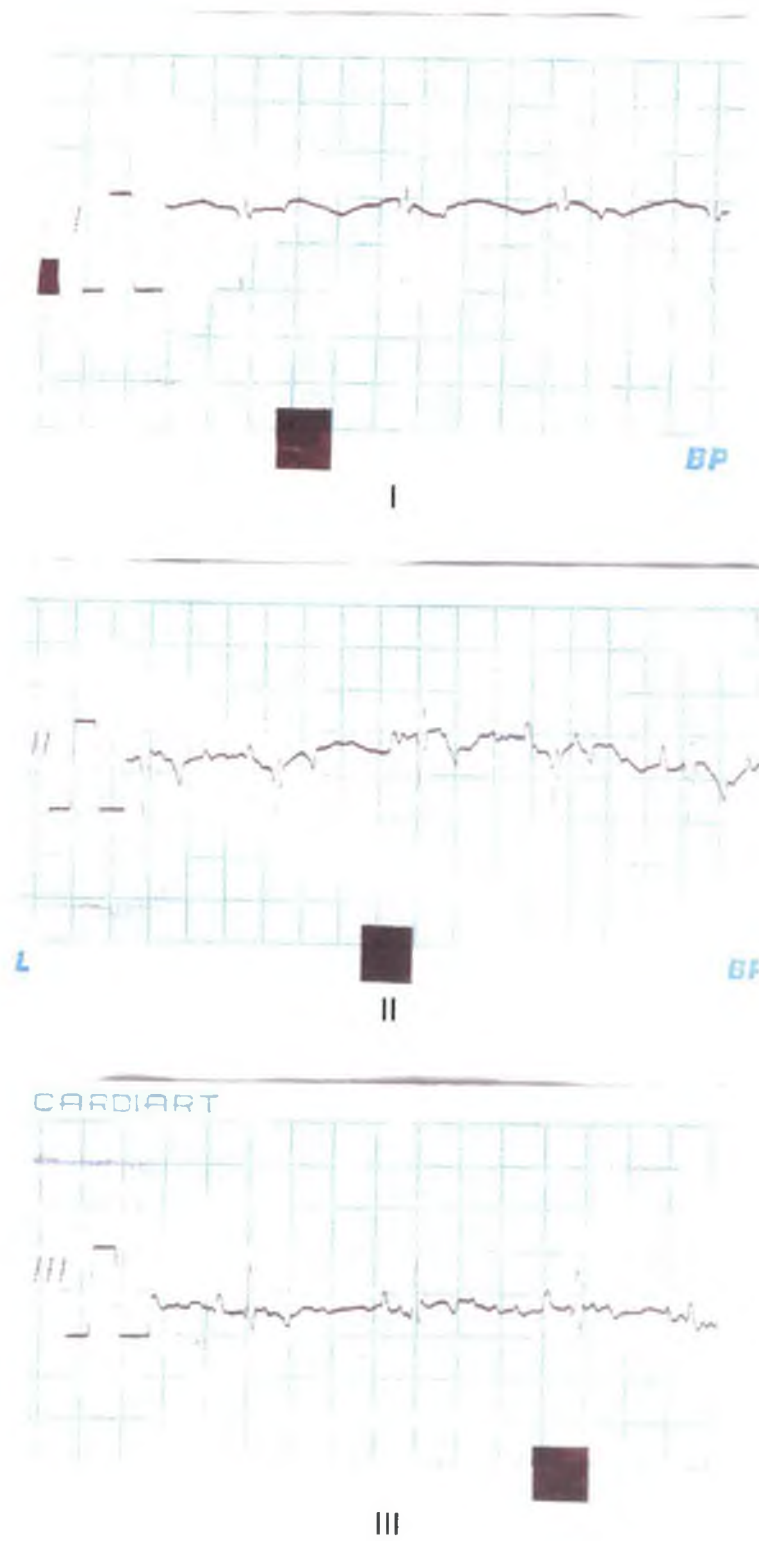


Plate 6. ECG of Dog: 8  
- Deep 'Q' waves  
- Deep 'S' waves  
'W' pattern of "QRS" complexes





**Plate 7. Ultrasonography of general abdomen in Dog: 7**  
**A. Echogenic floating particles in anechoic ascitic fluid (cellular ascites).**  
**B. Turbidity of ascitic fluid (cellular ascitic fluid) of Dog: 7**  
**C. Urinary bladder wall hypoechoic pseudo discontinuity (arrow).**



A



B

**Plate 8. Hepatic ultrasonogram of a dog with CHF**

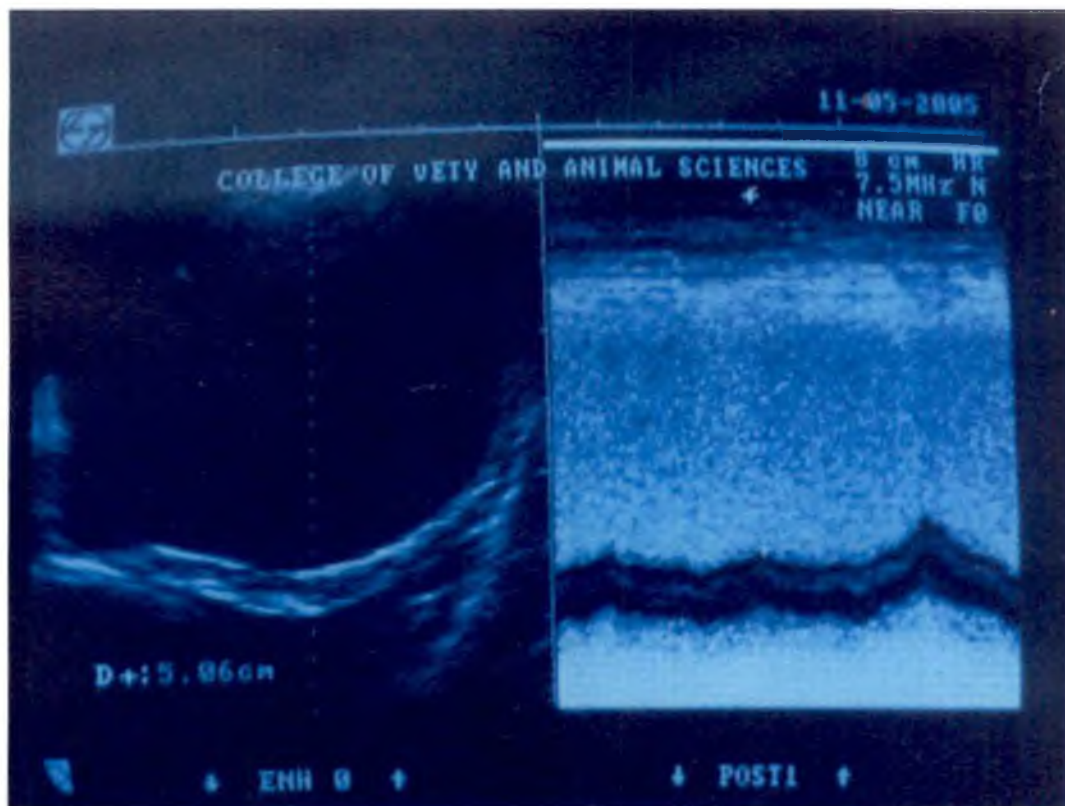
**A. Enlarged liver with rounded borders.**

**B. Dilated hepatic veins and caudal vena cava (longitudinal plane)**





A



B

Plate 9. Echocardiogram of a dog with DCM (Dog: 7)

A. Left parasternal short-axis view. Dilated ventricles and thin interventricular septum

B. Left parasternal short-axis view in B + M mode. Marked dilatation of ventricles



#### **4.3.4 Hemoglobin and Packed cell volume**

All the three dogs had normal hemoglobin concentration ranged from 11 g% to 13 g% and packed cell volume (PCV) ranged from 22% to 42% on the day of admission. On 10<sup>th</sup> day examination, the Hb and PCV values ranged from 10 – 12 g% and 38 – 45% respectively. On 21<sup>st</sup> day of examination, the PCV was 42% and the Hb was 12g% in dog: 6 (Table 5).

#### **4.3.5. Serum biochemistry**

The serum biochemical values of this group of dogs are given in Table 5.

##### ***4.3.5.1. Liver enzymes***

The liver enzymes (ALT and ALP) in the serum of affected dogs were within the reference range during the period of study.

##### ***4.3.5.2. Serum proteins***

On the day of admission, the total serum protein concentrations of ascitic dogs were 83 g/L, 52 g/L and 73 g/L respectively. On 10<sup>th</sup> day of examination, corresponding values were 70 g/L, 48 g/L and 61 g/L respectively. On the day of admission, the albumin concentration in serum of two dogs (Dogs 6 and 8) remained elevated (42 g/L and 53 g/L) respectively. But, one dog (Dog: 7) had hypoalbuminemia with an albumin concentration of 20 g/L. On 10<sup>th</sup> day of observation, serum albumin concentration ranged from 20 to 53 g/L.

On the day of admission, the globulin concentration in the serum of two dogs (dogs 6 and 7) was within the reference range. They had 44 g/L and 32 g/L of globulin respectively. One dog (dog: 8) was hypoglobulinemic, had a globulin concentration of 17 g/L. On 10<sup>th</sup> day, the serum globulin concentrations were 28 g/L, 28 g/L and 8 g/L respectively in dogs 6, 7 and 8. The A: G ratio of two dogs (dogs 6 and 7) remained normal on the day of admission. Dog: 8 had an A: G ratio of 3.3. On 10<sup>th</sup> day of observation, the A: G ratio ranged from 0.71 – 6.6.

#### ***4.3.5.2.1. Protein concentrations in ascitic fluid***

Invariably, all the three dogs had > 25 g/L of protein in ascitic fluids. Dogs 6, 7 and 8 had a protein concentration of 32 g/L, 27 g/L and 45 g/L respectively on the day of admission. The corresponding values were 18 g/L, 27 g/L and 43 g/L respectively on 10<sup>th</sup> day of examination. The ascitic fluid to plasma protein ratio ranged from 39 % to 62 % on the day of admission. It ranged from 25.71% to 70.5% on 10<sup>th</sup> day of observation.

#### ***4.3.5.3. Kidney function tests***

##### **4.3.5.3.1 Blood urea nitrogen (BUN)**

The BUN levels of the affected dogs were within the reference range during the period of study.

##### **4.3.5.3.2. Creatinine**

The serum creatinine concentrations of the affected dogs were within the reference range during the period of study.

##### **4.3.5.4. Sodium**

The serum sodium concentration was variable among these dogs. Dog: 6 was hypernatremic (167 mmol/l), dog:7 was normonatremic (148 mmol/l) and dog: 8 was hyponatremic (136 mmol/l) on the day of admission. On 10<sup>th</sup> day of observation, the corresponding values were 142 mmol/l, 133 mmol/l and 158 mmol/l respectively.

##### **4.3.5.5. Potassium**

On the day of admission, mild hypokalemia (4.2 mmol/l) was noticed in two dogs (dogs: 6 and 8). Mild hyperkalemia was observed in dog: 7 with serum potassium concentration of 5.6 mmol/l. On 10<sup>th</sup> day of examination, the serum potassium concentration ranged from 4.3 to 5.9 mmol/l.

Table 4. Electrocardiographic findings in dogs with ascites of cardiac origin

PARAMETERS	1 <sup>ST</sup> DAY			10 <sup>TH</sup> DAY			21 <sup>ST</sup> DAY		
	Case No:								
	6	7	8	6	7	8	6	7	8
'P' duration (sec)	0.06	0.04	0.08	0.06	0.08	0.08	0.04	-	-
'P' amplitude (mV)	0.3	0.2	0.4	0.2	0.2	0.5	0.3	-	-
P-R interval (sec)	0.12	0.12	0.13	0.16	0.12	0.16	0.16	-	-
QRS duration (sec)	0.06	0.04	0.08	0.04	0.04	0.08	0.04	-	-
'R' amplitude (mV)	3.3	0.9	2.5	3.0	0.9	3.2	2.4	-	-
'T' wave polarity	-ve	+ve, peak	-ve	-ve	+ve, peak	-ve	-ve	-	-
S-T segment	Slurring	Deep 'S'	Deep 'S'	Normal	Normal	Deep 'S'	Normal	-	-
MEA ( degrees)	+60°	+60°	+60°	+60°	Electri- cally vertical	+60°	+60°	-	-
Heart rate (bpm)	210	120	120	168	120	128	164	-	-

N :

Normal

+ve : Positive

-ve : Negative

- : Animal died during the course of study

Table 5. Haematological and serum bio-chemical parameters in dogs with ascites of cardiac origin

PARAMETERS	1 <sup>ST</sup> DAY			10 <sup>TH</sup> DAY			21 <sup>ST</sup> DAY		
	Case No:								
	6	7	8	6	7	8	6	7	8
Hb (g %)	11	13	12.5	10	12	12	12	-	-
PCV (%)	22	38	42	38	43	45	42	-	-
ALT (U/L)	29	47	19	32	475	37	37	-	-
ALP (U/L)	52	83	99	47	160	115	57	-	-
Total protein (g/L)	83	52	73	70	48	61	73	-	-
Albumin (g/L)	39	20	56	42	20	53	51	-	-
Globulin (g/L)	44	32	17	28	28	8	22	-	-
A:G ratio	0.89	0.63	3.3	1.5	0.71	6.6	2.32	-	-
Ascitic fluid Protein (g/L)	32	27	45	18	27	43	13	-	-
AFPP ratio (%)	39	51	62	25.71	56.25	70.5	17.8	-	-
BUN (mg/dl)	22	38	14	18	20	23	21	-	-
Creatinine (mg/dl)	1.7	2	1	1.2	1.1	2.1	0.8	-	-
Sodium (mmol/l)	167	148	136	142	133	158	140	-	-
Potassium (mmol/l)	4.2	5.6	4.2	4.3	5.2	5.9	4.3	-	-

AFPP ratio- ascitic fluid to plasma protein ratio

#### 4.4. ASCITES OF RENAL ORIGIN

Two dogs (dog: 9 and 10) were diagnosed having renal protein loss and associated ascites. Dachshund and Doberman pinschers were the breeds involved. The ages of affected dogs (dogs 9 and 10) were six and three years respectively. Both the dogs were males.

##### 4.4.1. Clinical findings

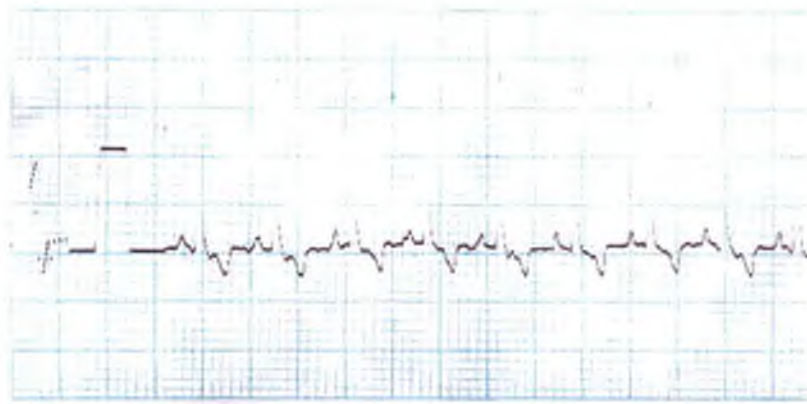
Inappetance and abdominal distension were the owner's complaints in both the dogs. Hind limb edema was observed in dog: 10 and scrotal and ventral abdominal edema was observed in dog: 9. Lethargy was noticed in both the dogs. Dog: 10 was reluctant to walk. They were vaccinated and dewormed as per schedule.

Dog: 10 was having moderate distension of abdomen and it had been treated with furosemide and ranitidine by a local veterinarian. Dog: 9 had severe abdominal distension and respiratory distress was marked. Dog: 10, on auscultation of heart revealed dull heart sounds and a coarse murmur. Pulse was strong in that dog. Rectal temperature was 102°F in both the dogs. Dog: 10 was having blanched conjunctival mucous membrane and had a pulse rate of 160 /minute.

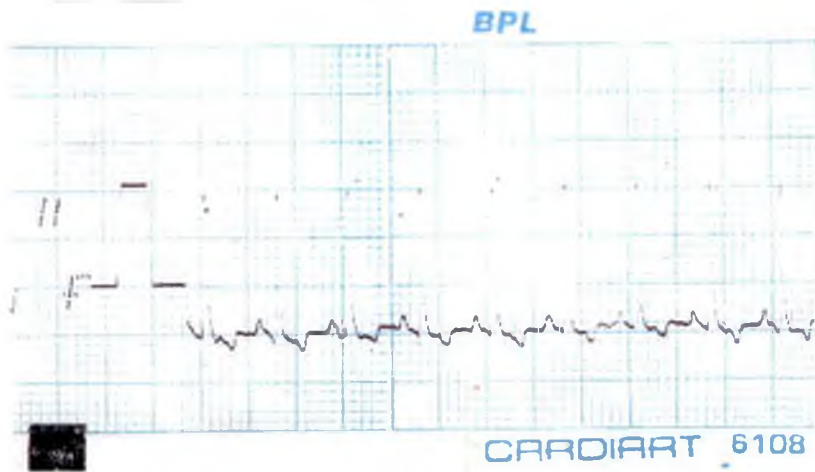
##### 4.4.2. Electrocardiographic findings

The ECG findings of these two dogs are presented in Table 6. 'P' duration, 'P' amplitude and 'QRS' duration were within the reference range. The 'R' amplitude was variable in these two dogs. Dog: 9 had 'R' amplitude of 1.4 mV and dog: 10 had 0.8 mV. The P-R interval was slightly prolonged in dog: 10 (0.16 mm / s). But, the P-R interval of dog: 9 was normal (0.8 sec).

The polarity of 'T' wave was negative in dog: 9 and it was positive in dog: 10. S-T slurring was observed in dog: 9 and elevated S-T segment seen in dog: 10. The mean electrical axis was +60° in both the dogs. The heart rate was 210 and



I



II

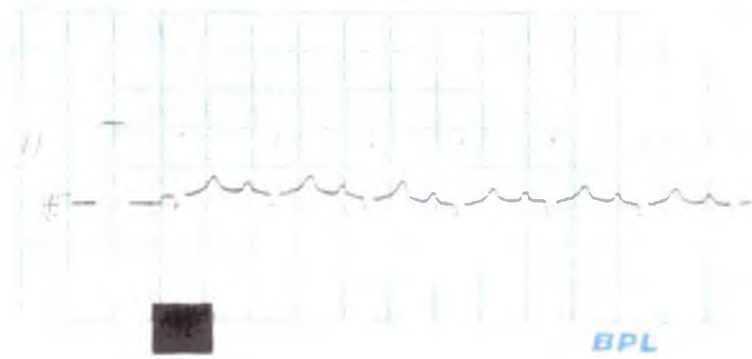


III

**Plate 10. ECG of Dog: 9 (Nephrotic syndrome)**  
**Normal sinus rhythm**  
**Slight slurring of S-T segment**



I



II



III

**Plate 11. ECG of Dog: 10 (Nephrotic syndrome)**  
**Normal sinus rhythm**  
**S-T elevation**  
**Tall 'T' waves**

150 bpm in dogs: 9 and 10 respectively. On 10<sup>th</sup> day of observation, 'P' duration, 'P' amplitude, QRS duration and 'R' amplitude did not change significantly from the first day observation. S-T segment changes were sustained in both the dogs on 10<sup>th</sup> day of observation (PLATES 10 and 11).

#### **4.4.3. Ultrasonographic findings**

##### **4.4.3.1. Ascitic fluid**

Both the dogs had virtually anechoic fluid in the abdominal cavity. Urinary bladder wall hypoechoic discontinuity was observed in both the cases and the urine was anechoic.

##### **4.4.3.2. Liver**

The ultrasonographic examination of liver parenchyma revealed normal echogenicity in both the cases. No change in the portal and hepatic vasculature observed in these two cases. No marked changes were observed in the gall bladder of these two dogs.

##### **4.4.3.3. Kidney**

Ultrasonographically, the kidneys remained normal in both the cases. There were no abnormalities in the cortico – medullary distinction and cortical echogenicity (PLATE 12).

##### **4.4.3.4. Heart**

Dog: 9 had no cardiac abnormality in both 2-D echocardiography and M-mode echocardiography. The myocardial and valvular kinesis was normal in that dog. But, dog 10 had dilatation/ eccentric hypertrophy of the left ventricle on 2-D echocardiography. Right ventricular echocardiogram yielded no abnormal findings. Though the left ventricle was dilated, myocardial contractility remained normal in dog: 10.





**Plate 12. Sagittal image of left kidney of Dog: 10 (Nephrotic syndrome)  
Normal echotexture (hypoechoic renal cortex to the surrounding tissues)**

#### **4.4.4. Hemogram**

On the day of admission, dog: 9 was having normal hemoglobin concentration and hematocrit value. The hemoglobin concentration was 12 g% and PCV was 38%. Dog: 10 was severely anaemic with hemoglobin concentration of 3.9 g% and a PCV of 10%. On 10<sup>th</sup> day of observation, Hb and PCV values were 12 g% and 40% respectively in dog: 9. The corresponding values were 8 g% and 22% respectively in dog: 10 (Table 7).

#### **4.4.5. Serum biochemistry**

The serum biochemical values of these two dogs are presented in Table 7.

##### ***4.4.5.1. Liver enzymes***

The serum concentration of ALT and ALP remained within the reference range in both the dogs during the period of observation. On the day of admission, serum concentrations ALT in dogs 9 and 10 were 45 U/L and 54 U/L respectively. No marked changes in serum concentrations of liver enzymes observed on 10<sup>th</sup> and 21<sup>st</sup> days of examination.

##### ***4.4.5.2. Serum proteins***

Both dogs were hypoproteinemic. The total protein concentrations were 43 g/L and 42 g/L in dogs 9 and 10 respectively. The total serum protein concentration was 48 g/L and 32 g/L respectively in dogs 9 and 10 on 10<sup>th</sup> day of examination. On 21<sup>st</sup> day of observation, dog: 9 had 55 g/L of total serum protein.

Dog: 9 was severely hypoalbuminemic (5 g/L) and dog: 10 also remained hypoalbuminemic (13 g/L). The serum albumin concentrations were 4 g/L and 11 g/L in dogs 9 and 10 respectively on 10<sup>th</sup> day of observation.

The serum globulin concentration remained normal in both the dogs. The values were 38 g/L and 29 g/L on the day of admission. On 10<sup>th</sup> day, the serum globulin concentrations were 44 g/L and 21 g/L in dogs 9 and 10 respectively. The A: G ratios were 0.13 and 0.45 in dogs 9 and 10 respectively on the day of admission.

On the day of admission, the estimated total protein concentration of ascitic fluid was 0.9 g/L and 1.8 g/L respectively in dog 9 and 10. The protein concentrations in ascitic fluid were 3 g/L and 12 g/L in dogs 9 and 10 respectively on 10<sup>th</sup> day of observation. On the day of admission, the ascitic fluid to plasma protein ratios of dog: 9 and 10 were 2.09 % and 4.28 % respectively. The corresponding values were 6.25% and 37.5% on 10<sup>th</sup> day of observation.

#### ***4.4.5.3. Kidney function tests***

##### **4.4.5.3.1. Blood urea nitrogen (BUN)**

The BUN concentrations were within the reference range during the period of observation. The BUN concentrations were 28 mg/dl and 12 mg/dl in dogs 9 and 10 respectively on 10<sup>th</sup> day of observation.

##### **4.4.5.3.2. Serum creatinine**

The serum creatinine concentration remained within the reference range in both the dogs (dogs 9 and 10) during the period of study. The values were 0.85 mg/dl and 0.6 mg/dl respectively on the day of admission. Serum creatinine concentrations were 0.7 mg/dl and 0.8 mg/dl in dogs 9 and 10 respectively on 10<sup>th</sup> day of observation.

#### **4.4.5.4. Sodium**

Dog: 9 was severely hypernatremic and dog: 10 was normonatremic. The serum sodium concentration was 190 mmol/l and 148 mmol/l respectively on the day of admission. The serum sodium concentrations were 141 mmol/l and 152 mmol/l in dogs 9 and 10 respectively on 10<sup>th</sup> day of observation.

#### **4.4.5.5. Potassium**

Both the dogs were hyperkalemic. They had a serum concentration of 6.0 mmol/l and 5.8 mmol/l of potassium respectively on the day of admission. Serum potassium concentrations were 5.9 mmol/l and 6.2 mmol/l respectively in dogs 9 and 10 on 10<sup>th</sup> day of examination.

Table 6. Electrocardiographic findings in dogs with ascites of renal origin

PARAMETERS	1 <sup>ST</sup> DAY		10 <sup>TH</sup> DAY		21 <sup>ST</sup> DAY	
	Case No:					
	9	10	9	10	9	10
'P' duration (sec)	0.04	0.04	0.04	0.04	0.04	-
'P' amplitude (mV)	0.1	0.1	0.2	0.2	0.1	-
P-R interval (sec)	0.08	0.16	0.09	0.16	0.08	-
QRS duration (sec)	0.04	0.04	0.04	0.06	0.04	-
'R' amplitude (mV)	1.4	0.8	1.6	1.3	1.2	-
'T' wave polarity	-ve	+ve	-ve	+ve, peak	-ve	-
S-T segment	Slurring	Elevated	Slurring	Elevated	Normal	-
MEA ( degrees)	+60°	+60°	+60°	+90°	+60°	-
Heart rate (bpm)	210	150	200	120	200	-

N : Normal

+ve : Positive

-ve : Negative

- : Animal died during the course of study

Table 7. Haematological and serum bio-chemical parameters in dogs with ascites  
of renal origin

PARAMETERS	1 <sup>ST</sup> DAY		10 <sup>TH</sup> DAY		21 <sup>ST</sup> DAY	
	Case No:					
	9	10	9	10	9	10
Hb (g %)	12	3.9	12	8	14	-
PCV (%)	38	10	40	22	32	-
ALT (U/L)	45	54	73	13	51	-
ALP (U/L)	70	50	170	55	163	-
Total Protein (g/L)	43	42	48	32	55	-
Albumin (g/L)	5	13	4	11	22	-
Globulin (g/L)	38	29	44	21	33	-
A:G ratio	0.13	0.45	0.09	0.52	0.67	-
Ascitic fluid Protein (g/L)	0.9	1.8	3	12	0.1	-
AFPP ratio	2.09	4.28	6.25	37.5	1.8	-
BUN (mg/dl)	33	12	28	12	20	-
Creatinine ( mg/dl)	0.85	0.6	0.7	0.8	0.8	-
Sodium (mmol/l)	190	148	141	152	143	-
Potassium ( mmol/l)	6.0	5.8	5.9	6.2	5.4	-

AFPP ratio- Ascitic fluid to plasma protein ratio

#### 4.5. DIETARY MANAGEMENT

In all the cases, salt- restricted food was advised. In dogs with ascites of cardiac and renal origin, boiled- egg feeding was advised.

#### 4.6. THERAPEUTIC RESPONSE

Animals were observed for a period of 30 days from the day of admission. Treatment protocol varied depending on the confirmatory diagnosis. Administration of diuretic, furosemide @ 2 mg/ kg or (Lasilactone<sup>®</sup> 2 mg/kg PO) was done in all the cases of ascites. With the aforesaid therapeutic regimen, six dogs survived beyond 30 days and one dog (dog: 5) died on the 9<sup>th</sup> day of admission and three dogs (dogs: 7, 8 and 10) were died during the third week of observation.

##### 4.6.1. Ascites of hepatic origin

In addition to diuretic therapy, oral administration of prednisolone (Wysolone<sup>®</sup>) @ 1 mg/kg on tapering pattern, silymarin (Silybon-70<sup>®</sup>) @ 10 mg/kg BID and Liv-52<sup>R</sup> syrup ( 1 teaspoon b.i.d PO) was instituted for one month.

Dogs 1, 4 and 5 received corticosteroid therapy. All the five dogs received oral administration of silymarin for 20 days. Liv- 52<sup>®</sup> syrup was administered in dogs 2, 3, and 5. Dogs 1 and 4 were administered oxytetracycline (@ 10 mg/kg for 5 days) injection intravenously and doxycycline orally for nine days as they were suffering from subclinical ehrlichiosis characterized by reduced platelet count. Urso deoxycholic acid (Ursofalk<sup>®</sup>) was given to dog: 4 @ 10 mg/kg B.W PO for 10 days.

Appetite was normalized by the second day of institution of therapy in all the 5 dogs. A marked reduction in the abdominal distension was observed on the 10<sup>th</sup> day of examination. The changes in the serum biochemistry are presented in

Table 3. Among the five dogs, four dogs survived beyond 30 days. Although the dog: 5 was improved from his initial presentation, on its 9<sup>th</sup> day, it was reportedly anuric and died on next day. Post- mortem of the animal could not be conducted.

#### **4.6.2. Ascites of cardiac origin**

On first day, intravenous administration of furosemide (Lasix<sup>®</sup>) was carried out in all the three cases. Thereafter, Lasilactone<sup>®</sup> tablets were given for remaining days. All the dogs were treated with digoxin (Lanoxin<sup>®</sup>) @ 0.02 mg/kg BW b.i.d P.O. Dog: 6 was given enalapril (Enapril<sup>®</sup>) @ 0.5 mg/kg B.W PO for 20 days.

With the above mentioned therapeutic and mangemental protocol, one dog (dog: 6) survived beyond 30 days and one dog (dog: 7) died on 20<sup>th</sup> day and dog: 8 died on 15<sup>th</sup> day of clinical presentation.

#### **4.6.3. Ascites of renal origin**

Both the dogs (9 and 10) were treated with furosemide (@ 2 mg/kg b.i.d) tablets and prednisolone @ 1 mg/kg for five days. Dog: 10 was subjected for whole- blood transfusion two times as it was severely anaemic. Dog: 10 was comatosed at 12<sup>th</sup> day and intravenous administration of dextrose 25% (200 ml) was carried out for two days. Among the two dogs, one dog (dog: 9) survived beyond 30 days and dog: 10 died on 18<sup>th</sup> day of admission. Post- mortem examination revealed pale kidney and dilated left ventricle. Other visceral organs remained normal.



## *Discussion*

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## 5. DISCUSSION

In the present study, ten animals with ascites were studied in detail and the results are discussed here.

### 5.1. ASCITES OF HEPATIC ORIGIN

Among the ten dogs, five of them were confirmed having liver diseases as the cause of ascites. Age of the affected dogs ranged from one to six years. The predisposition of middle- aged and young adults for liver diseases has been reported by Thornburg and Moody (1981) Leib (1997), and Szatmari *et al.* (2002). The average age of occurrence of hepatic diseases was 5.1 years. The highest incidence was recorded during four to six years. Four out of five dogs were males. This may be because of over representation of males in the canine population (Vijayakumar *et al.*, 2003). Spitz, Dachshund, Labrador retriever, Rottweiler and Boxer were the breeds affected with liver diseases in the present study. No breed predisposition could be stated as each breed represented equal number to the total number of cases.

All the five dogs were inappetent. It is in agreement with the findings of Boer *et al.* (1984) and Sevelius (1995). Diseases of liver usually affect the appetite (Strombeck and Guilford, 1991). Inappetance and lethargy were the most common owner's complaints in dogs with hepatic disease (Sevelius, 1995).

Invariably all the five cases had marked abdominal distension and fluid thrill on tactile percussion of abdomen as suggested by Kelly (1984).

#### 5.1.1. Hepatitis due to sub clinical ehrlichiosis

Two dogs (Dogs: 1 and 4) developed ascites due to the subclinical ehrlichiosis. Ultrasonographic changes, serum biochemistry and lowered platelet counts and the response to therapy with doxycycline suggest subclinical ehrlichiosis as the cause of ascites.

Dog: 1 was a five -year- old male, Spitz presented to the clinic with the complaint of abdominal distension for the past three weeks and poor appetite. Animal was lethargic and pyretic (Temperature. 104.2°F). This was probably a febrile reaction associated with canine ehrlichiosis (Greig *et al.*, 1996).

Dog: 4 was a one- year -old female Rottweiler with the complaint of abdominal distension and inappetance. On physical examination, the rectal temperature was 103.8 ° F and the mucous membrane was pale roseate. The owner observed inappetance for the past two weeks. Similarly, inappetance was reported in various hepatic disorders. Sevelius (1995) observed inappetance in dogs with chronic hepatitis and cirrhosis. Nottidge *et al* (2003) observed inappetance in a dog with cirrhosis. The primary manifestation of hepatic dysfunction was inappetance (Renjith, 2003). Rychlik *et al.* (2005) observed inappetance in a dog with lobular dissecting hepatitis. Ascitic distension was visible and fluid thrill was observed on tactile percussion of abdomen (Kelly, 1984).

On the day of admission, ECG parameters of dog: 1 was normal and within the reference range as stated by Bolton (1975) but the 'R' amplitude was reduced (1.1 mV). This reduction of 'R' amplitude was probably because of the excessive accumulation of ascitic fluid, which reduced the electrical conductivity to the body surface. This opinion coincided with the findings of Jones (1985).

On 10<sup>th</sup> and 21<sup>st</sup> days of examination 'R' amplitude was increased to 2.0 mV and 2.1 mV respectively which implied the facilitated conduction of electrical impulse due to the reduction in quantity of ascitic fluid following diuretic therapy. The other ECG parameters remained unchanged during the course of treatment. Boer *et al.* (1984) observed no remarkable change in the ECG of dogs with liver diseases.

'P' duration was slightly prolonged (0.08 sec) without notching in dog: 4. The increase in the 'P' duration is usually associated with left atrial hypertrophy (Bolton, 1975). As the author stated, the criteria for left atrial hypertrophy like

notching of 'P' wave and prolongation of P-R interval were not observed in this case. The other ECG parameters were normal and within the reference range. This ruled out the possibility of cardiac involvement in the origin of ascites of the present study. On 10<sup>th</sup> day of observation all the ECG parameters were within the reference range.

Ultrasonography of the abdomen revealed a large amount of anechoic ascitic fluid in both the dogs suggestive of transudate or modified transudate (Spaulding, 1993). The absence of echogenicity or septation in the abdominal fluid ruled out the possibility of exudation in the peritoneal cavity (Spaulding, 1993). The hypoechoic bladder wall discontinuity observed in these cases is in agreement with Douglass and Kremkau (1993).

The generalized increase in the liver parenchymal echogenicity observed in dog: 1 was because of the progressive hepatitis and the specks of hyperechoic areas observed were the fibrotic walls of portal veins. Bressler *et al.* (2003) observed vasculitis and portal vein thrombosis in ehrlichiosis. The inflammation of the portal vein and thrombosis could have resulted in specks of echogenic/ hyperechoic areas in the liver parenchyma.

No abnormalities could be seen in the portal system and hepatic vasculature of dog: 4. Rychlik *et al.* (2005) observed no change in the hepatic ultrasonogram of a dog with ascites due to lobular dissecting hepatitis. Increase in the parenchymal echogenicity was observed by Yamagami *et al.* (2002) in a dog with lymphangiomatosis. Bressler *et al.* (2003) observed no change in liver echotexture except the portal vein thrombosis on hepatic ultrasonogram of dogs. The generalized echogenicity observed in this case probably because of the through transmission associated with large amount of ascitic fluid (Pennick, 1995).

Transverse, longitudinal and sagittal planes of ultrasonography of right and left kidney in both the dogs revealed no marked change in the cortical and medullary

echogenicity and the cortico-medullary distinction was clear. Findings in the present case are in agreement with Konde *et al.* (1984) and Nyland *et al.* (1995). Echocardiography revealed no marked changes in the chamber dimension and myocardial contractility in both the cases. Left and right parasternal views were taken to study the right and left sides of the heart. Echocardiographic findings were in agreement with the ECG findings.

One dog (dog: 1) had reduced Hb of 6.7 g% and PCV of 25%. Another dog (dog: 4) had Hb of 9.5 g% and PCV of 31% on the day of admission. Anaemia is a consistent finding in various liver diseases. (Hunt *et al.* 1993; Sevelius, 1995; Varshney *et al.*, 2002). Codner and Farris – Smith (1986) observed normal PCV in dogs with subclinical phase of ehrlichiosis. Greig *et al.* (1996) observed reduced PCV in three out of ten dogs with a granulocytic ehrlichiosis. The findings in the present cases are in agreement with Greig *et al.* (1996).

Reduction in PCV is a common finding in hydremic conditions like pregnancy, hypoproteinemia and iatrogenic overhydration (Goldston *et al.*, 1980). A slight increase in the Hb concentration of dog: 4 was observed on the 21<sup>st</sup> day of observation. The PCV was 46% and Hb was 11g%. This denotes the clinical and hematological reversal with the therapy instituted.

Buffy coat and peripheral blood smear examination did not reveal ehrlichia organism. In chronic/ sub clinical ehrlichiosis, it is not common to find the organism in the peripheral blood smear. Codner and Farris-Smith (1986) confirmed the subclinical ehrlichiosis by serologically positive titres. The response to therapy with doxycycline and prednisolone is suggestive of chronic ehrlichiosis as suggested by Bressler *et al.* (2003). The platelet count of dog: 1 was 80,000/ cu. mm and dog: 4 had platelet counts of 93,000/ cu.mm and 1, 35,000/ cu.mm on 1<sup>st</sup> and 10<sup>th</sup> day of observations. The thrombocytopenia observed in these cases is suggestive of sub clinical ehrlichiosis. Thrombocytopenia is a constant feature in canine ehrlichiosis (Codner and Farris-Smith, 1986; Greig *et al.*, 1996 ). Thrombocytopenia might have

resulted either due to the direct action of parasite on thrombocytes or immune-mediated disorders of thrombocyte.

The total protein levels in the serum were within the reference value in both the dogs. Hypoalbuminemia (20 g/l) with hyperglobulinemia (45 g/l) were observed in the serum of dog: 1 on the day of admission. The A: G ratio was low (0.44). Hyperglobulinemia and hypoalbuminemia was marked on the 10<sup>th</sup> day of examination. The hyperglobulinemia and hypoalbuminemia in this case might be due to chronic immunological reaction as stated by Greig *et al.* (1986). The author observed hypoalbuminemia in dogs with granulocytic ehrlichiosis. But the total protein level was reduced only in 20% of dogs with granulocytic ehrlichiosis. In these two dogs, the total serum protein level was normal.

The albumin level was markedly reduced (19 g/l) in dog: 4. The globulin level was 36 g/l and remained normal. The A: G ratio was slightly reduced (0.53). The hypoalbuminemia and normoglobulinemia in this case is because of the reduced albumin synthesis due to liver disease as stated by Strombeck and Guilford, (1991).

Codner and Farris – Smith (1986) observed hyperglobulinemia and normal albumin concentrations in dogs with subclinical ehrlichiosis. This opinion is in contrast to the present finding. On 21<sup>st</sup> day of examination, the total protein level was similar to that of the day of admission. A slight increase in the albumin and slight decrease in globulin level was observed. This is probably because of the response to therapy with doxycycline and prednisolone. The A: G ratio though increased from the first day of admission, did not reach the normal reference range.

Hunt *et al.* (1993) observed ascites in three young dogs with non-fibrosing liver disease. Two dogs had low protein ascites (5 g/l) associated with pre-hepatic portal hypertension and one dog had post hepatic hypertension. The protein content of ascitic fluid of dog: 4 was 6 g/l and A:FP ratio was 11%. On 10<sup>th</sup> and 21<sup>st</sup> days of observation, the ascitic fluid protein was 2 g/l and 13 g/l respectively. These

findings suggest a low- protein ascites associated with pre-hepatic portal hypertension (Hunt *et al.*, 1993). A: G ratio was low (0.53), compared to reference range.

The low protein ascites seen in this case is suggestive of pre- hepatic portal hypertension associated with the portal vein lesions. The lower AFPP ratio was found in two dogs with non fibrosing liver disease (Hunt *et al.*, 1993). The lower AFPP ratio obtained in the present study rule out the possible contribution of protein by hepatic lymph to the ascitic fluid.

Varshney *et al.* (2002) studied seven refractory cases of low protein ascites in dogs. The protein concentration of ascitic fluid ranged between 3 g/l to 20 g/l. The author concluded that ascitic distention was because of the portal venous hypertension due to cirrhosis. The findings in the present case suggest pre-hepatic portal hypertension associated with liver diseases.

The ALT concentration of dog: 1 was 393 U /L and ALP was 289 U/L. These values were above the reference range. Cirrhosis (Rutgers *et al.*, 1993; Sevelius., 1995; Speeti *et al.*1995; Varshney *et al.*, 2002; Ertekin *et al.*, 2003), Chronic active hepatitis (Leib, 1997), Hepatic arteriovenous fistula (Koide *et al.*,2003), Chronic non specific hepatitis (Sevelius, 1995) are the situations which lead to increased liver enzyme concentrations in the serum. In the present case, elevated ALT and ALP concentrations may be due to liver damage associated with chronic/ sub clinical ehrlichiosis. This is in agreement with Greig *et al.* (1996).

The serum ALT and ALP concentrations in dog: 4 were 144 U/L and 328 U/L respectively. The slight increase in ALT and marked elevation in ALP concentration is a consistent finding in sub clinical ehrlichiosis. These findings coincide with that of Greig *et al.* (1996). On 10<sup>th</sup> day of examination, the serum ALT and ALP concentrations were 123 U/L and 482 U/L respectively. The reduction in ALT suggests clinical improvement and increased concentration of ALP on 10<sup>th</sup> day

of observation would have resulted from corticosteroid therapy given to this dog. This is in agreement with Cornelius (1997). Reduced ALT concentration following doxycycline therapy was not in agreement with the findings of Vijayakumar *et al.* (2004 a). The author observed increased ALT concentration following therapy with oxytetracycline. In contrast to the findings in this case, Codner and Farris-smith (1980) observed normal ALT and ALP concentrations in dogs with subclinical ehrlichiosis. The variation observed in the present study may be due to the variation in the degree of severity of disease.

The BUN and creatinine concentrations were within the reference range in both the dogs. These findings are in contrast to the findings of Sevelius (1995) and Leib (1997). There was reduction in the BUN value in chronic progressive hepatitis, chronic non specific hepatitis, chronic cholangiohepatitis and cirrhosis. Varshney *et al.* (2002) is also of the same opinion. The author observed reduced BUN (5.0 to 10.0 mg/dl) in seven refractory cases of ascites associated with cirrhosis. The findings of the present case are in agreement with the observations made by Codner and Farris- Smith (1986) in subclinical ehrlichiosis, who observed normal concentrations of BUN.

The serum creatinine level was within the reference range on 10<sup>th</sup> and 21<sup>st</sup> day of examination. Similar observations were made by Codner and Farris- Smith (1986) in sub clinical ehrlichiosis. The slight increase observed on the day of admission could have resulted from dietary changes (Swanson *et al.*, 2004).

The serum sodium concentrations were within the reference range on 1<sup>st</sup> and 10<sup>th</sup> day of examination. But it was slightly elevated on 21<sup>st</sup> day of examination. The serum sodium concentrations observed in dog: 4 were 152 mmol/l, 137 mmol/l and 148 mmol/l on 1<sup>st</sup>, 10<sup>th</sup> and 21<sup>st</sup> day of observation. The serum sodium concentration was on its upper limit on the day of admission, suggestive of mild sodium retention. On 10<sup>th</sup> day of observation, mild hyponatremia observed could be because of furosemide diuresis (Jacobs, 1989a; Gross, 1995). The estimation of serum sodium



concentration might not be accurate as the intravascular fluid volume determines the sodium concentration (DiBartola, 2000).

Dog: 1 had a potassium concentration of 3.70 mmol/l, 4.20 mmol/l and 4.80 mmol/l on 1<sup>st</sup>, 10<sup>th</sup> and 21<sup>st</sup> day of examination respectively. Hence, there is no sodium and potassium retention in this case. This is in contrast to the 'Overflow theory' stated by Leib (1997).

The serum potassium concentration was within the reference range in dog: 4, is in disagreement to the findings made by Codner and Farris-Smith (1986). The authors observed retention of sodium and potassium in dogs with subclinical ehrlichiosis. The variation may be because of hydremic condition associated with ascites.

On the day of admission, these dogs were treated with furosemide @ 2 mg/kg BW b.i.d and oxytetracycline @ 10 mg/kg BW i.v for five days followed by doxycycline @ 10 mg/kg b.i.d PO for 10 days and silymarin @ 10 mg/kg b.i.d. PO for 20 days. Martin *et al.* (1984) and Vijayakumar *et al.* (2004b) used silibinin and silymarin in the treatment of hepatic disorders successfully. Liv-52<sup>®</sup> Vet syrup was given @ 1 teaspoon b.i.d. PO for one month. Umesh (2000) stated that Liv-52<sup>®</sup> vet syrup was effective in dogs with ascites of hepatic origin. Apart from this conventional therapy, prednisolone was given @ 1 mg/kg BW for 10 days. Prednisolone was administered to dogs with chronic active hepatitis by Thornburg and Sumerlin (1981).

Increased liver enzyme activity, reduced protein fraction, reduced platelet count (93,000/ cu.mm) suggested ehrlichiosis (Greig *et al.*, 1996). Diuresis was achieved by administration of Lasilactone<sup>®</sup> orally @ 2 mg/kg PO b.i.d. Silymarin (10 mg/kg BW PO b.i.d for 20 days) and prednisolone (1mg/kg BW PO for 5 days). A marked reduction in the abdominal distension and resumption of appetite was evident on 10<sup>th</sup> day of examination. Complete recovery of the animal was observed

on 21<sup>st</sup> day. Prednisolone and silymarin were effective in the present case, is in agreement with Thornburg and Sumerlin (1981) and Vijayakumar *et al.* (2004b)

On 10<sup>th</sup> day of examination, liver echogenicity turned normal and the concentrations of liver enzymes were reduced from the initial observation. This suggested clinical improvement. Vijayakumar *et al.* (2004 b) observed a pronounced increase in the erythron and a significant reduction in the leukogram, ALT, ALP, serum bile acids and bilirubin following therapy with silymarin. The author also observed reduction in the initial hyperechogenicity of liver parenchyma. On 21<sup>st</sup> day of observation, animal recovered from illness with markedly reduced abdominal distension.

### **5.1.2. Cirrhosis**

Dogs 2 and 3 were diagnosed as having ascites associated with pre-hepatic portal hypertension and liver cirrhosis. Dog: 2 was a three-year-old male Dachshund presented with complaints of inappetance, lethargy and severe abdominal distention. Temperature was 103.6°F and mucous membrane was pale roseate.

Dog: 3 was a four- year- old male Labrador retriever presented to the clinic with a past history of ascites and treatment for the same. It was anorectic and severely dehydrated. It was under treatment for the past two weeks with furosemide and Liv- 52<sup>®</sup> Vet syrup. Inappetance and lethargy were the predominant owner's complaints in dogs with liver diseases as stated by Boer *et al.* (1984), Hunt *et al.* (1993) and Sevelius (1995). On clinical examination, the rectal temperature was 101°F and the conjunctival mucous membrane was congested. The dog was severely dehydrated and skin tenting lasted for more than five seconds. Animal was lethargic and there was no visible abdominal distension. The congestion of mucous membrane could be because of the intensive diuresis and dehydration by furosemide administration previous to the clinical presentation (Jacobs, 1989a). The heart rate was 180 bpm and the animal was panting. Thoracic auscultation was unremarkable.

Abdominal palpation revealed freely moving intestinal loops and no appreciable enlargement of the liver.

The heart rate and pulse rate and quality remained normal. Respiratory distress was noticed in dog: 2. Kelly (1984) stated that respiratory distress was common in abdominal distension.

On the day of admission, ECG parameters in lead II were normal in both the dogs. (Table 2). But, the 'R' amplitude was reduced markedly (0.9mV) in dog: 2. and dog: 3 (1.0 mV). Low voltage 'QRS' complexes were common in ascitic animals (Jones, 1985).

The ECG tracings of dog: 2 revealed increased 'R' amplitude from the initial 0.9 mV to 1.4 mV and 1.7 mV respectively on the 10<sup>th</sup> and 21<sup>st</sup> days of examination. This is probably because of the reduction in the amount of abdominal fluid by diuretic therapy. No ECG changes relevant to heart diseases were seen in this case. This is in agreement with Varshney *et al.* (2002). The author observed no marked changes in the ECG of dogs with low- protein ascites.

Heart rate was slightly elevated in dog: 3 (180 bpm), probably because of the compensation to reduced preload associated with diuresis (Jacobs, 1989b) Cardiac preload is largely determined by the intravascular blood volume. No marked changes observed in the electrocardiograms on 10<sup>th</sup> and 21<sup>st</sup> day as the prime organ affected was liver (Varshney *et al.*, 2002).

Abdominal ultrasonography revealed anechoic, free abdominal fluid and floating organs in both the dogs as described by Hunt *et al.* (1993). The absence of echogenicity in the peritoneal fluid suggests a transudate (Spaulding, 1993). A hypoechoic pseudo discontinuity observed in the urinary bladder wall is due to sound wave refraction on the curved wall. This is in agreement with Douglass and Kremkau (1993). As per the method described by Szatmari *et al.* (2004a), dog: 2 had a 'large' amount of ascitic fluid as it was easily detectable on physical examination

itself. Dog: 3 had 'moderate' amount of abdominal effusion. In this case there was no visible distension of abdomen but ultrasonographic detection of ascitic fluid was easy. Abdominal ultrasonography revealed floating organs with reduced acoustic enhancement as there is moderate distention.

In these two dogs, ultrasonography of the liver revealed diffused hyperechogenicity of the liver parenchyma and tortuous portal vessels. The diffused pattern of hyperechogenicity observed in this case is probably because of the increased fibrous tissue (Nyland *et al.*, 1995; Vijayakumar, 2002). Yamagami *et al.* (2002) observed increased parenchymal echogenicity of liver in hepatic lymphangiomas. The increased echogenicity of liver observed in this case is not seen in the case described by Hunt *et al.* (1993) with non fibrosing liver diseases.

Ultrasonography of liver in dog: 3 was effectively carried out through substernal approach. Reduced fluid content of the abdomen did not hinder the liver sonography (Jian – Xin WU and Carlisle, 1995). The present case had no marked change in the hepatic and portal vasculature. Gall bladder was distended with bile and the contents were mixed echoic. Vijayakumar *et al.* (2004 a) observed increased liver parenchymal echogenicity following oxytetracycline therapy.

The left and right kidney was scanned while the animal was on standing position. This method was efficient in reducing the pseudohyperechogenicity observed due to through transmission. Nyland *et al.* (1995) opined that the kidneys of the ascitic animal could efficiently be scanned while the animal was in standing position. No marked changes were observed in the kidney of these two dogs. The left renal cortex was hypoechoic to spleen and surrounding tissue (Konde *et al.* 1984; Nyland *et al.*, 1995) and the cortico- medullary distinction was clear. Ultrasonographically, the kidneys were normal in both the cases.

Left and right parasternal short-axis and long-axis views revealed no abnormal findings in the heart. The myocardial contractility and the chamber

dimensions were within the normal reference range (Su *et al.*, 2003). Echocardiographic findings were in agreement with ECG findings in these dogs.

Reduced PCV (25%) and normal Hb (9.2 g %) were observed in dog: 2 on the day of admission. This shows a mild degree of anaemia. Similar observations were made in various hepatic diseases (Hunt *et al.*, 1993; Sevelius, 1995; Varshney *et al.*, 2002). The increase in PCV on subsequent sampling is probably indicative of clinical improvement (Vijayakumar *et al.*, 2004 b) or hemoconcentration associated with diuretic therapy (Benjamin, 1998).

Similarly, Hb and PCV values were reduced in dog: 3 also. Reduction in PCV and Hb has been reported in various liver diseases (Sevelius, 1995; Varshney *et al.*, 2002; Nottidge *et al.*, 2003).

The serum total protein was reduced (37 g/l) and hypoalbuminemia (15 g/l) and hypoglobulinemia (22 g/l) were also seen in dog: 2. The ascitic fluid protein was 26 g/l and AFP ratio was 70%. These findings suggest high- protein ascites associated with post- sinusoidal obstruction. This type of post- sinusoidal hypertension with out visible obstruction of caudal vena cava was observed in a dog by Hunt *et al.* (1993). The author diagnosed that case as post- sinusoidal portal hypertension associated with functional obstruction of portal blood flow. Similarly, hypoalbuminemia, hypoproteinemia and high- protein ascites were observed in the study.

On the day of admission, the serum total protein content was slightly reduced (51 g/l) and hypoalbuminemia (19 g/l) and normal globulin concentration (32 g/ l) were observed in dog: 3. The hypoproteinemia and hypoalbuminemia observed in this case is also seen in most of the liver diseases such as idiopathic hepatic fibrosis (Rutgers *et al.*, 1993), cirrhosis and chronic progressive hepatitis (Sevelius, 1995), primary hypoplasia of portal vein (Van den Ingh *et al.*,1995) and chronic active hepatitis (Leib, 1997). In the present case, globulin level remained normal but the

presence of hypoproteinemia and hypoalbuminemia suggested impaired synthesis of albumin in the liver (Strombeck and Guilford, 1991; McGrotty and Knottenbelt, 2002).

Hepatic ultrasonogram of dogs 2 and 3, could not reveal the portal vascular obstruction even at the level of caudal vena cava. High- protein in the ascitic fluid observed in the present case might be contributed by the hepatic lymph in post-sinusoidal obstruction (Maddison, 1990).

The protein content of ascitic fluid of dog: 3 was 20 g/l, suggestive of low protein ascites, but the AFPP ratio was 39% which implies a post- sinusoidal portal hypertension with leakage of hepatic lymph (Hunt *et al.*, 1993). The probable reason for the development of ascites in this case is cirrhosis associated with post-sinusoidal portal hypertension. (Hunt *et al.* 1995; Sevelius, 1995). A: G ratio was (0.5) normal in this case. On 10<sup>th</sup> day of observation, total protein was slightly increased (52 g /l), hypoalbuminemia was worsened (14 g/l) and globulin was 38 g/l. A: G ratio was low (0.36). The reduction in A: G ratio could be because of the relative increase in globulin concentration.

The serum ALT and ALP concentrations in the dog: 2 were 381 U/L and 160 U/L respectively on the day of admission. The serum concentrations of liver enzymes were elevated in dog: 3 (ALT – 126 U/L. ALP – 270 U/L). This finding is in coincidence with the findings of Hunt *et al.* (1993). The author observed elevated serum ALT and ALP concentrations in a dog with of post- sinusoidal portal hypertension associated with non fibrosing liver disease.

The elevated concentrations of liver enzymes suggestive of liver disorders and hepatocellular damage (Maddison, 1990). Sevelius (1995) opined that ALT, ALP concentrations were markedly elevated in chronic progressive hepatitis, chronic non- specific hepatitis and chronic cholangiohepatitis. Findings in these two

cases are in contrast to the findings of Varshney *et al.* (2002) and Nottidge *et al.* (2003). They observed low serum levels of ALT in cirrhotic dogs.

On 10<sup>th</sup> day of observation, dog: 2 had an ALP concentration of 173 U/L and ALT of 102 U/L. The values were reduced from the day of admission after therapy with silymarin (Vijayakumar *et al.*, 2004 c). On the 21<sup>st</sup> day ALT was 84 U/L and ALP 73U/L. This suggests a decrease in the severity of the disease. Vijayakumar *et al.* (2004 c) observed a reduction in the serum ALP and ALT concentrations following silymarin therapy for 21 days. The finding in the present case is in agreement with Vijayakumar *et al.* (2004 c).

Boer *et al.* (1984) reported elevation in the serum levels of ALT and ALP in a dog with hepatic fibrosis. In the dog: 3, ALT was slightly elevated from the normal reference range of 21 – 102 U/L (Kaneko *et al.*, 1997). The ALP concentration was elevated markedly (270U/l). Experimental induction of cirrhosis resulted in marked elevation of ALT and ALP (Ertekin *et al.*, 2003). On 10<sup>th</sup> day of examination, ALT was 130 U/L and ALP was 344 U/L. A further increase in the liver enzyme level could be because of the corticosteroid therapy given in this case. Elevation in the serum liver enzyme concentration was a common observation in dogs after corticosteroid therapy (Cornelius, 1997). On 21<sup>st</sup> day of examination, this animal had an ALT concentration of 136 U/L and ALP of 240 U/L. ALP level decreased from the 10<sup>th</sup> day observations. This is probably because of the withdrawal of corticosteroid after its course.

These animals had normal BUN concentration. But BUN level was usually reduced in cirrhosis and other types of hepatitis (Sevelius *et al.*, 1995; Varshney *et al.*, 2002). Reduced BUN was observed in seven refractory cases of low- protein ascites with cirrhosis and portal hypertension by Varshney *et al.* (2002). Similar opinion was reported by Hunt *et al.* (1993) and Sevelius (1995) in cirrhosis and non fibrosing liver diseases. Nottidge *et al.* (2003) observed normal BUN and creatinine concentration in a dog with liver cirrhosis. The present case findings are in

agreement with that of Nottidge *et al.* (2003). The serum creatinine level of dog: 2 was within the normal reference range.

The present cases had normal sodium content in the serum. But, as per the “Overfill theory” sodium retention was the principle force in retaining / increasing the fluid volume in the body (Leib, 1997). This dog was normonatremic and not in agreement with the findings of Leib (1997). Estimation of serum sodium concentration cannot provide the direct information about the total sodium concentration of body. (DiBartola, 2000). The serum sodium concentration of dog: 3 were 146 mmol/l, 135 mmol/l and 136 mmol/l respectively on 1<sup>st</sup>, 10<sup>th</sup> and 21<sup>st</sup> day of observation. The serum sodium level was normal on the day of admission. But, mild hyponatremia observed on 21<sup>st</sup> day might be because of furosemide therapy (Jacobs, 1989a).

On 1<sup>st</sup>, 10<sup>th</sup> and 21<sup>st</sup> day of examination, the serum levels of potassium in dog: 2 were 4.40 mmol/l, 4.60 mmol/l and 6.20 mmol/l respectively. On 1<sup>st</sup> and 10<sup>th</sup> day of observation, animal was normokalemic. But on 21<sup>st</sup> day, there was increase in potassium concentration. Potassium-sparing diuretic used might also be contributed to the hyperkalemia observed in the present study. Pseudohyperkalemia observed in conjunction with the lysis of thrombocytes (DiBartola and de Moraes, 2000). The serum potassium concentration of dog: 3 were 3.70 mmol/l, 5.50 mmol/l and 4.70 mmol/l respectively on 1<sup>st</sup>, 10<sup>th</sup> and 21<sup>st</sup> day of observation. The serum potassium levels were within the reference range.

This dog: 2 was treated with Lasilactone<sup>®</sup> @ 2 mg b.i.d PO, Liv-52<sup>®</sup> vet syrup @ one teaspoon b.i.d PO for 20 days and silymarin @ 10 mg/kg BW PO for 20 days. As soon as the animal developed hyperkalemia on 21<sup>st</sup> day, diuretic was switched over to furosemide. The animal resumed food intake on 2<sup>nd</sup> day. Marked reduction in the abdominal distension was observed on 10<sup>th</sup> day of observation. On 10<sup>th</sup> day of treatment, there was reduction in the echogenicity of the liver. This is in



accordance with Vijayakumar *et al.* (2004b). Following therapy with this regimen, the animal recovered from the illness uneventfully.

On the day of admission, the dog: 3 was treated with silymarin, Liv-52 vet<sup>®</sup> syrup and Lasilactone<sup>®</sup> as per the above schedule. As an anti-fibrotic agent, prednisolone (@ 1 mg/kg BW PO for 5 days in tapering pattern) was given. Watson (2004) observed clinical cure in cirrhotic dogs following therapy with prednisolone. Thornburg and Sumerlin (1981) observed a clinical recovery in a dog with chronic active hepatitis when treated with prednisolone. Silymarin and Liv-52 vet<sup>®</sup> syrup administration was effective in restoring the lost appetite (Umesh, 2000; Vijayakumar *et al.*, 2004b). Urso deoxycholic acid (Ursofalk<sup>®</sup>) was given @ 10 mg/kg BW b.i.d PO for 10 days. A clinical recovery was evident on 21<sup>st</sup> day of examination.

Based on sonographaphic findings, serum biochemical assays and the presence of increased AFPP ratio, these two cases were concluded as post-sinusoidal portal hypertension associated with cirrhosis was responsible for ascites. Confirmation needs biopsy of liver, selective angiography and portal pressure measurement.

### **5.1.3. Chronic active hepatitis**

Dog: 5 was diagnosed to be suffering from chronic active hepatitis resulted in ascites. Six- year- old male Boxer was admitted to the clinic with the complaint of abdominal distention for the past two months. It was reportedly not taking food since two days. The animal voided deep- yellow coloured urine and it had no history of deworming and vaccination. This dog was previously treated by a local veterinarian with ranitidine, cephalosporin, B-complex, chlorpheniramine, metaclopramide and furosemide. On physical examination, the rectal temperature was 104.4°F and the mucous membrane was congested. Elevation of temperature and congestion of mucous membrane suggest a febrile reaction associated with

concurrent liver disease. Popliteal lymph node was slightly enlarged and no abdominal organ could be palpable because of the ascitic distension. The dog was found to have ascitic distension, scrotal edema and respiratory distress.

The ECG parameters were within the reference range as described by Bolton (1975). No change in the rhythm and the mean electrical axis observed in all the six leads. Ultrasonographic examination of the abdomen revealed cellular ascites with multiple echogenic floating particles (Mattoon and Nyland, 1995). Ascitic fluid was comparatively echogenic than the urine in the bladder. Spaulding (1993) stated that the mildly echogenic ascitic fluid could be observed in chronic right heart failure, pericardial diseases, caval syndrome, Budd-chiari like syndrome or chronic hepatic failure.

Mildly echogenic ascitic fluid suggested the presence of transudate or modified transudate (Spaulding, 1993). As per the classification of ascites described by Szatmari *et al.* (2004a), this case had a 'large' amount of ascitic fluid.

A diffused hyperechoic liver with uneven borders was observed in this case. The increased echogenicity of liver suggested cirrhosis, hepatic fibrosis or lymphangiomatosis (Yamagami *et al.*, 2002). Ultrasonographic examination of portal system was not giving useful information as no differentiation could be observed between the liver parenchyma and portal vessels. Jian – Xin –WU and Carlisle (1993) stated that the portal veins were surrounded by hyperechoic connective tissue compared to hepatic veins, which in general were not. Gall bladder was normal,

The findings of this case is contradictory to the opinion of Jian – Xin –WU and Carlisle (1993) as no portal vein could be well visualized due to increased liver parenchymal echogenicity. In the present case ultrasonographic structure of kidney remained normal without any change in the parenchymal echotexture.

Two dimensional echocardiography and M mode echocardiography revealed no abnormality in the chamber dimension, myocardial contractility and wall thickness. The echocardiographic imaging and the measurements were within the physiological norms as stated by Su *et al.* (2002).

Animal was anemic (Hb 8g % and PCV 36%). Most of the liver diseases resulted in normocytic anaemia. It has been reported by Hunt *et al.* (1993), Sevelius (1995), Varshney *et al.* (2002) and Nottidge *et al.* (2003). Dyspnoea noticed in the clinical examination could be because of severe anaemia.

The serum total protein level was 59 g/l, albumin 22 g/l and globulin 37 g/l. The normal protein concentration in the serum, hypoalbuminemia, normoglobulinemia were observed in chronic liver diseases (Strombeck and Guilford, 1991). The transudative effusion (< 25 g/l) suggest a possible cause of hypoproteinemia but the AFPP ratio was 38 % (high- protein ascites), suggesting post- sinusoidal portal hypertension ( Hunt *et al.*, 1993).

The serum ALT concentration in this dog was elevated (216 U/L) compared to the reference value of 21 to 102 U/L (Kaneko *et al.*, 1997). The elevation in ALT concentration denotes an active hepatocellular damage which results in leakage of ALT into the serum (Maddison, 1990). The ALP concentration was markedly elevated (2343 U/L). The ALP elevation is usually associated with idiopathic hepatic fibrosis (Rutgers *et al.*, 1993), cirrhosis and chronic progressive hepatitis, chronic cholangiohepatitis (Sevelius, 1995) and idiopathic non-cirrhotic portal hypertension (De Marco *et al.*, 1998)

Koide *et al.* (2003) observed higher ALP concentrations (1513 U/L) in a dog with hepatic arteriovenous fistula, portosystemic shunt and aortic stenosis. This suggested that the development of ascites was due to the post- sinusoidal portal hypertension associated with biliary stasis.

The BUN value was at its upper limit (27 mg/dl) but within the range of reference value of 10 – 28 mg/dl (Kaneko *et al.*, 1997). The BUN value is usually reduced in various liver disorders like chronic progressive hepatitis, cirrhosis, chronic non specific hepatitis and chronic cholangiohepatitis (Sevelius, 1995). Serum creatinine concentrations were within the reference range during the period of study.

The serum sodium and potassium concentrations were within the reference range. A mild degree hyponatremia was observed (136 mmol/l), and was because of furosemide diuresis (Jacobs, 1989a). The serum potassium concentration within the reference range, is in disagreement to the findings made by Gross (1995). The author observed hypokalemia in dogs previously treated with furosemide. Potassium sparing diuretic (Lasilactone<sup>®</sup>) might be responsible for the normokalemia observed in the present case.

On the day of admission, Lasilactone , silymarin and prednisolone were given. Liv-52 vet<sup>®</sup> syrup was added to the therapeutic regimen from the second day. Thornburg and Moody (1981) effectively used prednisolone in the treatment of chronic active hepatitis. Animal resumed the food intake on the third day. On 8<sup>th</sup> day the dog was reportedly anorectic and anuric. It died on the next day. Post-mortem could not be conducted. Death could have resulted from the anuric state and uremia.

## 5.2. ASCITES OF CARDIAC ORIGIN

In three out of ten dogs, congestive heart failure was diagnosed as the cause of ascites. Biventricular affections were diagnosed in two cases. (Dog 6 and 7) and dog: 8 had isolated right- heart failure.

### 5.2.1. Hypertrophic cardiomyopathy

Dog: 6 was diagnosed as hypertrophic cardiomyopathy causing ascites. A three- year- old female Dachshund presented to the clinic with abdominal distension for the past three months. It was reportedly passing deep- yellow coloured urine. Thoracic auscultation revealed loud heart sounds suggestive of cardiac enlargement (Kelly, 1984). Animal was already treated with furosemide, amino acids and liver extracts. Animal had normal appetite and water intake. Femoral pulse was strong, suggestive of heart diseases. Kelly (1984) stated that changes in the normal qualities of the pulse were caused by structural or functional diseases of heart. The pulse rate was 160 / min. A slight degree of dyspnoea was noticed. Tactile percussion of abdomen revealed fluid thrill in this dog is in agreement with Ringheim (1975) and Kelly (1984).

The 'P' duration of 0.06 sec observed in ECG, suggestive of left atrial enlargement. 'P' amplitude was 0.3 mV and within the reference range. QRS duration was slightly increased and 'Q' waves were deep ( $> 0.4$  mV) in leads I, II and aVF suggestive of right ventricular enlargement.

The prolonged 'QRS' duration observed in this case is suggestive of right ventricular enlargement (Martin, 2002). The P- R interval and the polarity of 'T' wave remained normal. A mild degree of S-T slurring was observed. Martin (2002) stated that prolonged 'QRS' duration, S-T segment sagging / coving, shift of MEA to the left were the features of left ventricular enlargement. Prolonged QRS' duration, tall 'R' waves (3.3 mV), S-T slurring and deep 'Q' waves observed in this case was suggestive of biventricular enlargement. The mean electrical axis remained normal in this case. The MEA observed in this case is  $+60^{\circ}$ . Bolton (1975) stated that if both the ventricles are hypertrophied, the axis remained normal.

The heart rate was 210 bpm. The heart rate of normal toy breed should not exceed 180 bpm. This suggested tachycardia (Bolton, 1975; Martin, 2002). A mild

degree of sinus arrhythmia was observed in the ECG tracings of this dog but only small changes were observed in the R-R interval.

Ultrasonography of liver revealed no marked change in the size and echotexture. Nephrosonogram revealed slight increase in the cortical echogenicity but the cortico- medullary distinction was clear. A slight increase in the cortical echogenicity observed in this case could be due to the acoustic enhancement phenomenon (Pennick, 1995). Echocardiography revealed biventricular hypertrophy and septal thickening. Myocardial contractility and the valvular kinesis were normal. 2-D echocardiography revealed hypertrophy of papillary muscles. These findings are suggestive of hypertrophic cardiomyopathy (Moise and Fox, 1988).

Su *et al.* (2003) stated that normal Taiwanese dog had LVIDd of 2.87 cm and LVIDs of 1.90 cm and LVFS of 34%. The present case had LVIDd and LVIDs of 1.40 cm and 1.0 cm respectively. The LVFS was 28.57%. These findings suggest reduced left ventricular internal dimension due to cardiac hypertrophy.

Moise and Fox (1988) stated that left ventricular hypertrophy was associated with pressure overloads, hypertrophic cardiomyopathy, endomyocardial fibrosis, infiltrating myocardial diseases and hyperthyroidism. The authors also opined that right ventricular hypertrophy resulted from pulmonic stenosis, pulmonary thromboembolism and idiopathic pulmonary hypertension.

The Hb and PCV values were 11 g% and 22% respectively on the day of admission. A slightly reduced Hb and markedly reduced PCV would result from hemodilution associated with sodium retention in CHF (Davis, 1995). Ringheim (1975) observed slight reduction in PCV (33%) in dogs with heartworm associated ascites.

Hyperproteinemia (83 g/l), hyperalbuminemia (39g/l) and hyperglobulinemia (44 g/l) were observed in this case. A slight elevation in serum total protein and its

fractions might be because of lipaemia. McGrotty and Knottenbelt (2002) observed pseudohyperproteinemia in dehydrated and lipaemic animals. The A: G ratio was normal. Ascitic fluid had high protein content (32 g/l) and AFPP ratio was 39% suggestive of post-sinusoidal portal hypertension associated with CHF. High-protein ascites (> 25 g/l) is suggestive of post-hepatic portal hypertension and right-sided heart failure (Maddison, 1990).

Liver enzymes remained within the reference range and BUN was also normal. A slight increase in the creatinine concentration (1.7 mg/dl) could be because of dietary factors (Swanson *et al.*, 2004). Serum sodium was elevated (167 mmol/l). Activation of renin-angiotensin – aldosterone axis, natriuretic peptide and poor renal perfusion might be the reason for the elevation of serum sodium concentration (Leib, 1997). This is in concurrence with the findings of Davis (1995). Serum potassium concentration was within the reference range.

On the day of admission, along with Lasilactone<sup>R</sup>, enalapril (0.5 mg/kg BW PO) was given for ten days. A remarkable reduction in ascitic distension was observed on 10<sup>th</sup> day of examination. Enalapril treatment was an effective mode of CHF management (Allworth *et al.*, 1995). Digoxin is usually not advised in hypertrophic cardiomyopathy (Leib, 1997). Dog was treated with enalapril and clinical improvement was evident. The changes observed in serum biochemical parameters and physical examination findings denoted clinical improvement.

### **5.2.2. Dilated cardiomyopathy (DCM)**

According to the echocardiographic findings, two cases (dogs 7 and 8) were diagnosed as dilated cardiomyopathy leading to ascites. Dog: 7 was a seven-year-old male Boxer presented with a complaint of ascitic distension, reduced appetite and hind limb edema and difficulty in walking. Inappetance was observed since two days back. Previously, it was having normal appetite. Ascitic distension was noticed by the owner since one month.

Dog: 8 was a 15-year- old male Mongrel dog presented to the Veterinary College hospital, Mannuthy with a complaint of abdominal distension for the past two months. The owner reported that the urine production was low in this dog. Animal was severely dehydrated. Food intake was very much reduced. Cachexia was observed. Animal was already treated with furosemide and liver extracts for the last two weeks.

Inappetance was observed in both the cases. Moneva-Jordan (2003) opined that inappetance could be directly related to congestive heart failure or related manifestations like fatigue, malaise, dyspnoea and abdominal discomfort associated with ascitic distension and hepatomegaly. The inappetance observed in these dogs has been reported in CHF. The author also opined that cardiac cachexia is a poor prognostic indicator.

Clinical examination of these two dogs revealed respiratory distress and ascitic distension showing fluid thrill on tactile percussion of abdomen (Kelly, 1984). Animal was very weak and cachectic. Cardiac cachexia is a usual complication of congestive heart failure. Cardiac cachexia has been reported in various diseases of heart. Cardiac cachexia is most commonly seen in dogs with dilated cardiomyopathy, especially those with right- sided heart failure. Cardiac cachexia might have resulted from malabsorption, maldigestion, increased energy requirements and hypermetabolic state associated with CHF (Moneva-Jordan, 2003). On physical examination both the dogs revealed loud heart sounds and cardiac palpitation. The precordial thrill and loud heart sounds observed were suggestive of cardiomegaly (Kelly, 1984). Auscultation of the lung revealed no abnormality. Other clinical parameters remained normal and within the physiological norms in both the dogs.

ECG findings observed in dog: 7 were normal 'P' duration (0.04 sec) and 'P' amplitude (0.1 mV). They were within the reference range as described by Bolton



(1975). The 'QRS' duration was 0.04 sec and 'R' amplitude was 0.9 mV. The reduced 'R' amplitude suggested the poor conductivity of electrical impulse to the surface of the body due to the ascitic fluid (Jones, 1985). P-R interval was 0.12 sec and within the reference range of 0.06 – 0.13 sec. Deep 'S' waves were observed in leads I, II, III and aVF. Presence of deep 'S' waves in leads I, II, III and aVF is suggestive of right ventricular enlargement (Bolton (1975); Bond (1997)). The mean electrical axis (MEA) was  $+60^{\circ}$  and within the normal range of electrical axis. The heart rate was 120 bpm. A significant increase in the heart rate ( $183 \pm 49$  bpm) was observed in dogs with dilated cardiomyopathy (Tidholm *et al.*, 2001).

The ECG tracings of dog: 8 revealed a 'P' duration of 0.08 sec and 'P' amplitude of 0.4 mV. 'P' duration is increased and 'P' amplitude was at the maximum height suggesting bi-atrial enlargement (Bolton, 1975). The 'QRS' duration was increased (0.08 sec) and the 'R' amplitude was 2.5 mV. Deep 'S' waves were observed in leads I, II, and III suggestive of right ventricular enlargement (Bolton (1975); Ringheim (1975); Bond (1997)). But the mean electrical axis and heart rate (120 bpm) remained normal. S-T slurring observed in this case suggestive of left ventricular hypertrophy, myocardial hypoxia, myocarditis and electrolyte imbalances such as hypocalcaemia as suggested by Bolton (1975).

Ventricular premature contractions were evident in dogs with dilated cardiomyopathy (Calvert and Jacobs, 2000). Calvert *et al.* (1982) observed sinus rhythm in 80% of dogs with congestive cardiomyopathy. The findings of the present cases did not exhibit the presence of dysrhythmias and it was in contrast to the findings of Calvert and Jacobs (2000). The ventricular tachycardia observed in Doberman pinschers affected with dilated cardiomyopathy (Calvert *et al.*, 1982) was not noticed in these cases.

Ultrasonography of abdomen of both the dogs revealed mildly echogenic ascitic fluid (cellular ascites), suggestive of modified transudate (Spaulding, 1993). The cellular ascites observed in this case might be because of the dispersed RBCs in

the ascitic fluid. Centrifugation of the ascitic fluid revealed RBC sediment in dog: 7. The hypoechoic pseudo discontinuity of urinary bladder wall was observed in these cases (Douglass and Kremkau, 1993).

The hepatic ultrasonogram in both the dogs revealed no abnormality in the echotexture. Gross enlargement of liver with dilated hepatic veins was observed. These findings were reported by Renjith (2003) in right-sided heart failure. The presence of dilated caudal vena cava and hepatic veins, and enlarged and rounded liver or ascites was suggestive of right sided heart failure (Moise and Fox, 1988). Abdominal paracentesis of dog: 8 revealed blood-tinged ascitic fluid. Nephrosonogram was unremarkable in both the cases.

Echocardiography of dog: 7 revealed severely dilated right and left ventricles. Thinning of interventricular septum and hypokinetic walls of the ventricles were observed. The left ventricular internal dimensions were markedly increased and the EF and FS were markedly reduced suggestive of dilated cardiomyopathy (Moise and Fox, 1988).

Echocardiography of dog: 8 revealed no marked abnormality in the left ventricle. But the right ventricle was dilated to a greater extent. The internal dimensions of right ventricle were not measured in this case. Hidaka *et al.* (2003) observed heartworms in the right atrium and right ventricle, and flattening of the interventricular septum in the right parasternal short-axis view of the left ventricle. Moise and Fox (1988) opined that dilatation of the right ventricle was usually associated with right ventricular volume overload resulting from tricuspid insufficiency, atrial septal defects, pulmonic insufficiency and DCM. In the present case, valvular insufficiency could not be confirmed by 2-D echocardiography.

Echocardiographic findings in dilated cardiomyopathy in Doberman pinschers were decreased shortening fraction, decreased per cent systolic thinning of septal and left ventricular free wall and increased E-point septal separation (EPSS)

(Calvert and Brown, 1986; Moise and Fox, 1988). The present case findings are coinciding with the findings of Calvert and Brown (1986) and Moise and Fox (1988).

The normal Hb concentration of 12.5 g% and the PCV of 42% were observed in dog: 8 on the day of admission. The PCV and the Hb were within the reference range on 10<sup>th</sup> day of observation also. The concentrations of liver enzymes were within the reference range in both the dogs. Hidaka *et al.* (2003) observed increased concentration of liver enzymes in dogs with heartworm caval syndrome. The hypoproteinemia, hypoalbuminemia, normoglobulinemia and normal A: G ratio observed in this case has been reported in congestive heart failure by Ringheim (1975) and McGrotty and Knottenbelt (2002). This might be due to inappetance, hypermetabolic state, malabsorption (protein-losing enteropathy) and maldigestion associated with CHF (Moneva- Jordan, 2003).

A slight hyperproteinemia (73 g/l), hyperalbuminemia (56 g/l) and hypoglobulinemia (17 g/l) were observed on the day of admission. A clinical dehydration could have resulted in hyperproteinemia (McGrotty and Knottenbelt, 2002). Hypoglobulinemia might be resulted from protein-losing enteropathy or blood loss in conjunction with hypoalbuminemia.

The protein content of ascitic fluid was 27 g/l and the AFPP ratio 51% in dog: 7. The high protein content of ascitic fluid and increased AFPP ratio suggested post-sinusoidal portal hypertension (Maddison, 1990 and Hunt *et al.*, 1993).

In dog: 8, the A: G ratio was increased (3.3). This might be because of the relative increase in albumin concentration, which could change the ratio. Ascitic fluid was blood-tinged suggesting leakage of RBC into the abdominal fluid. The protein content of ascitic fluid was 45 g/l and AFPP ratio was 62% suggesting high-protein ascites resulting from post-sinusoidal portal hypertension (Hunt *et al.*,

1993). High- protein ascites is a common finding in right- sided congestive heart failure (Maddison, 1990).

The BUN concentration was 38 mg/dl and creatinine 2 mg/dl in dog: 7 on the day of admission. An elevated BUN and serum creatinine suggested prerenal azotemia. Increased BUN concentrations were observed in congestive heart failure associated with heartworm caval syndrome (Hidaka *et al.*, 2003). The BUN and creatinine concentrations were normal in dog: 8. Ringheim (1975) observed a normal BUN level in three dogs with heartworm disease.

Dog: 7 had a serum sodium concentration of 148 mmol/l and potassium 5.6 mmol/l. The normonatremia and mild hyperkalemia might be associated with the variation in volume of distribution of sodium and potassium (DiBartola, 2000). Moneva- Jordan (2003) stated that CHF was usually associated with retention of sodium and the author also opined that hypokalemia might be a feature of diuretic therapy which is not noticed in this case. Mild hyponatremia (136 mmol/l) and a normokalemia (4.2 mmol/l) were observed in dog: 8. Hyponatremia might be resulted from previous therapy with furosemide (Gross, 1995 and DiBartola, 2000).

On the day of admission, the dog: 7 was treated with Lasix<sup>®</sup> (1mg/kg BW) intravenously. From the 2<sup>nd</sup> day, tab. Lasix<sup>®</sup> was given @ of 1 mg/kg BW b.i.d PO. Dextrose (25%) was given to the animal (200 ml i.v). From the 4<sup>th</sup> day, oral digoxin therapy was started. Condition of the animal was stable though the animal could not eat by its own. On 10<sup>th</sup> day the dose of furosemide was increased to 2 mg/kg BW. Uechi *et al* (2003) stated a dose- dependent increase in the diuresis for furosemide therapy. On 20<sup>th</sup> day, the animal died. Death might be due to the cachexia and weakness associated with dilated cardiomyopathy and low- output state of heart.

On the day of admission, the dog: 8 was treated with Lasilactone<sup>R</sup> @ 2 mg/kg BW b.i.d PO. From the 2<sup>nd</sup> day, oral digoxin therapy was started. From the beginning, there was no marked improvement in the condition of the animal. On

10<sup>th</sup> day of examination, though there was reduction in the abdominal distension, the animal was anorectic. Therapy was unsuccessful and the animal died on 15<sup>th</sup> day. Carcass was not available for post- mortem.

### 5.3. ASCITES OF RENAL ORIGIN

Two ascitic dogs (dogs 9 and 10) were affected with nephrotic syndrome. The absence of sonographic changes in liver, kidney and heart, progressing hypoalbuminemia, proteinuria, non responsiveness to therapy and normal concentrations of liver enzymes were suggestive of nephrotic syndrome.

#### 5.3.1. Nephrotic syndrome

Dog: 9 was a five- year- old male Dachshund presented with a complaint of abdominal distension and inappetance. Animal was reportedly voiding deep- yellow coloured urine. Dog was not feeding for the past two days. Dog: 10 was a three- year – old male Doberman pinscher presented with a complaint of abdominal distension, edema of hind limbs and weakness. DiBartola *et al.* (1980) observed non specific signs like weakness, lethargy, weight loss and ascites in dogs with proteinuria associated with nephrotic syndrome. Animal was inappetent. Vaccination and deworming was done in both the dogs as per schedule.

Severe distension of abdomen and fluid thrill was observed in both the cases on tactile percussion of abdomen (Kelly, 1984). Respiratory distress was noticed on lung auscultation. Scrotal and ventral abdominal subcutaneous edema was observed in dog: 9. Bown (1977) stated that nephrotic syndrome was characterized by ascites and subcutaneous edema. Other clinical parameters were normal and within the physiological norms.

Physical examination of dog: 10 revealed, pale mucous membrane and the rectal temperature of 102°F. Pulse rate was 150 / minute. A moderate abdominal

distension was observed. Auscultation of the precordium revealed dull heart sounds and murmurs. Animal was severely anemic with a PCV of 10% and an Hb of 3.9 g%. The murmur and strong pulse observed in this dog could be because of anemia (Kelly, 1984).

Electrocardiography revealed no marked abnormalities in the measurements of complexes in both the nephrotic dogs. A slight S-T slurring was observed in dog: 9. S-T slurring is usually associated with electrolyte imbalances such as hypocalcaemia (Bolton, 1975). The heart rate of dog: 9 was 210 bpm. Tachycardia could arise out of excitement (Kelly, 1984). The mean electrical axis remained normal ( $+60^{\circ}$ ) and within the reference range.

The S-T segment was slightly elevated in dog: 10. The elevation of S-T segment implies myocardial hypoxia or myocarditis (Bolton, 1975). As the animal was anemic, the oxygen carrying capacity of blood would have got reduced and myocardial hypoxia resulted. On 10<sup>th</sup> day of examination, peaking of 'T' waves in lead II was observed, suggestive of hyperkalemia (Bolton, 1975). In this dog, the serum potassium concentration was 6.2 mmol/l on 10<sup>th</sup> day of observation.

Ultrasonography of abdomen revealed anechoic ascitic fluid suggestive of transudate (Spaulding, 1993) in both the nephrotic dogs. Hepatic ultrasonogram did not reveal any abnormality in echotexture and the portal and hepatic vasculature. Nephrosonogram was unremarkable in both the dogs. These findings are in agreement with the findings of Leib (1997).

Leib (1997) stated that nephrosonogram of dogs with nephrotic syndrome did not reveal useful information to diagnose the disease. The author also pointed out that no specific change in the echogenicity of the renal parenchyma could be observed in glomerulonephritis and renal amyloidosis. Echocardiographic findings in dog: 9 were unremarkable and coincided with the reference range stated by Su *et al.* (2003)

Echocardiography of dog: 10 revealed dilated left ventricle and normal size of other chambers. The myocardial contractility remained normal. The left ventricular dilatation observed in this case would be a secondary manifestation of anemia which lead to high- output state (Moise and Fox, 1988). The animal was severely anemic with an Hb of 3.9 g% and a PCV of 10%. Anemia was a clinical feature of nephrotic syndrome associated with glomerular disease and renal amyloidosis as suggested by Cowgill (1983).

Dog: 9 had an Hb concentration of 12 g% and a PCV of 38%. These values were within the reference range as stated by Kaneko *et al.* (1997). On 10<sup>th</sup> and 21<sup>st</sup> days of observation, the PCV and Hb remained normal. A mild hypoproteinemia (43 g/l), severe hypoalbuminemia (5 g/l) and normoglobulinemia (38 g/l) were observed on the day of admission. Serum biochemical assay of dog: 10 revealed hypoproteinemia (42 g/l), hypoalbuminemia (13 g/l) and normoglobulinemia (29 g/l) on the day of admission. Hypoproteinemia and hypoalbuminemia were the consistent features of nephrotic syndrome (Cowgill, 1983; Fleming *et al.*, 1989; Choi and Lee, 2004). These findings concur with the findings of Bown (1977) and DiBartola *et al.* (1980).

The ascitic fluid contained low protein (0.9 g/l and 1.8 g/l respectively in dogs 9 and 10), suggestive of pre-hepatic portal hypertension (Maddison, 1990; Hunt *et al.* 1993). The reduced AFPP ratio (2.09 % and 4.28% respectively in dogs 9 and 10) suggested of pre-sinusoidal portal hypertension. These findings confer with that of Hunt *et al.* (1993). On 10<sup>th</sup> day of examination, protein content of ascitic fluid increased to 37.5%. This followed two times whole blood transfusion in dog: 10.

McGrotty and Knottenbelt (2002) stated that in the presence of concurrent hypoalbuminemia and low- protein ascites, urinary protein: creatinine ratio was essential to confirm protein- losing nephropathy. Choi and Lee (2004) observed hypoproteinemia and hypoalbuminemia in dogs with induced nephrotic syndrome.

On qualitative analysis of urine, proteinuria was observed in both the nephrotic dogs. Proteinuria in the absence of lower urinary tract infections is highly suggestive of protein-losing nephropathy (Cowgill, 1983; Fleming *et al.*, 1989). Urinary protein: creatinine ratio was the most accurate measurement of proteinuria (McGrotty and Knottenbelt (2002). Hypoproteinemia, hypoalbuminemia, normoglobulinemia, proteinuria, low-protein ascites, normal liver function tests, negative ECG and echocardiographic findings suggest loss of protein through urine or faeces.

The serum concentrations of liver enzymes were within the reference range. The BUN level was slightly elevated (33 mg/dl) and serum creatinine was normal (0.85 mg/dl). DiBartola *et al.* (1980) observed increased BUN (>80 mg/dl) and creatinine (>2 mg/dl) in proteinuric dogs with amyloidosis and glomerular disease. The level BUN and creatinine concentration might be normal or variably elevated depending on the degree of impairment of glomerular filtration rate (GFR) in dogs with nephrotic syndrome (Cowgill, 1983). A slight elevation in the BUN level and normal creatinine concentration observed in the present case is in agreement with the findings of Cowgill (1983).

The serum sodium concentration of dog: 9 was elevated on the day of admission (190 mmol/l). Cowgill (1983) stated that nephrotic patients retained sodium and fluid. In the present case, hypernatremia and hyperkalemia (6.0 mmol/l) were noticed. A normonatremia and slight hyperkalemia observed in the present case is in contrast to the findings of Cowgill (1983). On 10<sup>th</sup> day of examination hypernatremia and hyperkalemia were observed as described by Cowgill (1983).

Histopathological examination of renal parenchyma, immunopathological and elution studies were essential to conclude the existence of glomerular diseases (DiBartola *et al.*, 1980).



Urine protein – creatinine ratio the sensitive test in diagnosing proteinuria (DiBartola *et al.*, 1981 and McGrotty and Knottenbelt, 2002). Urinary protein: creatinine ratio was not studied in the present case. Presence of hypoproteinemia, hypoalbuminemia, negative ECG and echocardiographic findings, absence of hepatic abnormality in sonography, normal liver function test and absence of lower urinary tract diseases suggestive of protein loss through urine or feces. In protein-losing nephropathy both the fractions of protein (albumin and globulin) will be reduced.

This dog: 9 was treated with Lasix<sup>®</sup> @ 2 mg/kg BW b.i.d PO and prednisolone (1 mg/kg BW PO) from the 2<sup>nd</sup> day onwards. Diuresis was effective with furosemide. Prednisolone was indicated in dogs with nephrotic syndrome (Forrester, 1997). The distension of the abdomen was reduced on 10<sup>th</sup> day. Because of the continuous loss of protein through urine, the owner was advised to give boiled egg everyday and other vegetable protein- rich commercial foods. On 21<sup>st</sup> day of examination, though the animal was ascitic, distension was reduced to a greater extent than the previous observation. Owner was advised to continue the therapy for one more month.

On the day of admission, dog: 10 was treated with Lasix<sup>®</sup> @ 2 mg/kg BW b.i.d PO for 10 days. Though the abdominal distension got reduced, the general health of the animal did not improve. Whole blood was transfused two times (200ml each time). Prednisolone @ 1 mg/kg BW PO was administered for 10 days (Forrester, 1997). Animal showed slight improvement following blood transfusion and injection of dextrose 25% solution. But, the clinical improvement was transient. On 15<sup>th</sup> day, the dog became comatosed and died. Death would have resulted from chronic anemia and hypoproteinemia resulted from protein- losing nephropathy.

Post mortem findings revealed pale kidney and other visceral organs were normal. Leib (1997) observed pale kidney as the only pathognomonic finding in nephrotic syndrome.

## *Summary*

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## 6. SUMMARY

The study entitled "Clinical and Ultrasonographic investigation of Ascites in Dogs" was conducted to understand the etiopathogenesis of ascites in dogs. Based on electrocardiography, ultrasonography of liver, kidney and heart, abdominal paracentesis and serum biochemical assays, ascitic dogs were classified into ascites of hepatic, renal and cardiac origin.

Among the ten dogs with ascites, five cases had hepatic origin, three cardiac origin and two renal origin. Nine out of 10 cases had visible abdominal distension and one dog had a past history of ascites and treatment for the same with diuretics, did not have visible abdominal distension.

All the five dogs with liver diseases elicited lethargy and inappetance. Subclinical ehrlichiosis was the cause of ascites in two dogs. Two dogs developed ascites due to post- sinusoidal portal hypertension associated with cirrhosis. One dog developed ascites owing to post- sinusoidal portal hypertension associated with chronic active hepatitis.

None of the five dogs with liver diseases showed electrocardiographic and echocardiographic changes. The hyperechogenicity of liver was observed in all the five cases. The liver borders were uneven in two dogs with cirrhosis and one dog with chronic active hepatitis. Ultrasonography of abdomen revealed anechoic fluid in four out of five cases. One dog had cellular ascites. Nephrosonogram was unremarkable in all the five cases.

All the five dogs were anaemic. Mild to marked elevation in the serum concentrations of ALT and ALP was observed in all the five dogs. Hypoproteinemia and hypoalbuminemia were seen in all the five dogs. Serum creatinine and BUN levels were within the reference range.

Three out of ten dogs developed ascites of cardiac origin. Two dogs suffered from DCM and one dog was affected from hypertrophic cardiomyopathy. Two dogs had bi-ventricular failure and one dog had isolated right sided heart failure. Strong femoral pulse, precordial thrill, loud heart sounds and cardiac cachexia were the physical examination findings.

Deep 'Q' waves in leads I, II and aVF, prolonged 'QRS' duration, S-T slurring, tall 'R' waves, mild sinus arrhythmia and S<sub>1</sub>, S<sub>2</sub> and S<sub>3</sub> pattern were the abnormal ECG findings in dogs with CHF.

Ultrasonography of abdomen revealed cellular ascites. One dog had anechoic ascitic fluid suggestive of transudate. Dilated hepatic vessels were observed in two cases suggestive of right- sided heart failure. Echocardiography revealed hypertrophic cardiomyopathy (HCM) in one dog and dilated cardiomyopathy in two dogs. Isolated right- sided cardiac dilatation was observed in one of the three dogs. Haemoglobin, PCV and liver enzymes were within the reference range.

High- protein ascites was observed in all the three dogs. Blood urea nitrogen and serum creatinine were within the reference range. Serum sodium concentration was variable in all the three dogs. Hypokalemia in two dogs and hyperkalemia in one dog were observed.

Two dogs were affected by nephrotic syndrome. Hind limb edema and subcutaneous abdominal edema and inappetance were present along with ascites. S-T segment changes were observed in the ECG of both the dogs. ECG was unremarkable.

Ultrasonography revealed transudate and no abnormalities in the liver, kidney and heart. One dog had dilated left ventricle with normal contractility as it was anaemic. Severe hypoproteinemia, hypoalbuminemia and low protein ascites were observed in both the cases. Slightly elevated BUN was noticed in one of the



two dogs. Serum sodium concentrations were variable in both the dogs. Both the dogs were hyperkalemic.

Six out of 10 dogs survived beyond 30 days following therapy with diuretics, tetracyclines, corticosteroids, silymarin, Liv- 52 vet syrup, digoxin, enalapril as the case may be. Four dogs died during the course of study.

From this study, it was concluded that

1. Usual scanning planes of liver in normal dogs could not be efficiently used. Imaging the liver through the intercostal spaces was efficient in ascitic dogs.
2. Renal ultrasonography could be efficiently done when the animal is in standing position in dogs with ascites.
3. No marked changes were observed in the kidneys of dogs with nephrotic syndrome ultrasonographically.
4. S1, S2, S3 pattern and deep 'Q' waves were the consistent findings in ECG of dogs with right sided heart failure.
5. Protein content of ascitic fluid and Ascitic fluid to plasma protein ratio (AFPP ratio) could be utilized as a criterion for differentiation of pre- sinusoidal and post- sinusoidal portal hypertension.
6. Therapy with silymarin in liver diseases was effective in restoring the clinical condition.
7. Serum sodium and potassium concentrations were highly variable in dogs with ascites.

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\* Originals not consulted

# **CLINICAL AND ULTRASONOGRAPHIC INVESTIGATION OF ASCITES IN DOGS**

**JEGAVEERA PANDIAN. S.**

**Abstract of the thesis submitted in partial fulfilment of the  
requirement for the degree of**

## **Master of Veterinary Science**

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## ABSTRACT

Study entitled "Clinical and Ultrasonographic Investigation of Ascites in Dogs" was conducted in ten dogs. The study aimed at understanding the etiopathogenesis of ascites in dogs. The parameters observed were signalment, history and detailed clinical examination, electrocardiography, ultrasonography of liver, kidney and heart, course of illness, estimation of haemoglobin concentration packed cell volume(PCV), total plasma protein, albumin, A: G ratio, liver enzymes like alanine amino transferase (ALT) and alkaline phosphatase (ALP), protein content in ascitic fluid, ascitic fluid to plasma protein ratio, blood urea nitrogen (BUN), serum creatinine, sodium and potassium.

Inappetance and lethargy were observed in dogs with liver diseases. Cardiac palpitation, loud heart sounds and strong femoral pulse were noticed in dogs with CHF. Non-specific and vague signs were noticed in dogs with nephrotic syndrome. Deep 'Q' waves in leads I, II and aVF, prolonged 'QRS' duration, S-T slurring, tall 'R' waves, mild sinus arrhythmia and S<sub>1</sub>, S<sub>2</sub> and S<sub>3</sub> pattern were the abnormal ECG findings in dogs with CHF. No marked changes could be observed in the ECG of dogs with ascites of hepatic and renal origin.

Ultrasonography of liver revealed hyperechogenicity of parenchyma, specks of hyperechogenicity and mildly echogenic gall bladder contents in three out of five dogs with ascites of hepatic origin. Two dogs had uneven and eroded borders along with hyperechoic liver parenchyma in dogs with ascites of hepatic origin. Nephrosonogram was unremarkable in all the ten dogs. Ultrasonographic findings and serum biochemical findings were coinciding with each other. Ultrasonography was an efficient tool in studying the changes of liver parenchyma and portal vasculature. ECG in cardiac diseases was complementary to echocardiography. Echocardiography was efficient in diagnosing DCM (two dogs) and HCM (one dog).

All the dogs with liver diseases had mild to marked elevation in serum levels of ALT and/ or ALP. Hypoproteinemia and hypoalbuminemia were observed in dogs with liver and kidney diseases. Liver and kidney function tests were unremarkable in dogs with nephrotic syndrome and heart diseases.

Treatment regimen involved administration of furosemide and/ or furosemide + spironolactone, silymarin, Liv- 52 Vet, enalapril, digoxin and prednisolone as the case may be. Six out of 10 dogs survived beyond 30 days following the therapy instituted.

Nephrotic syndrome in dogs could be concluded by progressing hypoproteinemia especially hypoalbuminemia, low- protein ascites, negative ECG and echocardiographic findings and non- responsiveness to therapy. Nephrotic syndrome can be confirmed by biopsy and / or urine protein: creatinine ratio. Liver diseases can be confirmed and characterized only with biopsy.