Ultrasonographic evaluation of liver disorders in dogs

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By

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CERTIFICATE – I

This is to certify that the thesis entitled **"Ultrasonographic evaluation of liver disorders in dogs"** submitted in partial fulfilment of the requirements for the award of the degree of **Master of Veterinary Science** in the subject of **Veterinary Surgery and Radiology** to the Odisha University of Agriculture and Technology, Bhubaneswar is a faithful record of bonafide and original research work carried out by **Varun S N** under my guidance and supervision. No part of this thesis has been submitted for any other degree or diploma.

It is further certified that the assistance and help received by him from various sources during the course of investigation has been duly acknowledged.

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CHAIRMAN
ADVISORY COMMITTEE

CERTIFICATE – II

This is to certify that the thesis entitled **"Ultrasonographic evaluation of liver disorders in dogs"** submitted by **Varun S N** to the Odisha University of Agriculture and Technology, Bhubaneswar in partial fulfilment of the requirements for the degree of **Master of Veterinary Science (Veterinary Surgery and Radiology)** has been approved/disapproved by the student's advisory committee and the External Examiner.

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LIST OF ABBREVIATIONS

%	:	% Percent
gm/dl	:	Gram/deciliter
mg/dl	:	Milligram per decilitre
mg/L	:	Milligram per litre
min.	:	Minute
ml	:	Milliliter
et al.	:	et alibi or ‘And others’
°F	:	Degree Fahrenheit
mg	:	Milligram
kg	:	Kilogram
b. wt.	:	Body weight
mg/kg	:	Milligram per kilogram
viz.,	:	videlicet or ‘Namely’
etc.	:	et caetera or ‘And others of the like/kind’
n or N	:	Numero or ‘Numbers’
i.e.,	:	id est (Latin) or ‘That is’
P	:	Probabilty
<	:	Less than
>	:	More than
mm	:	Millimeter
mm^3	:	Cubic millimeter
WBCs	:	White Blood Cells
RBCs	:	Red Blood Cells
Hb	:	Hemoglobin
TEC	:	Total Erythrocyte Count
TLC	:	Total Leucocyte Count
DLC	:	Differential Leucocyte Count
L	:	Lymphocyte
M	:	Monocyte
E	:	Eosinophils

B	:	Basophils
N	:	Neutrophils
PCV	:	Packed Cell Volume
ALT	:	Alanine Amino Transferase
AST	:	Aspartate Amino Transferase
GGT	:	Gamma glutamyltransferase
ALP	:	Alkaline phosphatase
Hz	:	Hertz
TVCC	:	Teaching Veterinary Clinical Complex
Fig.	:	Figure
Cm	:	Centimeter
VD	:	Ventrodorsal
No.	:	Number
SE	:	Standard Error
CR	:	Computed radiography
mA	:	Milli ampere
e.g.	:	Example
B- mode	:	Brightness or, 2-D mode
OUAT	:	Odisha university of agriculture and technology
±	:	Plus-minus
/	:	Per
MHz	:	Mega hertz

ABSTRACT

The present study was conducted on 46 clinical cases of dogs having hepatic disorder and six apparently healthy dogs as control group at Department of Veterinary Surgery and Radiology, College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar. Preliminary abdominal ultrasound examination of suspected cases of hepatic disorder was done for evaluation of liver parenchyma and cases were selected for study. Detailed history, physical examination findings were recorded and were subjected to haemato-biochemical examinations, radiography and detailed ultrasonographic examination of liver. Among these, diffuse, focal and multifocal parenchymal hepatic abnormalities were diagnosed in 31, 6 and 9 dogs, respectively. The occurrence was more common in middle aged and large breeds of dogs. Lethargy was the common symptom followed by anorexia, vomiting, weight loss and melena. Weak body condition and abdominal distention were seen in nearly half of the cases. Conjunctival mucous membrane was icterus and pale in 1/3rd of cases each. In some of the animals of with diffuse hepatic disorder and multifocal hepatic disorder conditions dehydration and abdominal ballottement were evident. Physiological parameters were within normal physiological range. Haematological leukocytosis with nutrophilia and lymphocytopenia were evident. Liver enzymes viz., ALT, AST, ALP and GGT values were increased and TPP and albumen levels were decreased. Survey radiography of lateral and ventrodorsal thoraco-abdominal radiographs showed size, shape and location of liver in dog without ascites. Liver density was not distinct in ascetic conditions. Ultrasonography was provided saggittal section images of liver parenchyma. The comparative echogenicity with renal cortex was hypoechoic in 20% to 33% of dogs with hepatic disorder. Uniformly isoechoicparenchyma was evident in diffuse parenchymal abnormalities, whereas, focal and multifocal hepatic parenchymal abnormalities were easily identifiable. Assessment of liver location, margins and size was possible in all animals. Ascites conditions provided better visibility of individual lobes. Color Doppler was useful in assessment of vasculature in liver parenchyma and focal/multifocal abnormalities. Ultrasonography was also found useful in accurate biopsy to aid in confirmatory diagnosis.

CHAPTER I

INTRODUCTION

Liver is the largest and very crucial parenchymal organ in the body, which maintains homeostasis by carrying out more than 1500 biochemical functions.Few of these important processes include metabolism of lipids, carbohydrates and amino acids; storage of energy as glycogen; synthesis of plasma proteins, fats and various clotting factors; extramedullaryhaematopoiesis; secretion of bile for proper digestion and detoxification or excretion of drugs and toxins.To cause hepatic dysfunction, hepatic injury must be significant, recurrent or chronic because liver has good regenerative capability and vast functional reserves. Since the liver is associated with so many diverse metabolic functions in the body, any insult that alters normal physiology significantlywill often produce hepatic damage. Such damage may result from infectious, degenerative, metabolic, toxic, congenital or neoplastic diseases.Many drugs have also been found to adversely affect the functioning of the liver (Johnson, 1994).Many times there is an overzealous medication by the owner himself in an attempt for better health care of their petresults in hepatic injury.

Diagnosis of primary hepatobiliary diseases in suspected small animal cases is difficult because of its phenomenal storage, vast functional reserves (about 80%) and magnificent regenerative capabilities. Clinical signs occur only when the reserves are exhausted by progressive disease and are often non-specificandvague, especially in mildor earlystages of liver diseases. Because of these factors and diverse biochemical functions of liver, accurate identification of hepatic disease or its underlying cause using a single test is not possible, hence, requires a combination of tests to assess hepatobiliary system.

Extrahepatic diseases resulting in hepatic dysfunction mimic primary hepatic diseases posing problems in diagnosis. Hepatic changes in response to extrahepaticdiseases are being recognized with increasing frequency (Meyer and Twedt, 2000).

Several studies have been conducted to assess the utility and clinical value of different diagnostic test for diagnosis of liver disease. Diagnosis is a complex process

because no single diagnostic test currently available can be used to accurately diagnose heptobiliary disease in small animal patients (Hess and Bunch, 2000).

Complete blood count, plasma biochemical profile, survey abdominal radiography and ultrasonography is being considered of screening value. In both dogs and catsserum hepatic enzymesactivities are analysed as markers for assessinghepatobiliary disease.Haemato-biochemical findings in hepatopathies are many times deceptive leading to a diagnostic error (Chaudhary *et al.,* 2008). In some liver diseases there is dramatic increase in enzyme activity with normal functional indices while some have subtle changes in enzymatic activity with severe functional disturbance, thus complicating the diagnosis. Some of the liver specific enzymes are also synthesized in other tissues resulting in marked elevations of serum activities of hepatic enzymes above the control range in various non-hepatic conditions (Steiner, 2010).

Both radiography and ultrasonography are used complementary to each other in diagnosis of abdominal affections but lack of contrast in abdominal radiographs limits the precision of diagnosis of abdominal disorders (O' Brien, 1978). Morphologic abnormalitiesin shape, size, density (mineralization/radiolucencies) and position of the liverand presence of abdominal effusioncan be evaluated by using survey abdominal radiographs, but much of the liver is silhouetted by the stomach, right kidney and diaphragm,which makes it difficult to evaluate the entire liver (Konde and Pugh, 1996).

Ultrasonography is choicest modality, which overcomes the disadvantages of radiographyfor identifying the various forms of hepatic disease in canines. The advantages includesdetailednon-invasive assessment of the internal architecture of the liver and its adjacentstructures, including the portal vein with less time consumption.Patients with presence of free peritoneal effusionandelevated liver enzymesare indicated for hepatic ultrasound. Liver size,parenchymal uniformity,parenchymal echogenicity, vascular structures and biliary structures are the features that are typically evaluated during abdominal ultrasound examination (Kemp *et al.,* 2013). Salvekar*et al.* (2010) reported that ultrasonography is helpfulin diagnosis of liver diseases in dogs with altered echotexture of liver parenchyma like hepatic abscess, cyst, fatty liver, cirrhosis and hepatic neoplasia and also suggested

that best results were obtained by combination of both clinico-pathological and ultrasonographic evaluation for correct diagnosis.

Doppler ultrasonography, beingnon-invasiveandrapid, is usedfor the evaluation of hepatic vessels (Brinkman-Ferguson and Biller, 2009) and vascularity of focal hepatic lesions (Harvey and Albrecht, 2001).The absence of ultrasonographic changes does not eliminate the existence of disease (Nyland and Park, 1983 and Feeny*et al.*, 1984), as the ultrasound can detect only the echo textural changes, which may not be present in all the diseases.

Dogs are considered to be popular pets and distinctly occupies a place in a family as a beloved member.In fast changing world of increasing urbanization, environmental pollution, stress and unscientific feeding, like humans, dogs are also becoming more susceptible to hepatobiliary diseases owing to increased concern of the owner regarding their pets. Various studies have been conducted on hepatobiliary affections of dogs over last few years in other parts of India but any systematic study in this part of the country is lacking.

Therefore, the present study was undertaken to evaluate the relative value of transabdominal ultrasonography and to correlate with history, physical examinations, clinical pathology and radiography in arriving at final diagnosis with the following specific objectives.

1. To study the incidence of liver disorders in dogs.
2. To study echogenicity and echotexture of liver parenchyma in liver disorders of dogs.
3. To evaluate the efficacy of ultrasound in the diagnosis of liver disorders in comparison with available diagnostic tests.

CHAPTER II

REVIEW OF LITERATURES

2.1 EPIDEMIOLOGY

Strombeck and Gribble (1978) studied the incidence of chronic active hepatitis (CAH) and it was found to be 8.12 per cent that accounted for 18% of all cases of hepatobiliary diseases in dogsof mean age of 5.3 years.

Bishop *et al.* (1979) reported an association between Chronic Active Hepatitis and Leptospira infection in dogs for the first time.

Hardy (1983) observed that hepatobiliary diseases accounted for three per cent of all animal diseases.

Bergman (1985) studied the prevalence of nodular hyperplasia on necropsy examination. The prevalence was found to be 70 per cent and was related with the age. Around 6 to 8 years age group at which the nodules were found earliest.

Crow (1985) stated that in dogs primary hepatic tumors were found to be uncommon and accounted for <1 per cent of all hepatic tumors

George *et al.* (1986) studied on dogs showing signs of jaundice with reference to age, sex and breed and found males and female with incidence of 59.7 per cent and 40.3 per cent respectively. Alsatian, Dachshund and Pomeranians were frequently affected breeds of dog.

Speeti and Ihantola (1989) opined that young or middle aged Doberman Pinschers were highly predisposed to CAH and treatment was difficult.

Anderson and Sevelius (1991) observed that female predisposition in Labrador retriever, male predisposition in American and English cocker spaniels, and no sex difference in West Highland white terriers for chronic hepatic diseases in dogs. They found hereditary association between chronic liver disease and liver cirrhosis,and breeds.

Johnson (1992) reported incidence of cholelithiasis and cholecystitis in dogs as 0.03 to 1.3 per cent and 0.16 to 0.4 per cent respectively.

Hammer and Sikkema (1995) reported that the incidence of primary hepatic neoplasms in dogs and cats were not common and accounted for only 0.08-2.3 per cent of all neoplasms. Secondary hepatic neoplasms due to metastasis to liver was much more common accounting for 7-36 per cent of dogs having cancer.

Tiwari (2002) observed that 51.9 months as mean age of dogs with hepatic diseases. The ratio (M/F) in hepatitis and cirrhosis was found to be < 1 and also opined that Pomeranian and German Shepherds were over presented.

Boomkens*et al.* (2004) reported that in the given canine population approximately one per cent of the animals were suffering from hepatitis. They also opined that next to human beings and experimental animals, dogs were affected more frequently with hepatitis.

DeNicola (2005) opined that the incidence of primary liver disease are found to be less than 10 per cent or even less. According to a survey conducted by one of the Breed Club,the incidence of liver diseases was estimated to be 2% and opined that there is increase in the incidences of liver diseases in the recent years (Halstead, 2007).

Chohan*et al*. (2007) reported that the primary liver disorders and reactive hepatopathies were 26 (74%) and 9 (26%) respectively in a study consisting of 35 dogs suffering from liver dysfunctions. Amongst primary liver disorders, the acute hepatitis(42%) formed the largest group followed by chronic hepatitis (19%), hepatic venous outflow disorders (15%), cholecystitis (12%) and others conditions such ashepatic abscess, hepatic tumor and hepatic lipidosis (4%) each. Out of all liver dysfunction cases presented 51% were female and 49% were males. They also observed that primary hepatopathies (61.5%) were found to be more common in females whereas males suffered more from reactive hepatopathies (77.8%). The mean of presentation of cases was 5.83 years and it was 7.1 years for cirrhosis. Spitz breed constituted the largest group with 34.3% followed by Labrador retriever as 22.9% of the presented cases of liver dysfunction.

d'Anjou(2008) observed that the commonly encountered hepatic disorders in dogs, include chronic liver disease, neoplasia, hepatic abscess, parenchymal masses, and conditions involving common bile duct andgall bladder etc.

Choudhary*et al.* (2008) in a study of 200 clinical cases of suspected hepatic involvement, reported that German Shepherd, Labrador, Pomeranian, Doberman, LahsaApso and Dalmation were the most commonly affected breeds.

Poldervaart*et al.* (2009) in a histological study of 101 dogs between 2002 and 2006 observed that primary hepatitis occurrence in 0.5% dogs. In the same study, chronic and acute hepatitis were diagnosed in 67and 21 dogs, respectively.

2.2 BREED, AGE AND SEX PREDILECTION

Strombeck and Gribble (1978) stated that the mean age of dogs with CAH was 5.3 years.

Patnaik*et al*. (1981) reported that the prevalence of primaryhepatobiliarytumours were mostly reported in older dogs of 10 to 11.1 years age.

Trigo (1982) observed no sex predisposition in dogs with primary hepatobiliary tumours.

Thormburg*et al.* (1983) observed a wide variation in the age of dogs (8 months to 10 years), diagnosed with cirrhosis.

Rutgers and Haywood (1988) observed chronic hepatitis in Doberman pinschers of 2 to 10 years of age due to hepatic copper accumulation.

Strombeck*et al.* (1988) conducted a retrospective study where the incidence level CH in Doberman pinschers found to be four times greater than the expected level and they also reported no significant difference in the occurrence of CH among males and females.

Mondelli*et al.* (1988) reported increased incidence of CAH in females as compared to males.

In dogs Anderson and Sevelius (1991) conducted studies on epidemiological aspects of chronic liver diseases and found male predisposition in American and English cocker spaniels, female predisposition in Labrador retriever and no sex difference in West Highland white terriers. On the basis of their study, they found hereditary association between chronic liver disease and liver cirrhosis, and breeds. The age of dogs with histologically confirmed chronic (active / progressive) hepatitis was found to be five to seven years up on presentation.

Anderson and Sevelius (1991) and Fuentealba*et al.* (1997) observed no sex difference in West highland terriers, over representation of males in American and English cocker spaniel, and among Labradors females were predominantly affected.

Martin (1993) reported congenital shunts in some breeds of dogs including Yorkshire terriers and Miniature Schnauzers with less than 5 per cent seen in mixed breed dogs.

Rutgers *et al.* (1993) observed high incidence ofidiopathic hepatic fibrosis in German Shepherds were appeared to be associated with young age groups.

Speeti*et al.* (1996) reported that increased incidence of chronic hepatitis in Doberman bitches.

Twedt(2004) observed that theinherited metabolic defect in Beddlington terriers predisposed them for the development of CH and it had been estimated that 25 to 50 per cent might be carriers.

Mandigers*et al*. (2004) reported high incidence of CH in Doberman pinschers in comparison with other breeds and also observed dogs of four to six years age are more affected from liver diseases.

Chohan*et al.* (2007) stated that 51% female and 49% males were affected among 35 dogs suffering from liver dysfunctions. They also found that female suffer more from primary hepatopathies (61.5%) and males suffer more from reactive hepatopathies (77.8%).

Shih *et al*. (2007) observed predisposition ofLabrador retrievers for the development of chronic hepatitis which may progress to liver failure.The Labrador

hepatopathywas commonly observed in middle age to old age dogs with a mean age of 7.3 years (range 3.9 to 14 years).

Ranjith (2007) reported a higher incidence of liver diseases in mongrels (33.33 per cent), followed by Doberman and others breeds.

Choudhary*et al*. (2008) studied 200 clinical cases of suspected hepatic involvement in dogs and reported that German Shepherd, Labrador, Pomeranian, Doberman, LahsaApso and Dalmation were the main breeds affected.

Poldervaart*et al.* (2009) in their study of cases with primary hepatitis, observed an over representation ofAmerican andEnglish Cocker spaniel, followed by Golden andLabrador retrievers, White and Jack Russell terriers,West highland and German pointers. In their study of cases with primary hepatitis, and they also reported that females are over represented than males.

Pooja*et al.* (2010) observed the increased prevalence of hepatitis and cirrhosis in females and intrahepatic PSS in males. The mean age of dogs suffering from hepatic disease was higher than the mean age of dogs with intra hepatic PSS. They also opined that primary or secondary hepatopathies were over represented in Pomeranians as they were the predominant breed among the local canine population.

2.3 CLINICAL PRESENTATION

Witte *et al.* (1971) reported that in most cases of cirrhosis both hepatic lymph (high protein) and mesenteric lymph (low protein) were produced at an increased rate and failure of return of lymph to systemic venous circulation compared to its production results in the development of ascites.

Hardy (1983) reported that accumulation bilirubin in plasma or tissues resulted in development of jaundice, but it is rarely evident until the serum concentration reaches 3 mg/dl or greater.

Rutgers and Haywood(1988)reported that clinical signs exhibited by dogs suffering from chronic hepatitis were in general found to be nonspecific, or may be asymptomatic. Vague symptoms such as anorexia, weight loss, depression, diarrhoea and vomiting were also noted. In advanced disease condition clinical signs such as

ascites, icterus, polyuria and polydipsia as well as neurological signs indicative of hepatic encephalopathy are seen.

Conn and Bircher (1988) reported that hepatic encephalopathy was developed due toreduction of functional liver mass and/ in combination with porto-systemic shunting of blood and the condition was characterised by exhibition ofcomplex of neurological signs.

Wrigley *et al.* (1988a) observed that in cases of canine haemangiosarcomathere was lethargy, anorexia, weakness, depression, pale mucus membrane, and abnormal palpable masses as the most frequent clinicalsigns.

Forrester *et al.* (1992) observed symptoms such as anorexia, fever, vomition, icterus, abdominal discomfort, depression, dehydration, and hepatospleenomegaly in a dog suffering with cholangiohepatitis. Histological examination of biopsy specimens revealed suppurativecholangiohepatitis and splenitis.

Rutgers *et al.* (1993) in dog with hepatic fibrosis observed signs of hepatic encephalopathy, jaundice and portal hypertension predominately rather resulting in ascites.

Batt and Twedt (1994) suspected that failure of liver to clear the endotoxins from blood resulted in vomiting due todirect stimulation of the vomiting centerin the fourth ventricles via the CTZ.

Sevelius (1995) reportedascites as the most common clinical findings in chronic hepatitis which was predominantly associated with cirrhosis and chronic active hepatitis.

Wadhwa*et al.* (1995) in five cases of ascites observed average body temperature of 38.86 ± 0.9 ^{0}C, tachycardia (118.5 ± 5.25/min) and tachypnoea (38.0 ± 5.83/min).

Farrar *et al.* (1996), in a study of 14 dogs diagnosed with hepatic abscess the most common complaints were reported to be anorexia and lethargy followed by vomition and diarrhoea. Physical examination findings included signs of abdominal pain, fever, hepatomegaly, dehydration, and bleeding diathesis. From the study he

opined that hepatic abscesses were uncommon in dogs, but the clinical signs presented were similar to other inflammatory hepatic diseases.

Schwarz *et al.* (1998) observed clinical signs such as vomiting, diarrhoea,lethargy, trembling, fever, polyuria, polydipsia, colic, dehydration and tachypnoea in dogs diagnosed with hepatic abscess.

Downs *et al.* (1998) reported history of vomitingand depression since past two days in a dog with liver abscess andliver lobe torsion.Physical examination of the dog revealed lethargy,rectal temperature of 105.4 ºF, rapid irregular heartbeatand weak femoral pulses.

Rothuizen and Meyer (2000) observed icterus, hepatomegaly, spleenomegaly, ascites, and pale mucous membrane were common findings while petechiation of skin or mucous membrane was extremely infrequent and opined that physical examination was informative in only a minority of dogs with liver disease.

Hess and Bunch(2000) observed that many liver diseases resulted in hepatic encephalopathy. Clinical signs indicative of hepatic encephalopathy included circling, aimless wandering, head pressing, weakness, blindness, ataxia, ptyalism, aggression, seizures, dementia and coma.

Sonnenfield*et al.* (2001) in a 12 year old, male neutered dog diagnosed with liver lobe torsion reported the history of inappetence and lethargy that had been slowly improving. The dog was alert and bright and palpationof cranial abdomen revealed a large firm non-painful mass.

Twedt (1998), Rudgers and Haywood(1998) and Sterczer*et al.*(2001) described that the clinical signs observed were in correspond with the extent of hepatic damage and vomiting, diarrhoea and poor appetite or anorexia were the early signs; whereas ascites, jaundice and hepatic encephalopathy were result of the disease progression.

Vijaykumar (2002) observed vomiting, cough, anorexia, lethargy, abdominal distention, breathing difficulty, groaning while lying down,abdominal discomfort,episodic weakness and fever, scrotal or penile oedema, weight gain in dogs with ascites.

Varshney and Hoque (2002) reported varied clinical signs including jaundice, mild anaemia, nausea/vomition, abdominal distention, constipation, diarrhoea, convulsion, hypersalivation, head pressing, muscle tremor and melena in a study of 24 clinical cases of canine hepatopathy. Other nonspecific signs like chronic anorexia, weakness, emaciation and depression were occur in most of the dogs.

In a retrospective study,Liptak*et al.* (2004) reported that during physical examinationabdominal mass was palpable in 19 dogs out of 48 dogs with hepatocellular carcinoma. Ascites was detected in 2 dogs, signs of pain were observed in 7 dogs and weight loss was observed in eleven dogs under study while icterus was detected in none of the dog.

Tiwari *et al.*(2005) observedthe clinical symptoms of prolonged anorexia, pale/icteric mucus membrane, nausea/vomiting, weakness and pain in epiastric region etc. though vague and nonspecific, are strong indicators of hepatobiliary diseases.

Shih *et al.*(2007) stated that most of the dogs with hepatic diseases had vague clinical signs of decreased appetite, lethargy, vomiting, weight loss but some were asymptomatic except for increases in serum liver enzymes

Vijayanand and Nagarajan (2007) observed clinical signs such as anorexia, depression, lethargy, weight loss, polydipsia,polyuria, vomiting, and diarrhoeain a dog suffering from cirrhosis.

Chaudhary *et al.* (2008) in dogs with hepatic diseases recorded nausea, vomiting, mild anaemia, constipation, polydipsia, pyrexia, jaundice, ascites, polyuria, muscle tremors, weakness, debility, anorexia etc. They opined that history of prolonged anorexia and weakness may be suspected for liver involvement.

Mirceau*et al.* (2008) in dogs suffering with hepatobiliary diseaseobserved inappetence, vomiting, lethargy, abdominal distention, weight loss, polydipsia, cutaneous lesions, fever etc.

Pooja*et al.* (2010) reported that the clinical signs were vague and varying from decreased appetite, anorexia, weakness, weight loss, nausea, vomiting, ascites, , bilateral hind limb edema, polyuria, polydipsia, pale mucosa, epigastric pain,

pyrexiaconstipation, diarrhoea, melena, icterus, encephalopathy to depression in different combination.

Salvekar*et al*. (2010) opined that, it posed difficulty to appreciate liver disorders in early stages due gradual development of symptoms which are relatively nonspecific.

Saravanan*et al.* (2012) reportedlethargy, anorexia, distended abdomen in a dog with ascites. They opined these signs as non-specific due to varietyof causes results in ascites condition.

Kumar *et al*. (2013) observed dullness, depression, inappetence, anorexia, anaemia, vomiting, abdominal pain, ascites, diarrhoea, pedal edema, icterus and swollen lymph nodes in dogs with hepatobiliary dysfunction.

Elhiblu (2015) observed inappetence, halitosis, polyuria, polydipsia, dehydration, icterus, melena, hematochezia, weight loss and abdominal distentionin dogs with cirrhosis of liver.

2.4 HAEMATO-BIOCHEMICAL PARAMETERS

2.4.1 HAEMATOLOGY

Patnaik*et al*. (1981) observed that nonspecific hematologic abnormalities were common in hepatic diseases and included mild to moderate anaemia and leucocytosis.

Wadhwa*et al.* (1995) reported low haemoglobin (7.8 ± 0.22 g/dl) and packed cell volume (24.5 ± 2.95%), whereas total differential leucocyte counts were almost unaffectedin five cases of ascites.

Farrar *et al.* (1996) reported leucocytosis (11 dogs), neutrophilia (11 dogs) with left shift (8 dogs), mild lymphopenia (5 dogs),mild monocytosis (2 dogs), mild to moderate thrombocytopenia (7 dogs) and mild anemia (8 dogs) among 14 cases of liver abscesses.

Center (1996) observed anaemia, abnormal erythrocyte morphology reduced platelet number or function and lipaemic plasma in cases of liver pathology.

Fuentealba*et al.* (1997) opined that haematological values were nonspecific in case of chronic hepatitis.

Downs *et al.* (1998) recorded moderate neutrophilicleucocytosis with left shift andmarked thrombocytopenia with increased mean platelet volumein a dog diagnosed with liver lobe torsion and liver abscess.

Schwarz *et al*. (1998) in dogs with hepatic abscess observedneutrophilia with left shift, monocytosis, anaemia and thrombocytopenia.

Hess and Bunch (2000) reported variety of CBC abnormalities in various hepatobiliary diseases and alsoobserved microcytosis without anaemia in dogs and cats affected with congenital PSS.

Besso*et al*. (2000) observed leucocytosis with mature neutrophilia in a dog with gallbladder mucocele.

Howe *et al*. (2000) in cases of portosystermic shunts reported that there might be a mild, non-regenerative anaemia, microcythemiawith normochromic erythrocytes and target cell formation.

Johnson (2000) opined that anaemia present in hepatic tumours might be associated with anemia of chronic disease or excessive haemorrhage from neoplasm.

Bush (2002) reported that neutrophilicleucocytosis in hepatic disorders was opined due to acute inflammatory conditions.

Tiwari (2002) reported significantly reduced mean haemoglobin level (7.36 g %) as compared to that in healthy dogs (11.33 g %) in dogs suffering from different hepatic diseases. The clotting time was slightly higher in dogs with hepatic cirrhosis, but was within normal range and did not differ significantly from that of healthy group.

Liptak*et al.* (2004) in a study of 41 dogs having hepatocellular carcinoma recorded anemia in 22 dogs,leucocytosis in 11 dogs, thrombocytosis was diagnosed in 18 dogs, microcytosis in 12 dogs and PCV ranged from 35% to 39% in 13 of these dogs.

Thushara*et al*. (2006) reported low Hb (8 g/dl), leucocytosis (19x103 /µl) and differential leucocyte count of 72 % Neutrophils, 26% Lyphocytes and 2%Eosinopils in a case of hepatic cirrhosis associated ascites in a dog.

Tiwari *et al*. (2007) reported significantly low Hb and PCV in a study of liver in 58 dogs suffering from diseases of non-hepatic origin.

Vijayakumar*et al*. (2008) studied dogs with hepatic disorders. They observed decreased erythrocytecount in diffuse parenchymal diseases with ascites, hepatitis due to leptospirosisandoxytetracycline induced hepatitis, while neutrophilic leucocytosis was noticed in all the hepatic diseased groups.

Sarma *et al.* (2009) observed low levels of Hb (9.48 ± 0.30 g/dl) and PCV (34.50 ± 1.26%), whereas TLC and DLC were almost unaffected in a study of 15 cases of dogs suffering from ascites.

Chohan*et al.* (2009) observed significant decrease in haemoglobin in acute and chronic hepatitis while in acute hepatitis, chronic hepatitis and cholecystitisneutrophilic leucocytosis was observed.

Tomar*et al.* (2011) observed elevated levels of total leucocyte count and neutrophililia, while decreased levels of haemoglobin and PCV in dogs suffering from cholecystitis.

Kumar *et al*. (2013) reported significantly lower values of TEC and lymphocytes, and significantly increasedTLC with neutrophilia in dogs suffering from hepatic disease. Normocytic and normochromic anaemia was the most common finding in hepatic disease often associated with insufficient use of systemic iron store.

Tantary*et al*. (2013) found nonsignificantly decreased Hb, PCV, TEC and platelet count, unaltered TLC and increased clotting time in dogs suffering from ascites due to chronic hepatitis.

Tantary*et al.* (2014) recorded decreased Hb, PCV, TEC and total platelet counts; significantly increased clotting time, non-significantly increased TLC and slight neutrophiliain dogs with hepatic disorders.

Elhiblu*et al.* (2015) in dogs affected with liver cirrhosis,observed decreased values of haemoglobin, lymphocytes, PCV, platelet count, fibrinogen and increased values of TLC and neutrophilic counts.

2.4.2 BIOCHEMICAL PROFILE

Price and Sammons (1976) reported that elevation of serum ALP was a result of intrahepatic and extrahepatic cholestasis. They also opined that increased serum ALP in a patient older than one year was usually of hepatic origin unless the patient had the bone disease.

Strombeck*et al.* (1976) observed normal blood chemistry, haemogram and serum electrolyte values but decreased total plasma protein (5 gm/dl) in chronic active hepatitis. Electrophoresis revealed that hypoproteinemia occurred mainly due to hypoalbuminemia.

Strombeck (1979) observed substantial elevation of serum ALT as the most consistent finding in severe acute hepatocellular injury and necrosis.

Singh and Nigam (1980) observed marked elevation in values plasma ALT levels with its peak between $7^{th}$to 10^{th}days of experimentally induced biliary obstruction in dogs.

Patnaik*et al*. (1981) reported that in dogs with hepatocellular carcinoma, liver enzymes serum concentration(especially ALT and AST) were significantly increased. In most of the cases of hepatocellular carcinoma they observed high serum activities of more than one liver enzymes.

Guelfi*et al.* (1982) reported that total bilirubin was less sensitive than ALP, the activity of which increased rapidly and persistently but it was not very specific to liver, and suggested that serum GGT levels should be used in conjunction to confirm the cholestaticorigin of increase in ALP.

Hall (1985) reported decreased albumin production associated with liver disease resulted in hypoproteinaemia and could be used to differentiate acute from longterm disease.

Inghe*t al.*(1986) observed thatextrahepatic cholestasis and intrahepatic cholestasis can be differentiated by studying plasma GGT levels, degree of icterus and presence of alcoholic faeces and histological examination of liver biopsy was of reliable diagnostic value.

Rothuizen and Van Den Brom (1987) reported that hyperbilirubinemia(both conjugated and unconjugated) in dogs and cats as a result of liver dysfunction with cholestasis and increased production of bilirubin due to primary hepatic and haemolytic disease.

Valentine *et al*. (1990) reported that acute hepatocellular injury and necrosis resulted in significant increase in serum ALT levels.

Center(1996) and Valentine *et al*. (1990) observed that in cases of hepatic disorders ALT activity have the highest sensitivity (more than 80%) but in cases of hepatic congestion, neoplasia, and portosystemic vascular anomalies have less sensitivity (under 60 %).

Valentine *et al.*(1990) reported that the increases in activity of either ALT or AST indicate leakage of the enzymes and hepatocellular membrane damage. AST serum rising in the absence of increased ALT activity, show an extrahepatic problems, like muscle injury.

Center*et al.* (1992) in 270 cases of confirmed liver diseases in dogs,observedgreater increase in ALP than GGT in both frequency and magnitude. Further they concluded that Alkaline Phosphatase had higher sensitivity but lower specificity than Gamma GlutamylTransferase as an indicator of liver disease.

Sevelius and Anderson (1994) observedchanges in GGTand ALP was almost similarmanner in cholestasis and opined thathypoalbuminemia could also be considered as indicator of liver function.

Solter*et al.* (1994) reported alkaline phosphatase as a selective indicatorin most of the mammalian species including dogs for cholestasis.

Farrar *et al*. (1996) reported elevated levels of ALP (14 dogs) and ALT activities (11dogs) in 14 cases of hepatic abscesses. They also reported

hypoproteinemia (8 dogs), hypoalbuminemia (11 dogs), high bilirubin concentration (5 dogs) and prolonged coagulation (5 dogs) values.

Tennant (1997) reported that chronic hepatic disorders such as cirrhosis and portosystemic vascular abnormalities resulted in serum hypoalbuminemia.

Schwarz *et al*. (1998) observed increased levels of ALP, AST and ALT along with increased total bilirubin concentration in a study of 5 dogs with hepatic abscess.

Daniel and Marshall (1999) and Rosalki and Mcintyre(1999) reported that the hepatic synthetic and secretory capacities are large; only severe and usually prolonged liver disease, such as cirrhosis, demonstrably impairs albumin and prothrombin synthesis.

Prause and Twedt(2000) observed that the liver function tests were found to be normal with increase in serum ALP activity but serum ALT activity was less commonly found to be increasedin the cases of nodular hyperplasia.

Johnson (2000) stated that ALT was cytosolic enzyme; the magnitude of hepatic disease was in proportionwith the number of hepatocytes injured resulting in increased concentration of ALT in serum.

Varshney and Hoque (2002) studied 24 cases of hepatopathies in dogs and reported that levels of total serum protein, serum albumin, BUN and blood glucose were considerably low in cases of intrahepatic porto-systemic shunt (PSS) and cirrhosis. The activities of serum alkaline phosphatase and alanine amino transferase were also lower side in porto-systemic shunts. However, in chronic active hepatitis serum alkaline phosphatase, alanine aminotransferase and arginase value were high.

Vijaykumar (2002) reported decreased levels of cholesterol and albumin, elevated levels of ALT, AST, ALP, bilirubin and bile acids in liver diseases and low levels of glucose in hepatic disease of ascitic dogs.

Vijaykumar*et al.* (2004) studied biochemical profile of 18 dogs with diffuse hepatic parenchymal disorder and reported significantly elevated levels of ALT, SAP, bile acids and bilirubin in affected dogs when compared with healthy control group in comparison the levels of TP, albumin and glucose were markedly low in dogs with hepatic dysfunctions.

Liptak*et al.* (2004) opined that change in serum concentration of alanine aminotransferase and aspartate aminotransferase levels were found in proportionwith the extent of hepatocellular injury, high serum levels of ALT and AST were indicative of poor prognosis of hepatocellular carcinoma.

Arambulo*et al.* (2004) in a study of malignant histiocytosis, malignant histiocytoma and malignant fibrous histiocytoma in dogs reporteddecreased levels of total protein, albumin and calcium; elevated levels of ALP and total bilirubin.

Tiwari *et al.* (2005) reported markedly elevated level of ALT, SAP and GGT in canine hepatosis as compared to that of healthy dogs.

Tiwari *et al.* (2007) reported reactive hepatopathy in 58 dogs with diseases of extra-hepatic origin (babesiosis, ehrlichiosis and mixed infection of both, canine viral diarrhea simulating to parvo, epilepsy, trypanosomosis, pyometra and hydronephrosis) and found significantly low albumin whereas bilirubin and activities of ALT and ALP were significantly higher in diseased dogs.

Vijayanand and Nagarajan (2007) in a dog with cirrhosis,reported increased levels ofserumalkaline phosphatase,alanine aminotransferase, and decreased levels of albumin.

Bandyopadhyay*et al.* (2008) reported liver specific enzymes like ALT, ALP and GGT activity and biochemical parameters like total cholesterol, triglyceride, total and direct bilirubin, were found to be markedly elevated in serum of the dogs with mucocele or cholecystitis.

Chaudhary *et al.*(2008) observed that dogs diagnosed with hepatitis and cirrhosis, levels of TP, serum albumin, blood glucose found to be decreased whereas values of alkaline phosphatase, alanine aminotransferase increased.

Mircean*et al.* (2008) in a study of focal hepatic lesions such as hepatic abscessand hemangiosarcomaobserved elevated of serum hepatic enzymes.

Chohan*et al.* (2009) observed that activities of alanine aminotransferase, aspartate aminotransferase, alkaline phosphatase and gamma glutamyltransferase enzymes were significantly higher in acute and chronic hepatitis as well asin

cholecystitis. Total bilirubin also increased significantly but the change was pronounced in acute hepatitis.

Salvekar*et al.* (2010) correlated the findings of serum biochemical parameters and ultrasonography in dogs suffering from various liver disorders and reported that increase in liver specific enzymes were insignificant and were within normal physiological range in many cases.

Xenoulis and Steiner(2010) and Lakner*et al.*(2011) opined that hypoalbuminemia asa insensitive marker for hepatic insufficiency and were of the opinion that hypoalbuminemia is likely to be seen in patients with portosystemic shuntsoradvanced chronic liver disease.

Tomar*et al.* (2011) observed elevated serum ALT and bilirubin levelsin dogs suffering from cholecystitis.

Kumar *et al.* (2013) studied the therapeutic management of hepatobiliary dysfunction in canines and found significantly elevated ALT, AST and GGT levels in dogs.

Tantary*et al.* (2013) observedsignificant increase in serum concentrations ofAST, AST, ALP, GGT, BUN and bilirubin; and significantly decreased total protein, globulin, albumin, albumin/globulin ratio, blood glucose and cholesterol levels in dogs with hepatic disorders.

Tantary*et al*. (2014) recorded significant increase in values of ALT, AST, ALP, GGT, total bilirubin and BUN, and creatinine; and significantly decreased mean values of plasma glucose, plasma cholesterol, total protein, albumin, globulin, and albumin/globulin ratio in dogs with hepatic disorders.

Elhiblu*et al.* (2015) reported significantly lower levels of glucose, total protein, albumin, albumin-globulin ratio and fibrinogen; significantly increased values of aspartate amino transferase, alanine amino transferase, activated plasma thrombin time and prothrombin time in dogs affected with cirrhosis of liver.

Kozat and Sepehrizadeh (2017) reported that ALP activity measurement hadlowspecificity (50%), but high sensitivity (80%), for hepatobiliary diseases.Changes in serum gamma-glutamyltransferase (GGT) activity generally in

parallel with those in serum ALP activity. Specificity for liver disease increases up to 90% if elevated ALP activity is noted with a concurrent increase in serum GGT activity.Severe raising in GGT activity have importance in diseases of the biliary epithelium such as bile duct obstruction and cholecystitis (Center, 1996).

Kozat and Sepehrizadeh(2017) described that ALT is metabolized in the liver,liver disease in the dog results longer serum half-life and persistent increases of ALT are characteristic of chronic hepatitis in the dog. ALT increases should be investigated when they are greater than twice the normal or persistently abnormal.

2.5 HEPATIC IMAGING

2.5.1 RADIOGRAPHY

Root (1974) and O‟Brien (1978) reported that diffuse hepatomegalies resulted in substantial portion of caudal liver margins to project caudal to the costal arch, indicating an obvious increase in liver size and rounding of costal liver edges in lateral radiographs.

O‟Brien (1978) and Pechman Jr. (1998) observed that visualization of gall bladder as a separate structure was not possible on the survey radiographs as it had the same radiographic density as that of hepatic parenchyma.

O‟Brien(1978)opined that with the stage and severity of the disease radiographic appearance of cirrhosis varied and reported that a small, dense liver with irregular nodular surface with or without pneumoperitoniograph as the most common radiographic appearance of the cirrhosed liver.

Suter(1982)reported thatof reduced liver size (microhepatica)is more difficult to appreciate radiographicallythan hepatomegaly. In dogs with ascites, abdominal effusion interferes with the radiographic evaluation of the liver and other abdominal organs resulting in loss of detail withits classic “ground glass” appearance.

Godshalk*et al.* (1988) and Barr (1992) reported that the radiographic evaluation of alteration in liver size was subjective and was insensitive to subtle changes and accurate evaluation of mild generalized changes in liver size was difficult.

Lamb (1991) reported that choleliths were radiographically recognized at cranial right ventral shadow as discrete rounded radiopacities.

Partington and Biller (1995) opined that the diseased liver may change in size, position, shape or opacityradiographically. Focal hepatic enlargement could be detected by alteration in the hepatic margins or the localized displacements of the gastric body, fundus, pylorus, cranial duodenal flexure, right kidney, transverse colon and diaphragm orhead of the spleen.Contrast studies such as cholecystography, portography, peritoneography, and arteriography may be performed to aid in the radiographic diagnosis.

Pechman Jr. (1998) reported that the enlargement of the liver lobe resulted in displacement of the body and pyloric region of the stomach dorsally and to the left.

Konde and Pugh (1996) reported that thesurvey abdominal radiographs (both lateral and ventrodorsal views) are useful for the evaluation of the morphologic abnormalities in shape, size, position and density (radiolucencies/mineralization) of the liver and presence of abdominal effusion but as much of the liver is silhouetted by the stomach, diaphragm and right kidney, evaluation of the entire liver is difficult.

England (1996) reported that, assessment of liver size was inexact and subjective. Evaluation of the liver borders and the position of adjacent structures like costal arch, right kidney, stomach and duodenal flexure was needed for the assessment of alteration in liver size. Theyalso opined that, radiographic measurement of liver length was a consistent parameter for the assessment of liver sizethat might be used by comparison with a standard anatomical land mark such as vertebral body length.

Farrar *et al.* (1996) performed abdominal radiography in which 9 of 14 dogs had hepatic abscesses. Radiography revealed hepatomegaly and splenomegaly in six and three dogs respectively and three had poor abdominal detail. Only one dog was suspected to have hepatic mass on radiograph.

Downs *et al.* (1998) in a survey abdominal radiograph of dog with liver lobe torsion and liver abscessobserved a mottled soft tissue and gas opacity caudal to the stomach and cranial to the transverse colon.

Kull *et al.* (2001) opined identification of extrahepatic abnormalities that affect the liver may be possibleradiographically. However,information regardingt the hepatic parenchymal changes is limited.

Vijaykumar(2002)observedhazy opaque abdominal cavity with a classic ground glass appearance on lateral abdominal radiographs of dogs suffering from ascites.

Liptak*et al.* (2004) studied abdominal and thoracic radiographs in 34 dogs out of the total 48 dogs having hepatocellular carcinoma. Pulmonary metastasis was not detected in any dog while in all the 9 dogs on which abdominal radiography was performed cranial abdominal mass, displacing the stomach caudolaterally, was detected.

Chaudhary *et al*. (2008) observed displacement of caudal liver edges beyond the rib cage in dogs with hepatomegaly.

Adel (2012) recorded normal hepatic size without hepatobiliary calcification on a lateral abdominal radiograph of a dog with polypoid lesions in the gallbladder.

Elhiblu*et al.* (2015) could not get any information to diagnose cirrhosis in dogs through radiographs.

2.5.2 ULTRASONOGRAPHIC FEATURES

Hepatic lymphosarcomaappeared with varied sonographic features such as diffuse, increased ordecreased echogenicity in a normal to enlarged liver (Feeney *et al.,* 1984),multifocal poorly circumscribed hypoechoic areas or well-circumscribed hypoechoic nodules surrounded by areas of hyperechogenicity (target lesions) (Nyland, 1984 and Lamb, 1991), solitary hyperechoic mass (Whiteley*et al.,* 1989).

Nyland and Hager (1985) also reported decreased hepatic echogenicity in dogs with hepatic lymphoma.

Nyland and Hager (1985) has reported diffused parenchymal abnormalities in hepatomegaly with normal, decreased orincreased parenchymal echogenicity wherein visualisation of the discrete hepatic borders were not possible in cases of acute hepatic failure in dogs.

Nyland and Hager (1985) and Lamb (1991) reported the layered appearanceof thickened gall bladder wallincholecystitiswas due to visualization of both the outer andinner walls, giving double rim effect.

Nyland*et al.* (1989) opined that ultrasonography was helpful in detection of focal hepatic, disorders of the biliary system andvascular abnormalities but when the liver was diffusely affected without parenchymal abnormalities it was less helpful. Percutaneous ultrasound guided biopsy helped definitive diagnosis of these lesions.

Lamb (1990) observed that primary hepatic neoplasiaultrasonographicallyhad a variable appearanceranging from a large, moderate circumscribed, infiltrating mass with an echogenicity slightly more mixed than normal liver bulging beyond the normal liver margins.

Stowater*et al.* (1990) and Voros*et al*. (1991) studied cases of cirrhosis in dogs and reported multiple hepatic nodules due tomacronodular regeneration along withhyperechogenic hepatic parenchyma and decreased liver lobe size.

Biller *et al.* (1992) reported cirrhosis with increased echogenicity of hepatic parenchyma, less distinct appearance of the portal vein margins and increased beam attenuation resulting in decreased distal visualization.

Biller *et al.* (1992) opinedultrasonographyas an important diagnostic toolfor the evaluation of diffuse hepatic parenchymal diseases. Ultrasonographically diffuse liver diseasesappear as change in the liver echogenicity when compared to the spleen or renal cortex as either hypoechoic due to suppurative hepatitis,congestion and lymphoma orhyperechoic due to cirrhosis,steroid hepatopathy, andfatty change,.

Yeager and Mohammad (1992) and Partington*et al.* (1992) observed increased hepatic echogenecity in disease conditions like cirrhosis, steroid hepatopathy,hepatic lipidosis, long term cholengiohepatitis,lymphosarcoma and in some of the toxic hepatopathies.

Yeager *et al.* (1992) in animals suffering from obesity, hepatic lipidosis or diabetes mellitus, fatty infiltration of liver parenchymaresulted in increased attenuation ofbeam in the deeper portions of the liver parenchyma.

Penninck*et al.* (1993) reported that, in veterinary medicine fine needle aspiration and tissue core biopsy of liver parenchyma were the commonlyperformed procedures under ultrasound guidance.Ultrasound guided fine needle aspiration method helped in preoperative diagnosis of hepatic nodular lesions of the liver and also in differentiating between malignant from benign tumours.

Lamb and Mahoney (1994) opinedthat it was difficult to diagnose the portal hypertension with standard ultrasonography and were of the view that enlarged main portal as well as extra hepatic portal veins, presence of multiple portosystemic collateral vessels, ascites, splenomegaly and abnormal liver echogenicity were the associated features with portal hypertension whereas Johnson (1994) reported ultrasound as an effective diagnostic tool for the evaluation of the presence of portal hypertension and for the assessment of its effects.

Carlisle (1995)observed no difference in the visibility of hepatic and portal vessels between dogs with different body conformation (shallow or deep thorax) or with different amounts of fat.

Selcer (1995) reported difficulty in identification of the focal lesion when it is less than 2 cm in diameter or when the parenchyma is isoechoic with normal tissue.

Hughes and King (1995) in cases of acute hepatic failure in dogs, reported varied sonographicfindings ranging from normal to decreasedordiffusely mottled hepatic echogenicity.

Lamb (1995) reported that normal liver lobe has sharp edges whereas large liver lobes in hepatomegaly often had rounded margins.

Partington and Biller (1995) and Nyland and Park (1983) observed choleliths as hyperechoic foci with some degree of acoustic shadowing within the dependent lumen of the GB.

Farrar *et al*. (1996) studied on dogs having hepatic abscesses and the appearance varied fromhypoechoic, heteroechoictohyperechoicultrasonographically. Early stages of abscess with central region of liquefactiveor coagulativenecrosis appeared as hypoechoicmasses while heteroechoic masses represented established

abscess. The hyperechoicappearance wasdue to as caseous suppuration and fibrosis of the abcess over time.

Schwarz *et al.* (1998) reportedultrasographic findings of 13 dogs with hepatic abscessation. Hepaticabscessation was characterized by number, shape, size, locationandechogenicity. Solitary lesions which are greater than 3 cm in size were more common than multiple ones. The shape of the lesionsvaried from round to oval or even irregular.The abscesses were most commonly observed as poorly echogenic lesions, often with central cavitation.

Saunders (1998) observedthat noncysticcavitary lesions (e.g. abcesses, hematomas, cavitated neoplasms,hepatic nodular hyperplasia) are mixed echoic structures with either well or poorly defined borders resulting in edge shadowing that is less apparent than that occurring from well defined cysts.

Hill *et al.* (2000) reported abnormal heterogeneous hepatic echogenecity(mottled appearance) and abnormally dominant marginal echoes of the intra hepatic branches of portal vein, suggestive of periportal fibrosis in cirrhotic liver.

Spaulding (1993) and Stieger and Url (2001) diagnosed cholecystitiswithhyperechoic GB wall thicker than 3-3.5 mm and 4 mm were of the opinion that thickened GB wall might be isoechogenic with hepatic parenchyma due to inflammatory processes.

Sonnenfield*et al.* (2001) reported a case of liver lobe torsion with a mass in the cranial abdomenin a dog. Radiography was indicative of splenic origin of the mass whereas ultrasonography showed that the mass originating from right side of the liver. Ultrasonographically the mass was continuous with the liver, along withhyperechoic walled vessels suggestive of portal vessels were identified within the mass, two aspirates under ultrasound guidance were also taken from the mass for further diagnosis.

Hoque and Varshney (2001) classified hepatic abnormalities as hepatitis, cirrhosis, intrahepatic portosystemic shunts and gall bladder abnormalities cases either alone or in combination in aultrasonographic study of 35 clinical cases of hepatobilliary involvement in dogs.

Vijayakumar*et al.* (2001) in a dog with cholecystitisobservedthe thickened gall bladder wall and presence of echogenic bile using ultrasosnography.

In dogs suffering from cirrhosis,Varshney and Hoque (2002) observed diffuse hyperechoic and bright echotexture of liver with prominent vasculature.

Vijaykumar (2002) in their study over therapeutic management of ascites in dogs and reported hyperechoic to mixed echogenicity, nodular pattern or porto-cavalshunt of liver in hepatic diseases, dilated hepatic vasculature in right congestive heart failure and floating abdominal organs in the ascitic fluid.

Cuccovillo and Lamb (2002) studied the cellular features of ultrasonographictarget lesions of the liver in dogs. On ultrasound the lesions had hyperechoicinner zone, hypoechoicouter zonewhen compared to the echogenicity of surrounding parenchyma. It was concluded in the study, that finding of one or more target lesions in the liver or spleen had a positive predictive value for malignancy of 74% cases. The finding of multiple target lesions in one organ had 81% positive predictive value for malignancy. Benign lesions associated with target lesions were nodular hyperplasia of liver, cirrhosis, pyogranulomatous hepatitis and chronic active hepatitis.

Doppler ultrasound evaluation of focal liver lesions has been used in an effort to differentiate benign from malignant diseases and to characterize various tumour types (Nyland and Mattoon, 2002).

Tiwari *et al.* (2003) conductedultrasonographic survey of canine gall bladder diseases. Distension of gall bladder, presence of sludge, thickening (>3.5 mm) of gall bladder wall, cholelith and biliary obstruction were observed in 25, 17, 15, 1 and 2 dogs respectivelyeither alone or in combination.

Tiwari *et al.* (2005) observed biliary sludge in 13 cases (15.95%) in an ultrasonographic study of 82 dogs with liver diseases. Biliary sludge was detected in association with thickened gall bladder wall and/or with distended gall bladder.

Vijayanand*et al.* (2006) carried out ultrasonographicstudies on biliary obstruction in experimental dogs by four transducer positions as described by Finn-Bondner*et al.* (1998). Cystic duct was viewed by 24 hours post ligation in right or left

transverse oblique views. Common bile ductwas appreciated the by 7thday post ligation in right lateral transverse view whereas ventral transverse view was not useful in identifying both the ducts.

Vijayanand and Nagarajan (2007)in a dog suffering from cirrhosis,observed irregular hepatic margins, regenerative nodules, micro hepatica, increased parenchymal echogenicity,less distinct periportal parenchymal echoes due to fibrosis and ascites.

Chaudhary *et al.* (2008) diagnosed hepatic disorders like cyst, abscess, tumor, gallbladder distension, sludge in gallbladder, thickened gallbladder wall, choleliths and mucocele with changes in the echotexture of liver and gallbladder. Hepatic cysts were diagnosed by multiple focal anechoic changes in liver parenchyma.

Choudhary*et al.* (2008) conducted ultrasonographic examination of 200 dogs suspected for hepatopathy and confirmed hepatobilliary changes in 66 (33%) cases. It revealed diffuseand focallesions in 45 and 21 cases, respectively. They classified hepatic abnormalities sonographicallyas cirrhosis/fibrosis,hepatitis and gall bladder abnormalities as cholecystitis, choleliths, gall bladder sludge,gall bladder distention, and mucocele.

Mircean*et al*. (2008) studied the comparative efficacy of ultrasonographic and laboratory findings in the diagnosis of liver diseases in dogs and concluded that, the intensity of the biochemical changes were not always correlated with the severity of liver lesions that were found during ultrasonography and diffuse hepatic lesions such as cholangiohepatitis and cirrhosiswere associated with similar enzymatic abnormalities and opined that ultrasonography could be useful in differential diagnosis.

Brinkman-Ferguson and Biller (2009) advocated the use of ultrasonography through the right lateral intercostal space to scanlarge and deep chestdog breeds, and dogs having a large amount ofperitoneal effusions, gas orwith microhepatica. Visualization of portahepatiswas possible through this window and vessels were easily distinguished by their doppler characteristics. Evaluation of hepatic veins with spectral doppler from right 9ththrough 11thintercostal spaces helped in diagnosis of liver and heart diseases.

Salvekar*et al.* (2010) demonstrated that the ultrasonography was a valuable tool for diagnosis of liver diseases in dogs. Altered echotexture of liver parenchyma could be visualized to confirm various liver diseases like, hepatic abscess, cyst, fatty liver, cirrhosis and hepatic neoplasia. In the presence of ascitic fluid ultrasonography was found best to scan liver. They suggested that best results could be obtained by combination of both clinico-pathological and ultrasonographic evaluation for the correct diagnosis of the liver diseases.

Tomar*et al.* (2011) noted double walled gallbladder, suggestive of increased thickness in dogs with cholecystitis.

Saravanan*et al*. (2012) confirmed ascites in dogs with anechoic areas and hyperechoic cirrhotic liver by abdominal ultrasound. They correlated the ultrasonographic findings of hyperechoic /cirrhotic liver with high gradient ascites with >1.1g/dl of serum ascites albumin gradient.

Kumar *et al.* (2013) reported hyperechoic bright liver with normal size in dogs with chronic hepatitis. However, hyperechoic small liver with irregular margins in hepatic cirrhosis and diffuse hyperechoic liver parenchyma with less distinct portal vessels and peritoneal fluid in chronic hepatic cirrhosis or hepatitis.

Tantary*et al.* (2013) found hepatomegaly associated with diffuse hyperechoic liver parenchyma with less distinct portal vessels and accumulation of peritoneal fluid, in dogs affected with chronic hepatitis.

Kumar and Srikala (2014) reported ascites with right heart failure in a dog and found floating viscera in the anechoic peritoneal effusion with distended hepatic vasculature, presence of areas of hyper echoic to mixed echogenicity and rounding of liver lobes indicative of hepatic disease.

Tantary*et al.* (2014) reported that in acute hepatitis, reduced parenchymal echogenicity with enhanced visualization of portal vessels; hyper-echoic bright liver with normal size in chronic hepatitis and hyperechoic and small liver with irregular margins in hepatic cirrhosis. They considered ultrasonography as a more reliable method in the diagnosis of liver disease in dogs.

Elhiblu*et al.*(2015) studied on ultrasonography ofdogs with cirrhotic liver andrevealed generalized and diffuse hyperechoic hepatic parenchyma, rounded and irregular liver margins, micro hepatica and distended gall bladder and abundant free anechoic effusion in the abdominal cavity. They also concluded that, ultrasonography along with haemato-biochemical alterations might be used as a diagnostic tool in complementary to each other for evaluation of livercirrhosis in dogs.

2.6 ULTRASOUND GUIDED PERCUTANEOUS LIVER BIOPSY

Farrar *et al.* (1996) performed ultrasound guided percutaneous aspiration biopsy in a dog with hepatic abscesses. On cytological evaluation they observed acellular transudate with degenerated neutrophils and bacteria.

Ultrasound-guided fine needle aspiration was performed by Schwarz *et al.* (1998) in 10 of 13 dogs with hepatic lesions confirming abscessation. Ultrasound-guided percutaneous drainage of abscesses was performed in four dogs along with medical management.

Sonnenfield*et al.* (2001) performed ultrasound guided fine needle aspiration biopsy of hepatic mass in a 12 year old, neutered male dog presented with nonpainful cranial abdominal mass in which cytology revealedkaryolytic and degenerated neutrophils indicative of an infected or necrotic lesionsand these findingsare consistent with surgical findings showing torsion of quadrate lobe of the liver that had resulted in complete necrosis with evidence of diminished blood flow.

Weiss *et al*. (2001) performed fine needle aspiration cytology of liver in 51 cases. Overall sensitivity for diagnosis of inflammatory liver disease was 93%. For chronic active hepatitis (n = 13), the sensitivity and specificity of cytological diagnosis was 100% and 93%respectively. For suppurative hepatitis (n = 14), the sensitivity and specificity was 100% and 95%respectively. Sensitivityand specificity for lymphocytic hepatitis (n = 3), were 33% and 100% respectively.

Cuccovillo and Lamb (2002) performed fine needle aspiration of liver in animals having target like lesions under ultrasonography for the assessment of the diagnostic significance of the target lesions in relation to malignancy. On cytology malignant lesions of adenocarcinoma in was observed in two animals, lymphoma in

two animals and hepatocellular carcinoma in one animal. Among all only one animal had benign lesion, on cytology which was found to be lymphoid hyperplasia.

Under ultrasound guidanceBurton *et al.* (2005) obtained fine needle aspirates from the abdominal mass in a 14 year old, neutered male Bearded Collie. On the basis of histopathological examination of the aspirate,it was diagnosed as hepatocellular carcinoma in the mesentery which revealed several groups of epithelial cells with cytological features of hepatic origin.

Faverzani*et al.*(2006) opined that Ultrasound-guided liver biopsy as gold standard test for the diagnosis of hepatic masses, but he also mentioned that sometimes it may be difficult to perform a biopsy, especially when the site of lesion is deep in the liver or whenhaemangiosarcoma is suspected

In 31 animals (27 dogs and 4 cats) having single or multiple focal lesionsultrasonographically,Faverzani*et al.* (2006b) performed ultrasound-guided fine needle biopsy liver. He observed no complication during and after biopsy procedure and concluded that ultrasound-guided fine needle aspiration biopsy as a safe and harmless procedure even in conscious dogs.

Vijaykumar*et al*. (2012) opined that liver biopsy is often required for the definitive characterisation of the nature and severity of the liver disease. It can be alsobe used in differentiatiatingchronic from acute disorders, to stage neoplastic disease and to assess response to therapy.

CHAPTER III

MATERIALS AND METHODS

The present study was conducted on 46 clinical cases of dogs atDepartment of Veterinary Surgery and Radiology,College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, during the period from October-2019 to March-2020.

3.1 EXPERIMENTAL DESIGN

3.1.1 PATIENT SELECTION

The clinical cases of dogs presented to the Dept. of Veterinary Surgery and Radiology and to the Teaching Veterinary Clinical Complex, College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, having hepatic disorder and underwent abdominal ultrasound examination for evaluation of liver parenchyma were selected for the study.

Six apparently healthy dogs of either sex belonging to different breed and aged between 3 to 7 years that werepresented for routine health check-up were alsoevaluated as control group to serve as base line referral values for comparison of various parameters.

3.1.2 GROUPING

Apparently healthy dogs as control group were designated as Group A. On ultrasonography of liver, dogs with diffuse parenchymal hepatic abnormalities were categorized as Group B, dogs with focal parenchymal hepatic abnormalities were categorized as Group C and dogs with multifocal parenchymal hepatic abnormalities were categorized as Group D.

Table 1: Grouping of animals

Groups	Type of hepatic parenchymal abnormality on ultrasonography	No. of animals
Group A	Control group	6
Group B	Diffuse parenchymal hepatic abnormalities	31
Group C	Focal parenchymal hepatic abnormalities	6
Group D	Multifocal parenchymal hepatic abnormalities	9

3.2 PATIENT EVALUATION

All the dogs were evaluated individually on the basis of the following parameters.

3.2.1 SIGNALMENT

The data regarding age, breed,sex,body weightand type of nutrition were recorded for the study of occurrence.

3.2.2 HISTORY

Patient history related to duration of illness,observations of animal owner regarding associated clinical signs viz., anorexia, vomiting, melena, lethargy, and weight loss were collected for analysis. Any previous treatment and presence of other illness were also recorded.

3.2.3 VISUAL OBSERVATION

a. General condition of theanimalswere observed from a distance and recorded as normal, obese or weak body condition.

b. Abdominal distention was observed from a distance and recorded as normal, tucked up or distended.

3.2.4 PHYSICAL EXAMINATION

a. Conjunctival or oral mucus membrane was observed and recorded as normal, pale, congested or icterus.

b. Dehydration statuswas assessed by skin turgor test at dorsal neck region and recorded as normal or dehydrated.

c. Abdominal palpation was undertaken to assess abdominal ballottement or fluid thrill and abnormalities in palpable abdominal organs.

3.2.5 PHYSIOLOGICAL PARAMETERS

a. Rectal temperature was measured using digital thermometer (Fig. 1) and recorded as degree Fahrenheit (°F).
b. Heart rate was measured by auscultation of chest using a stethoscope and recorded as beats per minute (beats/min).
c. Respiratory rate was measured by observing number of times the chest expands and contracts and was recorded as breaths per minute (breaths/min).

3.2.6 HAEMATOLOGICAL STUDIES

Two ml of blood was collected (Fig. 2) in sterile EDTA vial and analysed in automated blood cell counter for evaluation of Total Erythrocyte Count (TEC) (millions/cmm), Packed Cell Volume (PCV) (%),Haemoglobin (Hb) (gm/dl) and Total leucocyte count (TLC) (thousands/cmm). A thin blood smear on glass slidewas prepared and stained with Giemsa stain for Differential Leucocyte Count (DLC) (%).

3.2.7 BIOCHEMICAL STUDIES

Threeml of blood was collected in sterile clot activator vial.Plasmaand serum were separated by centrifugation at 3000 rpm for 10 minutes and was used for biochemical analysis.Alanine aminotransferase (ALT) (IU/L), Alkaline Phosphatase (ALP) (IU/L),Aspartate Aminotransferase (AST) (IU/L), Gamma GlutamylTransferase (GGT) (IU/L), Total Plasma Protein (TPP)(g/dl) andplasma albumin (g/dl)levels were analysed as per the standards in the laboratory.

3.2.8 RADIOGRAPHY

All the canine patients were subjected to plain radiography of thoracic and abdominal region (Fig. 3) using the 700 mA X-Ray machine of Allengers 625. Animals were restrained physically. Right lateral view and ventro-dorsal view of thoracic and abdominal region weretaken for visualization of liver. The exposure factors were dictated by the size of the subjectwith a constant focal film distance (ffd) of 90 cm. The radiographs were processed by using computerized radiography (CR) system and observations were made as follows:

a. Visibility of hepatic margin: Clearly visible margin of hepatic density was recorded as well defined, partly visible margin of hepatic density was marked as obscured and in cases of ground glass appearance of abdominal content due to peritoneal fluid completely obstructing visibility of hepatic density was recorded as ascites.
b. Location of hepatic density was recorded as either within costal arch, extending slightly beyond costal arch or extending up to mid-abdomen.
c. Size of the liver was recorded as normal, small or enlarged based on location of hepatic density.
d. Shape of the hepatic density was recorded based on visibility of border of the lobes as pointed borders of lobes or rounded edges.

3.2.9 ULTRASONOGRAPHY

In the present study ultrasonography was carried out using Make-Wipro GE, Model- Logiq F8 Expert ultrasound machine (Fig. 5) in real time B mode and Doppler mode with a 8C Micro Convex multi-frequency probe of 6.0-10.0 MHz, 3C Macro Convex multi-frequency probe of 3.0-5.0 MHz, L6-12 Linear multi-frequency, probe of 6.0-12.0 MHz in awake animals. The selection of probe and frequency was dictated by size of the animal. For small to medium sized dogs 6-8 MHz, and for medium to large sized dogs 4-6 MHz frequency probes were used. Real time B-mode to evaluate liver parenchyma and Doppler ultrasonography was used for differentiating vascular structures and to determine extent of blood supply. Frozen images and video loops were recorded for evaluation.

Preparation and restraining of the animal

The ventral body area was prepared by clipping and shaving hair from right and left lateral thoraco-abdominal region (Fig. 4). The scanning area was cleaned with surgical spirit. The animals were restrained on dorsal, right or left recumbency as per the requirement on examination table with one assistant securing the rear legs while another one assisted to maintain the required positioning by restraint of the front legs and head. The ultrasound coupling gel was liberally applied over the scanning area to increase the skin transducer contact. The transducer of appropriate frequency was selected depending on the size and body weight of the patients.

Ultrasound scanning procedure

The transducer was placed on the sub-xiphoid regionwith firm but gentle pressure to image transverse section of the liver in the mid sagittal planeby angling sound beam cranio-dorsally. The image was oriented with right side of the animal to leftof the viewer on transverse scan and the cranial portion of the animal to left of the viewer on sagittal scans. The liver was then scanned from left to right by sweeping the transducer in an arc through the entire liver. Dorsal and ventralangling of the beamwas made in successive sweeps to image the entire liver. The beam orientation is changed to mid-transverse plane and the liver is scanned in an arc from ventral to dorsal. Transducer was angled to the left and right of midline in successive sweeps to make sure the entire liver is imaged. The liver is also imaged along the caudal aspect of the costal archby moving the transducer laterally to the left and right of midlinein both planes(Fig. 6). In patients that haveexcessive bowel gas,small liver or a deep chest,10^{th} to 12^{th} left and right intercostal spaceswere used for scanning the liver. Animals were examined in standing position whenever an air-filled stomach or intestine gas impairs visualization from ventral position.

During the ultrasound scanning, liver was evaluated for:

a. Comparative echogenicity with renal cortex as isoechoic, hyperechoic or hypoechoic.
b. Echogenicity within hepatic parenchyma was recorded as either uniformly isoechoic or hyper/ iso/ hypo echoic of the lesions compared to surrounding hepatic parenchyma.
c. Location of the liver was recorded as within costal arch or beyond costal arch.
d. Margins/borders of the liver were recorded as either smooth or irregular.
e. Presence of peritoneal effusion resulting in separation of individual liver lobes were recorded as either present or absent.
f. Vasculature in the hepatic parenchyma or of the lesions within the hepatic parenchyma was recorded using colour Doppler as either normal, increased or decreased.

3.3. ULTRASOUND GUIDED PERCUTANEOUS LIVER BIOPSY

Attempts were made for ultrasound guided biopsy of liver parenchymal lesions in cases of dogs wherein animal owner was willing for the procedure and the patient body condition was stable.

Anaesthesia

The dogs selected for ultrasound guided percutaneous biopsywere anaesthetisedby injecting Atropine (@ 0.04mg/kgbw.), Xylazine(@1mg/kgbw.) and Ketamine(@5mg/kgbw.) mixture intramuscularly. Ringer's Lactate was administered through intra venous catheterisation of cephalic or saphenous vein throughout the procedure until the animal recovered from anaesthesia. Maintenance dose of plain ketamine if required was administered through IV route.

Biopsy procedure

The site was aseptically prepared. Preliminary scan was performed to visualise the lesion at minimum possible depth for biopsy. An 18 gauge Tru-cut biopsy needle was inserted at an angle guided by the ultrasound(Fig. 7). When the needle enters the middle of the lesion, cutting action of the needle was employed to obtain hepatic tissue sample for biopsy and the needle was removed. The sample obtained within the specimen notch of the needle was transferred into 10% neutral buffered formalin. The biopsy sample was subjected to histopathological studies. The patient was observed ultrasonographically for evidence of haemorrhage at the site of biopsy. Animal was kept under observation until its recovery from anaesthesia.

3.4 STATISTICAL ANALYSIS

The quantitative data were presented as Mean ± Standard Error (SE) and qualitative data as frequency or percentage. All the required statistical calculations were done using SPSS (Statistical Package for Social Science) statistical analysis package version – 15. For hypothesis testing $P<0.05$ was considered and the level of significance was noted (Snedecor and Cochran, 1994).

Fig. 1: Clinical examination in a dog with hepatic disorder with concurrent skin allergy.

Fig. 2: Blood sample collection for hematobiochemical analysis.

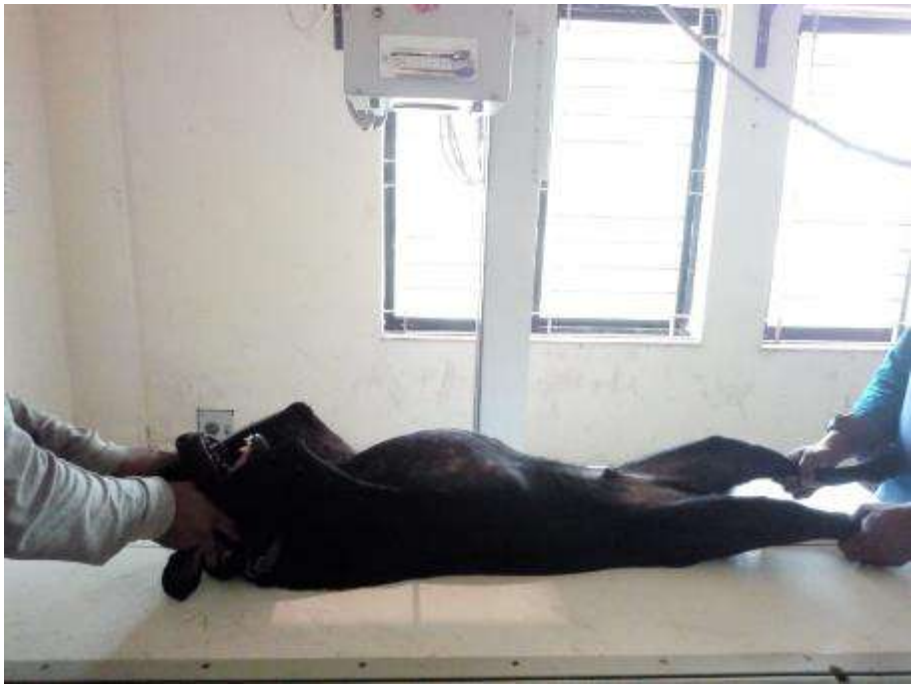


Fig. 3: Radiographic positioning for ventro-dorsal view of thoraco-abdominal region.

Fig. 4: Dog with distended abdomen prepared for ultrasound scanning.

Fig. 5: Logiq F8 Expert ultrasound machine.

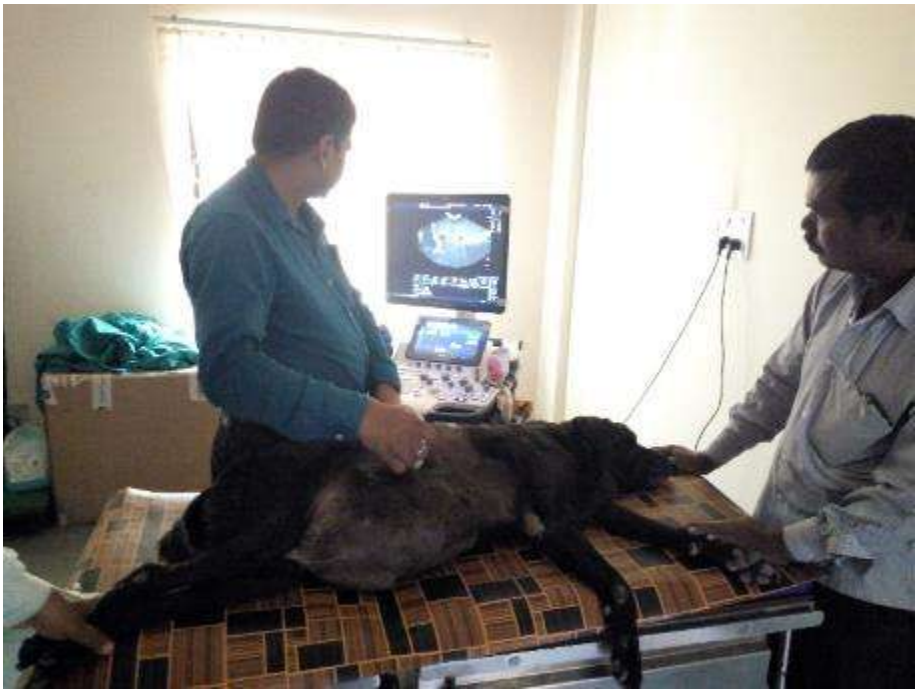


Fig. 6: Ultrasound scanning in a dog on lateral recumbency.

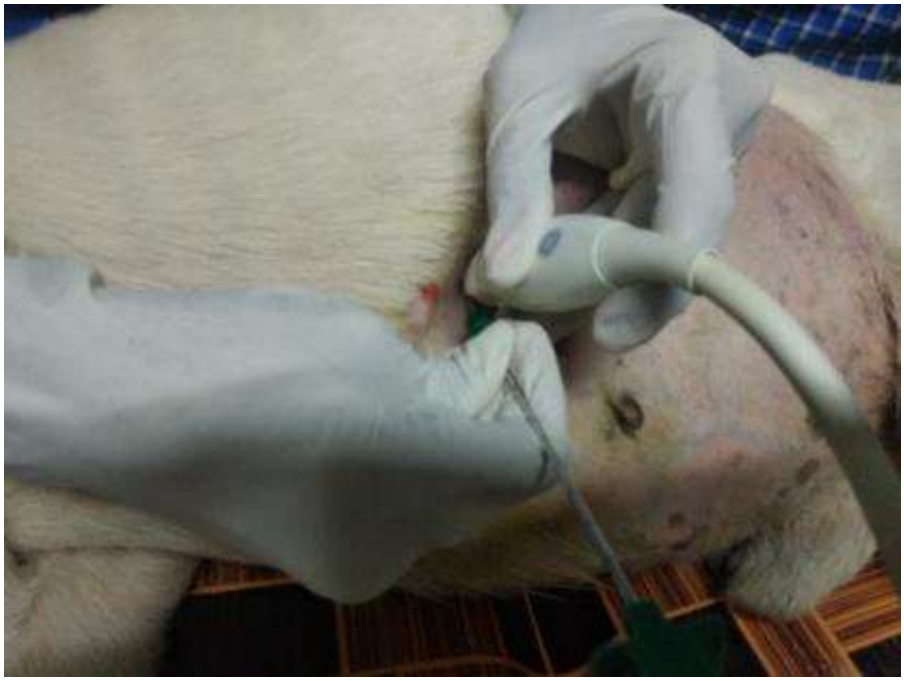


Fig. 7: Ultrasound guided percutaneous biopsy of hepatic lesions.

CHAPTER IV

RESULTS

The present clinical study of "Ultrasonographic evaluation of liver disorders in dogs" was conducted on 46 clinical cases presented with hepatic disorders to the Department of Veterinary Surgery and Radiology and to the Teaching Veterinary Clinical Complex, College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, during the period from October 2019 to March 2020, in an attempt to correlate the findings of ultrasonography with other clinical diagnostic tests like haematological, biochemical and radiographic techniques to establish diagnosis of hepatic disorders and to aid in therapeutic management.

4.1 PATIENT SELECTION

The clinical cases of dog presented with clinical signs associated with hepatic disorder and underwent abdominal ultrasound examination for evaluation of liver parenchyma were selected for the study. On ultrasonography of liver, dogs with diffuse parenchymal hepatic abnormalities were categorized as Group B, dogs with focal parenchymal hepatic abnormalities were categorized as Group C and dogs with multifocal parenchymal hepatic abnormalities were categorized as Group D. Six apparently healthy dogs were alsoevaluated as control Group to serve as base line referral values for comparison of various parameters and were as Group A.

4.2 PATIENT EVALUATION

4.2.1 OCCURENCE

The distribution of occurrence of hepatic disorder in 46 clinical cases of dogs was assessed from the data collected.

The occurrence of hepatic disorder was more common in middle aged dogs, aged between 6years to 10years (56%). Occurrence in young animals aged less than five years and old animals aged more than 10years was 22% each (Fig. 8).

Occurrence in male and female animals was equally distributed (Fig. 9).

Breed wise distribution of hepatic disorder in the present study was highest in Labrador retriever (41%), followed by Spitz (22%), German Shepherd (20%), Rottweiler (5%), Golden retriever (4%), non-descript (4%), Great Dane (2%) and Doberman (2%) (Fig.10).

Body weight of dogs with hepatic disorder ranged from 2kg to 45kg with Mean±SE of 23.91±1.59. Dogs weighed more than 20kg (70%) were affected more than dog with body weight of less than 20kg (30%) (Fig. 11).

4.2.2 HISTORY

Nutrition of the animals included homemade food and/or commercial dog food. 37% of dogs had a nutrition of both homemade and dog food. 35% of dog were on only commercially available dog food and only 17% of dogs were maintained on homemade food.

Duration of illness, before presentation of dogs for the treatment, ranged from one day to 25days with a mean duration of 5.6days. Out of 46 cases of dogs, 25dogs were treated symptomatically before presentation. Six dogs (13.04%) had other illness like skin allergy, mammary tumour, cutaneous tumour and skin lesions.

Percentage of animals having clinical symptoms of anorexia, vomiting, melena, lethargy and weight loss was 74.19%, 54.83%, 22.58%, 90.32% and 32.25% respectively in animals of Group B having defuse hepatic parenchymal disorder which was highest when compared to animals of Group C and Group D. Percentage of animals having clinical symptom of melena, lethargy and weight loss was more in animals Group D, animals with multifocal hepatic parenchymal lesions (22.22%, 77.77% and 22.22% respectively) when compared to Group C, animals with focal hepatic parenchymal lesions (16.66%, 66.66% and 16.66% respectively) (Fig. 12).Basic data and history of animals of various Groups are represented from Table 2-5.

4.2.3 VISUAL OBSERVATION

a. General condition of the all the dogs of control Group A were normal. However, 54.83% of dogs of Group B, 33.33% of dogs of Group C and

Group D were weak in body condition. Further, except two dogs in Group B, all the other dogs were in normal body condition.

b. Abdominal distention was visibly normal in all the dogs of control Group A and Group C with focal hepatic parenchymal abnormalities. Percentage of dogs having distended abdomenin Group B was 51.61% and in Group D was 22.22%. Tucked up abdomen was seen in three dogs of Group B and two dogs of Group D. Rest all the dogs had normal abdominal appearance.

4.2.4 PHYSICAL EXAMINATION

a. Conjunctival or oral mucus membrane was normal light pink in all the dogs of control Group A. In Group B dogs, mucous membrane was pale in 38.71% and icterus in 32.25%. In Group C dogs, mucous membrane was pale in 16.67% and icterus (Fig. 20) in 16.67%. In Group D dogs, mucous membrane was pale in 33.33% and icterus in 11.11%. In rest of the dogs, mucous membrane was normal in appearance.

b. Skin turgor test for assessment of hydration status showed good hydrated condition in dogs control Group A, dehydration in 61.29% of dogs in Group B, dehydration in 33.33% of dogs in Group C and dehydration in 55.55% of dogs of Group D.

c. Abdominal palpation of dogs of control Group A showed absence of abdominal ballottement and there was no abnormal palpable masses. In Group B dogs, abdominal ballottement was present in 41.93% of dogs indicating ascites and there was palpable abdominal masses suggestive of enlarged liver in 29.03% of dogs. In Group C dogs, there was no signs of abdominal ballottement and palpable abnormal abdominal masses. In Group D dogs, 22.22% of dogs had abdominal ballottement indicative of ascites and there was no palpable abnormal abdominal masses, except pain on palpation in two dogs.

Visual and physical examination findings of various Groups are represented from Table 6-9 and Fig. 15-21.

4.2.5 PHYSIOLOGICAL PARAMETERS

a. The Mean±SE value of rectal temperature (°F) in Group A, Group B, Group C and Group D dogs were 101.62±0.23, 102.49±0.19, 102.47±0.34 and 102.37±0.33 respectively. However, the mean values of different Groups with hepatic disorder was not statistically significant from control Group and the mean values were within normal physiological range.
b. The Mean±SE value of heart rate (beats/min) in Group A, Group B, Group C and Group D dogs were 96.67±1.98, 95.68±1.13, 94.83±2.77 and 103.11±3.97 respectively. The mean values of Group D differedstatistically significantly from the Group A. However, the mean values of the all the Groups were within normal physiological range.
c. The Mean±SE value of respiratory rate (breaths/min) in Group A, Group B, Group C and Group D dogs were 17.33±0.84, 16.97±0.17, 16.67±0.33 and 17.44±0.63 respectively. However, the mean values of different Groups with hepatic disorder was not statistically significant from control Group and the mean values were within normal physiological range.

Mean±SE values of physiological parameters in various Groups are represented in Table 10.

4.2.6 HAEMATOLOGICAL STUDIES

a. The Mean±SE value of total erythrocyte count (millions/cmm) in Group A, Group B, Group C and Group D dogs was 5.26±0.12, 4.57±0.22, 5.94±0.22 and 5.98±0.23 respectively. The mean values of Group B was lower than other Groups and differed statistically significantly from control Group. However, the mean values of the all the Groups were within normal physiological range.
b. The Mean±SE value of packed cell volume (%) in Group A, Group B, Group C and Group D dogs was 41.17±0.70, 34.93±1.24, 43.92±1.64 and 44.22±1.72 respectively. The mean values of Group B was lower than other Groups and differed statistically significantly from control Group. However, the mean values of the all the Groups were within normal physiological range.

c. The Mean±SE value of haemoglobin (gm/dl) in Group A, Group B, Group C and Group D dogs was 12.45±0.11, 8.86±0.40, 11.73±0.58 and 11.99±0.68 respectively. The mean values of Group B was lower than other Groups and differed statistically significantly from control Group. However, the mean values of the all the Groups were within normal physiological range.

d. The Mean±SE value of total leucocyte count (thousands/cmm) in Group A, Group B, Group C and Group D dogs was 12.27±0.14, 18.60±1.11, 14.16±0.62 and 17.28±0.79 respectively. The mean values of all Groups with hepatic disorders were higher than control Group and the mean values of Group B and Group D was statistically significant from the control Group.

e. Differential leukocyte count

   The Mean±SE value of neutrophils (%) in Group A, Group B, Group C and Group D dogs was 76.00±0.67, 82.26±1.12, 76.33±1.93 and 80.22±1.08 respectively. The Mean±SE value of lymphocytes (%) in Group A, Group B, Group C and Group D dogs was 18±0.72, 14.29±1.03, 17.67±2.11 and 14.44±0.88 respectively. Mean±SE value of monocytes (%) in Group A, Group B, Group C and Group D dogs was 2.67±0.17, 1.55±0.19, 5.00±0.37 and 1.67±0.33 respectively. Mean±SE value of eosinophils (%) in Group A, Group B, Group C and Group D dogs was 4.67±0.17, 1.74±0.19, 1.17±0.31 and 1.89±0.39 respectively. Mean±SE value of basophils (%) in Group A, Group B, Group C and Group D dogs was0.00±0.00, 0.13±0.06, 0.00±0.00 and 0.78±0.28 respectively. The mean values of neutrophils with hepatic disorders were higher than control Group and the mean values of lymphocytes in Groups with hepatic disorder were lower than the control Group. The mean values neutrophils and lymphocytes of Group B and Group D animals were statistically significant from control Group.

Mean±SE values of haematological parameters in various Groups are represented in Table 11.

4.2.7 BIOCHEMICAL STUDIES

a. The Mean±SE value of Alanine aminotransferase (IU/L) in Group A, Group B, Group C and Group D dogs was 37.15±0.35, 137.66±13.64, 83.67±15.84 and 120.78±16.09 respectively. The mean values of Groups of hepatic disorder were higher than the control Group and were statistically significant.
b. The Mean±SE value of Aspartate Aminotransferase (IU/L) in Group A, Group B, Group C and Group D dogs was 13.71±0.11, 79.26±18.14, 19.50±3.58 and 37.67±15.07 respectively. The mean values of Groups of hepatic disorder were higher than the control Group and the mean values of Group B and Group D were statistically significant from control Group.
c. The Mean±SE value of Alkaline Phosphatase (IU/L) in Group A, Group B, Group C and Group D dogs was 72.67±5.46, 265.38±24.84, 93.50±14.87 and175.89±15.07 respectively. The mean values of Groups of hepatic disorder were higher than the control Group and the mean values of Group B and Group D were statistically significant from control Group.
d. The Mean±SE value of Gamma GlutamylTransferase (IU/L) in Group A, Group B, Group C and Group D dogs was 3.46±0.59, 26.84±2.64, 15.00±1.84 and 28.67±3.44 respectively. The mean values of Groups of hepatic disorder were higher than the control Group and were statistically significant.
e. The Mean±SE value of Total Plasma Protein (g/dl) in Group A, Group B, Group C and Group D dogs was 7.65±0.22, 6.52±0.27, 6.58±0.47 and 6.62±0.48 respectively. The mean values of Groups of hepatic disorder were lower than the control Group, however, there was no statistically significant difference from the control Group.
f. The Mean±SE value of plasma albumin (g/dl) in Group A, Group B, Group C and Group D dogs was 2.90±0.07, 1.76±0.09, 2.35±0.08 and 1.82±0.10 respectively. The mean values of Groups of hepatic disorder were lower than the control Group and the mean values of Group B and Group D were statistically significant from control Group.

Mean±SE values of biochemical parameters in various Groups are represented in Table 12.

4.2.8 RADIOGRAPHY

Positioning of animals for radiography by physical restraint was found to be sufficient for radiography. Computerized radiography provided contrast adjustment and better visibility of abdominal structures.

a. Visibility of hepatic margin: Hepatic margin was clearly visible and well defined in all the dogs of Group A. In Group B animals, hepatic margin was well defined in 45.16% dogs, obscured in 16.13% dogs and hepatic margin was not visible due to large quantity of peritoneal fluid in 38.71% dogs. In Group C animals, all the dogs had well defined hepatic margin except one dog wherein it was obscured (16.67%). In Group C, 22.22% dogs had poor visibility of hepatic margin due to peritoneal fluid and rest 77.77% dogs had well defined hepatic margin.
b. Location of hepatic density on radiographs was assessed in radiographs wherein hepatic margin was visible. In Group A, the liver density was within costal arch or slightly beyond costal arch. The hepatic density extended up to mid-abdomen in 41.93% dogs of Group B, in 50% dogs of Group C and in 22.22% dogs of Group D.
c. Size of liver was assessed in radiographs wherein hepatic margin was visible. In Group A, liver size appeared normal in all dogs. In Group B, enlargement of liver was noticed in 41.93% dogs and small sized liver was noticed in 6.45% dogs. In Group C, hepatic enlargement was noticed in 50% dogs. In Group D, 22.22% showed hepatic enlargement and 11.11% dogs had small liver size.
d. The shape of the liver was assessed in radiographs where in hepatic margin was visible. In Group A, all the dogs had pointed borders of lobes. In Group B, 41.93% dogs had rounded edges and 3.22% dogs had irregular margin of hepatic margins. In Group C, 50% dogs had rounded edges of hepatic margin. In Group D, 33.33% dogs had rounded borders, 33.33% had irregular border and in one dog had large round left lateral lobe.

Survey radiographic appearance of liver in various Groups of animals are represented from Table 13 – 16.

4.2.9 ULTRASONOGRAPHY

Positioning of the dog by physical restraint was found to be adequate for preparation and ultrasound scanning. Shaving of the ventral abdomen region and lateral caudal costal region provided adequate window for scanning of hepatic parenchyma. The results of parameters studied is shown in table no.17-20.

a. Comparative echogenicity with renal cortex was either isoechoic or hyper echoic in dogs of control Group A. Hyperechoic liver parenchyma was seen in 58.06% dogs of Group B, 66.66% dogs of Group C and 33.33% dogs of Group D. Hypoechoic liver parenchyma was seen in 22.58% dogs of Group B, 33.33% dogs of Group C and 66.66% dogs of Group D. Isoechoic liver parenchyma was seen only inGroup B of test Groups and it was 19.35% (Fig. 13).
b. Echogenicity within hepatic parenchyma was uniformly isoechoicin dogs of control Group A. In Group B, uniformly isoechoic was noticed in 83.87% dogs and mixed echogenicity was noticed in 16.13% dogs. Group C, focal lesions were noticed in liver parenchyma wherein the lesions were hyperechoic 50% dogs, hypoechoic in 33.33% dogs and mixed echogenicity in 16.67% dogs when compared to surrounding liver parenchyma. In Group D, the multifocal lesions were noticed in liver parenchyma wherein the lesions werehyperechoic in 33.33% dogs, hypoechoic in 11.11% dogs and mixed echogenicity in 55.56% dogs when compared to adjacent liver parenchyma (Fig. 14).
c. Location of the liver was within costal arch in all the animals of Group A and Group C. 51.61% dogs of Group B and 66.66% dogs of Group D had location of liver extending beyond costal arch.
d. Margins/borders of the liver was smooth and pointed in all the dogs of Group A and Group C. 41.93% dogs of Group B and 66.66% dogs of Group D had irregular margins (Fig. 22).
e. Presence of peritoneal effusion was assessed by observing separation of individual liver lobes with anechoic fluid (Fig.38). Peritoneal effusion was absent in all the dogs of control Group. Peritoneal effusion was present in

54.83% dogs of Group B, 16.67% of Group C and 22.22% dogs of Group D.

f. Vasculature assessment of liver parenchyma was made by colour Doppler (Fig.39). Vasculature of control Group was considered normal and the appearance of vasculature in dogs with hepatic disorder was compared with control Group. In Group B, 41.94% dogs had decreased vasculature and 38.71% had increased vasculature in liver parenchyma. In Group C, the vasculature within focal lesions were decreased in 33.33% dogs and increased in 33.33% dogs and absent in 16.67% dogs. In Group D, 44.44% dogs had decreased vasculature in the multifocal lesions, 44.44% dogs had increased vasculature in the multifocal lesions and 11.11% dogs had absent to decreased vasculature.

Based on ultrasonographic findings, the Group B dogs were tentatively diagnosed as Hepatitis (22.58%), Chronic Hepatitis (22.58%), Cirrhosis (29.03%), Steroid / Vacuolarhepatopathy (9.67%). The dogs of Group C and Group D and also six dogs (19.35%) of Group B which had diffuse mixed echogenicityrequired liver biopsy for diagnosis since many conditions appear similar ultrasonographically. Ultrasonography finding of hepatic parenchyma of various Groups of animals are represented from Fig. 22-39.

4.3 ULTRASOUND GUIDED PERCUTANEOUS LIVER BIOPSY

Ultrasound guided percutaneous liver biopsy (Fig. 7) was undertaken in two dogs viz., animal number C-3 and D-8. The anaesthesia protocol used provided good depth of anaesthesia and found adequate for the procedure. Trucut biopsy needle was easy to use and tissue penetration was clearly visible on ultrasonography. The sample collected from Trucut needle was found adequate for histopathological studies. There was no haemorrhage at the site of biopsy and both the dogs recovered uneventfully from anaesthesia and biopsy procedure.

The ultrasound guided percutaneous liver biopsy findings are as shown in table no. 21.

Table 2: Basic data and history of animals of Group A: Control group

Animal no.	Age	Breed	Sex	Body weight (kg)	Nutrition	Duration of illness (Days)	Previous treatment	Presence of other illness
A-1	4yr	Labrador Retriever	M	31	Dog food	Nil	Nil	Nil
A-2	6yr	Labrador Retriever	F	32	Dog food	Nil	Nil	Nil
A-3	3yr	German Shepherd	F	28	Dog food	Nil	Nil	Nil
A-4	3yr	Labrador Retriever	M	27	Homemade +Dog food	Nil	Nil	Nil
A-5	5yr	German Shepherd	M	34	Dog food	Nil	Nil	Nil
A-6	7yr	Spitz	F	7	Homemade +Dog food	Nil	Nil	Nil

Table 3: Basic data and history of animals of Group B: Diffuse hepatic parenchymal disorder

Animal no.	Age	Breed	Sex	Body weight (kg)	Nutrition	Duration of illness (Days)	Previous treatment	Presence of other illness
B-1	7yr	Spitz	M	8	Homemade +Dog food	7	Symptomatic	Nil
B-2	12yr	Labrador Retriever	F	35	Dog food	4	Symptomatic	Nil
B-3	4m	German Shepherd	M	7	Dog food	4	Symptomatic	Nil
B-4	4yr	Spitz	F	7	Dog food	14	Symptomatic	Nil
B-5	16m	Labrador Retriever	M	14	Homemade +Dog food	2	Nil	Nil
B-6	9yr	Golden Retriever	M	29	Dog food	1	Nil	Nil
B-7	6yr	German Shepherd	F	27	Homemade +Dog food	7	Symptomatic	Nil
B-8	4.5yr	Rottweiler	M	31	Dog food	3	Nil	Nil
B-9	4yr	Labrador Retriever	F	28	Dog food	2	Nil	Nil
B-10	6yr	Spitz	M	11	Homemade food	5	Symptomatic	Nil
B-11	5yr	Labrador Retriever	M	25	Homemade food	7	Symptomatic	Nil
B-12	7yr	Spitz	F	10	Homemade food	5	Symptomatic	Nil

B-13	14yr	Labrador Retriever	M	35	Homemade + Dog food	2	Nil	Nil
B-14	7yr	Labrador Retriever	M	31	Dog food	5	Symptomatic	Nil
B-15	8yr	Labrador Retriever	F	27	Homemade + Dog food	7	Symptomatic	Nil
B-16	7yr	Labrador Retriever	F	35	Dog food	3	Nil	Nil
B-17	7yr	Labrador Retriever	F	29	Homemade + Dog food	2	Nil	Nil
B-18	10yr	German Shepherd	M	24	Dog food	25	Symptomatic	Skin allergy
B-19	4yr	Spitz	M	7	Homemade food	2	Nil	Nil
B-20	10yr	Non-descript	F	19	Homemade food	10	Symptomatic	Nil
B-21	2m	Labrador Retriever	M	2	Dog food	5	Symptomatic	Nil
B-22	6yr	Spitz	F	9	Homemade food	2	Nil	Nil
B-23	7yr	Labrador Retriever	M	31	Dog food	5	Symptomatic	Nil
B-24	3yr	Labrador Retriever	F	26	Homemade + Dog food	3	Nil	Nil
B-25	4yr	Golden Retriever	F	29	Dog food	2	Nil	Nil
B-26	10yr	Labrador Retriever	F	36	Dog food	1	Nil	Mammary tumor
B-27	7yr	Labrador Retriever	F	34	Homemade + Dog food	7	Symptomatic	Nil
B-28	11yr	Labrador Retriever	M	27	Dog food	20	Symptomatic	Nil
B-29	7yr	Labrador Retriever	F	28	Homemade + Dog food	4	Nil	Nil
B-30	10.5yr	German Shepherd	M	34	Homemade + Dog food	1	Nil	Nil
B-31	10yr	Non-descript	M	21	Homemade food	10	Symptomatic	Nil

Table 4: Basic data and history of animals of Group C: Focal hepatic parenchymal disorder

Animal no.	Age	Breed	Sex	Body weight (kg)	Nutrition	Duration of illness (Days)	Previous treatment	Presence of other illness
C-1	12yr	German Shepherd	M	29	Homemade + Dog food	10	Symptomatic	Nil
C-2	11yr	Spitz	M	8	Homemade food	7	Symptomatic	Nil
C-3	7yr	Labrador Retriever	F	36	Homemade + Dog food	4	Symptomatic	Nil
C-4	10yr	German Shepherd	M	31	Dog food	2	Nil	Nil
C-5	6yr	Great Dane	F	45	Dog food	5	Nil	Nil
C-6	6yr	German Shepherd	F	32	Homemade + Dog food	4	Symptomatic	Nil

Table 5: Basic data and history of animals of Group D: Multifocal hepatic parenchymal disorder

Animal no.	Age	Breed	Sex	Body weight (kg)	Nutrition	Duration of illness (Days)	Previous treatment	Presence of other illness
D-1	12yr	Spitz	M	9	Homemade + Dog food	2	Nil	Nil
D-2	14yr	Spitz	F	7	Dog food	3	Nil	Mammary tumor
D-3	11yr	Spitz	F	11	Homemade food	1	Nil	Cutaneous tumor
D-4	9yr	Labrador Retriever	M	27	Dog food	12	Symptomatic	Nil
D-5	9yr	German Shepherd	M	32	Homemade + Dog food	3	Nil	Nil
D-6	12yr	Labrador Retriever	F	33	Homemade + Dog food	7	Symptomatic	Mammary tumor
D-7	11yr	Doberman	F	24	Dog food	10	Symptomatic	Nil
D-8	8yr	German Shepherd	F	27	Dog food	4	Nil	Skin lesions
D-9	7yr	Rottweiler	M	33	Homemade + Dog food	5	Symptomatic	Nil

Table 6: Visual and physical examination findings of animals of Group A:Control group

Animal no.	Visual examination		Physical examination		
	General condition	**Abdominal distension**	**Conjunctival/oral mucous membrane**	**Skin turgor test**	**Abdominal palpation**
A-1	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
A-2	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
A-3	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
A-4	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
A-5	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
A-6	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses

Table 7: Visual and physical examination findings of animals of Group B: Diffuse hepatic parenchymal disorder

Animal no.	Visual examination		Physical examination		
	General condition	Abdominal distension	Conjunctival/oral mucous membrane	Skin turgor test	Abdominal palpation
B-1	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-2	Normal	Normal	Normal	Dehydrated	Abdominal ballottement absent and palpable enlarged liver
B-3	Normal	Tucked up abdomen	Normal	Dehydrated	Abdominal ballottement absent, palpable enlarged liver and pain on palpation.
B-4	Weak	Distended	Pale	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-5	Weak	Tucked up abdomen	Pale	Dehydrated	Abdominal ballottement absent and palpable enlarged liver
B-6	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
B-7	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-8	Normal	Normal	Normal	Dehydrated	Abdominal ballottement absent and no abnormal palpable masses
B-9	Weak	Normal	Icteric	Normal	Abdominal ballottement absent and no abnormal palpable masses
B-10	Normal	Normal	Pale	Normal	Abdominal ballottement absent and no abnormal palpable masses
B-11	Weak	Distended	Pale	Normal	Abdominal ballottement present and no abnormal palpable masses
B-12	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-13	Normal	Normal	Normal	Normal	Abdominal ballottement absent, no abnormal palpable masses and pain on palpation

B-14	Normal	Normal	Normal	Normal	Abdominal ballottement absent, palpable enlarged liver and pain on palpation
B-15	Weak	Normal	Pale	Dehydrated	Abdominal ballottement absent and palpable enlarged liver
B-16	Normal	Normal	Normal	Dehydrated	Abdominal ballottement absent, no abnormal palpable masses
B-17	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-18	Weak	Tucked up abdomen	Icteric	Dehydrated	Abdominal ballottement absent, abnormal palpable mass with pain on palpation.
B-19	Weak	Distended	Pale	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-20	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-21	Normal	Distended	Pale	Normal	Abdominal ballottement present and no abnormal palpable masses
B-22	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-23	Weak	Distended	Pale	Normal	Abdominal ballottement present and no abnormal palpable masses
B-24	Normal	Normal	Normal	Normal	Abdominal ballottement absent and palpable enlarged liver
B-25	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-26	Obese	Distended	Pale	Dehydrated	Abdominal ballottement absent and palpable enlarged liver
B-27	Normal	Distended	Pale	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-28	Weak	Normal	Pale	Normal	Presence of mild fluid thrill on abdominal palpation
B-29	Obese	Distended	Pale	Normal	Abdominal ballottement absent, palpable enlarged liver and pain on palpation
B-30	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
B-31	Normal	Normal	Normal	Normal	Abdominal ballottement absent, palpable enlarged liver and pain on palpation

Table 8: Visual and physical examination findings of animals of Group C: Focal hepatic parenchymal disorder

Animal no.	Visual examination		Physical examination		
	General condition	**Abdominal distension**	**Conjunctival/oral mucous membrane**	**Skin turgor test**	**Abdominal palpation**
C-1	Weak	Normal	Icteric	Dehydrated	Abdominal ballottement absent, no abnormal palpable masses and pain on abdominal palpation.
C-2	Weak	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
C-3	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
C-4	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
C-5	Normal	Normal	Pale	Normal	Abdominal ballottement absent and no abnormal palpable masses
C-6	Normal	Normal	Normal	Dehydrated	Abdominal ballottement absent and no abnormal palpable masses

Table 9: Visual and physical examination findings of animals of Group D: Multifocal hepatic parenchymal disorder

Animal no.	Visual examination		Physical examination		
	General condition	Abdominal distension	Conjunctival/oral mucous membrane	Skin turgor test	Abdominal palpation
D-1	Normal	Normal	Normal	Dehydrated	Abdominal ballottement absent and no abnormal palpable masses
D-2	Normal	Tucked up abdomen	Normal	Dehydrated	Abdominal ballottement absent and pain on palpation
D-3	Normal	Normal	Pale	Normal	Abdominal ballottement absent and no abnormal palpable masses
D-4	Weak	Distended	Icteric	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
D-5	Normal	Normal	Pale	Dehydrated	Abdominal ballottement absent and no abnormal palpable masses
D-6	Weak	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
D-7	Weak	Distended	Pale	Dehydrated	Abdominal ballottement present and no abnormal palpable masses
D-8	Normal	Normal	Normal	Normal	Abdominal ballottement absent and no abnormal palpable masses
D-9	Normal	Tucked up abdomen	Normal	Normal	Abdominal ballottement absent and pain on palpation

Table 10: Mean±SE values of physiological parameters in various groups

Groups	Rectal temperature (°F)	Heart rate (beats/min)	Respiratory rate (breaths/min)
Group A	101.62±0.23[a]	96.67±1.98[a]	17.33±0.84[a]
Group B	102.49±0.19[a]	95.68±1.13[a]	16.97±0.17[a]
Group C	102.47±0.34[a]	94.83±2.77[a]	16.67±0.33[a]
Group D	102.37±0.33[a]	103.11±3.97[b]	17.44±0.63[a]

Values bearing common superscript do not differ significantly with control group A (P≤0.05)

Table 11:Mean±SE values of haematological parameters in various groups

Groups	TEC (millions/ cmm)	PCV (%)	Hb (gm/dl)	TLC (thousands/ cmm)	DLC (%)				
					Neutrophils	Lymphocytes	Monocytes	Eosinophils	Basophils
Group A	5.26±0.12[a]	41.17±0.70[a]	12.45±0.1[a]	12.27±0.14[a]	76.00±0.67[a]	18±0.72[a]	2.67±0.17[a]	4.67±0.17[a]	0.00±0.00[a]
Group B	4.57±0.22[b]	34.93±1.24[b]	8.86±0.40[b]	18.60±1.11[b]	82.26±1.12[b]	14.29±1.03[b]	1.55±0.19[a]	1.74±0.19[a]	0.13±0.06[a]
Group C	5.94±0.22[a]	43.92±1.64[a]	11.73±0.58[a]	14.16±0.62[a]	76.33±1.93[a]	17.67±2.11[a]	5.00±0.37[a]	1.17±0.31[a]	0.00±0.00[a]
Group D	5.98±0.23[a]	44.22±1.72[a]	11.99±0.68[a]	17.28±0.79[b]	80.22±1.08[b]	14.44±0.88[b]	1.67±0.33[a]	1.89±0.39[a]	0.78±0.28[a]

Values bearing common superscript do not differ significantly with control group A(P≤0.05)

Table 12:Mean±SE values of serum biochemical parameters in various groups

Groups	ALT (IU/L)	AST (IU/L)	ALP (IU/L)	GGT (IU/L)	TPP (g/dl)	plasma albumin (g/dl)
Group A	37.15±0.35[a]	13.71±0.11[a]	72.67±5.46[a]	3.46±0.59[a]	7.65±0.22[a]	2.90±0.07[a]
Group B	137.66±13.64[b]	79.26±18.14[b]	265.38±24.84[b]	26.84±2.64[b]	6.52±0.27[a]	1.76±0.09[b]
Group C	83.67±15.84[b]	19.50±3.58[a]	93.50±14.87[a]	15.00±1.84[b]	6.58±0.47[a]	2.35±0.08[a]
Group D	120.78±16.09[b]	37.67±15.07[b]	175.89±15.07[b]	28.67±3.44[b]	6.62±0.48[a]	1.82±0.10[b]

Values bearing common superscript do not differ significantly with control group A($P \leq 0.05$)

Table 13: Survey radiographic appearance of liver in animals of Group A: Control group

Animal no.	Visibility of hepatic margin	Location	Size	Shape
A-1	Well defined	Extending just slightly beyond costal arch	Normal	Pointed borders of lobes
A-2	Well defined	Extending just slightly beyond costal arch	Normal	Pointed borders of lobes
A-3	Well defined	Within Costal arch	Normal	Pointed borders of lobes
A-4	Well defined	Extending just slightly beyond costal arch	Normal	Pointed borders of lobes
A-5	Well defined	Within Costal arch	Normal	Pointed borders of lobes
A-6	Well defined	Extending just slightly beyond costal arch	Normal	Pointed borders of lobes

Table 14: Survey radiographic appearance of liver in animals of Group B: Diffuse hepatic parenchymal disorder

Animal no.	Visibility of hepatic margin/ Ascites	Location	Size	Shape
1	Ascites	-	-	-
2	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
3	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
4	Obscured	Extending up to mid-abdomen	Enlarged	Rounded edges
5	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
6	Well defined	Extending just slightly beyond costal arch	Normal	Pointed borders of lobes
7	Ascites	-	-	-
8	Obscured	Extending up to mid-abdomen	Enlarged	Rounded edges
9	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
10	Well defined	Within costal arch	Small	
11	Ascites	-	-	-
12	Ascites	-	-	-
13	Well defined	Within costal arch	Small	Irregular borders
14	Obscured	Extending up to mid-abdomen	Enlarged	Rounded edges
15	Well defined	Within costal arch	Normal	Pointed borders of lobes

16	Well defined	Within costal arch	Normal	Pointed borders of lobes
17	Ascites	-	-	-
18	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
19	Ascites	-	-	-
20	Ascites	-	-	-
21	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
22	Ascites	-	-	-
23	Ascites	-	-	-
24	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
25	Ascites	-	-	-
26	Obscured	Extending up to mid-abdomen	Enlarged	Rounded edges
27	Ascites	-	-	-
28	Obscured	Within costal arch	Normal	Round edges
29	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
30	Ascites	-	-	-
31	Well defined	Extending up to mid-abdomen	Enlarged	Pointed borders of lobes

Table 15: Survey radiographic appearance of liver in animals of Group C: Focal hepatic parenchymal disorder

Animal no.	Visibility of hepatic margin	Location	Size	Shape
1	Well defined	Extending up to mid-abdomen	Enlarged	Pointed borders of lobes
2	Obscured	Within costal arch	Normal	Round edges
3	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
4	Well defined	Extending up to mid-abdomen	Enlarged	Rounded edges
5	Well defined	Within costal arch	Normal	Pointed borders of lobes
6	Well defined	Within costal arch	Normal	Pointed borders of lobes

Table 16: Survey radiographic appearance of liver in animals of Group D: Multifocal hepatic parenchymal disorder

Animal no.	Visibility of hepatic margin	Location	Size	Shape
1	Well defined	Extending just slightly beyond costal arch	Normal	Irregular border
2	Well defined	Extending just slightly beyond costal arch	Normal	Rounded and irregular border
3	Well defined	Within costal arch	Small	Rounded border
4	Ascites	-	-	-
5	Well defined	Extending just slightly beyond costal arch	Normal	Irregular border
6	Well defined	Extending just slightly beyond costal arch	Normal	Irregular border
7	Ascites	-	-	-
8	Well defined	Extending up to mid-abdomen	Enlarged	Large round left lateral lobe
9	Well defined	Extending up to mid-abdomen	Enlarged	Rounded border

Table 17: Ultrasonography finding of hepatic parenchyma in animals of Group A: Control group

Animal no.	Comparative echogenicity with renal cortex	Echogenicity within hepatic parenchyma	Location	Borders	Peritoneal effusion	Vasculature on Color Doppler
A-1	Isoechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal
A-2	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal
A-3	Isoechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal
A-4	Isoechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal
A-5	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal
A-6	Isoechoic	Uniformly isoechoic	Within costal arch	Smooth and pointed	Absent	Normal

Table 18: Ultrasonography finding of hepatic parenchyma in animals of Group B: Diffuse hepatic parenchymal disorder

Animal no.	Comparative echogenicity with renal cortex	Echogenicity within hepatic parenchyma	Location	Borders	Peritoneal effusion	Vasculature on Color Doppler
1	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth	Present	Decreased
2	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
3	Isoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
4	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Present	Increased
5	Hyperechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
6	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Absent	Decreased
7	Hyperechoic	Mixed echogenesity	Within costal arch	Smooth	Present	Normal
8	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Present	Increased
9	Hyperechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Normal
10	Isoechoic	Uniformly isoechoic	Within costal arch	Irregular	Absent	Decreased
11	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
12	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth	Present	Normal
13	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
14	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Present	Increased

15	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth	Absent	Decreased
16	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Absent	Decreased
17	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
18	Isoechoic	Mixed echogenesity	Extending beyond costal arch	Irregular	Absent	Increased
19	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
20	Hyperechoic	Mixed echogenesity	Extending beyond costal arch	Smooth	Present	Increased
21	Isoechoic	Uniformly isoechoic	Extending beyond costal arch	Irregular	Absent	Increased
22	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
23	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
24	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
25	Hyperechoic	Uniformly isoechoic	Within costal arch	Smooth	Present	Normal
26	Hypoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Present	Normal
27	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
28	Isoechoic	Mixed echogenesity	Extending beyond costal arch	Irregular	Present	Decreased
29	Isoechoic	Uniformly isoechoic	Extending beyond costal arch	Smooth	Absent	Increased
30	Hyperechoic	Uniformly isoechoic	Within costal arch	Irregular	Present	Decreased
31	Hyperechoic	Mixed echogenesity	Extending beyond costal arch	Smooth	Absent	Normal

Table 19: Ultrasonography finding of hepatic parenchyma in animals of Group C: Focal hepatic parenchymal disorder

Animal no.	Comparative echogenicity with renal cortex	Echogenicity within hepatic parenchyma	Location	Borders	Peritoneal effusion	Vasculature within focal lesion on Color Doppler
1	Hyperechoic	Hyperechoic focal lesion about 2cm diameter in right lateral lobe	Within costal arch	Smooth	Absent	Decreased
2	Hyperechoic	Hyperechoic focal lesion about 1.3cm diameter in left medial lobe	Within costal arch	Smooth	Present	Normal
3	Hyperechoic	Hypoechoic circular focal lesion about 1.5 cm diameter in left medial lobe	Within costal arch	Smooth	Absent	Absent
4	Hypoechoic	Hyperechoic focal lesion about 3.2cm diameter in right lateral lobe	Within costal arch	Smooth	Absent	Increased
5	Hypoechoic	Mixed echogenic irregular border focal lesion about 2.7cm diameter in right lateral lobe	Within costal arch	Smooth	Absent	Increased
6	Hyperechoic	Hypoechoic irregular border focal lesion about 3.1cm diameter in right medial lobe	Within costal arch	Smooth	Absent	Decreased

Table 20: Ultrasonography finding of hepatic parenchyma in animals of Group D: Multifocal hepatic parenchymal disorder

Animal no.	Comparative echogenicity with renal cortex	Echogenicity within hepatic parenchyma	Location	Borders	Peritoneal effusion	Vasculature within lesions on Color Doppler
1	Hypoechoic	Hyperechoic multiple irregular border lesions varying from 3cm to 4cm diameter	Within costal arch	Irregular	Absent	Decreased
2	Hypoechoic	Multiple mixed echogenic nodules	Extending beyond costal arch	Smooth	Absent	Decreased
3	Hypoechoic	Multiple uniformly hyperechoic lesions	Extending beyond costal arch	Irregular	Absent	Decreased
4	Hyperechoic	Multiple target lesions varying from 0.5cm to 2cm	Extending beyond costal arch	Smooth	Present	Increased
5	Hyperechoic	Mixed echogenic multiple lesions of irregular shape	Within costal arch	Irregular	Absent	Increased
6	Hyperechoic	Mixed echogenic numerous nodules	Within costal arch	Irregular	Absent	Increased
7	Hypoechoic	Hyperechoic numerous nodules	Extending beyond costal arch	Irregular	Present	Decreased
8	Hypoechoic	Mixed echogenic multiple nodules	Extending beyond costal arch	Irregular	Absent	Increased
9	Hypoechoic	Multiple Hypoechoic vacuolar lesions in right lateral and medial lobes	Extending beyond costal arch	smooth	Absent	Absent to decreased

Table 21: Ultrasound guided percutaneous liver biopsy findings

Animal no.	Site of sample collection	Size of the lesion	Diagnosis
C-3	Left medial lobe	About 1.5cm diameter	Hepatic abscess
D-8	Right medial lobe	About 3cm diameter	Hepatic nodule

Fig. 8: Age wise distribution of hepatic disorders

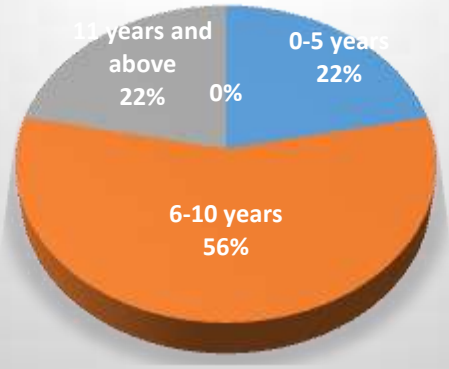


Fig. 9: Sex wise distribution of hepatic disorders

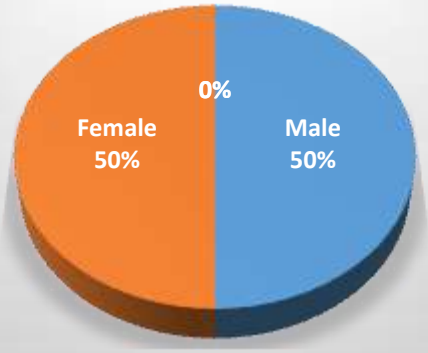
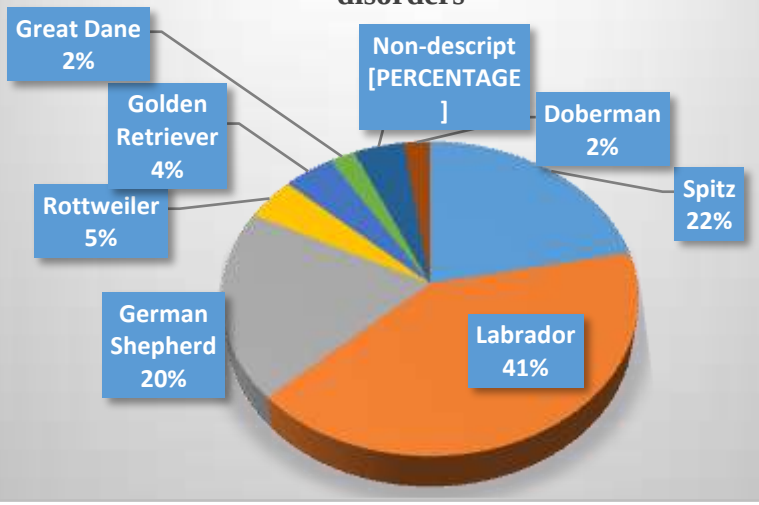


Fig. 10: Breed wise distribution of hepatic disorders



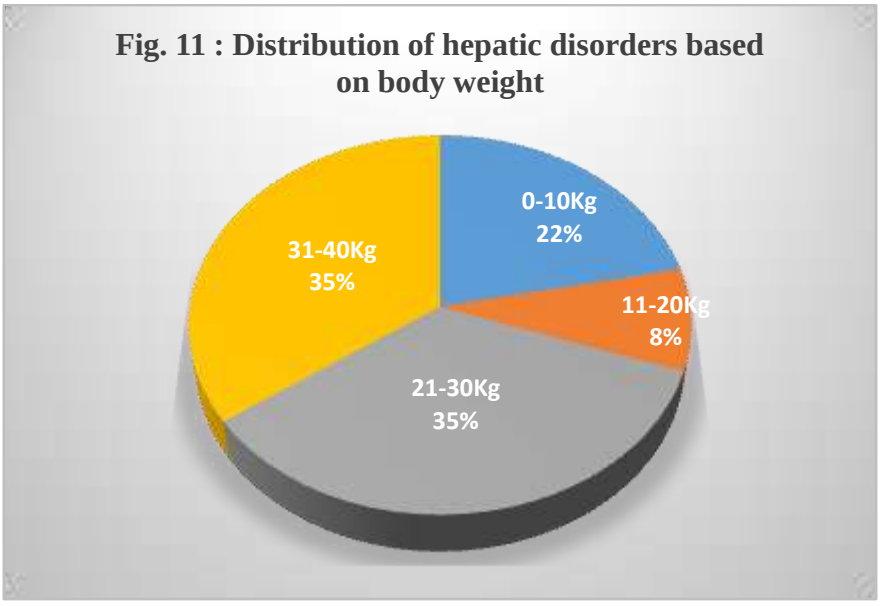


Fig. 11 : Distribution of hepatic disorders based on body weight
0-10Kg
22%
11-20Kg
8%
21-30Kg
35%
31-40Kg
35%

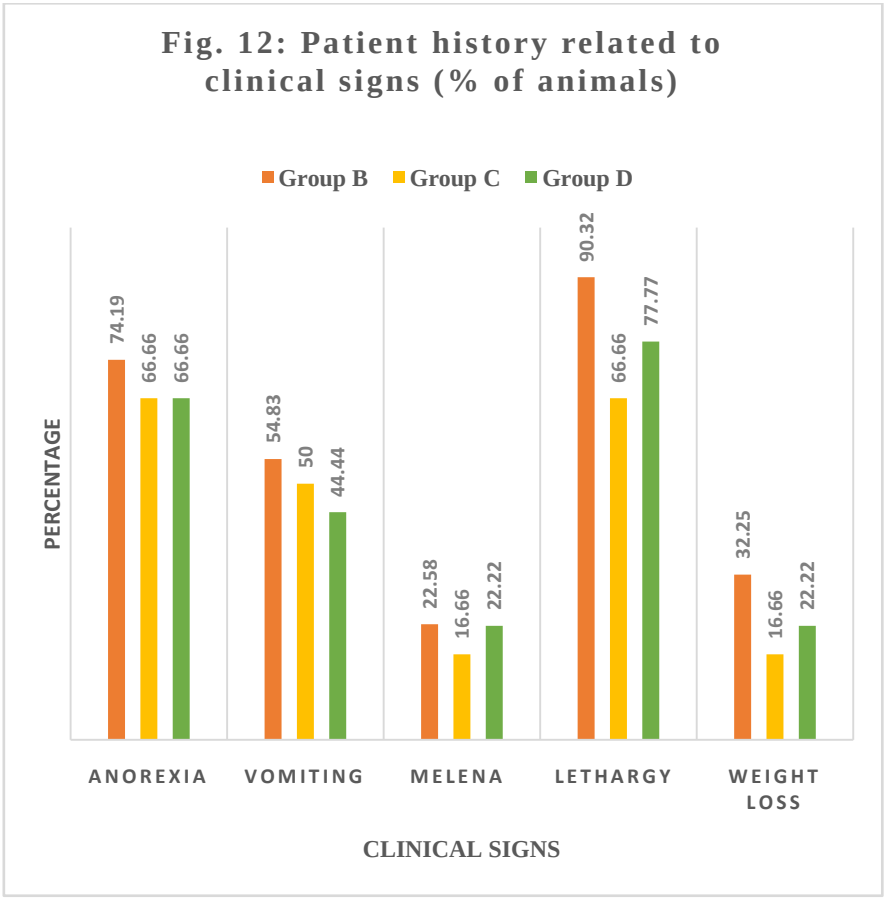


Fig. 12: Patient history related to clinical signs (% of animals)
Group B
Group C
Group D
PERCENTAGE
74.19
66.66
66.66
54.83
50
44.44
22.58
16.66
22.22
90.32
66.66
77.77
32.25
16.66
22.22
ANOREXIA
VOMITING
MELENA
LETHARGY
WEIGHT LOSS
CLINICAL SIGNS

Fig. 13: Comparative echogenicity of hepatic parenchyma with renal cortex in various groups

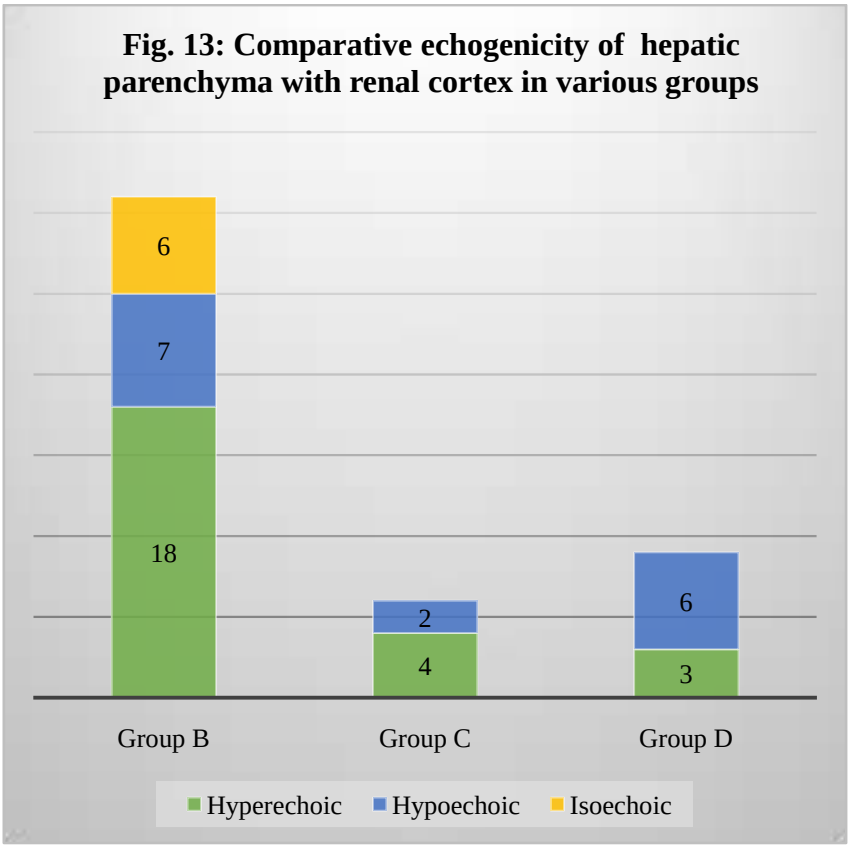


Fig. 14: Echogenicity within hepatic parenchyma

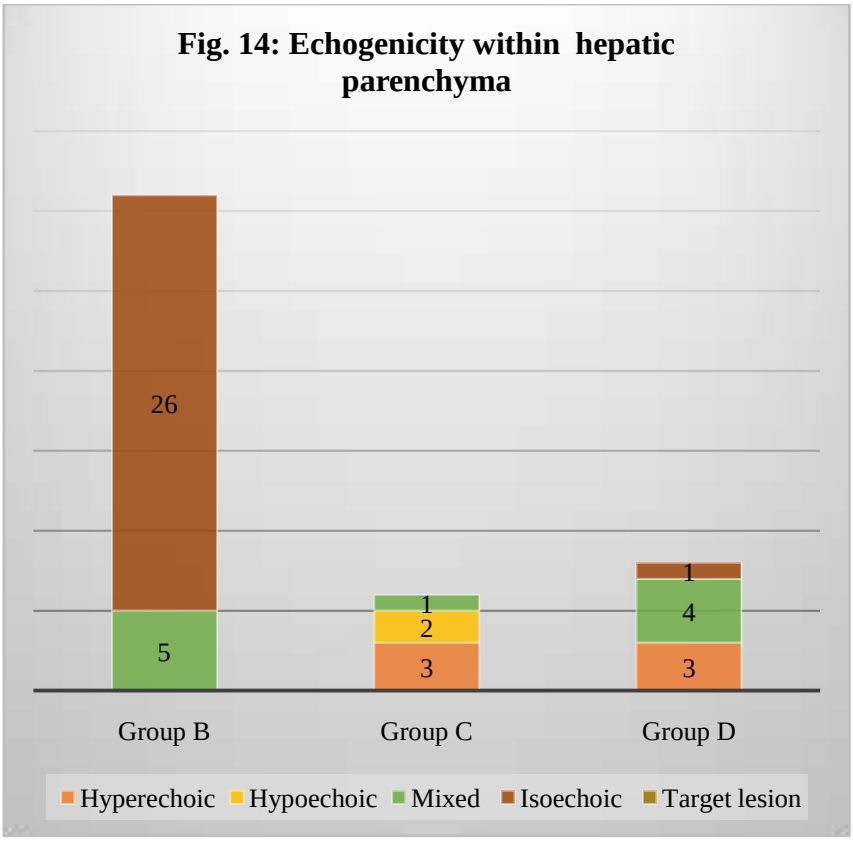


Fig. 15: Dog with distended abdomen due to peritoneal effusion.

Fig. 16: Cachectic condition in a dog.

Fig. 17: Male four month old GSD with history of vomiting, melena and anorexia.

Fig. 18: Ten year male GSD with weakness

Fig. 19: Twelve year male Spitz dog with tucked up abdomen and weakness

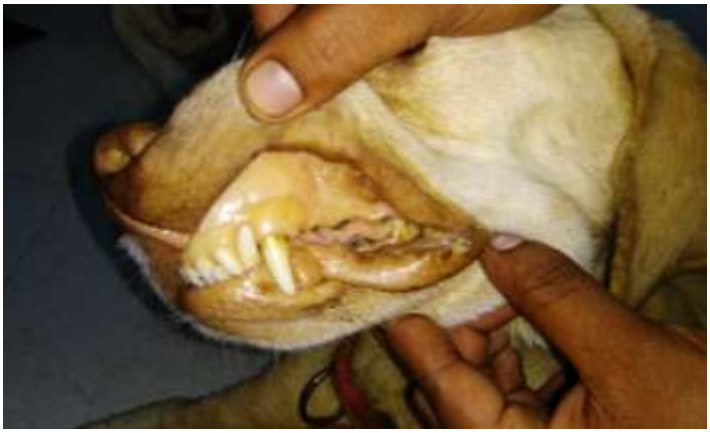


Fig. 20: Icteric oral mucous membrane in a dog

Fig. 21: Cutaneous tumor in a dog with hepatic disorder

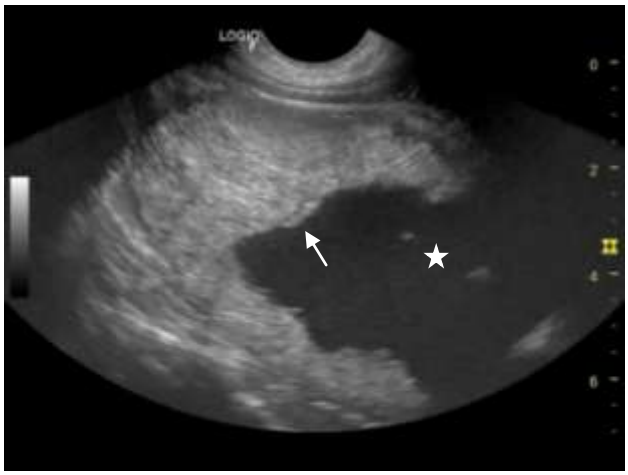


Fig. 22: Sagittal image of liver in a 5 year male Labrador showing small, hyperechoic, irregular contour of liver (arrow) with lack of normal portal vascular markings and an anechoic effusion (*). These findings are consistent with hepatic cirrhosis and fibrosis.

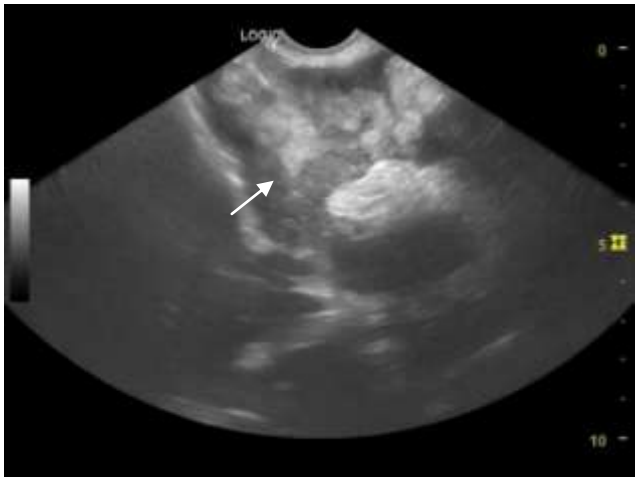


Fig. 23: Right sagittal image of liver in a 7 year old male Spitz with regenerative nodules (arrow) in end-stage cirrhosis in dogs.

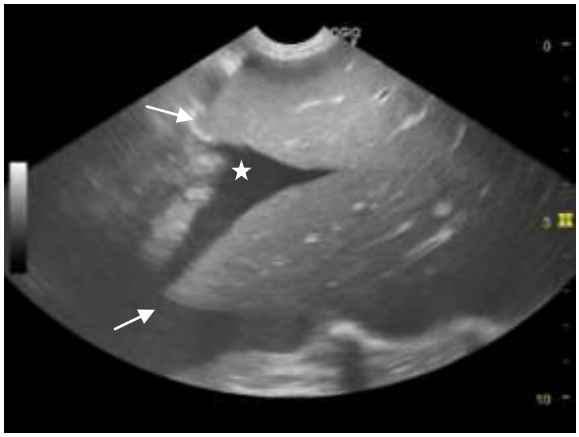


Fig. 24: Rounded liver edges (arrow) indicating hepatomegaly and presence of anechoic peritoneal effusion (*).

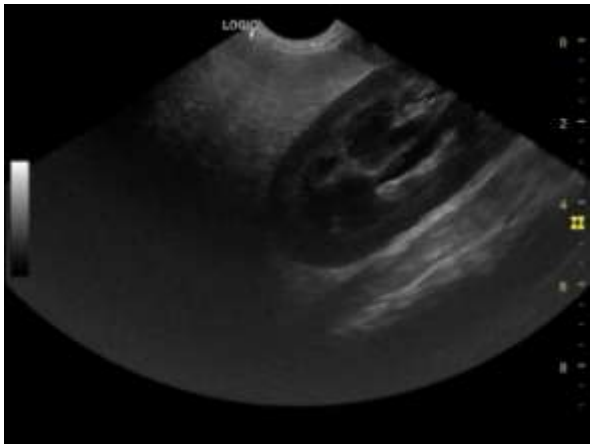


Fig.25: Hepatomegaly, hyperechoic liver parenchyma with ultrasound-beam hyperattenuation in a 16 month old male Labrador consistent with steroid hepatopathy. The margins of the portal veins are decreased in prominence because of an overall increase in hepatic echogenicity.

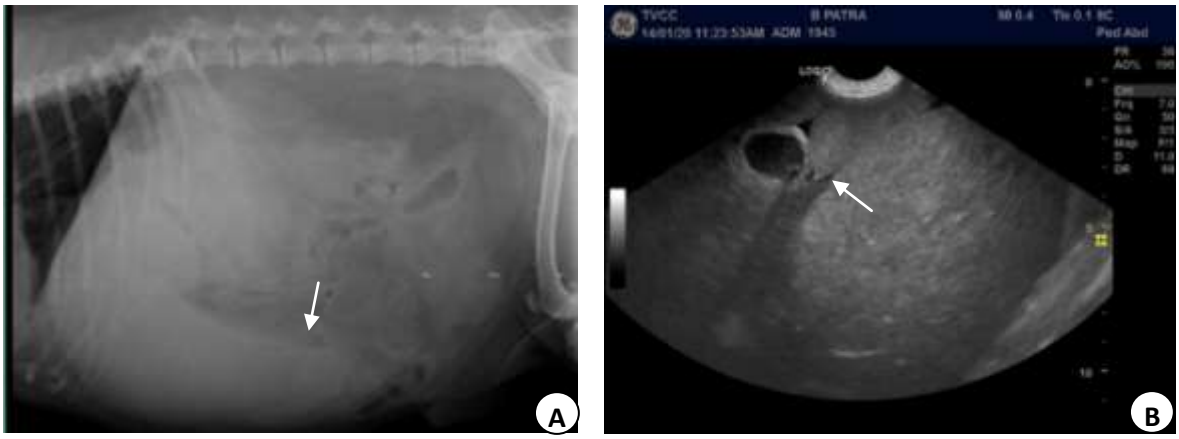


Fig. 26: (A) Right lateral survey abdominal radiograph of a 7year old male Labrador showing hepatomegaly with decreased abdominal detail. There is caudal protrusion of liver lobes (arrow) beyond costal arch up to mid abdomen displacing intestines, rounding of liver edges.(B) Hyperechoic, homogenous hepatic parenchyma with rounded edges (arrow) and peritoneal effusion.

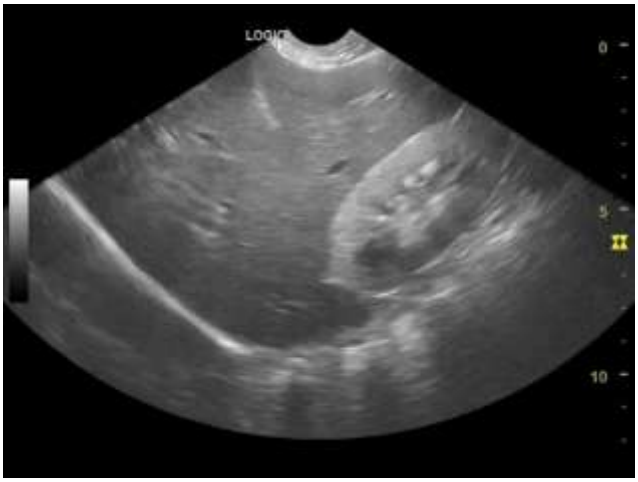


Fig. 27: Hypoehoic liver parenchyma extending beyond right kidney indicating hepatomegaly in a 10year old German Shepherd.

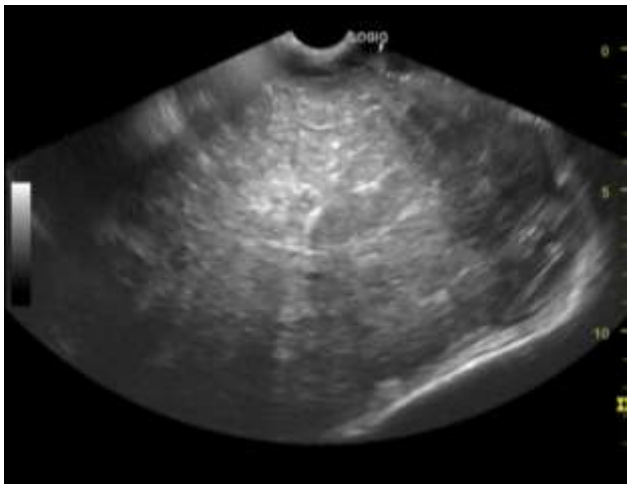


Fig. 28: Diffuse mixed echogenicity of liver parenchyma in an 11year old male Labrador dog.

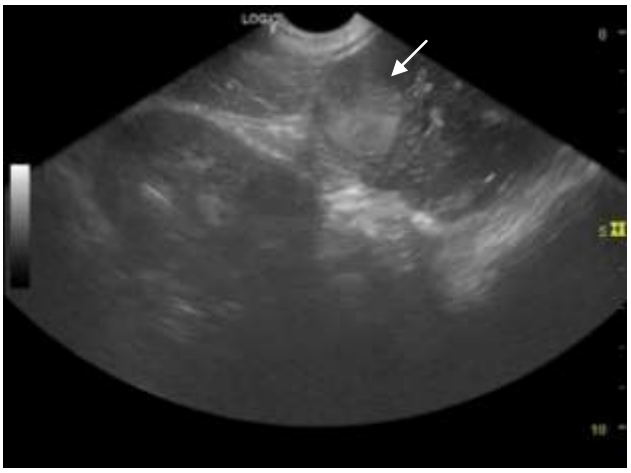
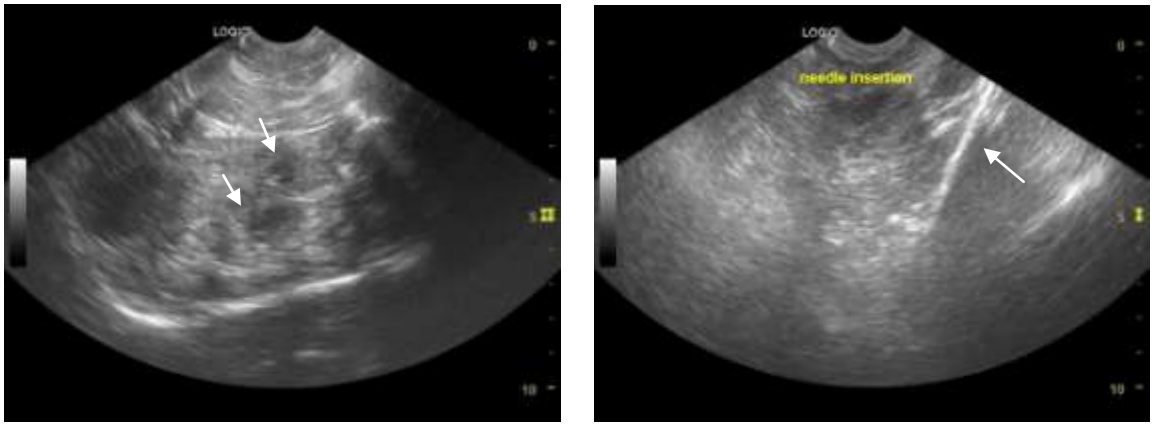


Fig. 29: Focal hyperechoic lesion (arrow) in a 10year old male German shepherd.



A. B.

Fig. 30: (A) Multifocal cavitory lesions (arrow) with a hyperechoic parenchyma in a 10year old Mongrel dog.

(B) Ultrasound guided percutaneous biopsy of multifocal cavitory lesion.

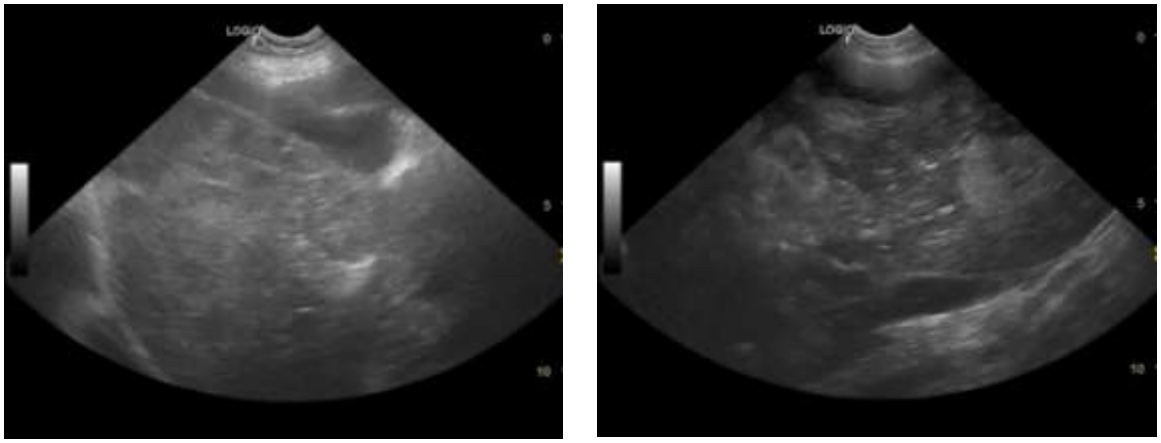


Fig. 31: Sagittal image of diffuse heterogenous or mottledliver parenchyma with mixed echogenicity in a 6year old female German shepherd.

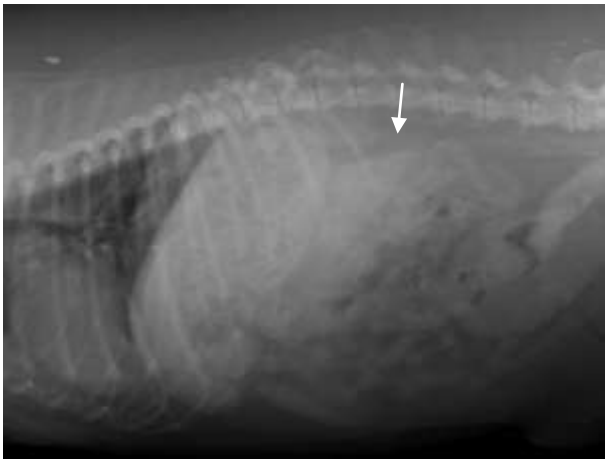


Fig. 32: Right lateral survey abdominal radiograph of a 14year old male Labrador dog with cranial displacement of the stomach and a reduced distance between the stomach and the diaphragm indicating reduced liver size. There also presence of soft tissue opacity (arrow) caudal to stomach displacing intestines caudally.

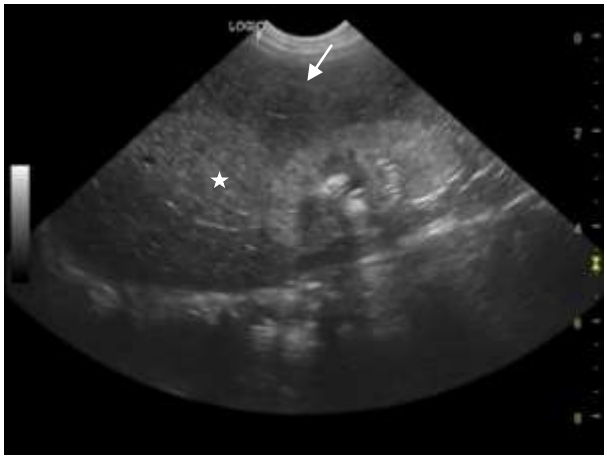


Fig. 33: Hypoehoic enlarged caudate lobe reaching beyond right kidney. Presence of both hyper (*) and hypoechoic (arrow) multifocal lesions.

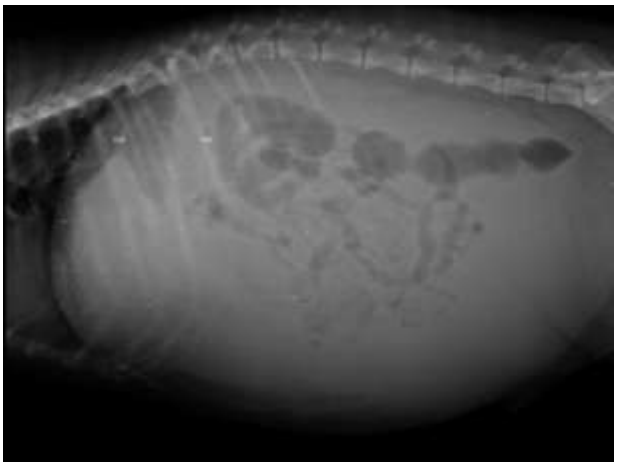


Fig. 34: Survey abdominal radiograph of a Spitz with ground glass appearance with loss of serosal detail representing ascites.

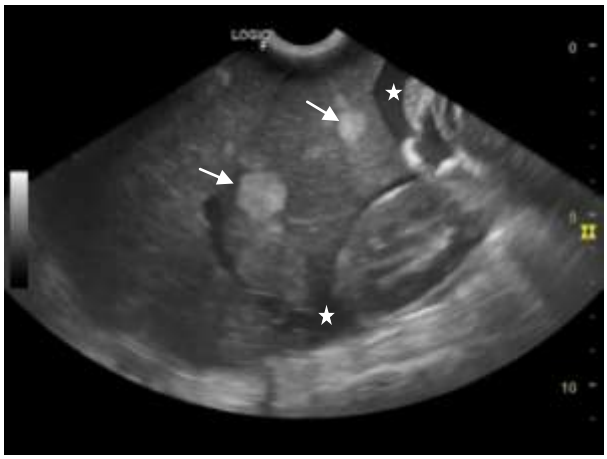


Fig. 35: Isoechoic liver parenchyma with irregular borders and multifocal hyperechoic nodules (arrow) in a 10year old Labrador dog.

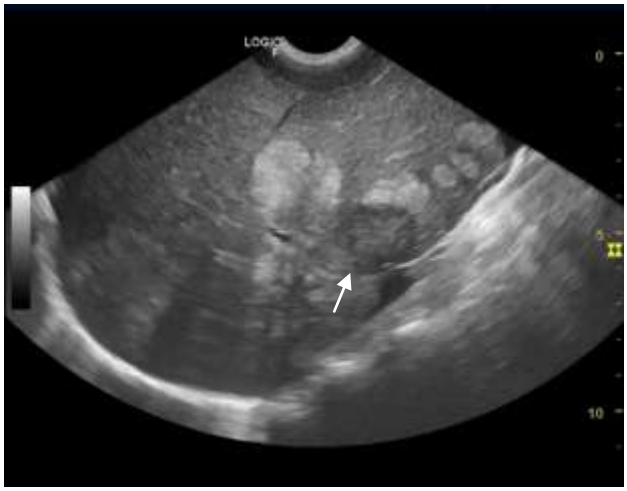


Fig. 36: Presence of target lesion (arrow) along with multifocal hyperechoic nodules.

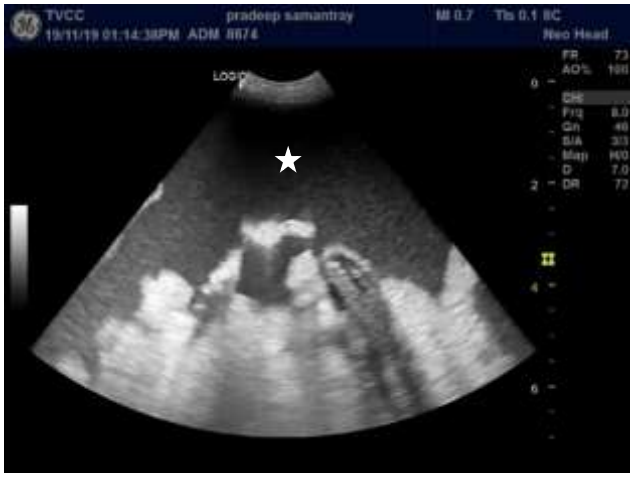


Fig. 37: Presence of hypoechoic peritoneal effusion (*).

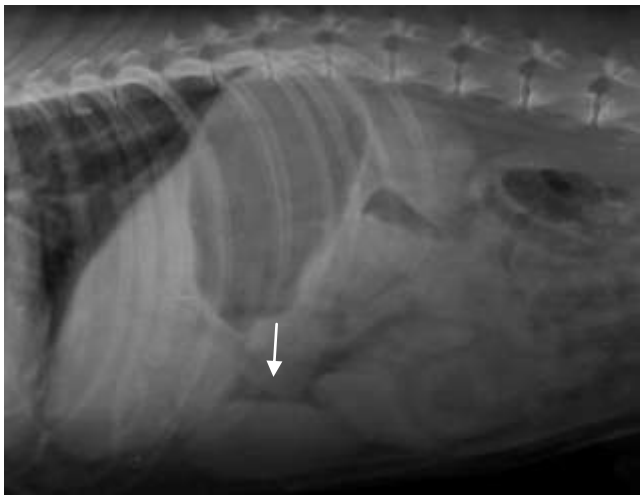


Fig. 38: Survey abdominal radiograph of a 10year old, male, German Shepherd showing radio opaque mass (arrow) with soft tissue density caudo-ventral to liver displacing spleen caudally.

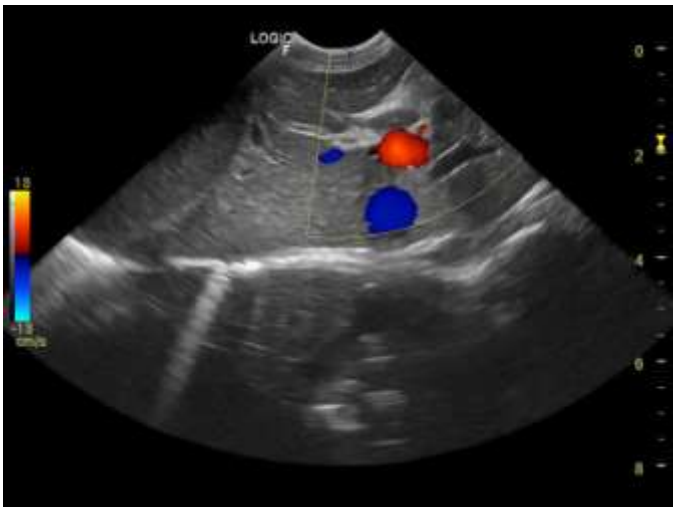


Fig. 39:Doppler ultrasound image of liver showing portal vein (red) and hepatic vein (blue).

CHAPTER V

DISCUSSION

The present study on “Ultrasonographic evaluation of liver disorders in dogs” was conducted in 46 clinical cases of dogs with hepatic disorder and underwentultrasonographic examination. The results of patient evaluation were compared with control group consisting apparently healthy dogs.

5.1 PATIENT SELECTION

The dogs selected in the present study were clinically presented for treatment of hepatic disorder. The Ultrasonographic evaluation of hepatic parenchyma was performed and abnormalities were recorded as diffuse, focal and multifocal as described byGaschen (2009). Further, the study groups were classified as per ultrasonographic findings as control group A, diffuse parenchymal hepatic abnormalities as Group B, focal parenchymal hepatic abnormalities as Group C and multifocal parenchymal hepatic abnormalities as Group D.

5.2 PATIENT EVALUATION

5.2.1 OCCURRENCE

Chandlin (1968) observed hepatic disorders in 3% of clinical cases of dogs. Similarly Vijaykumar*et al.* (2003) reported 3.01 % of incidence of hepatic disorders in dogs. In the present study, middle aged dogs aged between 6years to 10years had higher incidence than young dogs less than 5years or older dogs aged more than 10years. The incidence in both sexes was similar and among the breeds recorded Labrador retriever had highest incidence followed by Spitz, German Shepherd, Rottweiler, Golden retriever, non-descript, Great Dane and Doberman. Large size breeds were more affected compared to small sized breeds of dogs. High incidence of hepatic disorders in breeds of Labrador retriever and Dobermann Pinscher was also reported by Anderson and Sevelius (1991) and Sevelius (1995). The incidence of hepatic disorders in various breeds of dogs in the present study may also be influenced by the popular breeds in this region.

5.2.2 HISTORY

In the present study, nutrition given to the animal consisted homemade food and/or commercial dog food. The incidence was more in dogs given commercial dog food alone or with homemade food. However, further long term study required to assess the effect of nutrition on hepatic disorders. Dog with hepatic disorder were present early with mean duration of 5.6 days after onset of symptoms. Concurrent diseases noticed are skin allergy, mammary tumour, cutaneous tumour and skin lesionin 13.04% dogs with hepatic disorder. Clinical symptoms as noticed by animal owners related to hepatic disorder were anorexia, vomiting, melena, lethargy and weight loss, of which lethargy was the common symptom followed by anorexia, vomiting, weight loss and melena. The decreased production of thrombin activatable fibrinolysis inhibitor in patients with liver disorders leads to increased fibrinolysis resulting in gastrointestinal ulcers or coagulopathies which results in melena and hematochezia (Prins*et al.,* 2010).Further, inadequate nutrition due to inappetence and increased cellular metabolism results in gradual weight loss (Hess and Bunch, 2000).

5.2.3 VISUAL OBSERVATION

Weak body condition was noticed in 45.65% dogs with hepatic disorder. Abdominal distention was noticed in half of the cases with diffuse parenchymal disorder and nearly one-fourth of the cases with multifocal hepatic disorder. Weak body condition may be attributed to poor nutritional status due to decreased apatite. At the same time ascites results in distention of abdomen (Hess and Bunch, 2000).

5.2.4 PHYSICAL EXAMINATION

Conjunctival or oral mucous membrane was either normal, pale or icterus. Pale and icterus mucous membrane was noticed in one third of the cases each in the present study. Dehydration was noticed more commonly in diffuse and multifocal focal hepatic parenchymal disorder compared to focal hepatic parenchymal disorders. Abdominal ballottement indicating ascites and abnormal abdominal mass was evident in some animals of group B and group D, whereas it was absent in group C dogs on abdominal palpation.Similar signs of icterus and dehydration in patients with hepatic disorders were also reported Kearns (2009) and Kanemoto*et al.*(2011). According to

Kanemoto*et al*,(2011), since of jaundice and icterus are pathognomic signs of hepatic disorders.Other clinical manifestations reported in hepatic disorders are diarrhoea, polyuria, polydypsia and dehydration (Kearns, 2009 and Westgren*et al.*, 2014). Association of hypoalbuminemia with asciteswas observed in the present study and similar observations were also reported by Saravanan*et al.* (2012) and Elhiblu*et al.* (2015). Further, due to hypoalbuminemia there will be variation in intravascular and extravascular oncotic pressure resulting ascites and hindlimb oedema, seen as common complication in chronic hepatic dysfunction in dogs (Raffan*et al.*, 2009).

5.2.5 PHYSIOLOGICAL PARAMETERS

In the present study, the mean values of various physiological parameters viz., temperature, heart rate and respiratory rate were observed in normal reference range in all the groups. However, the mean values of heart rate of group D were significantly higher compared to control group A. This may be attributed to anaemia and hypoalbuminemia condition in the present study.

5.2.6 HAEMATOLOGICAL STUDIES

In the present study the mean values of various haematological parameters viz., haemoglobin, packed cell volumeand total erythrocyte count were observed in normal reference range, however, the values were lower than control group. The leukocyte count was higher in all the groups with hepatic disorder when compared to control group and the values were significantly higher in group B and group D dogs with neutrophilia and lymphocytopenia. Reduced portal blood flow increases transient time of erythrocytes in spleen and at the same time high level of bile acids results in fragility of erythrocytes which results in increased erythorlysis and anaemia in chronic hepatic dysfunction (Chikazawa*et al*., 2013; Bush, 2002 and Rothuizenand Meyer, 2000). Poor nutritional status and decreased availability of micronutrients for synthesis of haemoglobin may also reduce haemoglobin value in blood (Bush 2002). However, decrease in production of erythropoietin by the liver in hepatic disorders could lead to decrease in production of erythrocytes thus total erythrocyte count may be affected as per Washabau (2010). Decreased values of haematological parameters in hepatic disorders were also reported by Tantary*et al.* (2014), Saravanan*et al.* (2014) and Elhiblu*et al.* (2015).

Reports of mild leucocytosis in some cases of hepatic disorders were attributed to stress. Whereas, neutrophilia and leucocytosis with left shift was considered to be an indicator of chronic inflammatory response in chronic hepatitis leading to chirrhosis. Both anaemia and leucocytosis are the findings in hepatic disorders in dogs and similar findings were reported by Shaker and Khalifa(2012), Chikazawa*et al*. (2013) and Font (1989). According to by Poldervaart*et al*. (2009), neutrophilic leucocytosis was a characteristic feature of ongoing inflammatory conditions.

The reduction in the mean values of PCV can be attributed to dehydration status seen in the dogs affected with various hepatic disorders.Significant decrease in the mean values of packed cell volume in focal parenchymal disorders and a non-significant decrease in diffuse parenchymal disorders with ascites, without ascites and biliary tract disorders were reported by Tantary*et al.* (2014).

5.2.7 BIOCHEMICAL STUDIES

In the present study, serum biochemical studies showed increased mean values of Alkaline Phosphatase, Gamma GlutamylTransferase,Aspartate Aminotransferase andAlanine Aminotransferasein dogs suffering from hepatic disorder and was significantly higher when compared to control group. Mean values of total plasma protein and serum albumin levels in dogs with hepatic disorders were lower than the values of healthy dogs in the present study and were statistically significant from the control group. The values in individual animals ranged from normal to higher than the physiological range.

The normal findings in values of gamma glutamyltransferase,aspartate aminotransferase andalanine aminotransferase may be seen in liver cirrhosis or fibrosis of hepatocytes resulting in decreased production of enzymes (Font*et al.*, 1989). Although increase in alkaline phosphatase may indicate primary hepatic disease, it is not a liver-specific. Hence, alkaline phosphatase values may be elevated by extrahepatic sources (Chapman and Hostutler, 2013). Therefore, high levels of alkaline phosphatase values in the present study was not considered as indicator of hepatic disorder.

According to Kramer and Hoffman(1997) inflammation, altered cellular membrane permeability and cellular necrosis of hepatocytes my result in increased values of Aspartate Aminotransferase andAlanine Aminotransferase. Lidburg and Steiner (2013) also opined that evaluation of Aspartate Aminotransferase andAlanine Aminotransferaseconcentrations inserum were considered as indicator of hepatocellular leakage and they are most commonly measured markers in diagnosis of hepatocellular disorder in dogs. Significant elevation of ALP in hepatocellular diseases in dogs were also observed by Saravanan*et al.* (2014), Tantary*et al.* (2014) and Elhiblu*et al.* (2015). However, estimation serum gamma glutamyltransferaseconcentration in evaluation of hepatic disorder when associated with hypoalbuminemia can be a regular parameter when compared to Aspartate Aminotransferase andAlanine Aminotransferase estimation. Meyer and Rothuizen(2013) observed elevation of serum concentration of Alanine Aminotransferase and gamma glutamyltransferase concentration in hepatic disorder patients when drugs like corticosteroids were administered especially in patients with cholestatic disorder. Therefore, they were considered as cholestatic markers, which are synthesized in response to the retained bile (Webster, 2010).

Total protein values were decreased in the dogs of hepatic disorder in the present study. Since liver is the primary site for protein synthesis and also degradation, liver cirrhosis and portosystemic vascular abnormalities result in hypoproteinemia (Tennant, 1997). Most of the plasma proteins were synthesized and catabolised by the liver, it can be a vulnerable indicator of compromised hepatic function.Webster(2005) also considered that decrease in the total serum protein could be due to the primary role of liver in the synthesis of major plasma protein as well as the site of degradation and synthesis of many other proteins that is influenced by hepatic diseases in many ways. Prasse*et al.*(1983) opined that low levels of serum albumin concentration in hepatic disorders indicate chronic hepatopathies. Decreased food intake in patients with hepatic disorders is also associated with decreased serum albumin levels. Similar findings of hypoproteinemia and hypoalbuminemia in hepatic disorders in dogs was also reported by Kanemoto*et al*. (2013), Bexfield*et al*. (2011) and Kumar *et al*. (2012). Significant decrease in serum protein levels in dogs with hepatic dysfunction were also observed by Rutgers *et al*. (1993), Tantary*et al.* (2014), Saravanan*et al.*(2014), Lathamani and Nalinikumari (2015) and Elhiblu*et al*. (2015).

5.2.8 RADIOLOGY

Radiography of liver was found to be having limited use in the study of hepatic parenchymal disorders.Based on visibility of hepatic margins, location, size and shape of the hepatic borders were analysed. In dogs with ascetic fluid, the border of the liver was indistinct, thus study of liver on radiographs in dogs with ascites was found to be non-diagnostic. In the present study, hepatic margins was well defined in 45% of dogs of group B, whereas, nearly 80% dogs had well defined borders in group C and group D animals. This indicated diffuse hepatic parenchymal lesions have higher incidence of Ascites compared to focal and multifocal lesions. Location of the liver was useful in assessment of size and shape of the liver. Hepatic enlargement was seen in 42% of dogs in group B, 50% of dogs in group C and 20% of dogs in group D. Hepatic enlargement was more obvious in focal hepatic abnormalities when compared to diffuse and multifocal abnormalities. However, values not indicative as studies cannot be undertaken in ascites condition.According to Root (1974) and O‟Brien (1978),diffuse hepatomegaliesresults in significant portion of caudal liver mass to project caudal to the costal arch, indicating an obvious increase in liver size and rounding of costal liver edges in lateral radiographs. O‟Brien (1978) also reported that radiographic opacity of cirrhotic liver varied with the stage and severity of hepatic disorder. Increased liver density with irregular marginwere the most common radiographic appearance of the cirrhosed liver. However, according to Suter (1982) appreciation of radiographic appearance of reduced liver size in microhepatica is difficult to when compared to hepatomegaly.Radiographic evaluation give subjective assessment of alteration in liver size and it will not demarcate subtle changesin hepatic parenchyma and also has limitation in accurate evaluation of generalized changes in liver size (Godshalk*et al.*,1988 and Barr,1992).Therefore, radiography alone is not a suitable diagnostic tool in the assessment of hepatic disorder in dogs.

Radiographic evaluation of abdominal region were useful in assessment of size, shape and radiographic opacity of the liver, however, subtle changes in the liver were not were not appreciable (Partington and Biller, 1995). Cranial displacement of gastric axis can be considered as microhepatica and caudal extension of liver margin beyond costal arch with rounding of margin can be considered as hepatomegaly.

Further, in presence of ascites, visibility of distinct margins of visceral structures may be decreased or absent.

5.2.9 ULTRASONOGRAPHY

Ultrasonography was feasible to conduct on dogs by physical restraining. Assessment of echogenicity of liver parenchyma was useful in identification of diffuse, focal and multifocal abnormalities. Generalized and hypoechoic hepatic parenchymal abnormalities were indicative of diffuse infiltrative conditions like leukaemia, acute hepatitis, amyloidosis, lymphoma and passive congestion of the liver. Whereas, chronic hepatitis, steroid hepatopathy, cirrhosis and sometimes lymphoma may result in uniformly hyperechoic liver echogenicity. Mixed uneven echogenicity and coarse echo texture in part of the hepatic parenchyma or in whole liver structure may be caused in neoplastic, toxic and diffuse inflammatory conditions. However, determination of type of echogenicity in liver parenchyma and to ascertain the changes in echogenicity and echo texture as abnormal is difficult.

Ultrasonography of liver is useful identification of alternative diagnosis and other existing complications like ascites. Hence, for initial evaluation of the hepatic disorders in small animals, hepatic ultrasonography can be a preferred diagnostic imaging technique. By hepatic ultrasonography, size and shape of the liver; echotexture and echogenicity of liver parenchyma; and study biliary tract and blood vessels can be undertaken. However, in dogs with CH and in deep chested animals, wherein the liver is located cranially usually under the rib case possess challenge to the sonographer. Experience of the sonographer and the type of ultrasound scanning machine used, the results of published work vary (Bexfield*et al.*, 2011).

Small size of liverwas commonly reported in liver cirrhosis condition where in the liver parenchymal tissue is replaced with fibrous tissue (Kumar *et al.*, 2013; Elhiblu*et al*., 2015 and Bexfield*et al*., 2011). The findings of microhepatica condition in the present study may be attributed to hepatic fibrosis or cirrhosis. On ultrasonography,irregular liver border, small size of the liver, generalized or diffusely increased echogenicity and presence of lots of anechoic peritoneal fluid are typical findings in cirrhotic liverin dogs(Biller *et al*., 1992; Elhiblu*et al*., 2015; and Vijayanand and Nagarajan, 2007).Similar findings of ultrasonographic observations in

the present study may be attributed to cirrhosis condition of liver. The hepatic ultrasonographic results of chronic hepatitis in the present study was also similar to the findings of Tantary*et al*. (2014), who reported hyper echoic hepatic parenchyma in normal sized liver and also diffuse hyper echoic small sized liver associated with presence of peritoneal fluid.

The findings of focal hepatic parenchymal abnormalities and diffuse hepatic parenchymal abnormalities on hepatic ultrasonography in the present study were in accordance with the findings of Saravanan*et al.* (2014) who reported ultrasonographicevaluation in 72 dogs showing symptoms of ascites due to various hepatic disorders and found that 25% of dogs had focal hyperechoicabnormalities and 45.8% of dogs had diffuse hyper echoic abnormalities.

In present study hepatomegaly with increased parenchymal echogenicity and hyper ultrasound beam attenuation in the distantpart of liverwas observed which may be observed in fatty infiltration in hepatic parenchyma may be due to diabetes mellitus, steroid hepatopathy orin obesity (Yeager *et al.*,1992).

Observations of enlarged hypoechoicliver with rounded marginsandmarked visibility ofhepatic portal vessels may be attributed to acute hepatitis and these findings are in concurrence with Tantary*et al.* (2014) and Lamb (1995).

The target lesions in the hepatic parenchyma on ultrasound, had anhyperechoic centre zone surrounded with hypoechoic outer zone when compared to surrounding hepatic parenchyma. Similar findings were observed by Cuccovillo and Lamb (2002),they observed that 74% of cases with one or more target lesion in hepatic parenchyma and 81% of cases with multiple target lesions had malignancy. The non-malignant target lesions found in the liver were nodular hyperplasia, pyogranulomatous hepatitis, cirrhosis and chronic active hepatitis.

According to Cuccovillo and Lamb (2002) and Stockhaus*et al*.(2004), focal parenchymal lesions found either single or multiple should be consider for diagnosis of malignancy as focal parenchymal lesions present either single or multiple may or may not show typical finding to interpret as primary or metastatic hepatic neoplasia. Ultrasonographic appearance of hepatic tumours marked vary. Histologically different tumours may have similar finding on ultrasound image or ultrasonographically

different pattern of the lesions may have same type of tumour even within the same animal. Therefore, any presence of focal or multifocal hepatic lesions on ultrasonography requires confirmation by histopatholocal study of biopsy sample collected by ultrasound guided fine needle aspiration biopsy or tissue core biopsy taken with tru-cut needle. However, incidental finding of single focal lesion during routine scanning of healthy animal with normal appearance of liver on ultrasonography may be considered a possibility of benign lesion (Gaschen, 2009). In the present study, unwillingness of the many owners for biopsy collection posed challenge for diagnosis of focal and multifocal lesions as well as dogs with diffuse mixed echogenicity of liver parenchyma.

Hepatic abscesswere reported in both humans and animals. Both solitary and multiple abscess or complex abscess lesions seen on ultrasonography, however, solitary lesions are commonly encountered than multiple ones. On ultrasonography, the diameter, border of the lesion, echotexture and acoustic enhancement by the lesions will vary. The lesions may have finding of gas bubble within the abscess cavity, ascites, hyperechoicperi-hepatic adipose tissue and far enhanced artefact in most of the dogs. Far enhanced artefact on ultrasonography of liver may also be useful in identification of cavitary lesions containing fluid. Hence, sample collection from individual lesions is required with the help of ultrasonography. Although ultrasound guided sample collection will confirm the suspected abscess, it should be performed only in cases where it is safe to access.

Chronic hepatitis resulting in cirrhosis will have hyperechoic appearance on ultrasonography due to fibrosis or glycogen type vacuolation in liver parenchyma. As the condition advances, hypoechoic nodular structures interposed in heterogeneous hepatic parenchymal echotexture becomes evident. The pattern may vary from one lobe to the other liver lobe. At the same time size, shape, contour and echotexture of the liver may be affected by concurrent conditions like benign nodular hyperplasia, acute hepatitis, vacuolarchanges with glycogen or lipid (Cynthia *et al.*, 2019).

According to Kemp *et al*. (2013), 88% of hepatic neoplasia can be determined by ultrasonographic evaluation of liver and any finding of mass in hepatic parenchyma may be associated with neoplasia. Heterogeneous echotexturewhich is an indicator of malignant livertutors may also be found in intratumoral haemorrhage

and focal necrosis (Pradeep, 2017). However, presence of peritoneal fluid was not a sole indicator of malignancy condition in liver. (Cynthia *et al*., 2019).

5.3 ULTRASOUND GUIDED PERCUTANEOUS LIVER BIOPSY

The biopsy procedure required general anaesthesia and willingness of the animal owner. The procedure was found beneficial in biopsy from the lesion and also to monitoring of post biopsy complications like haemorrhage. Ultrasonography guided sample collection will help in collection of most accurate method of biopsy collection and will also help in sample collection with fine needle. Since ultrasonography appearance of different tumors types may be similar or similar tumors may have varying appearance on ultrasonography, it is essential to collect sample by fine needle aspiration or using tru-cut needle for tissue core biopsy for confirmation of type of lesion. Characterization of nature of lesion and severity of often requires biopsy from liver lesion. Ultrasound guided liver biopsy was also useful in differentiating acute hepatic disorder from chronic hepatic disorder and also for staging of neoplastic condition in animals to aid in therapeutic management (Vijay Kumar *et al.*, 2012).

In the present study, the ultrasound guided percutaneous liver biopsy was useful in diagnosis of hepatic nodule and hepatic abscess,the observations are found similar as described by Farrar *et al.* (1996) where they observed acellular transudate with degenerated neutrophils and bacteria.

Ultrasound guided percutaneous sample collection in patients with hepatic lesions on ultrasonography has confirmatory value especially in small focal lesions or nearly isoechoic hepatic lesions in hepatic parenchyma in dogs.

CHAPTER VI

SUMMARY AND CONCLUSION

The present clinical study of "Ultrasonographic evaluation of liver disorders in dogs" was conducted on 46 clinical cases presented with hepatic disorders to the Department of Veterinary Surgery and Radiology and to the Teaching Veterinary Clinical Complex, College of Veterinary Science and Animal Husbandry, OUAT, Bhubaneswar, during the period from October 2019 to March 2020, in an attempt to correlate the findings of ultrasonography with other clinical diagnostic tests like haematological, biochemical and radiographic techniques to establish diagnosis of hepatic disorder and to aid in therapeutic management.

1. Dogs with hepatic disorders and underwent ultrasound scanning of liver parenchyma were selected for the present study. On ultrasonography of liver, dogs with diffuse parenchymal hepatic abnormalities were categorized as Group B, dogs with focal parenchymal hepatic abnormalities were categorized as Group C and dogs with multifocal parenchymal hepatic abnormalities were categorized as Group D. Six apparently healthy dogs were also evaluated as control group to serve as base line referral values for comparison of various parameters and were as Group A.
2. The occurrence of hepatic disorder was more common in middle aged (56%), large breeds of dogs (70%) of either sex. Labrador retriever (41%) was most affected, followed by Spitz (22%), German Shepherd (20%), Rottweiler (5%), Golden retriever (4%), non-descript (4%), Great Dane (2%) and Doberman (2%).
3. Lethargy was the common symptom followed by anorexia, vomiting, weight loss and melena. Weak body condition and abdominal distention were seen in nearly half of the cases.
4. Conjunctival mucous membrane was icteric and pale in 1/3rd of cases each. In some of the animals of with diffuse hepatic disorder and multifocal hepatic disorder conditions dehydration and abdominal ballottement were evident.

5. Physiological parameters were within normal physiological range and were not significant indicator of hepatic disorder.
6. Haematological studies revealed decreased TEC, PCV and Hb, whereas, leukocytosis with nutrophilia and lymphocytopenia, indicative of affection in RBC production, dehydration and inflammatory process.
7. Serum biochemical studies revealed significant increase in liver enzymes viz., ALT, AST, ALP and GGT values, and decrease in TPP and albumen levels, indicative of hepatic disorder.
8. Survey radiography of lateral and ventrodorsal thoraco-abdominal radiographs showed size, shape and location of liver in dogs without ascites. Liver density was not distinct in ascitic conditions.
9. Ultrasonography was provided saggittal and transverse section images of liver parenchyma. The comparative echogenicity with renal cortex was hypoechoic in 20% to 33% of dogs with hepatic disorder. Uniformly isoechoic was evident in diffuse parenchymal abnormalities, whereas, focal and multifocal hepatic parenchymal abdnormalities were easily identifiable. Assessment of liver location, margins and size was possible in all animals. Ascites conditions provided better visibility of individual lobes. Color Doppler was useful in assessment of vasculature in liver parenchyma and focal/multifocal abnormalities.

Conclusion:

The conclusions of "Ultrasonographic evaluation of liver disorders in dogs" conducted on 46 clinical cases of dogs are as follows:

1. The occurrence of hepatic disorder was more common in middle aged, large breeds of dogs of either sex. Labrador retriever was most affected breed.
2. Lethargy was the common symptom followed by anorexia, vomiting, weight loss and melena. Icteric mucous membrane was seen in 1/3rd of cases.
3. Physiological and haematological parameters were within normal physiological range, whereas, serum biochemical parameters were suggestive of hepatic disorder.

4. Radiograph was useful only in dogs without ascites to assess location, size and margin.
5. Ultrasound was found useful in comparative assessment with other visceral organs; overall size, shape, margin and location assessment; visualization of liver parenchyma and its echogenicity in identification of diffuse, focal and multifocal parenchymal abnormalities; assessment of vasculature within the abnormal findings and useful in accurate biopsy to aid in confirmatory diagnosis.

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