RADIOGRAPHIC AND ELECTROCARDIOGRAPHIC CHANGES WITH SPECIAL REFERENCE TO THERAPEUTIC EVALUATION OF CONGESTIVE HEART FAILURE IN GERIATRIC DOG

ABSTRACT OF THE THESIS

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By

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Place _____

Date _____ (*Dr. Nikhil Raj*)

TABLE OF CONTENTS

Chapter	Title	Page
1	Introduction	1 - 3
2	Review of Literature	4 - 17
3	Materials & Methods	18 - 24
4	Results	25 - 64
5	Discussion	65 - 72
6	Summary & Conclusion	73 - 74
7	Literature Cited	75 - 92
	Brief Bio-data of the student	

Acquired Heart Diseases	AHD

Congestive Heart Failure	CHF
Dilated Cardio Myopathy	DCM
Mitral Valve Disease	MVD
Renin-Angiotensin System	RAS
Patent Ductus Arteriosus	PDA
Angiotensin-Converting Enzyme	ACE
Myocardial Oxygen Consumption	MVO_2
Systolic Arterial Blood Pressure	SAP
Diastolic Arterial Blood Pressure	DAP
Canine Heartworm	CHW
Vertebral Heart Size	VHS
Right Lateral	RL
Left Lateral	LL
Ventrodorsal	VD
Dorso – Ventral	DV
Renin–Angiotensin–Aldosterone System	RAAS
Packed cell volume	PCV
Point Of Maximum Intensity	PMI

ABBREVIATIONS

Introduction

Review of Literature

Materials & Methods

Results

Discussion

Summary & Conclusion

Literature Cited

A Tribute To My Late Grandfather

Department of Veterinary Medicine
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Title of the thesis : Radiographic and electrocardiographic changes with special reference to therapeutic evaluation of congestive heart failure in geriatric dogs

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ABSTRACT

The study on radiographic and electrocardiographic changes with special reference to therapeutic evaluation of congestive heart failure in geriatric dog was conducted in the department of veterinary medicine and veterinary clinical Complex of Bihar Veterinary College, Bihar Animal Sciences University for a period of 6 months from February 2021 to September 2021 Out of 1925 geriatric dogs presented to the clinical complex with 519 dogs were suspected for cardiopulmonary diseases. The detailed study of 50 geriatric cases was done on different parameters and 20 dogs suffering from cardiomegaly with congestive heart failure were divided into two therapeutic groups 10 each and hematobiochemical, radiographic and, ECG findings were studied. Data collected from apparently healthy geriatric dogs on various parameters served as a control value in the study. Clinical and screening study reveals that Labrador and German Shepherd breeds were highly susceptible and most of the dog in the age group of 7 to 9 are found (the reason for this may be that a greater number of cases are from this age group) to suffer from cardiac disease and also the male was more susceptible. An elevated level of creatinine, blood urea nitrogen, total protein, globulin, potassium, bilirubin, SAP, DAP, and CK-MB were found in dogs suffering from cardiac diseases. The lower level of albumin, sodium, hemoglobin, PCV and, platelets were found in dogs suffering from cardiac diseases. 40% of dogs were found to be positive for cardiac marker cardiac troponin I, whereas 13.3% and 56.6 % of geriatric dogs were found to be suffering from heartworm and E. canis infection. A therapeutic combination of pimobendan, ramipril, furosemide, and spironolactone were found better in the management of congestive heart failure in geriatric dogs.

Dr. Nikhil Raj **Dr. Pallav Shekhar**
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CERTIFICATE - I

This is to certify that the thesis entitled **"Radiographic and Electrocardiographic changes with special reference to Therapeutic evaluation of Congestive Heart Failure in Geriatric dog"** submitted in partial fulfilment of the requirement for the award of the degree of **Master of Veterinary Science** in the discipline of **Veterinary Medicine** of the faculty of Post-Graduate Studies, Bihar Animal Sciences University, Patna, Bihar is a bonafide research work carried out by **Dr. NIKHIL RAJ,** Registration No**.: VM0021/2019-20** under my supervision and guidance. No part of the thesis has been submitted for any other degree or diploma.

The assistance and help received during the course of investigation have been fully acknowledge

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CERTIFICATE – II

This is to certify that the thesis entitled **"Radiographic and Electrocardiographic changes with special reference to Therapeutic evaluation of Congestive Heart Failure in Geriatric dog"** submitted by **Dr. NIKHIL RAJ,** Registration No**.: VM0021/2019-20,** to the Bihar Animal Sciences University, Patna in partial fulfillment of the requirements for the degree of **Master of Veterinary Science** in the discipline of **Veterinary Medicine** has been approval by Student's Advisory Committee after an oral examination of the student in collaboration with External Examiner

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Introduction

Cardiac disease is definitely one of the most important diseases of geriatric dogs. This is the least studied disease in India. With the increasing canine population, more diseases are being reported to veterinarians in which cardiac diseases are very hard to diagnose. Parker *et al*. (2006) mentioned that canine cardiac diseases are common, complex, and terrifying to owners. As per Eichelberger and Seine (1996) cardiac disease in canines is considered the second most critical cause of death. It is considered a silent killer disease and can be identified by very specific cardinal signs, modern diagnostic techniques, and radiography. Canines heart diseases are reported at any age; however, age is the greatest risk factor for cardiac disease in both humans and dogs (Urfer *et al.* 2017). This disease is either congenital or acquired. Congenital heart disease has not been studied thoroughly and is found mostly in pups (Tilley 2008). The majority of cardiac disease is acquired, and the dogs are affected during their lifetime. There is similarity in age of human and dog. It is based on the body weight of the dog as the weight of dog above 40 kg, there is equivalency of 9 years to 1 year of human age. So, the definition of geriatric in dogs varied with the breeds. In general terms, geriatric patients are defined as those that have completed 75-80% of their expected life span. Great Danes and other giant breeds of dog could be considered geriatric at only six years of age and, alternatively, a toy Poodle or Jack Russell terrier may be older than 12 years before it is considered geriatric (Rush 2002)**.** Hayek and Devenport (1998) have suggested depending on the age a dog is considered older or geriatric in small breeds 9 to 13 years of age, in medium breeds 9 to 11 years, in large breeds 7 to 10 years and in case of giant breeds 6 to 9 years of age.

It is estimated that nearly 10-15% of all dogs are affected by cardiac diseases (Rush 2002). There are many age-related cardiac diseases in canines (Templeton *et al*. 1979). In aged dogs decline in the cardiovascular response of beta-adrenergic stimulation has been proven (Haidet 1993). However, unlike human elevation in blood pressure as primary problems for cardiac disease are not reported (Meurs *et al.* 2000). Furthermore, arteriosclerosis and related ischemic heart disease are exceptional in dogs, (Falk and Jonson 2000). Geriatric dogs are more susceptible to suffer from conduction problems (Carpenter *et al.* 2005). Many geriatric dogs have heart diseases like chronic valvular disease or cardiomyopathy. Chronic valvular heart disease is a commonest geriatric cardiac disorder. Heart diseases in aged dogs frequently result in CHF. Idiopathic dilated

cardiomyopathy (DCM) is more common in large and giant breed dogs. CHF is a clinical syndrome, caused in dogs primarily by myocardial or valvular changes (Roth 1993, Schlesinger and Rubin 1994, Buchanan 2000). These processes mainly cause tachycardia, peripheral vasoconstriction, and sodium and water retention (Awan and Mason 1996, Camacho 1996), increasing blood pressure and cardiac output, which favours tissue perfusion (Awan and Mason 1996, Camacho 1996).

There are several types of equipment for the diagnosis of cardiac disease. Thoracic radiographs, Electrocardiography, echocardiography is useful for confirmative diagnosis of CHF in geriatric dogs associated with acquired heart diseases.

There are many biochemical tests for the evaluation of cardiac and other functions of the body. AST, creatinine kinase, CK-MB are the cardiac marker test. Besides that, the Snap 4DX test help in the diagnosis of tropical diseases transmitted by ticks. Heartworm disease causes 13% of cardiac disease (Macpete 2018). The Snap 4dx plus test (IDEXX laboratories, USA) identifies antibodies to or infection with multiple tick-borne pathogens and canine heartworm antigen assay.

There are several therapeutic protocols for management of cardiac disease in canines. Recent studies advocate that pimobendan as a positive inotrope is very safe even after long-time therapy of CHF associated with DCM dogs (Justin *et al.* 2007). A chronic decrease in cardiac output leads to a reduction in blood pressure, activating compensatory mechanisms such as the renin-angiotensin system (RAS) and the sympathetic nervous system, which attempt to restore it (Snyder 1991, Knight 1992). Compensatory mechanisms, when acting chronically, contribute to the evolution of clinical signs observed in patients with cardiac failure. Sodium and water retention results in congestion, ascites, pleural effusion, pulmonary and limb edema. Other signs include fatigue, exercise intolerance, shortness of breath, tachycardia, the presence of a gallop rhythm, murmurs, and arrhythmias (Calvert 1991, Roth 1993, Camacho 1996, De Morais 2000). The compensatory mechanisms activated during CHF are responsible for the progression of the primary disease (Calvert 1991, Roth 1993, De Morais 2000). Survival time in dogs with CHF due to DCM, increased after the introduction of the ACE inhibitor (enalapril) (Ettinger *et al.* 1998). These drug decreases peripheral vasoconstriction caused by angiotensin II and inhibits sodium and water reabsorption stimulated by aldosterone production and release (Insel *et al.* 1989, Sisson 1991, Roth 1993). Other ACE inhibitors

have a similar pharmacological profile and can be used to treat CHF in dogs (Hamlin and Nakayama, 1998). Benazepril works for 24 hours and can be administrated once a day (king *et al*. 1995). It is excreted both renally and hepatically, requiring no dose adjustment in patients with renal failure (Kitagawa *et al.* 2000).

Keeping all the aspects in view and paucity of diagnostic and therapeutic evaluation of cardiac diseases in geriatric dogs, the study was planned to evaluate the clinical use of two combinations of medication with moderate to severe CHF, analysing the physical, serum biochemical, radiographic, and electrocardiographic characteristics and blood pressure values and prescribe package of practice for congestive heart failure in geriatric dogs.

The work was planned with following objectives:

- To evaluate the cardiac disease in geriatric dog with ECG and radiography.
- To study the endogenous cardiac marker in congestive heart failure in dogs.
- To evaluate the therapeutic efficacy of positive inotropic, diuretics and ACE inhibitors in congestive heart failure in dogs by ECG, radiographic and endogenous markers.

Review of Literature

Prevalence of cardiac disease in canine-

- Urfer *et al.* (2017) mentioned that age is greatest risk factor for heart disease in both people and dogs.
- Thirunavukkarasu (2019) reported Labrador and Spitz were more affected with cardiac diseases due to dilated cardiomyopathy (DCM) and mitral valve diseases (MVD) respectively and also older male dogs were found affected with Acquired Heart Diseases (AHDs).
- Macpete (2018) estimated that approximately 10-15% of all dogs are affected with heart diseases.
- Atkinson *et al*. (2009) reported 13.3% to 30% of cardiac diseases in geriatric dogs.
- Eichelberg and Seine **(**1996) stated cardiac diseases are the second most prevalent cause of death in the dog, accounting for a percentage of 16.3%.
- Rush (2002) estimated that approximately 10-15% of all dogs are affected with heart diseases.
- Rebecca (2008) found that males are more susceptible to certain cardiac diseases (e.g., male cocker spaniels to endocardiosis of the mitral valve, and large-breed males to dilated cardiomyopathy. sick sinus syndrome occurs in the female miniature schnauzer and PDA is more common in females than in males.
- Noszczyl-Nowak *et al.* (2017) found that pathological arrhythmias were generally found in male dogs and in German shepherds.
- Reetu *et al.* (2017) stated maximum incidence of cardiac disease was seen in Labrador retrievers and in male dogs. Geriatric dogs were found to be the most affected.
- Detweiler *et al.* (1968) noted that the incidence of the disease increased with progressing age so that approximately 10 per cent of 5- to 8-year-old dogs, 20 to 25 per cent of 9- to 12-year-old dogs and 30 to 35 per cent of dogs over 13 years of age shown murmurs.
- Detweiler (1964) found that incidence of congestive heart failure was six times higher in males as compared to females.
- Atkins *et al*. (2009) stated cardiac diseases in case of male dogs were 1.5 times more common than in females.
- Rao *et al*. (2008) suggested heart failure has been one of the commonest causes of sudden death in Indian dogs.

- Detweiler *et al*. (1961) stated it was found that occurrence of spontaneous heart disease in dogs used to be higher than generally known.
- Martin *et al.* (2009) observed several times the conditions are acknowledged too late until pronounced signs appear.

Clinical findings:

- Calvert *et al*. (1997) reported a history of syncope in the midst of 1 day and 6 weeks prior to the death of 46 Doberman dogs that die suddenly without progressing to CHF.
- Mc Ewan (2000) listed cold extremities, weak femoral pulse, pallor of the mucosa, exercise intolerance, syncope, cachexia, coughing, dyspnoea, ascites in dog suffering from CHF.
- Fascetti *et al*. (2003) reported that lethargy and anorexia were the commonest clinical abnormalities, and other clinical signs include cough, dyspnoea, weight loss, trembling, and collapse in dogs with CHF.
- Boujon and Amberger (2003) stated the clinical signs of CHF in a Boxer dog between 21-40 months. The dog was asymptomatic, developed clinical signs of exercise intolerance with syncope at 41 months.
- Tidholm (2006) stated that the clinical signs were labored breathing at rest, cough, exercise intolerance, inappetence, polydipsia, weight loss, and syncope in dog with CHF.
- Jeyaraja *et al*. (2008) witnessed cough and exercise intolerance inappetence depression weight loss and syncope in dogs with CHF.
- Martin *et al.* (2010) observed poor appetite, breathlessness, collapse, cough, dullness, exercise intolerance in dogs which were affected with CHF.
- Anju *et al.* (2011) listed exercise intolerance, dyspnoea, weight loss, abdominal distension, edema of hind limbs, and scrotum and fainting episodes in dog with CHF.
- Haggstrom (1998) found that the dogs suffering from mitral valve disease might be asymptomatic in the prodromal stage that was followed by pulmonary edema and ascites.
- Swenson *et al*. (1996) describe mitral regurgitation, probably the most prominent clinical finding was a systolic heart murmur. This heart sound originates from blood turbulence and associated vibrations that develop when ventricles expel blood into the atrium.
- Komitor (1976) witnessed that dogs with mitral valve disease were restless and unable to sleep exhibited by audible wheezing.
- Buchanan (1978) stated that generalized muscle weakness, exercise intolerance, syncope, and ascites were detected in dogs affected with mitral regurgitation.

- Snyder (1991) and Knight (1992) Stated that a chronic reduce in cardiac output results in a decrease in blood pressure, activating compensatory mechanisms such as the renin-angiotensin system (RAS) and the sympathetic nervous system, and that attempt to restore it.

Pathophysiology:

- Jan *et al.* (2018) reported an elevated level of creatinine up to 3.01 mg/dl and mean BUN 29.58 mg/dl in heart failure cases.
- Deferrari (2020) reported there is a co-existence of renal and cardiac dysfunction, where dysfunction in one organ induces acute or chronic dysfunction in the other.
- Alexa *et al.* (2014) reported hypoalbuminemia in elderly human patients with congestive heart failure which may be due to increased volume distribution, stasis in circulation and altered protein metabolism in the liver.
- Lourenco *et al.* (2009), Zamora *et al.* (2012) and Adlbrecht *et al*. (2008) stated cachexia, malnutrition, diffuse inflammation, increased urinary loss, plasma volume expansion may be the cause of hypoalbuminemia in case of CHF.
- Knight (1995) stated that a chronic reduce in cardiac output results in a decrease in blood pressure, activating compensatory mechanisms such as the renin-angiotensin system (RAS) and the sympathetic nervous system, and that attempt to restore it.
- Awan and Mason (1996) mentioned that the mechanisms primarily cause tachycardia, peripheral vasoconstriction, and sodium and water retention by increasing blood pressure and cardiac output, which favors tissue perfusion.
- De Morais (2000) mentioned that the compensatory mechanisms, when acting chronically, contribute to the evolution of clinical signs observed in patients with heart failure. Sodium and water retention lead to congestion, ascites, pleural effusion, and pulmonary and limb edema. Other signs include tiredness, exercise intolerance, dyspnea, tachycardia, presence of a gallop rhythm, murmurs, and arrhythmias
- De Morais (2000) mentioned that the compensatory mechanisms activated during CHF are primarily responsible for the progression of the primary disease
- De Morais (2000) mentioned that vasoconstriction, tachycardia and increased contractility intensify myocardial oxygen consumption (MVO2), decreasing the life of your cells. Neuroendocrine activation (renin-angiotensin and noradrenaline system) interferes with intracellular protein synthesis, causing hypertrophy and cell death, which increases the workload, accelerating the death of remaining cells

Etiology:

- Macpete (2018) mentioned heartworm disease reasons 13% of cardiac disease although it is entirely preventable.
- Aranda *et al*. (1998) mentioned (CHW), caused by *Dirofilaria immitis*, and transmitted by Aedes spp, Culex spp, and Anopheles spp of mosquitoes. Clinical signs of CHW disease depend upon severity and duration of infection and are mainly due to the effects of the adult nematodes on the lungs, the right ventricle of the hearts, and pulmonary arteries of infected dogs.
- Bowman and Atkins (2009) mentioned clinical signs of CHW include weight loss, exercise intolerance, cough, lethargy, dyspnea, and syncope.
- Yuasa *et al*. (2012) stated CHW disease has a global occurrence with greater prevalence in the tropic and subtropical climates and its spatial distribution patterns correlate with its vectors.
- Chandrashekar *et al*. (2010) stated the Snap 4Dx has been validated extensively for simultaneous detection of the antibody of *Anaplasma phagocytophilum*, *E. canis*, *E. ewingii*, *Borrelia burgdorferi* and *D. immitis* antigen.
- Liu *et al.* (2018) found that Snap 4DX was found to be 98% sensitive in detecting vector-borne disease in canines. *E. canis* infection in the present study would be responsible for cardiac problems in geriatric dogs.
- Diniz *et al.* (2008) stated *E. Canis* infection was a risk factor for cardiac injury associated with severe anemia.

Diagnosis:

- Detweiler *et al*. (1961) in a study on spontaneous occurrence of cardiovascular diseases on dogs show cardiac diseases in 10 percent of the dogs screened by electrocardiography, radiography and necropsy.
- Vengsarkar (1988) stated that it is very important that every dog considered susceptible to cardiac diseases should be examined for cardiac function during a routine examination.
- Devi *et al.* (2009) observed that the recognition of canine cardiac diseases has been delayed, and neglected on account of lack of awareness and knowledge by the owner and inadequate diagnostic facility in our country.

- Detweiler *et al.* (1961) mentioned that auscultation, electrocardiography, and roentgenography were the three most valuable diagnostic procedures utilized in recognizing heart diseases in dogs.
- Roth (1993) and Schlesinger and Rubin (1994) stated Congestive heart failure (CHF) is a clinical syndrome, caused in dogs mainly by myocardial or valvular changes.
- Haggstrom *et al*. (1995) stated that there was the association between the auscultatory intensity of cardiac murmurs and the severity of mitral regurgitation. Hence dogs might be screened for development and severity of chronic valvular disease by the use of cardiac auscultation.
- Ettinger and Feldman (2005) reported elevation of serum creatinine in geriatric dogs with congestive heart failure may be due to poor perfusion, arterial stenosis which reduces glomerular filtration, and thereby leading to accumulation of nitrogenous waste creatinine.

Radiography-

- Greco *et al.* (2008) concluded that among different diagnostic techniques, like electrocardiography or ultrasound, x-ray continues to be one of the most used equipment in assessing the cardiac changes in small animals.
- Baisan *et al*. (2016) mentioned that radiological examination of the cardiac silhouette is favored by the contrast with the lungs, latter showing a better radiolucency because of the amount of air within the alveolae enclosing the heart.
- Baisan *et al*. (2017) stated that radiographic interpretation can be done in various ways, subjectively, by gross examination or by using different measurements like cardiothoracic ratio, intercostal spaces or vertebral heart score. considerable differences in the cardiac shape can be found in diseases such as dilated cardiomyopathy or mitral valve disease.
- Baisan *et al*. (2017) mentioned that radiographic diagnosis of canine cardiac disease depends on recognition of signs, in addition to abnormal size or shape of the pulmonary vessels, presence of pulmonary edema and ascites. in dogs, the radiographic examination of cardiac diseases is concentrated on the evaluation of cardiac silhouette- in size and shape

- Buchanan (2000) mentioned that the vertebral heart size (VHS) method decides the cardiac dimensions relatives to length of thoracic vertebrae, using right lateral (RL), left lateral (LL) and ventrodorsal (VD) radiographic projections.
- Bonagura and Pipers (1983) reported cardiomegaly, pulmonary edema and aortic arch prominences were seen in thoracic radiography of dogs with aortic stenosis.
- Gooding *et al.* (1982) distinguished between concentric hypertrophy and excentric ventricular hypertrophy based on radiographical results in dogs that showed both electrocardiographic and radiographic evidence of ventricular enlargement.
- Tidholm and Johnsson (1997) in a retrospective study of DCM in dogs (189 cases) stated cardiomegaly (94%), pulmonary edema (86%), and pleural effusion (11%) on thoracic radiography.
- Dunn *et al*. (1999) observed that the DCM in dogs was normally characterized by generalized cardiomegaly although in some breeds (Doberman pinscher and Boxer) thoracic radiographs often seemed remarkably normal or with mild left atrial and left. Ventricular enlargement.
- Dukes-McEwan *et al*. (2003) reported the radiographic findings on thoracic radiography of dogs with DCM as cardiomegaly with the prominence of the left atrium, venous congestion, various degrees of interstitial or alveolar pulmonary edema, and sometimes pleural effusion and ascites.
- Jeyaraja *et al*. (2008) reported in a study of 21 dogs with DCM that radiographical proof of cardiomegaly was found in 94% of cases and out of that 28% cases showed the prominence of the left atrium.
- Martin *et al.* (2009) reported that cardiomegaly was evident in 289 out of 369 cases (80%) and pulmonary edema or pleural effusion was seen in 267 cases (74%) of dogs.
- Lefbon (2005) found on dorsoventral view, the left heart border normally became more convex and advanced toward the left chest wall, decreasing the space between the chest wall and the cardiac silhouette in dogs with mitral regurgitation. On the lateral view, the caudal cardiac border became more rounded or straighter than normal.
- Lombard and Spencer (1985) mentioned that the decisions of initiating cardiac treatment based on a radiographic diagnosis of left atrial enlargement should be made only after sufficient proof of enlargement was obtained, best by serial evaluation showing an increase of the size within a short period of time.

- Bonagura and Pipers (1983) described in dogs with aortic stenosis, cardiomegaly, pulmonary edema, and aortic arch prominences were in thoracic radiography and also mentioned that echocardiographically normal size of the left atrium was frequently diagnosed as enlarged in radiographs, such error was due to shape changes of the left atrium; The radiographic criteria for left atrial enlargement could be too sensitive resulting in false-positive diagnoses.
- Owens and Biery (1999) recommended radiographic rules of thumb such as normal cardiac silhouette in the dog usually ranged from 2.5 to 3.5 times the width of the intercostal spaces.
- Buchanan and Bucheler (1995) recommended the vertebral scale system to measure heart size in radiographs.
- Darke *et al.* (1996) reported the radiographic differences noticed in dogs with mitral regurgitation included well-marked left atrial enlargement, pulmonary congestion, enlargement of the caudal vena cava, and hepatic enlargement
- Kittleson (1998) found that in dogs left atrial enlargement was most reported with congenital or acquired mitral regurgitation. In severe left atrial enlargement, the large amount of blood in the left atrium resulted in the left atrium appears denser than the rest of the cardiac silhouette and appeared as a round radio dense region between the two mainstem bronchi.
- Kvart and Haggstrom (2000) reported chronic mitral valve insufficiency in dogs, the key structures to assess were left atrium, left ventricle, main stem bronchi, lung field, and pulmonary vessels. Dogs with a low degree of chronic mitral valve insufficiency normally had a normal heart size, normal lung field, and normal pulmonary vasculature, left atrial enlargement was one of the earliest and most consistent radiographic features of chronic mitral valve insufficiency, with progression, the left atrium and the left ventricle continued to enlarge.

Electrocardiography:

- Bolton (1975) mentioned electrocardiography is a non-invasive, within your means, easy to use diagnostic technique and is accepted as a mandatory part of special cardiac examination in small animals. Electrocardiography is a well-recognized useful diagnostic aid to identify cardiac arrhythmias, cardiac chamber enlargements, myocardial diseases, evaluation of drug therapy, evaluation or progression of cardiac disease and its prognosis.

- Kumar *et al*. (2003) stated ECG helps to diagnose heart failure, conduction defects, heart block, and pericardial effusions.
- Ettinger (1983) stated no hospital screening is as simple and rapid as electrocardiography which is easy in observation of treatment.
- Tilley (2008) mentioned that electrocardiography can serve two purposes that is diagnosing most cardiac arrhythmias and providing information on the status of the myocardium, since the P-Q R S-T deflection of the electrocardiographic tracing is often altered by either pathological or physiological reasons.
- Mukherjee *et al.* (2020) described electrocardiography (ECG), a non-invasive and inexpensive technique, is widely employed for the determination of heart rate, heart rhythm, conduction integrity, and mean electrical axis together with myocardial and pericardial affections together with tracking of certain non-cardiac anomalies like electrolyte imbalance, drug toxicity, and hormonal imbalances.
- Bonagura and Pipers (1983) stated sinus rhythm with first-degree atrioventricular block and variable periods of second-degree av block were detected in boxer with aortic stenosis.
- Sisson (1987) stated QRS complexes of abnormally high amplitude and left ventricular enlargement in dogs with subaortic stenosis.
- Lombard and Spencer (1985) in the study on the correlation of radiographic, echocardiographic, and electrocardiographic features of left heart enlargement in dogs with mitral regurgitation witnessed that left ventricular hypertrophy patterns of the electrocardiography did not correlate either with a radiographic diagnosis of left ventricular enlargement or with echocardiographic enlargement ratios. The occurrence of P-mitrale was 30 percent but this electrocardiographic abnormal condition when present, reliably acknowledged enlarged left atrial dimension.
- Tilley (1992) described that electrocardiogram had low sensitivity in detecting right atrial and right ventricular enlargement secondary to primary tricuspid valve insufficiency. Insignificant tricuspid valve insufficiency with pulmonary hypertension, the electrocardiographic changes might include evidence of right atrial enlargement and right ventricular enlargement.
- Haggstrom *et al.* (1992) found that the presence of sinus arrhythmia in advanced chronic mitral valve insufficiency indicated that heart failure was absent. Rapid and irregular heart rate was indicative of arrhythmia. The most common form of ectopy was atrial

premature contractions. In progressed chronic mitral valve insufficiency. Supraventricular tachycardia, atrial fibrillation, and ventricular premature contractions can be noticed.

- Darke *et al.* (1996) stated that electrocardiographic anomalies are associated with mitral valve diseases. The changes include widened P and R waves with increased amplitude, atrial premature complexes, and atrial fibrillation.
- Kittleson (1998) stated that the electrocardiographic findings in chronic mitral valve disease varied from normal tracings to well-marked abnormalities in rate, rhythm, and configuration of complexes. With the exception of documenting and classifying certain arrhythmias, the electrocardiogram was of restricted use in the diagnosis or management of chronic mitral valve disease
- Martin (2003) said prolonged and notched p wave with left atrial enlargement in dogs with mitral valve disease.
- Brown (2004) acknowledged sinus rhythm, prolonged P and QRS complexes as electrical conduction abnormalities in a dog with aortic endocarditis.

Cardiac Troponin:

- Collinson *et al*. (2001) Studies in laboratory animals and human clinical trials had found out that cardiac troponin proteins were the most specific and sensitive indicators of myocardial cell damage and that there was good correlation between myocyte damage and blood troponin levels.
- Oyama and Sisson (2004) reported that cardiac troponin-I was increased in dogs with cardiomyopathy, degenerative mitral valve disease, sub valvular aortic stenosis.
- Burgener *et al.* (2006) reported acute myocardial damage was observed in conditions like gastric dilatation and volvulus and blunt chest trauma. cardiac troponins increase compared to the normal values in such cases and cardiac troponin I appeared to be more sensitive in myocardial cell injury than cardiac troponin T.
- Adams *et al.* (1994) in a comparative study of muscle brain isoenzymes of creatine kinase and cardiac troponin-I, it was found that cardiac troponin I was more specific for damaged heart muscle.
- Schober *et al.* (1999) cardiac troponin I concentration was reported to be better correlated with myocardial damage than cardiac troponin T (cTnT) or creatinine kinase muscle brain protein (CK-MB)

- Sleeper *et al.* (2001) discovered preliminary normal levels of plasma of cTnI in healthy dogs and cats with myocardial damage or disease. the range of plasma cTnI concentration in dogs was 0.03-0.07 ng/ml with a mean of 0.02 ng/ml.
- De Francesco *et al.* (2002) in their study stated that dogs with asymptomatic dilated cardiomyopathy (DCM) had cTnI concentration below the level of detectability while in dogs with CHF secondary to valvular disease or DCM cTnI levels were raised compared with controls.
- Lobetti *et al.* (2002) and Schober *et al.* (2002) reported a limited number of clinical reports on cardiac troponins in dogs had been published in recent years.
- Spratt *et al.* (2005) stated that none had examined the connection between circulating troponin concentration and severity of heart failure.

Creatinine Kinase:

- Thrall *et al.* (2004) in their study in the cytoplasm of the muscle cells creatinine kinase freely found which is a muscle-specific leakage enzyme with three major isoenzymes: CK-MM (muscle type) CK-MB (myocardial type) CK-BB (brain type) isoenzymes.
- Schober *et al*. (1999) Stated there was a significant difference in the concentration of CK-MB, between normal and diseased myocardium depending on the disease state, patient's age, chronicity of myocardial stress, severity and duration of ischemia marked up-regulation of CK-MB synthesis in cardiac myocytes might be seen after myocardial injury despite a decrease in the absolute creatine kinase concentration. Such dynamic CK-MB alterations might cause a significant elevation of the relative increase of CK-MB in the ventricular myocardium within several weeks of the onset of disease.

Treatment:

- Hoque *et al.* (2019) stated that even though most forms of cardiac disease cannot be prevented, a cardiac disease caused by heartworms can be competently avoided with preventative medical therapy.
- Blackford *et al*. (1990), Holtz *et al*. (1990) and Hamlin (1996) *et al*. found in both experimentally induced and naturally occurring heart failure clinical trials of ACE inhibitors have been reported to improve hemodynamic parameters, clinical signs, and exercise tolerance in dogs.

- Webb *et al.* (1990) stated hereafter benazepril hydrochloride, described as benazepril, is a carboxylic acid ACE inhibitor.
- Colfer *et al*. (1992) describes benazepril has been proven to produce favorable effect on hemodynamic parameters, clinical symptoms, exercise tolerance, and survival times in human patients with congestive heart failure.
- King *et al.* (1995) stated benazepril has been proven to potently inhibit plasma ACE activity in dogs in vivo and to improve clinical signs of dogs with heart failure whenever used as monotherapy.
- Bowles and Fry (2011) the properties of Pimobendan alone or in combination with ß blockers and drugs like spironolactone having neuro-hormonal effects, improved quality of life and reduced heart insufficiency scores for patients with CHF due to dilated cardiomyopathy
- Todd and Benfield (1990) studied and found that ramipril improves cardiac function, metabolism, and exerts beneficial hemodynamic adjustments (improved preload and afterload) in acute ischemic left ventricular heart failure. Beneficial acute hemodynamic effects were detected after oral administration of ramipril in patients with congestive heart failure. There was a reduction in peripheral resistance as well as mean arterial pressure, associated with a reduction in filling pressure and accompanied by increased cardiac output. There was just a slight effect on heart rate. Severe first-dose hypotension would possibly occur with high doses.
- King *et al.* (1995) mentioned that benazepril works for 24 hours and can be administered once a day.
- Kitagawa *et al.* (2000) described that benazepril excreted both renally and hepatically, requiring no dose adjustment in patients with renal failure.
- Katz (1994) mentioned that the pharmacological blockade of ACE or their metabolic pathways prolongs the survival of patients with CHF.

Pimobendan:

- Takahashi and Endoh (2001) stated pimobendane is a benzimidazole derivative along with combined inotropic and peripheral vasodilating characteristics.
- Fitton and Brogden (1994) reported benzimidazole derivatives perform inotropic effects through a combination of inhibiting phosphodiesterase (PDE) iii and sensitizing cardiac myofilaments to intracellular calcium, and for this reason are generally termed calcium sensitizing agents

- Kanno *et al.* (2007) mentioned that pimobendane has a dual mechanism of action: it increases myocardial contractility through increasing calcium sensitization to troponin c and it encourages vasodilation by inhibiting PDE iii.
- Boswood (2010) stated that pimobendane is a drug with both inotropic and vasodilatory properties and is widely used for the treatment of heart failure in dogs. the best evidence regarding its efficacy is derived from various clinical studies in dogs with the two most common conditions that result in heart failure: dilated cardiomyopathy (DCM) and degenerative mitral valve disease (DMVD).
- Kanno *et al.* (2007) stated that pimobendane (Vet medicine) comes in 1.25mg and 5.0mg chewable tablets, pimobendane is the first drug of a new class of heart medicines called inodilators. Research have shown that when used with other cardiac medicines, pimobendane can be effective for dogs with congestive heart failure associated with either dilated cardiomyopathy (DCM) or degenerative mitral valve disease (MVD). This drug varies from other heart medicines because it supports the heart pump more efficiently. It opens up the blood vessels that take blood both to and from the heart so the heart doesn't need to work as hard. It also facilitates cut down pressure on the heart.
- Thomason *et al.* (2007) stated that pimobendane is safe and effective in dogs suffering from mitral valve disease and dilated cardiomyopathy at a dosage of 0.3mg/kg given orally every 12 hours. a clinical trial confirmed that Doberman pinschers with dilated cardiomyopathy treated with pimobendane, digoxin, enalapril, and furosemide survived significantly longer (median survival time 329 days) than those treated with digoxin, enalapril, and furosemide alone (median survival time 50 days).
- Ouellet *et al*. (2009) stated that a significant difference for the ejection fraction (EF) calculated with Sampson's rule, at day 30, with an EF significantly greater in PIMO group (80.8+14.1%, P-0.0064) compared with c group (69.0-2.75%).
- Chu *et al*. (1995) and Lefbon (2005) reported pimobendane, is a benzimidazole pyridazinone drug, was classified as an inodilator because of its non-sympathomimetic, non-glycoside positive inotropic and vasodilator characteristics.
- Ruegg (1986) reported ventricular tachyarrhythmias are concern in Dobermann pinschers and boxers but could occur in any dog with advanced dilated cardiomyopathy. Pimobendane’s effect on myocytes was conserved energy demand with small increases in intracellular calcium concentration which might reduce the chance of a proarrhythmic effect.

- Fujino *et al*. (1988) stated that pimobendane was a positive inotrope improving the affinity of myocardial troponin c to existing intracellular calcium. The result was better contractility without any increased myocardial oxygen or energy needs.
- Lubsen *et al*. (1996) mentioned that pimobendane resulted in a dose dependent increase in sinus rate in normal dog but had not more marked effect on heart rate in individual with heart failure. The electrocardiographic effect of pimobendane included enhanced atrioventricular conduction, a shortening of atrial, atrioventricular nodal and ventricular refractory periods.
- Iwasaki *et al*. (1999) reported pimobendane was a phosphodiesterase iii inhibitor resulting in balanced peripheral vasodilation thru exaggerated reflux of intracellular calcium from vascular smooth muscle. Additional properties of pimobendane included reversal of the sensitization of baroceptors, better cardiac relaxation, minimized platelet aggregation and anti-inflammatory effect.
- Iwasaki *et al*. (1999) stated pimobendan appeared to be well recognized in animals. Therapeutic doses in humans with chronic heart failure and preliminary indications in dogs suggested that in addition to contributing to a consistent improvement in exercise quality and quality of life, it was largely devoid of the proarrhythmic effects of phosphodiesterase iii inhibitors
- Kleemann *et al*. (1998) stated that Pimobendan is safe and effective in dogs having mitral valve disease and dilated cardiomyopathy at a dose rate of 0.3 mg/kg given orally every 12 hours.
- Fuentes (2004) stated pimobendane is indicated in dogs to treat congestive heart failure as a result of dilated cardiomyopathy or valvular insufficiency. Treatment was given in symptomatic patients that might have the benefit of positive inotropic action. The dosage range was 0.2 to 0.6 mg/kg daily divided into two doses given 12 hours apart.
- Smith *et al.* (2005) in Six-month trials demonstrated that dogs getting pimobendane and furosemide had lower negative outcomes (euthanasia, death, or drug withdrawal due to worsening of congestive heart failure) than dogs dosed with an ace inhibitor (ramipril) and furosemide.
- Chu *et al*. (1995) described pimobendane hadn't been verified in pregnant and lactating dogs. Pimobendane could be given to pregnant or lactating dogs only if the potential benefits outweigh the potential dangers. Although negative effects were uncommon, polyuria, polydipsia, vomiting, diarrhea and inappetence were possible.

- Rosenthal *et al*. (2006) mentioned that a dose-related sinus tachycardia could result, and as with any strong inotropic agent, ventricular tachyarrhythmias might develop or get worse while pimobendane is given.

- Thomason *et al.* (2007) stated that dogs with mitral valve disease pimobendane was recommended when overt congestive heart failure occurred in the face of acetyl cholinesterase inhibitor, spironolactone and amlodipine treatment.

- Haggstrom *et al.* (2008) stated that pimobendane is a new drug with phosphodiesterase-inhibiting and calcium-sensitizing effects and that increase myocardial contractility, promote arterial and venous dilatation and is valuable for the treatment of congestive heart failure in dogs.

- Ettinger *et al.* (1998) stated that suppression of the renin-angiotensin-aldosterone system (RAAS) with angiotensin-converting enzyme (ACE) inhibitors when added to other pharmacological treatments improves clinical status and prolongs life of dogs with congestive heart failure (CHF).

- Summerfield *et al.* (2012) mentioned that pimobendane is a benzimidazopyridazinone having a strong positive inotropic and a vasodilatory outcome. this combined effect of preload and afterload reduction, together with positive inotropic effect, could result in a reduction in cardiac size and filling pressures in Dobermans with preclinical DCM. such beneficial effects could result in lengthening of the preclinical stage of DCM.

Furosemide

- Atkins (2002) reported furosemide is a loop diuretic and that obstructs sodium and chloride reabsorption by the kidneys. With ace inhibitors, the oral diuretic dosage generally reduced by 50 per cent (1-2mg/kg body weight, twice daily) in treatment of DCM in dogs.
- Sisson *et al*. (2000) witnessed the most common side effects of furosemide in the treatment of DCM dogs had been RAAS activation, dehydration, excessive decrease of preload, decreased cardiac output, azotemia hypokalemia and hyponatremia.
- Tidholm (2006) reported that the average survival time in dogs treated with digoxin, furosemide along with propranolol was 126 days, with a survival rate at 1 year as 34 percent.

- Martin *et al.* (2010) in a retrospective study of DCM cases found that furosemide had slight signification in bettering survival in comparison to other drugs like enalapril and pimobendan.
- Ettinger *et al*. (1998) and CONSENSUS study, (1987) treatment with angiotensin conversion enzyme (ACE) inhibitors reduces angiotensin II and aldosterone production and has been shown to be of great clinical benefit in the treatment of heart failure in humans

Materials and Methods

Location of study:

The present study was conducted on clinical cases reported at the outpatient of Veterinary Medicine, Veterinary Clinical Complex of Bihar Veterinary College Patna.

Selection of cases:

(a) Duration of work: The duration of work will be 6 months from May to October.

(b) Diseased animals: 50 Geriatric dogs in the age group of 7and above showing any cardiopulmonary symptoms were screened for our study.

(c) Healthy Animals: 10 apparently healthy Medium sized middle-aged (7 to 9 years) dogs irrespective of sex and breeds were selected. These animals served as healthy control.

Parameters studied:

- **Detailed clinical examination:**

General and system-wise examinations were done as per guidelines given by Radostits *et al*. (2000). The general description of all the geriatric dogs presented to Veterinary Clinical Complex were recorded, which includes case no, owners name, pets name, address, date, breed, age, sex and body weight. Details are given in Table:2.

- **Anamnesis:**

The complete history of all the cases under study was collected from the animal owner. Which included the history of other diseases, duration of illness, chief complaint, feeding schedule, types of feed, history of vaccination and deworming. Results given in Table:3.

- **Clinical examination:**

Clinical examination was done as suggested by Kenney *et al*. (2010) and Radostits *et al*. (2000), which includes general examination and mucous membrane examination. Result given in Table:4 and 5.

- **Special Examination:**

Special examination of the respiratory and cardiovascular system was done as result given in Table no- 6 and 7.

- **Electrocardiographic findings:**

The electrocardiographic examination was done per the standard procedure described by Tilley and Smith (1997). Lead II was used to interpret electrocardiogram variables. All recordings of ECG were done at the paper speed of 25mm/sec. and amplitude 1mv(10mm) as per the method described by Tilley (2008).

- **Blood Pressure Measurement**:

Systolic arterial pressure (SAP), and Diastolic arterial pressure (DAP) were recorded as per Knaus and Wagner (1989).

The instrument used for the present study was the human wrist model automatic Oscillometeric sphygmomanometer.

1) Principle

Oscillometeric sphygmomanometer is based on detection of changes in oscillations produced by changes in arterial wall diameter. The cuff is connected to an oscillometric monitor and automatic measurement of SAP, DAP and pulse could be obtained at one-minute cycles. The cuff is automatically inflated to supra systolic pressure and then deflected in an incremental fashion. A microprocessor measures oscillation in the arterial wall. Systolic and diastolic pressure at which oscillation amplitude rapidly increases (Carr *et al.* 2000).

2) Position of the animal

The cuff is conveniently placed in the left forearm region of the animal. The transducer in the cuff is positioned on the medial aspect of the arm, over the median artery and the Velcro is wrapped around the foreleg.

3) Measuring the blood pressure

The start button of the instrument was pressed. The cuff was automatically inflated above the SAP and gradually deflated below the DAP. The values got displayed on the screen. Five reading were taken in each animal and average of the five readings was taken as the blood pressure value. Out-ranging values were neglected and further readings were taken.

- **Snap 4dx Test**:

This is a kit-based test used for the diagnosis of *Dirofilaria immitis* (Heartworm), *Ehrlichia canis*, *Ehrlichia ewingii*, *Borrelia burgdorferi*, and Anaplasmosis in canine. This test helped in the differential diagnosis of heartworm disease in canines. The kit was procured from IDEXX, USA.

- **Hematology:**

Two millilitres of blood was collected in a vacutainer containing EDTA as an anticoagulant to estimate Hb, PCV and Platelet count.

  - The values of hemoglobin were determined by the cyanmethemoglobin method.
  - Packed cell volume (PCV) by microhematocrit method.
  - Platelet count by hemocytometer.

- **Serum biochemistry:**

About 5.0 ml of blood were collected in a vacutainer without anticoagulant and serum were separated and stored at -20°C for further estimation of below mentioned parameter

  - Creatinine,
  - Blood urea nitrogen,
  - Bilirubin,
  - Total serum proteins,
  - Albumin,
  - Globulin,
  - Sodium (Na),
  - Potassium (K).

- **Serum Creatinine**:

The serum creatinine levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Serum Blood urea nitrogen:**

The serum blood urea nitrogen levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Total Serum Protein:**

  Estimation of total serum protein was carried out by direct biuret method (Doumas and Watson, 1971) using a total protein kit (Coral clinical systems Goa, India). The values were expressed in g/dl.

- **Albumin:**

  Albumin was estimated from serum by bromocresol green methodology using an albumin kit (Coral clinical systems Goa, India). The values were expressed in g/dl.

- **Globulin:**

  Globulin was calculated by subtracting the value of Albumin from the value of total protein and the values were expressed in g/dl.

- **Serum Sodium:**

  The serum sodium levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Serum Potassium:**

  The serum sodium levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Serum Calcium:**

  The serum sodium levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Serum Bilirubin:**

  The serum bilirubin levels were estimated by the procedure given in the leaflet along with the kit supplied by the Coral clinical systems Goa, India. The results were expressed in mg/dl.

- **Cardiac Troponin I:**

  Serum Cardiac Troponin I was estimated qualitatively by immune chromatographic assay with standard troponin -I kit (Supplied by CTK biotech, USA). It is expressed as either positive or negative in the given blood sample.

- **Creatine Kinase MB (CK-MB):**

The Creatine Kinase MB activity was estimated by Immuno-Kinetic Inhibition method given in the leaflet along with the kit supplied by Span Diagnostics Lab. CK-MB activity was expressed in UL.

- **Radiographic Measurements:**

a) Vertebral Heart Score:

Digital right lateral thoracic radiographic were recorded for measuring the VHS. The long axis (L) was measured from the ventral border of the left main stem of branches (carina) to the more distant point of the cardiac apex. The short axis (S) dimension was measured at the level of the caudal vena cava on a line perpendicular to the long axis. The measurements of the two axes were then compared to the vertebrae starting at the cranial edge of T4 Baisan *et al.* (2017).

b) Cardio-thoracic index:

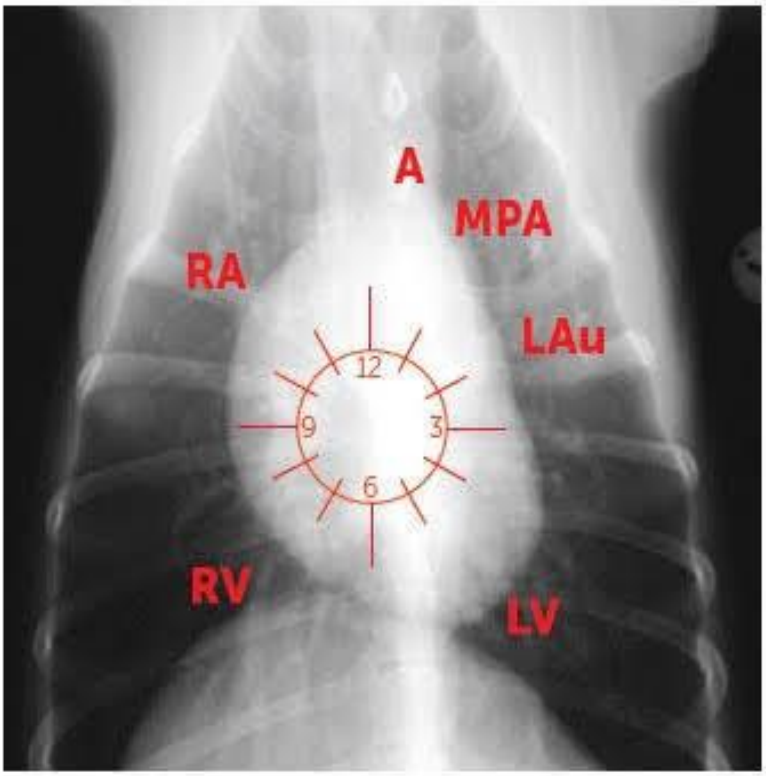
Cardio-thoracic index which is the width of the cardiac-silhouette is useful in measurement of cardiac enlargement. It is measured on the dorso-ventral (D-V) radiography by dividing the maximum diameter of heart and chest. The measurement should be 60-65% of the width of the thorax. Smith *et al*. (2009)

c) Clock face analogue of thoracic radiograph for chamber shape:

The clock face analogy is another method of assessing the cardiac silhouette or specific chamber enlargement. This method will be very useful for non-experienced radiologists and cardiologists. In this method the lateral and dorso - ventral views will be overlapped to the hours of the clock.

1) On the D-V- position the aorta is facing 12’ o clock the main pulmonary artery is located from the 1 to 2 o’clock, the left auricle is located between 2 and 3 O’clock, left ventricle between 3 to 6 O’clock right ventricle from 7 to 9 O’clock and the right atrium from 10 to 11 O’clock. (Figure no.1)
2) On lateral view: the left atrium is located at 1 to 2 O’clock, the left ventricle from 3 to 6 O’clock the rt ventricle from 7 to 8 O’ clock, the rt auricle between 9 and 10 O’clock and from 10 to 11 O’clock the aorta and pulmonary artery (Smith *et al.* 2009). (Figure no.2)

Fig no.1: Clock face analogy on the D-V view



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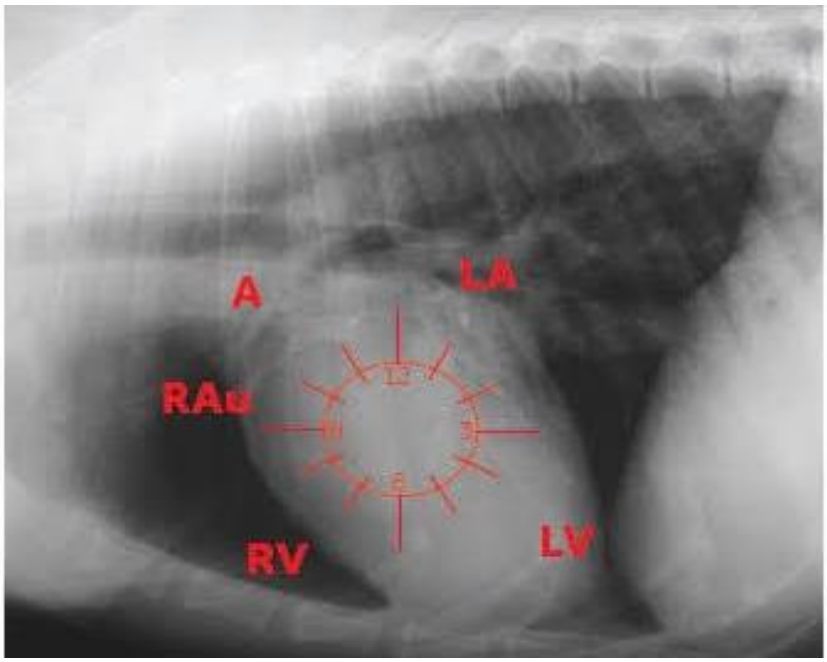


no.2: Clock face analogy on lateral view

- **Therapeutic trial:**

For the therapeutic trial, animals were divided into two clinical groups, each consisting of 10 geriatric dogs suffering from congestive heart failure. 10 dogs were kept as a healthy control for comparison. Two different combination groups of medication were used in each group of animals. The observation was done in 0,14- and 30-days periods. The combination of medicines was used as depicted in Table no.1:

Table 1: Treatment group in geriatric dogs suffering from CHF

Group	(Control)	T1	T2
	No treatment	Pimobenden @0.25mg/kg b wt Ramipril @ 0.125mg/kg b wt Furosemide @ 2-6mg/kg b wt Spironolactone @ 1-2 mg/kg b wt	Digoxin @ 0.125mg/kg b wt Benazepril @ 0.25-0.5mg/Kg b wt Furosemide @ (2-6mg/kg b wt

- Statistical analysis was done as per method described by Snecdecor and Cohran (1994) and with the help of software SPSS 12.

Results

Cardiac diseases in canines are the most important disease but it is least studied in terms of diagnosis and treatment. The present study on "***Radiographic and electrocardiographic changes with special reference to therapeutic evaluation of congestive heart failure in geriatric dogs"*** was conducted in the department of veterinary medicine from the clinical cases presented in the outpatient department of veterinary clinical complex, **Bihar veterinary college**. The research was completed in a 6-month period from February 2021 to September 2021. A total of 50 geriatric dogs (average age above 7) were screened showing any symptoms of cardio-pulmonary diseases. During the period of research from February to September 2021 out of 7812 cases of dogs presented to clinical complex 1925 geriatric dogs were found. Out of 1925 cases of geriatric dogs 519 (27%) dogs were suspected to be found to suffer from cardiac diseases.

The clinical trial study was conducted in three groups with one group kept as healthy control. The results of the study are presented in the tables.

Clinical presentation:

Out of the 50 geriatric dogs presented from February to September 2021, 50 dogs showed signs and symptoms of the cardio-pulmonary disease.

A perusal of table: 2 shows that out of 50 geriatric dogs screened with cardio-pulmonary signs, the breed Labrador was found highest (17) followed by the German shepherd (14). It was found that more than 50% of the dogs were between 7 to 9 years of age (the reason for this may be that a greater number of cases are from this age group) followed by 9 to 11 years of age. It was also found that the male (28) was more susceptible than the female (22) dogs for cardio-pulmonary diseases. (Fig: 3,4,5)

History of other diseases:

Out of 50 dogs screened for cardio-pulmonary signs 7 were presented with no other clinical signs whereas a maximum 23 had anorexia followed by fever (11) diarrhoea (06) and vomiting (03).

Duration of illness:

The history reveals that most of the dogs were brought for treatment after 15 and 30 days of illness. Only a few dogs were brought for early treatment (before 7 days).

Chief complaint:

Several types of complaints were noticed in dogs screened for diagnoses like coughing, nocturnal coughing, exercise intolerance, nasal discharge, weight loss, fainting, and anorexia. Out of this the exercise intolerance (14) and nocturnal coughing (11) were the most important chief complaints in geriatric dogs presented for cardio-pulmonary disease.

Feed and feeding schedule:

It is the important findings and it was found that 17 dogs with cardio-pulmonary signs were on commercial feed while 22 were on mixed (commercial and home food) while the rest 11 were exclusively on homemade food.

General impression:

A perusal of table no. 3 shows different general impressions, out of which some of the impressions are important for the diagnosis of cardiac disorder in dogs.

1. **Consciousness:**

   It was found that dullness was the important impression followed by stupor and coma in dogs.

2. **Behaviour:**

   There are certain behaviour changes that were noticed in dogs suffering from cardio-pulmonary diseases and these were restlessness, anxious and aggressiveness.

3. **Posture:**

   Postural changes are the important findings in dogs with cardio-pulmonary signs and it was found that long-standing (24) and long sitting posture with the inability to lie down or stand were the important postural signs.

4. **Gait:**

   In the screening of dogs for cardiopulmonary diseases, it was found that incoordination (8) and stiffness of gait (9) were the important findings in cardio-pulmonary diseases.

5. **Nutritional condition:**

In nutritional condition and body conditions, it was found that 11 geriatric dogs were found cachectic while 9 dogs were very obese both obesity as well as cachexia were found in dogs suffering from cardiac disease.

6. **Coat:**

Coat conditions were not the significant findings in geriatric dogs with cardio-pulmonary signs however, dullness (13) and alopecia (12) were the important findings.

7. **Body condition score:**

It was observed that the geriatric dogs with cardio-pulmonary dysfunction have lower body condition scores (21).

General examination:

A perusal of table no. 4 in the general examination, both costal (17) and abdominal types of respiration were observed, however, more costal (17) types of respiration were observed. The rhythm of the respiration in 13 dogs was found to be irregular. Heart rhythm in 12 dogs was found to be irregular with a murmur in 11, strong bonding in 08 and 04 dogs were suffering from changes in point of maximum intensity (PMI).

Mucous membrane:

Mucous membrane examination was a significant finding in the evaluation of cardiopulmonary diseases. 2 geriatric dogs were found to be cyanotic with a capillary refill time of more than 3 to 5 seconds. Dryness (17) of mucous membrane with Petechiae (09) was the important finding in screening. Lymph node swelling was found in 21 geriatric dogs with cardiopulmonary signs. (Table: 5)

Respiratory systems:

Epistaxis (6) were found in dogs under screenings while rails (6) whzeels (8) crackling (3) and dullness (4) were found in other dogs. Coughing (17) and respiratory distress were the important findings in dogs with cardiopulmonary signs. (Table: 6)

Cardiovascular examination:

Continuous laboured breathing (9) occasional (7) and laboured breathing during exercise (13) were the important findings in dogs with cardiopulmonary diseases. Nocturnal coughing (14) were represented from the dogs while Ascites (16) were the important finding during the screening. Edema in Limbs was found in 07 dogs and jugular vein pulsation in 14 dogs was present. (Table: 7 and Fig: 6,7)

Table 2: Clinical presentation of geriatric dog suspected for cardiopulmonary diseases

Breed (N=50)	Labrador	17
	German shepherd	14
	Doberman	8
	Belgium shepherd:	7
	Dalmatian	4
Age (in years) (N=50)	7-9	27
	9-11	18
	>11	5
Sex (N=50)	Male	28
	Female	22

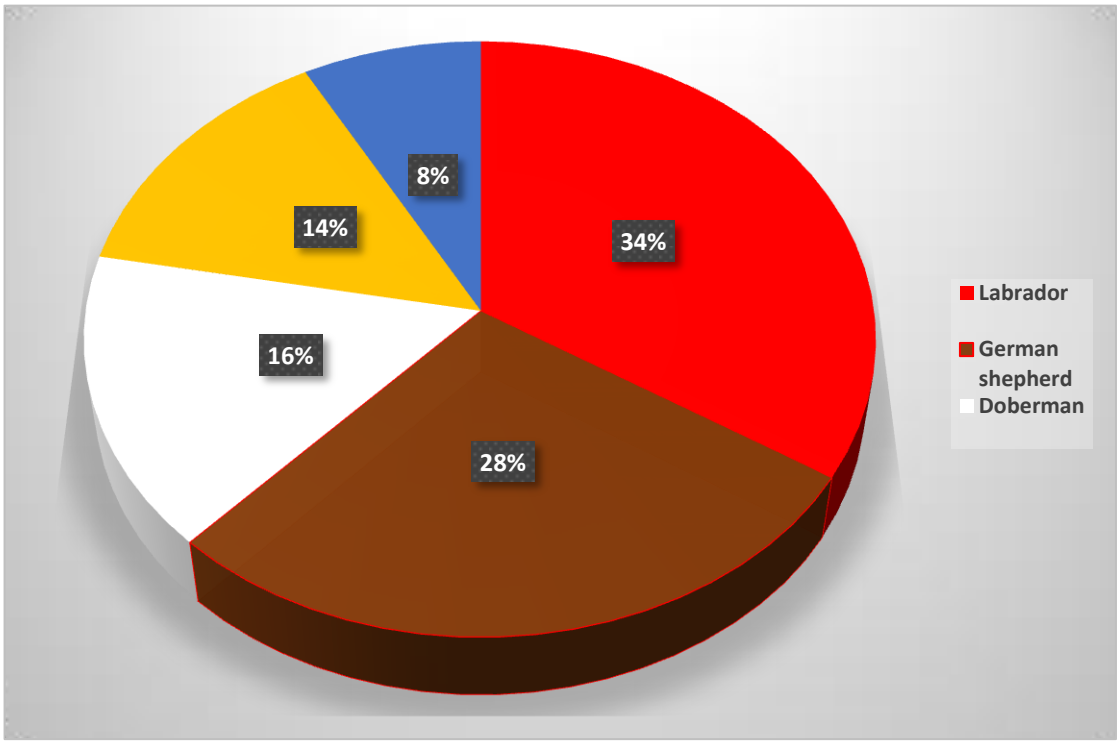
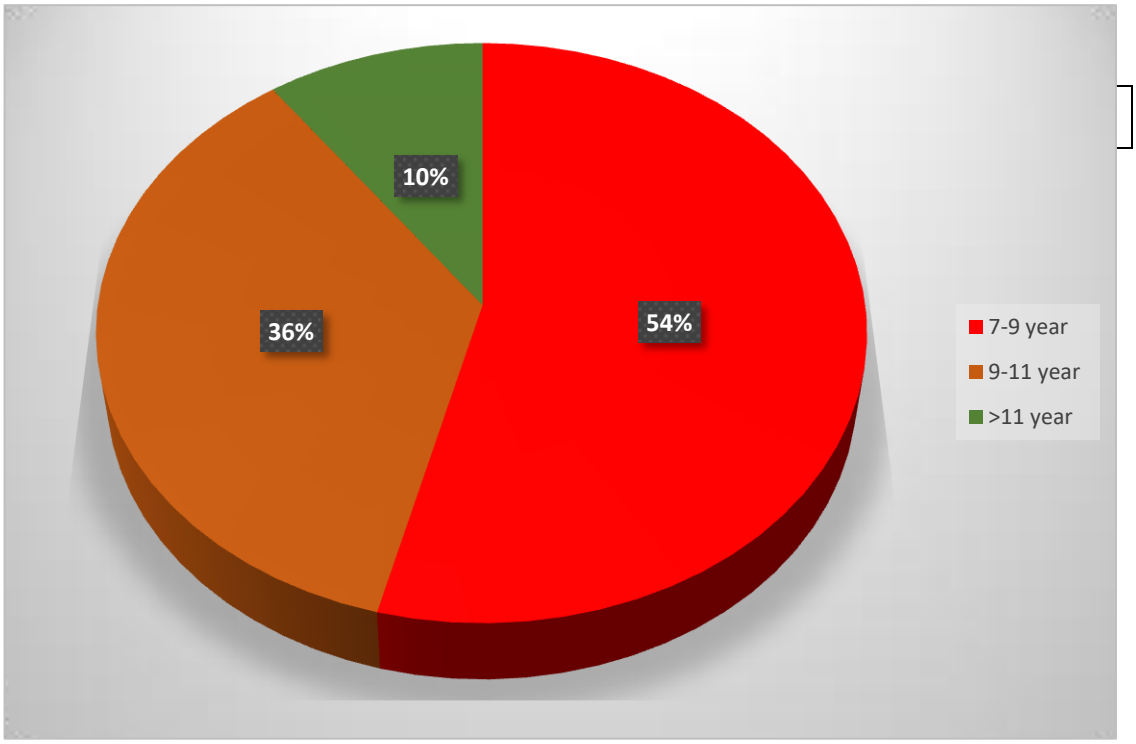


Fig 3: Breed wise incidence of cardiac disease

Fig 4: Age wise incidence of cardiac disease



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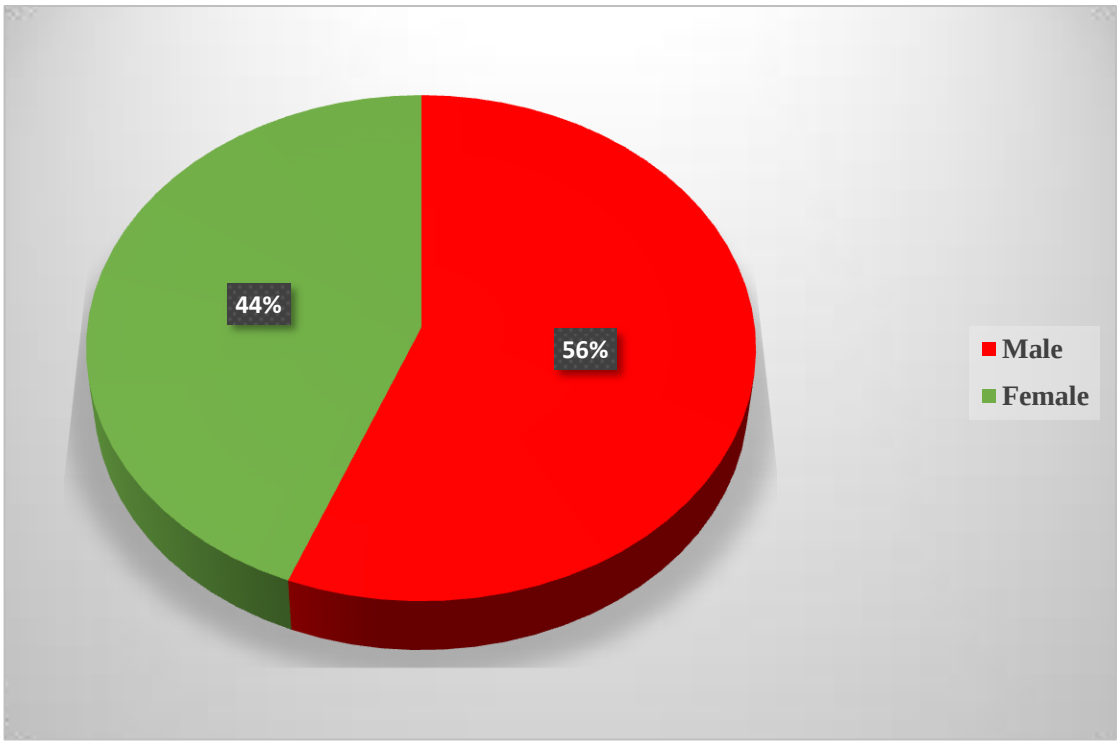


ig 5: Sex wise incidence of cardiac disease

Table 3: General impression of geriatric dog suspected for cardiopulmonary diseases

Behaviour (N=50)	Stupor	06
	Coma	02
	Alert	25
Posture (N=50)	Balanced	28
	Restless	07
	Anxious	08
	Aggressive	07
Gait (N=50)	Recumbent	08
	Sitting	11
	Standing	24
	Normal	07
Nutritional conditions (N=50)	Stiff	9
	Lame	02
	In coordination	08
	Normal	31
Coat (N=50)	Very obese	09
	Obese	12
	Moderate	18
	Poor	11
Body condition score (N=50)	Dull	13
	Thick	06
	Alopecia	12
	Normal	19
	05	11
	03	18
	02	21

Table 4: General examination of geriatric dog suspected for cardiopulmonary diseases

Type of respiration (N=50)	Costal	17
	Abdominal	08
	Pendulous	00
	Normal	25
Respiratory Rhythm	Regular	27
	irregular	13
	Normal	10
Heart Rhythm (N=50)	Regular	15
	Irregular	12
	Murmur	11
	Strong bonding	08
	Change in PMI	04
Murmurs (N=50)	Present	11
	Absent	39

Table 5: Mucous membrane examination of geriatric dog suspected for cardiopulmonary diseases

Colour (N=50)	Pink	29
	Pale	12
	Icteric	07
	Cyanotic	02
Moistness (N=50)	Dry	14
	Sticky	12
	Moist	24
Haemorrhage (N=50)	Petechiae	09
	Ecchymosis	06
	Normal	35
Lymph node (N=50)	Normal	29
	Abnormal	21

ry diseases

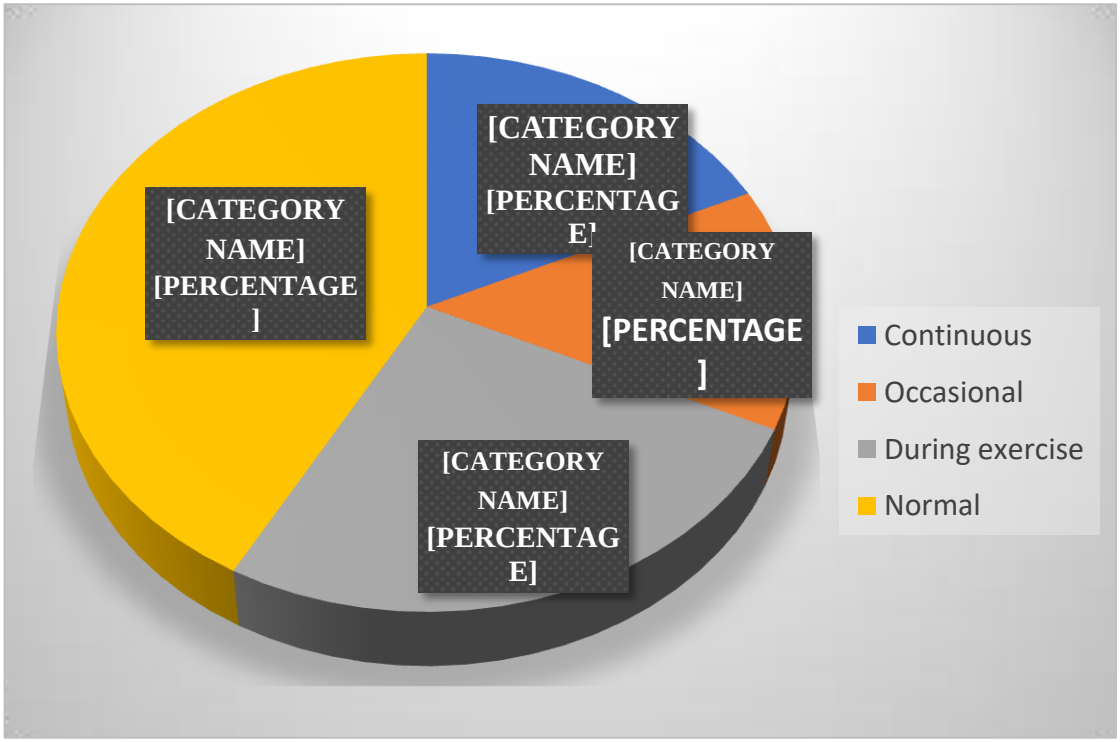
Table 6: Respiratory system examination of geriatric dog suspected for cardiopulmonary diseases

Auscultation of lungs (N=50)	Rales	06
	Whzeels	08
	Crackling	03
	Dull	04
Epistaxis (N=50)	06	
Nasal discharge (N=50)	09	
Coughing/sneezing (N=50)	17	
Respiratory distress (N=50)	18	

Laboured breathing (N=50)	Continuous	09
	Occasional	07
	During exercise	13
	Normal	21
Coughing (N=50)	Paroxysmal	06
	Occasional	10
	Nocturnal	14
	Absent	20
Fluid accumulation (N=50)	Ascites	16
	Pedal edema	07
	Hydrothorax	00
	Absent	27
Jugular vein pulsation (N=50)	Present	14
	Absent	36

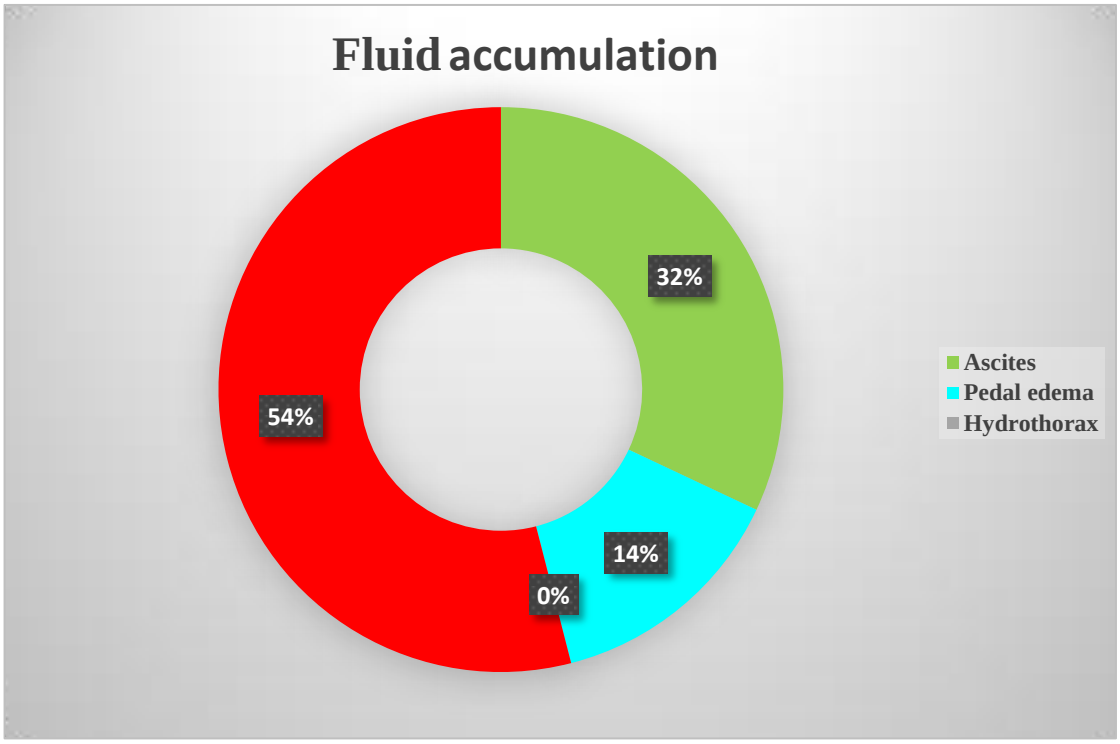
7: Cardiovascular examination of geriatric dog suspected for cardiopulmonary diseases

Fig no. 6: Respiratory system pattern of geriatric dog suspected



for cardiopulmonary diseases

Fig 7: Respiratory system pattern of geriatric dog suspected for c



nary diseases

Clinical trials:

Clinical trials of different drugs for the control of congestive heart failure in geriatric dogs were done with two groups of medication while the third group was kept as healthy control.

The duration of the treatment was up to 30 days and the observation period was 15- and 30-days from the start of treatment. The hematobiochemical observation in pre-and post-treatment was depicted in tabular form and the analysis of variation at a 5% level of significance was observed between the pre-treatment and in 15 and 30^{th} days of treatment.

Creatinine (mg/dl):

The creatinine which is the biomarker for renal disorder were found to be elevated in both treatment groups i.e., T-2 (1.76 ±0.33mg/dl) and T-3 (1.30 ±0.11mg/dl) at 0 days of treatment. Significantly high ($P<0.05$) creatinine was found in the treatment group (T-2 & T-3) than control group (T-1) (0.96 ±0.08mg/dl) which varied non significantly from T-3 (1.30± 0.11mg/dl) and control (0.96±0.08mg/dl) on the same days of treatment.

Significant reduction in serum creatinine level was observed in the T-2 group on the 15^{th} day (1.39±0.18mg/dl) from the 0 days of treatment which vary non significantly ($P>0.05$) from the 30^{th} day of treatment (1.32±0.15mg/dl). Similar trends were also observed in the T-3 group but the reduction was non-significant on the 15^{th} and 30^{th} days of treatment. Also, the level of creatinine in the T-2 group become comparable to the control on the 15^{th} day of treatment, and maximum reduction in serum creatinine was observed (1.76 to 1.39mg/dl) on 15 and (1.76 to 1.32mg/dl) on the 30^{th} day of treatment, respectively.

Thus, the treatment regimen in group T-2 was found to be better in reducing the serum creatinine level towards normal than the T-3 group.

Blood urea nitrogen (mg/dl):

Blood urea nitrogen in the control group T-1 varies from 18.93 mg/dl to 19.33 mg/dl on 0 to 30 days of treatment. Significantly ($P<0.05$) higher blood

urea nitrogen levels 41.44±9.24 mg/dl and 34.56±7.10 mg/dl were recorded in the T-2 - group on 0 days and 15 days which become comparable to control on the 30th day of treatment. Non-significant variation (P>0.05), although reducing trend was observed in the T-3 group on the 15th and 30th day of treatment. In between T-2 and T-3 groups significant (P<0.05) was observed on 0-day 15th day, while on 30th day non-significant (P>0.05) variation was observed.

Maximum reduction in blood urea nitrogen was observed in the T-2 group (41.44 to 34.56 mg/dl) and (41.44 to 30.41 mg/dl) on the 15th and 30th day of treatment respectively. Thus, the T-2 group was found to be better in comparison to T-1 in reduction of blood urea nitrogen in congestive heart failure cases.

Total serum protein (mg/dl):

Non-significant (P>0.05) variation in total serum protein level was observed between the healthy and control group of dogs. Non-significant but elevated level of total serum protein were observed both in T-2 and T-3 groups. Non-significant (P>0.05) decrement in total serum protein were observed in both group T-2 and T-3 on 15th and 30th day of treatment. Also, the level of total serum protein 0, 15 and 30 days vary non significantly (P>0.05) on 0, 15th and 30th day in between the control T-1 and T-2 groups. However, maximum reduction was observed in the T-2 group on the 15th and 30th days of treatment.

Albumin (mg/dl):

Non-significant(P>0.05) variation in albumin level was observed in dogs suffering from cardiac disease pre-and post-treatment and also from the healthy control group. However, a non-significant(P>0.05) decrease in albumin level was observed in groups T-2 and T-3 on 0 days which improves after treatment on 15th and 30th days.

Globulin (mg/dl):

The mean level of globulin in T-2 and T-3 was found to be higher than the healthy control group on 0, 15, and 30 days. Significant (P <0.05) decrement in globulin

levels was observed on the 15th and 30th day of the treatment in-group T-2 however this variation in mean globulin level was observed on the 30th day in group T-3.

Table 8: Mean ± S.E of Creatinine (mg/dl) in control and treatment groups

		0-day	15-day	30-day
M e a n	**T-1**	0.96±0.08 ^{ax}	0. 99±0.08 ^{ax}	0. 98±0.07 ^{ax}
	T-2	1.76±0.33^{bx}	1.39±0.18^{ay}	1.32±0.15^{by}
	T-3	1.30±0.11^{abx}	1.17±0.09^{ax}	1.16±0.08^{abx}

with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

	0-day	15-day	30-day
T-1	19.33±2.11 ^{ax}	18.93±1.45 ^{ax}	19.30±1.32 ^{ax}
T-2	41.44±9.24^{bx}	34.56±7.10^{by}	30.41±6.42^{ay}
T-3	22.12±3.39 ^{ax}	21.60±2.52 ^{ax}	20.73±2.29 ^{ax}

Table 9: Mean ± S.E of BUN (mg/dl) in control and treatment groups

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 10: Mean ± S.E of TSP (mg/dl) in control and treatment groups

M e a n w		0-day	15-day	30-day
	T-1	6.40±0.16 ^{ax}	6.43±0.15 ^{ax}	6.52±0.15 ^{ax}
	T-2	7.60±0.37 ^{ax}	7.25±0.28 ^{ax}	7.05±0.25 ^{ax}
	T-3	7.57±0.59 ^{ax}	7.46±0.51 ^{ax}	6.98±0.30 ^{ax}

ith different superscripts in column (a, b, c) differ significantly (p<0.05%)

	0-day	15-day	30-day
T-1	2.77±0.08 ^{ax}	2.76±0.11 ^{ax}	2.78±0.09 ^{ax}
T-2	2.57±0.08 ^{ax}	2.60±0.05 ^{ax}	2.67±0.08 ^{ax}
T-3	2.61±0.09 ^{ax}	2.63±0.07 ^{ax}	2.70±0.06 ^{ax}

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 11: Mean ± S.E of Albumin (mg/dl) in control and treatment groups

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 12: Mean ± S.E of Globulin (mg/dl) in control and treatment groups

	0-day	15-day	30-day
T-1	3.63±0.12 ^{ax}	3.67±0.94 ^{ax}	3.78±0.11 ^{ax}
T-2	5.71±0.33 ^{bx}	4.60±0.29 ^{by}	4.38±0.27 ^{by}
T-3	4.96±0.67 ^{bx}	4.38±0.53 ^{bx}	4.28±0.30 ^{by}

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Sodium (Na) mEq/L:

The mean level of sodium in the control group T-1 on 0, 15, and 30 days of treatment was found to be 145.84±2.78mEq/L, 144.53±2.80mEq/L, and 148.25±2.21mEq/L, respectively. Significantly lower ($P<0.05$) sodium level was observed in the T-2 and T-3 groups on 0 days, which became significantly ($P<0.5$) higher on the 15th and 30th days of treatment. However, non-significant ($P>0.05$) variation from the control level was observed in the 15th and 30th days. Significant ($P<0.05$) variation in mean sodium 145.25±1.81mEq/L was observed in the T-2 group which vary non-significantly ($P>0.05$) from 30 days 146.64±1.58 mEq/L.

Potassium (K) mEq:

The mean level of potassium in control group T-1 on 0, 15 and 30 days of treatment were 4.10±0.15 mEq/L, 4.05 ± 0.14 mEq/L and 4.18±0.09 mEq/L, respectively. Significantly ($P<0.05$) higher level of mean potassium level in T-2 and T-3 were observed on 0 days from the control group. On 15th day of treatment non-significant ($P>0.05$) decrease in potassium level was observed in T2 and T3 group from control. Similar, trend was observed on 30th day. Significantly ($P<0.05$) lower level of mean potassium was observed in T-2 and T-3 group on 15th and 30th days of treatment which did not vary significantly ($P>0.05$) among themselves. Maximum decrease in mean potassium level was observed in T-2 group from the 0-day level of potassium on 15th day (0.31mEq/L) and on 30th day (0.49mEq/L). Hence, group T-2 was found to be better in giving the result.

Calcium (Ca) mg/dl:

The mean calcium level in control geriatric dogs on 0, 15 day and 30 days in T-1 group were 11.71±0.52 mg/dl, 11.45±0.37 mg/dl and 11.54±0.34 mg/dl, respectively. Significantly, lower mean calcium level was found in T-2 and T-3 treatment group on 0-day which vary non- significantly (P>0.05) among themselves. Improvement in mean calcium levels was observed in both T-2 and T-3 on 15^{th} day and 30^{th} day which vary non-significantly (P>0.05) from control group. Maximum improvement in mean calcium level was observed in T-2 which increases from 8.48mg/dl to 10.42mg/dl on 15^{th} day and 8.48mg/dl to 10.58mg/dl.

Table 13: Mean ± S.E of Na (mEq/L) in control and treatment group

	0-day	15-day	30-day
T-1	145.84±2.78 ^{ax}	144.53±2.80 ^{ax}	148.25±2.21^{ax}
T-2	140.69±6.19 ^{bx}	145.25±1.81 ^{ay}	146.64±1.58 ^{ay}
T-3	141.36±2.59 ^{bx}	144.45±2.75 ^{ax}	143.02±3.39 ^{ax}

Mean

with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 14: Mean ± S.E of Potassium (mEq/L) in treatment group

	0-day	15-day	30-day
T-1	4.16±0.15 ^{ax}	4.05±0.14 ^{ax}	4.18±0.09 ^{ax}
T-2	4.50±0.14^{bx}	4.19±0.13^{ay}	4.0±0.1^{ay}
T-3	4.51±0.10^{bx}	4.27±0.10^{ay}	4.04±0.1^{ay}

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 15: Mean ± S.E of Ca (mg/dl) in control and treatment groups

	0-day	15-day	30-day
T-1	11.71±0.52 ^{ax}	11.45±0.37 ^{ax}	11.54±0.34 ^{ax}
T-2	8.48±0.47 ^{bx}	10.42±0.44 ^{ay}	10.58±0.41 ^{ay}
T-3	9.21±0.38 ^{bx}	10.37±0.38 ^{ay}	10.42±0.39 ^{ay}

Mean

with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Bilirubin (mg/dl):

The mean bilirubin level in control healthy group T-1 on 0,15-day and 30-day were 0.20±0.03mg/dl, 0.22±0.02mg/dl and 0.25±0.02mg/dl. Significantly, ($P<0.05$) higher mean bilirubin 0.41± 0.08 mg/dl and 0.56± 0.27 mg/dl were found on 0-day in T-2 and T-3 group. Non-significant decrease in mean bilirubin was observed on 15-day and 30-day in T-2 and T-3 group. Significant($P<0.05$) decrease in mean bilirubin 0.29± 0.03 mg/dl and 0.28± 0.02 mg/dl were observed in T-3 from 0 day.

Systolic Atrial Pressure (SAP) mmHg:

The mean SAP level in the geriatric dog in the control healthy group on 0,15 and 30-day were 128.40±03.33mmHg, 126.80±3.20mmHg, 127.10±2.84mmHg. Significantly ($P<0.05$), higher SAP was found in T-2 and T-3 groups on the 0, $15^{th,}$ and 30^{th} days. Significant ($P<0.05$) decrease in mean SAP level were found in T-2 and T-3 on the 30^{th} days of treatment from 0 and 15th-day levels. A maximum decrement of mean SAP was found in T-3 (7.50mmHg) in comparison to T-2 (8.30mmHg).

Diastolic Atrial Pressure (DAP) mmHg:

The mean DAP level in the geriatric dog in the control healthy group on 0,15 and 30-day were found to be 90.30±1.11 mmHg,88.20±1.09 mmHg, and 88.40±1.25 mmHg, respectively. Significantly ($P<0.05$) higher mean DAP level was found on 0, $15^{th,}$ and 30^{th} days in T-2 and T-3 groups from the control group. Significant reduction in mean DAP was observed in the T-2 group on the 15^{th} day, 105.00±1.23 mmHg and 30^{th} day 99.30±1.23 mmHg from 0-day level (110.30±1.06 mmHg). Unlike the T-2, in the T-3 group significant reduction was observed on the 30^{th} day (102.90±1.50 mmHg) from 0-day level (109.90± 1.43 mmHg), which vary non-significantly ($P>0.05$) from the 15th-day level 106.00±1.37 mmHg.

CK-MB (IU/L):

The mean CK-MB level in geriatric dogs in the control healthy group on 0,15 and 30-day was found to be 21.39±0.74 IU/L, 21.18±0.03 IU/L, and 21.13±0.07 IU/L respectively. Significantly ($P<0.05$) higher level of CK-MB was found in T-2 and T-3 on 0, $15^{th,}$ and 30^{th} days of treatment with respect to the control group. Significant ($P<0.05$) reduction in CK-MB 53.35±8.71 IU/L and 57.06±6.71 IU/L were found in T-2 and T-3 respectively

on the 30th day from 0 and 15th-day levels. However maximum decrease was found in the T-2 group from their 0-day level 18.7IU/L.

Hemoglobin (gm/dL):

The mean hemoglobin level of geriatric dogs in the control healthy group was found to be 14.54±0.53 gm/dL, 14.69±0.44 gm/dL, and 14.07±0.21 gm/dL, respectively on 0, 15th, and 30th days. Significantly ($P<0.05$), lower hemoglobin level was found in T-2 and T-3 on 0, 15th days from the control, while the level of hemoglobin becomes comparable and non-significant($P>0.05$) in both group T-2 and T-3 on 30th days of treatment. Significant ($P<0.5$) improvement in mean hemoglobin level was found on the 30th day in both treatment groups T-2 and T-3, 14.03±0.43 gm/dL and 14.07± 0.21 gm/dL, respectively which vary non-significantly ($P>0.05$) from the 15th day level in both groups. Similarly, non-significant ($P>0.05$) but higher hemoglobin level was found in both groups T-2 and T-3 on the 15th day from their 0 days level.

PCV (%):

The mean PCV level in the geriatric dogs in the control healthy group was found to be 43.60± 1.58, 44.03± 1.24, and 44.16± 0.96, respectively on 0, 15th, and 30 days of treatment. Significantly ($P<0.05$), lower mean PCV level was found in T-2 to and T-3 from the control group on 0, 15th, and 30th days of treatment. Significant ($P<0.05$) improvement in PCV was found in 30-day from 15th and 0- days in both T-2 and T-3 groups. Non-significant($P>0.05$) but higher improvements were observed in mean PCV from 0-day on 15th day of treatment in both group T-1 and T-2.

Platelets (Lakh/Cumm):

The mean platelet levels in geriatric dogs in the control healthy group were found to be 1.94± 0.04, 1.95± 0.05, and 1.91± 0.03 respectively. Significantly, lower platelets found in T-2 and T-3 on 0, 15th day of treatment from the healthy control group were found. However, non-significant ($P>0.05$) and higher mean platelets were found in T-2 and T-3 on the 30th day of treatment. Significant ($P<0.05$) improvement in the mean platelets was found in the T-2 group on the 15th day (1.59± 0.06) and 30th day (1.93± 0.08) from the 0-day level. Significant ($P< 0.05$) and higher improvement from the control was observed in T-3 on the 30th day of treatment.

Table 16: Mean ± S.E of Bilirubin (mg/dl) in control and treatment groups

	0-day	15-day	30-day
T-1	0.20±0.03 ^{ax}	0.22±0.02 ^{ax}	0.25±0.02 ^{ax}
T-2	0.41±0.08 ^{bx}	0.33±0.07 ^{ax}	0.34±0.06 ^{ax}
T-3	0.56±0.27 ^{bx}	0.29±0.03 ^{ay}	0.28±0.02 ^{ay}

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 17: Mean ± S.E of SAP (mmHg) in control and treatment groups

	0-day	15-day	30-day
T-1	128.40 ± 3.33 ^{ax}	126.80 ± 3.20 ^{ax}	127.10 ± 2.84 ^{ax}
T-2	156.60 ± 4.17^{by}	147.70 ± 3.73^{bxy}	143.10 ± 3.51^{bx}
T-3	155.4 0 ± 4.17^{by}	151.50 ± 2.14^{bxy}	147.10 ± 2.17^{bx}

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 18:

	0-day	15-day	30-day
T-1	90.30±1.11 ^{ax}	88.20±1.09 ^{ax}	88.40±1.25 ^{ax}
T-2	110.30±1.06 ^{bz}	105.00±1.23 ^{by}	99.30±1.23 ^{bx}
T-3	109.90±1.43 ^{by}	106.00±1.37 ^{bxy}	102.90±1.50 ^{bx}

Mean ± S.E of DAP (mmHg) in control and treatment groups

Mean with different superscripts in column (a, b, c) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 19: Mean ± S.E of CK-MB (IU/L) in control and treatment groups

	0-day	15-day	30-day
T-1	21.39±0.74 ^{ax}	21.18±0.63 ^{ax}	21.13±0.67 ^{ax}
T-2	72.05±9.71 ^{bx}	63.46±9.46 ^{bx}	53.35±8.71 ^{bx}
T-3	72.78±7.68 ^{bx}	65.41±7.20 ^{bx}	57.06±6.71 ^{bx}

Mean with different superscripts in column (a, b, c, d) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 20: Mean ± S.E of Hemoglobin (gm/dl) in control and treatment groups

	0-day	15-day	30-day

T-1	14.54±0.53^{bx}	14.69±0.44^{bx}	14.07±0.21^{ax}
T-2	10.80±1.14^{ax}	12.22±0.75^{axy}	14.03±0.43^{ay}
T-3	10.48±1.00^{ax}	12.18±0.63^{axy}	14.07±0.21^{ay}

Mean with different superscripts in column (a, b, c, d) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table 21: Mean ± S.E of PCV (%) in control and treatment groups

	0-day	**15-day**	**30-day**
T-1	43.60±1.58 ^{bx}	44.03±1.24 ^{bx}	44.16±0.96 ^{ax}
T-2	32.00±3.37 ^{ax}	36.90±1.81 ^{ax}	43.41±0.62 ^{ay}
T-3	32.50±2.58 ^{ax}	36.41±1.83 ^{ax}	42.15±1.11^{ay}

Mean with different superscripts in column (a, b, c, d) differ significantly (p<0.05%)
Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Fig 8: Mean ± S.E of Creatinine (mg/dl) in control and treatment groups

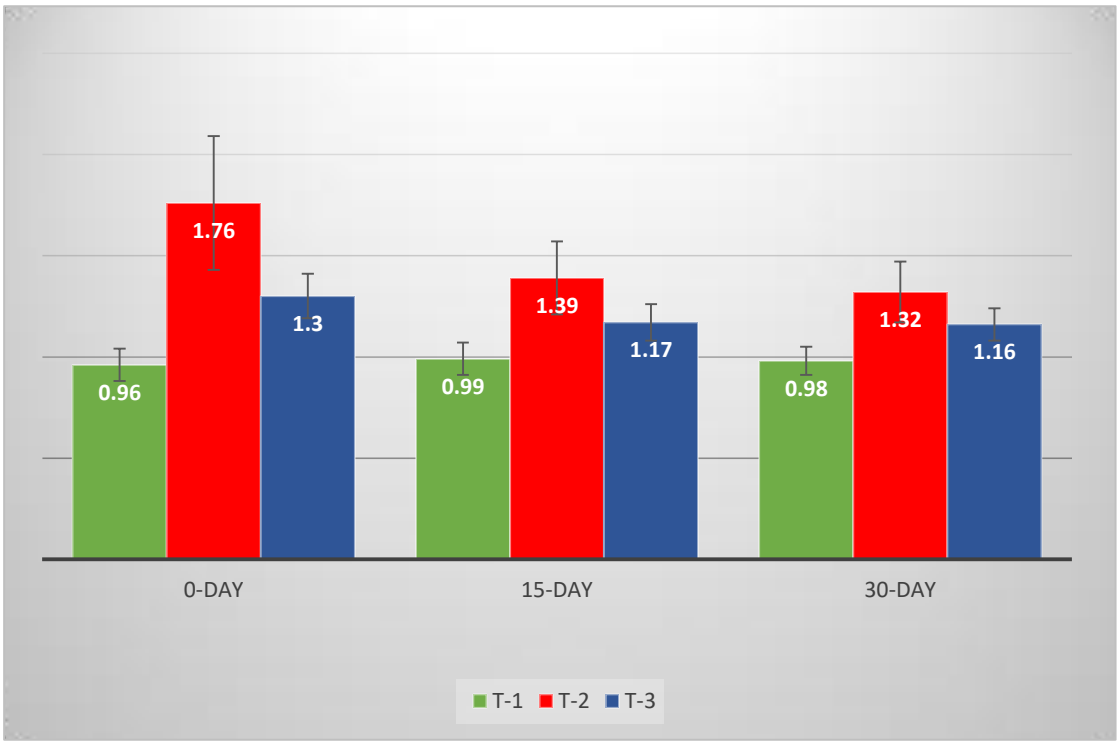


Fig 9: Mean ± S.E of BUN (mg/dl) in control and treatment groups

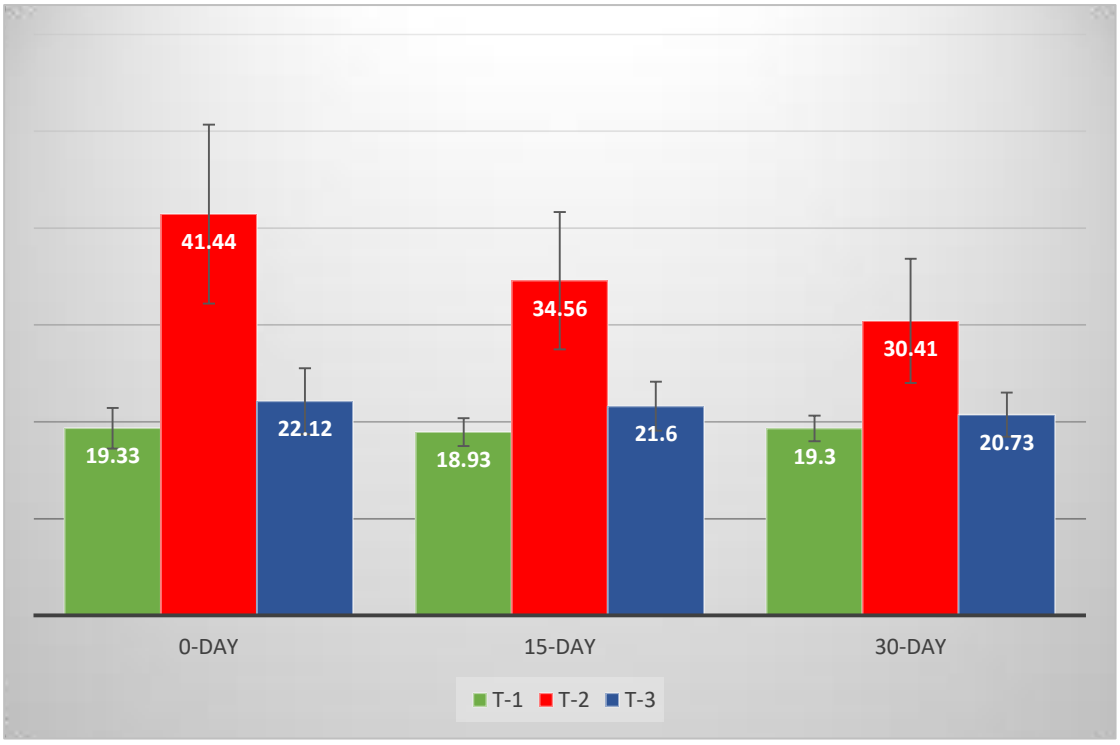


Fig 10: Mean ± S.E of TSP (mg/dl) in control and treatment groups

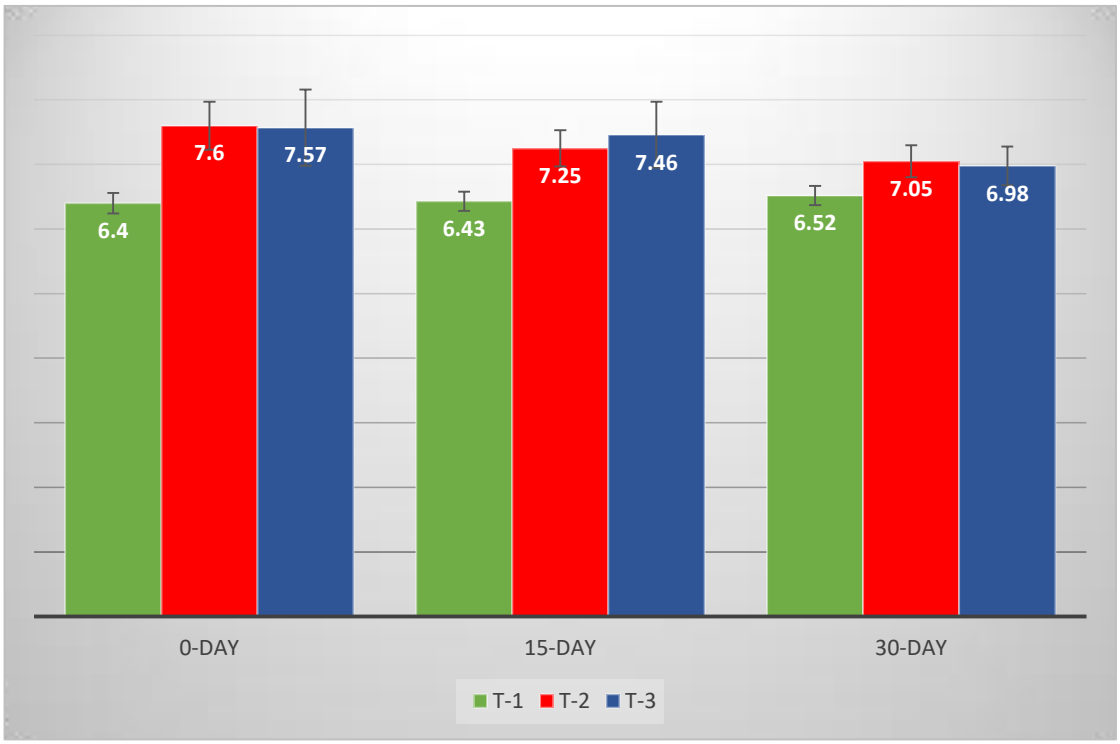


Fig 11: Mean ± S.E of Albumin (mg/dl) in control and treatment groups

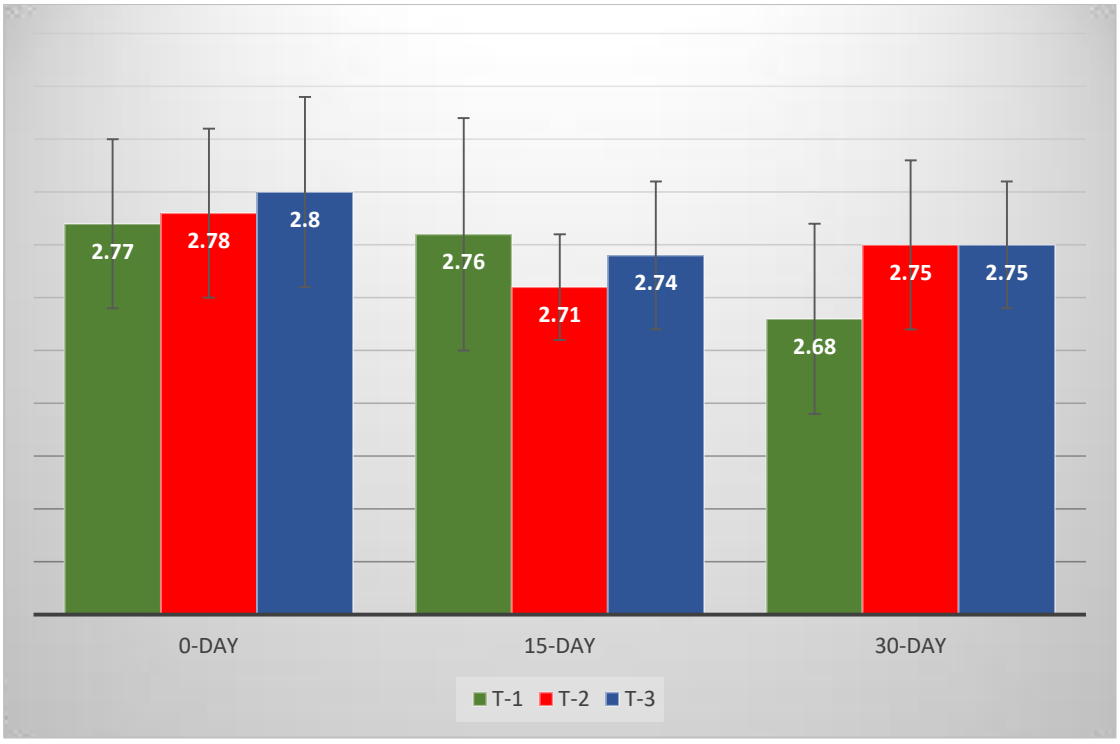
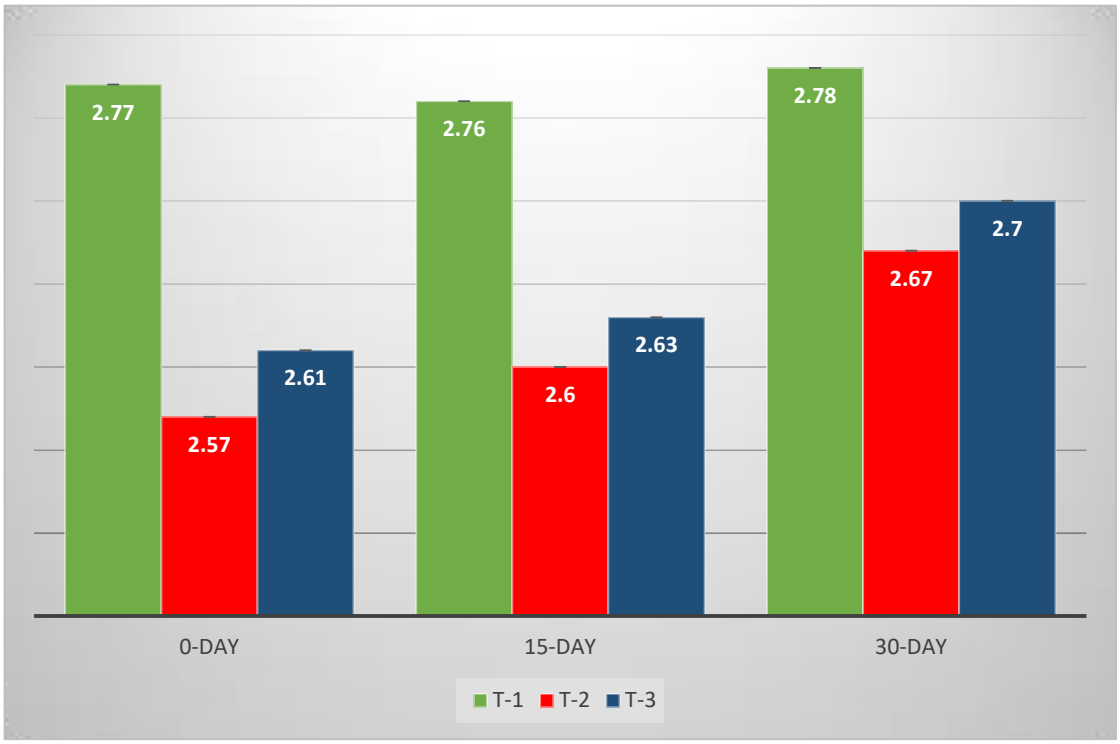


Fig 12: Mean ± S.E of Globulin (mg/dl) in control and treatment groups



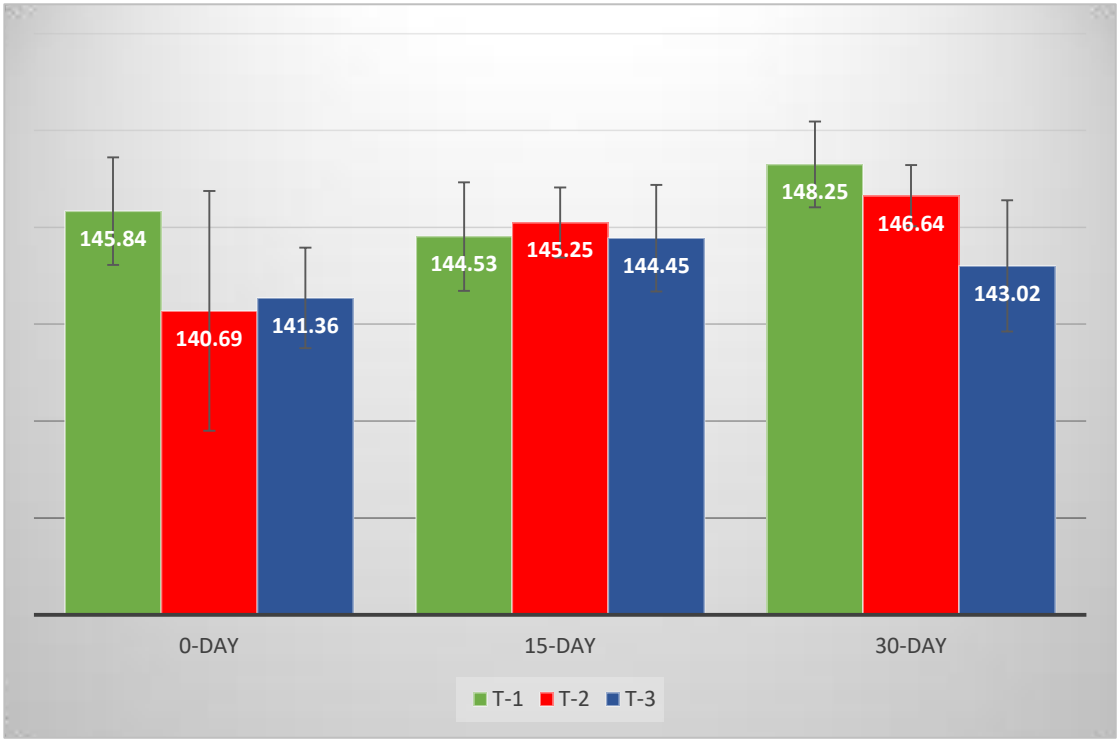


Fig 13: Mean ± S.E of Sodium (mEq/L) in control and treatment groups

Fig 14: Mean ± S.E of Potassium (mEq/L) in control and treatment groups

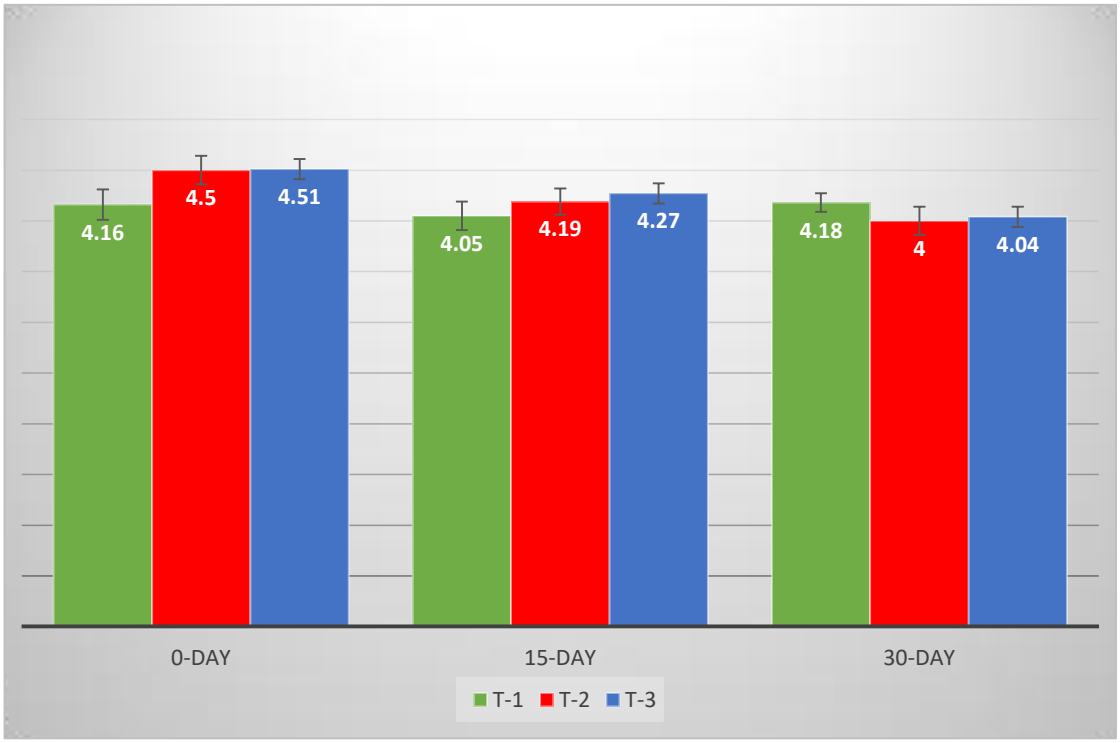


Fig 15: Mean ± S.E of Calcium (mg/dl) in control and treatment groups

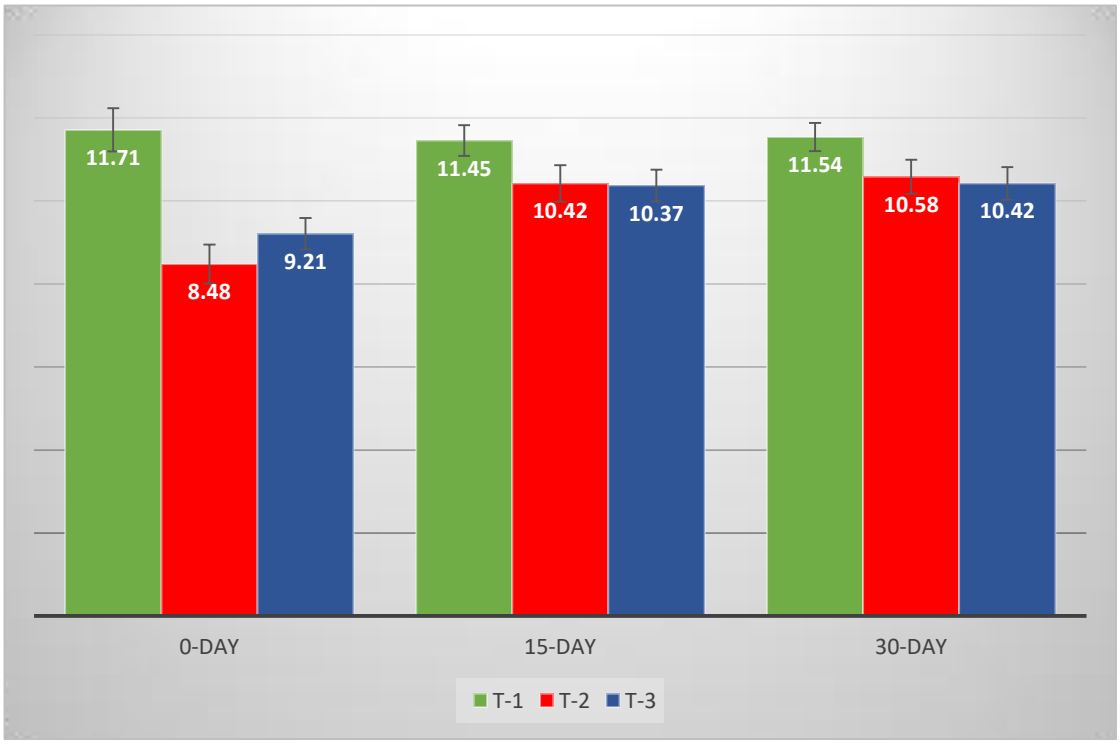


Fig 16: Mean ± S.E of Bilirubin (mg/dl) in control and treatment groups

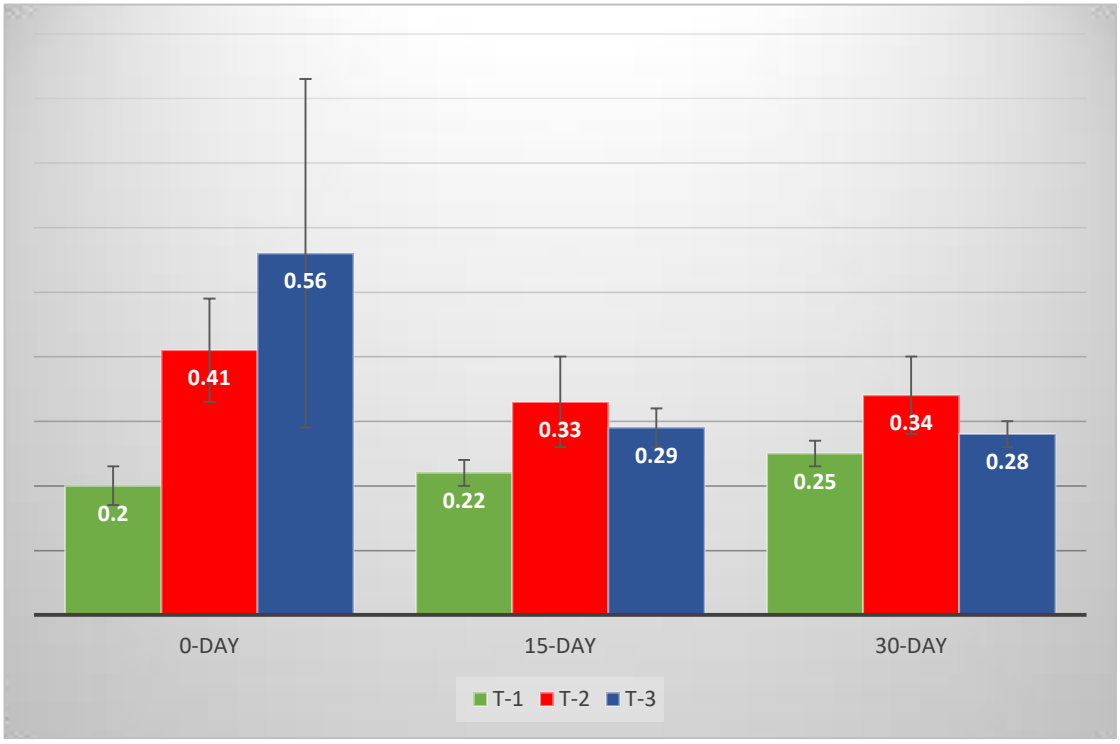


Fig 17: Mean ± S.E of SAP (mmHg) in control and treatment groups

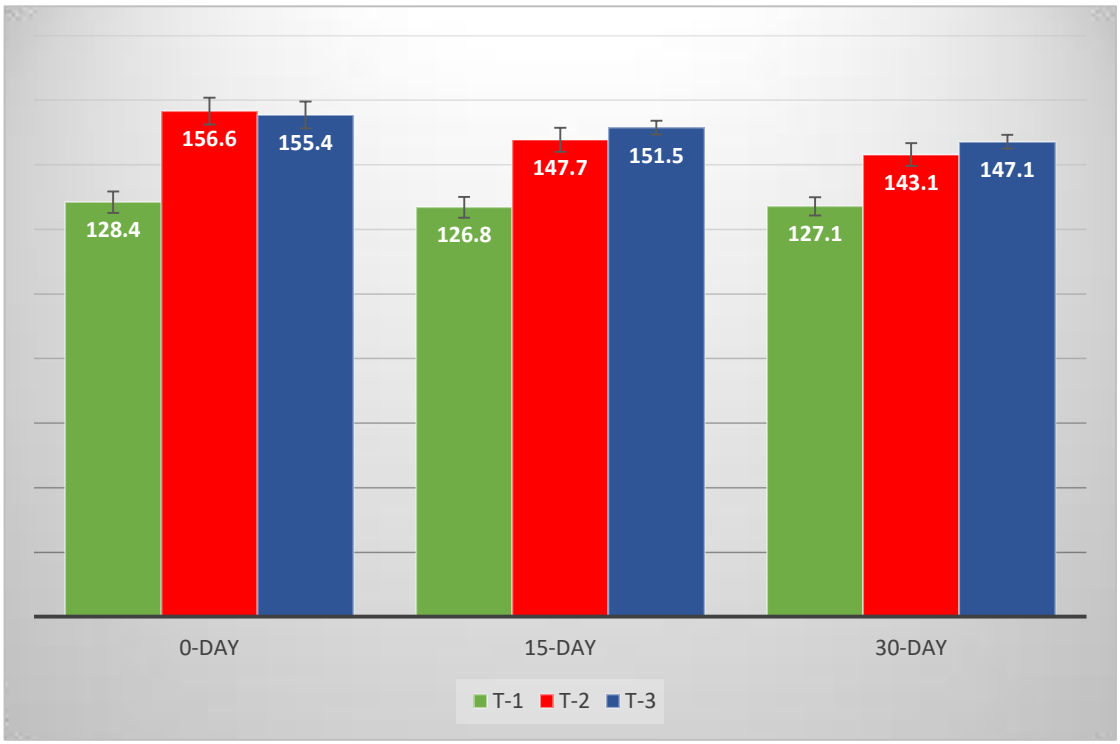


Fig 18: Mean ± S.E of DAP (mmHg) in control and treatment groups

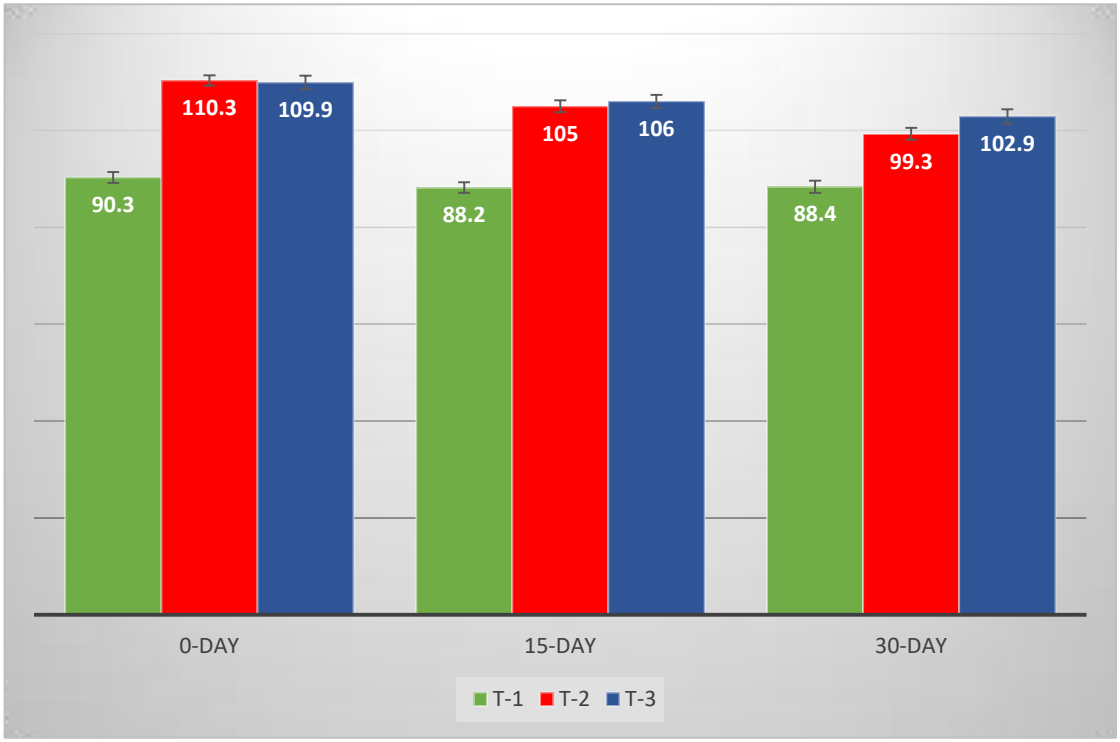


Fig 19: Mean ± S.E of CKMB (IU/L) in control and treatment groups

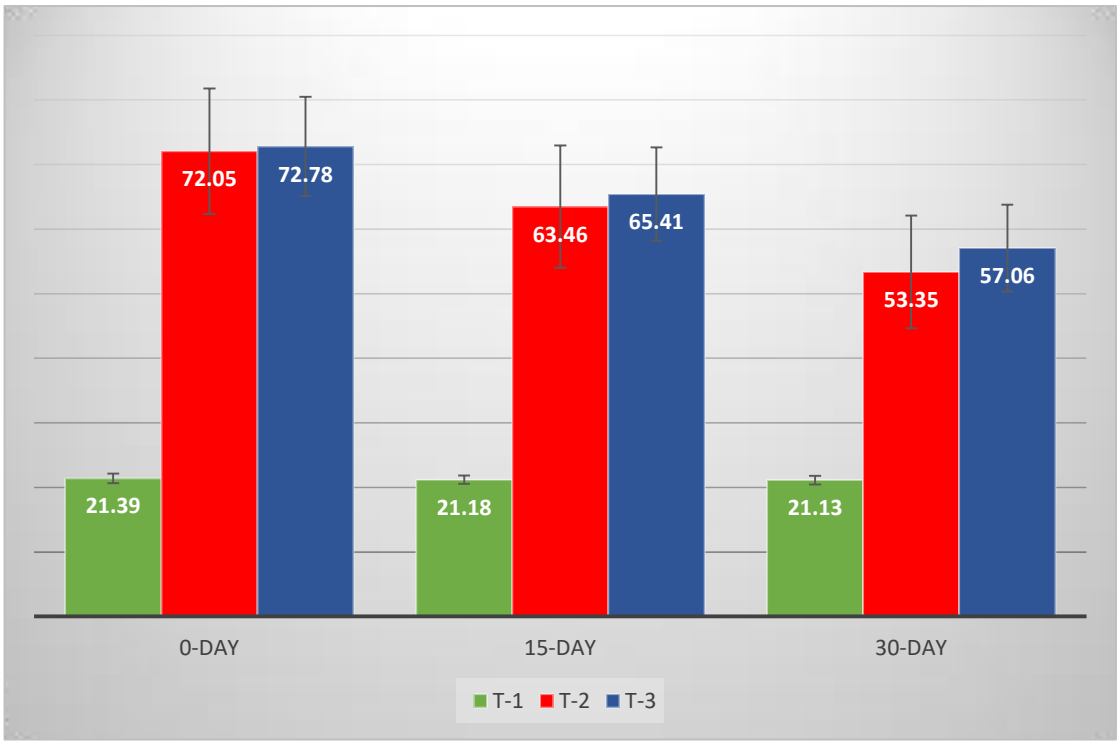


Fig 20: Mean ± S.E of Hemoglobin (gm/dl) in control and treatment groups

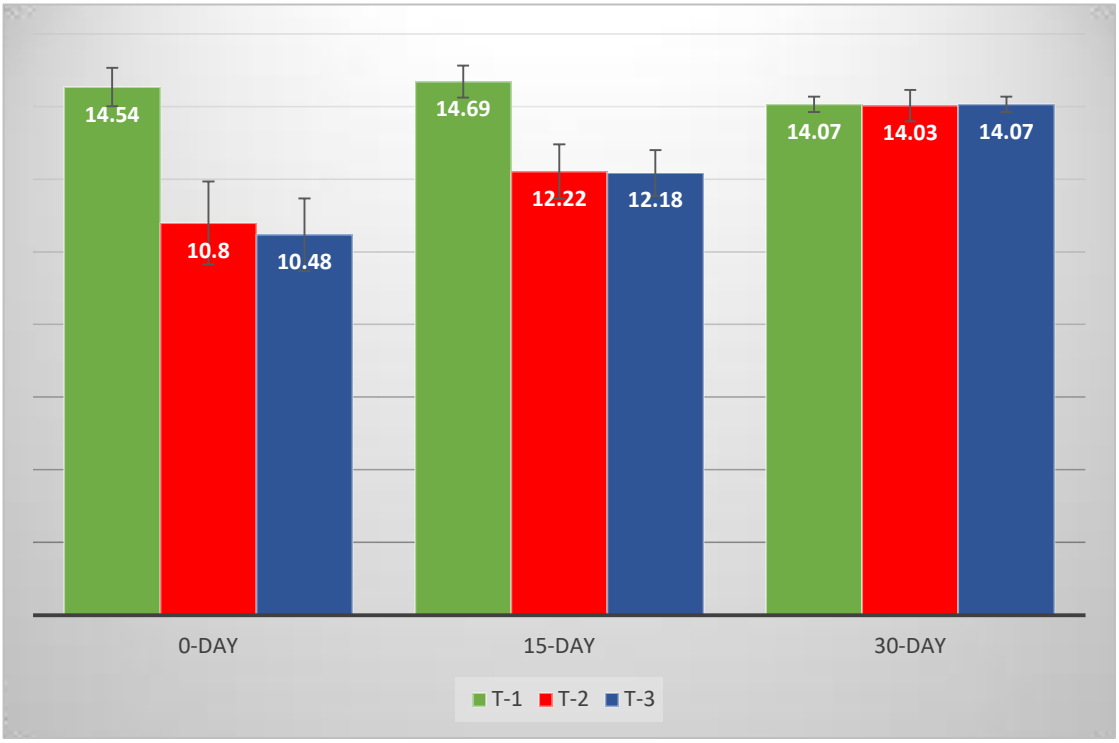


Fig 21: Mean ± S.E of PCV (%) in control and treatment groups

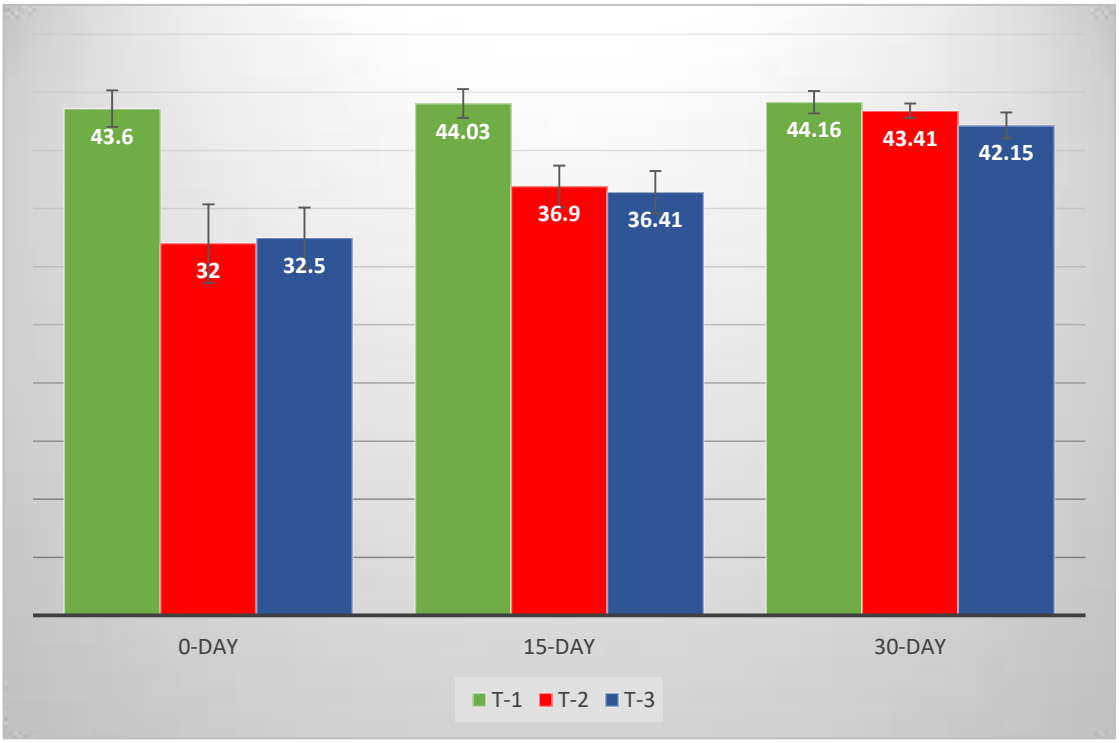


Fig 22: Mean ± S.E of platelets (Lakh/Cumm) in control and treatment groups

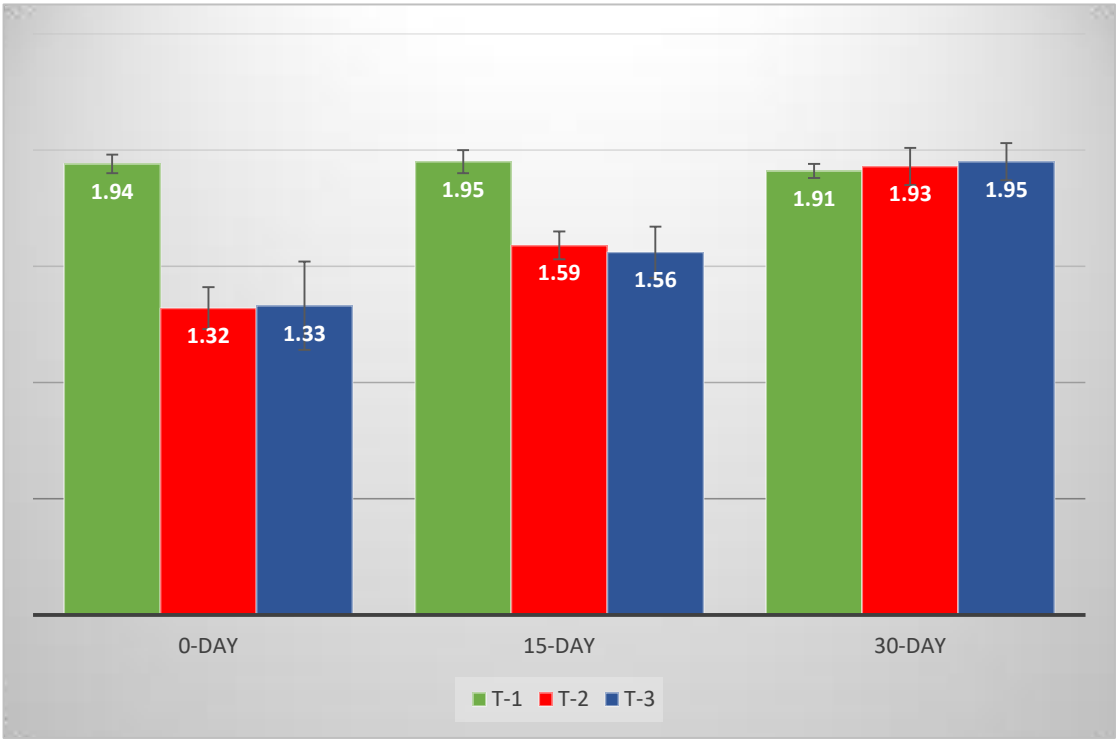


Table 22: Mean ± S.E of platelets (Lakh/Cu mm) in control and treatment groups

Mean with		0-day	15-day	30-day
	T-1	1.94±0.04^{bx}	1.95±0.05^{bx}	1.91±0.03^{ax}
	T-2	1.32±0.09^{ax}	1.59±0.06^{ay}	1.93±0.08^{az}
	T-3	1.33±0.19^{ax}	1.56±0.11^{axy}	1.95±0.08^{ay}

h different superscripts in column (a, b, c) differ significantly (p<0.05%)

Mean with different superscripts in row (x, y, z) differ significantly (p < 0.05%)

Table no. 23: Mean of amplitude and time interval of different waves in ECG of geriatric dog suspected for cardiopulmonary diseases

	Heart rate	P Wave Amplitude (mV)	P Wave Duration (sec)	P-R Intervals (sec)	QRS Duration (sec)	R Wave Amplitude (mV)	T wave Amplitude (mV)	T Wave Duration (sec)	Q-T Interval (sec)	Q-Deepening (mV)
Normal Range	70-170	0.15-0.4	0.02-0.04	0.08-0.12	0.04-0.05	1.2-2.8	0.15-0.5	0.04-0.08	0.11-0.23	0.05
Mean (N=50)	103.75	0.2	0.05	0.13	0.03	1.75	0.34	0.09	0.25	0.56

ECG:

The mean heart rate of genetic dogs brought with the symptom of cardio-pulmonary was found 103.75 beats per minute which are lower than the normal healthy range of 70 to 160 beats per minute. the mean P amplitude was found to be 0.20 (below) and the mean P duration to be 0.05 (above) which is below the normal range of P amplitude and duration <0.4 mv and <0.04 Second respectively.

The mean PR duration interval in our case is 0.13 sec which age Above and below the average PR interval that is 0.06 to 0.13 sec. the mean QRS duration was found to be 0.03 seconds which is below the normal range. the mean amplitude was found to be 1.75mv which is below the normal range of 2.5mv in the giant breed.

The mean T amplitude was found to be 0.3 mv and T duration 0.09 sec the main QT interval was found to be 0.25 sec which is below the normal / above the normal range the mean Q deepening was found to be 0.56 MV which is above the normal range of 0.5mv.

An electrocardiogram (ECG) is the recorded tracing produced by the electrocardiograph giving waveforms of PQRST. In the present study routine ECG was done in 50 dogs and dogs showing prudent variations in ECG is presented as result form. Apart from min level of all parameters of ECG the special cases were recorded and presented in the result form.

ECG 1 show mild arrhythmia and Q wave deepening suggestive of right ventricular enlargement. The variable R amplitude was also observed. Similarly, ECG-2 also shows arrhythmia and Q wave deepening up to 1mv. ECG 3 shows ventricular tachycardia. This type of heart rhythm disorder is caused by abnormal electrical signals in the lower chamber of the heart. It may develop in valvular heart disease. ECG 4 was indicative of low amplitude R-wave, Q-wave deepening, elevated ST-segment, and wide R-wave. Similarly, in ECG 5 S-T coving and the inverted T-wave is important findings. ECG 6 was indicative of low amplitude P-wave, S-T coving, and increased T-wave duration. Increased heart rate and increased amplitude of P- wave was noticed in ECG 7. ECG-8 indicated VPC, elevated T- amplitude, and S-T coving while ECG 9 and

10 were indicating flattening of PQRST wave while ECG 11 was indicative of deep S-wave.

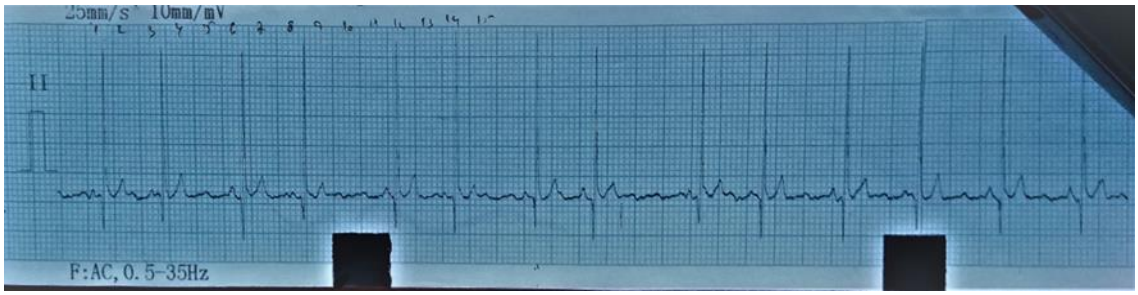


Fig 23: ECG-1 shows mild arrhythmia, and Q wave deepening suggestive of right ventricular enlargement the R amplitude is also variable.

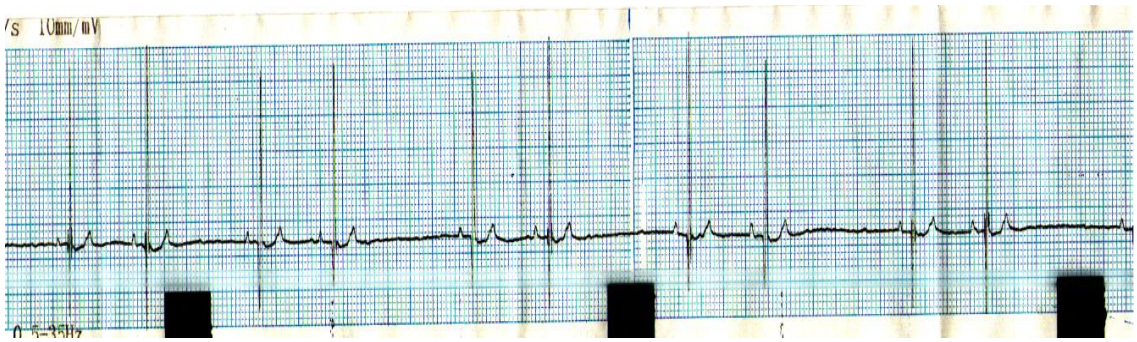


Fig 24: ECG-2 shows Arrhythmia, Q wave deepening up to 1mv suggestive of right ventricular enlargement

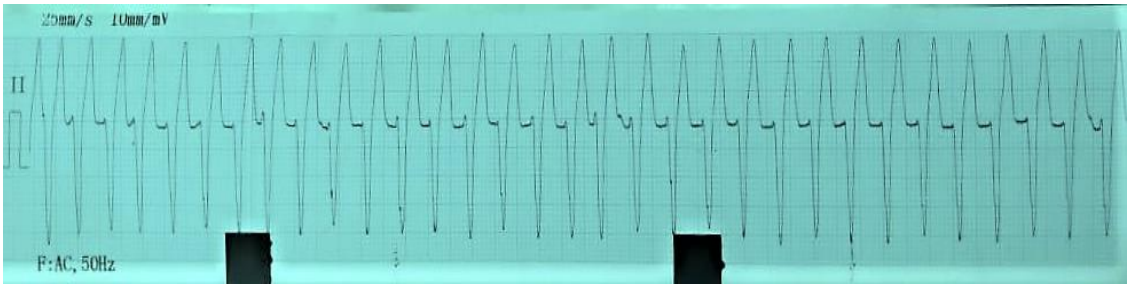


Fig 25: ECG-3 is suggestive of ventricular tachycardia.

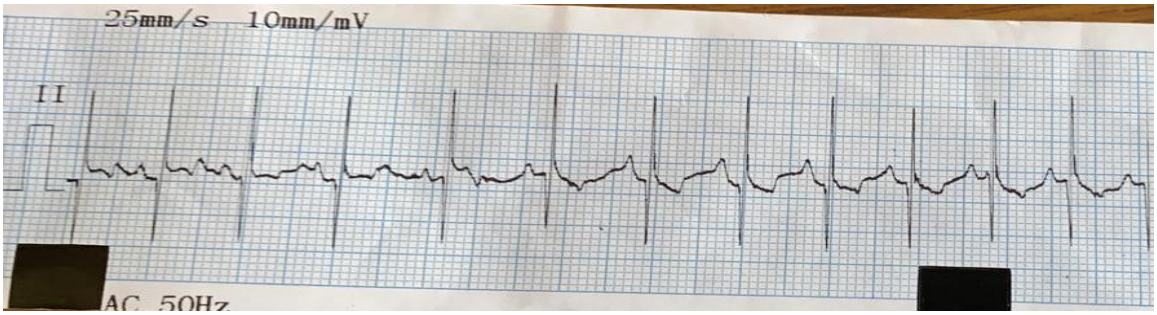


Fig 26: ECG-4 is indicating lower R amplitude, Q wave deepening, elevated S-T segment and wide P wave.

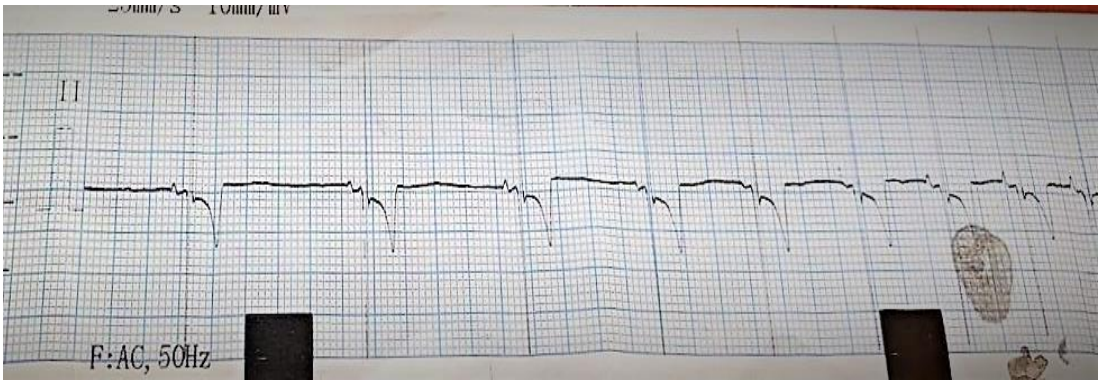


Fig 27: ECG-5 indicating deep Q wave and inverted T-wave and S-T coving.

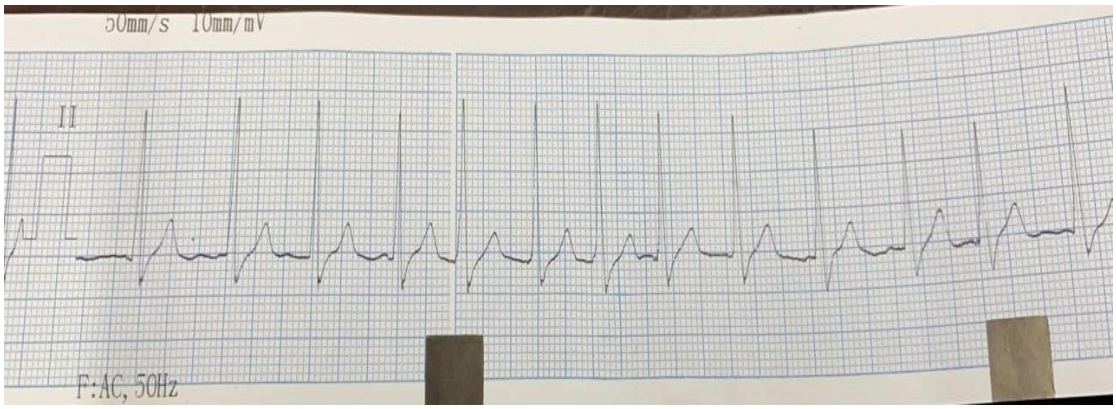


Fig 28: ECG-6 indicating low P wave amplitude, S-T coving and increased T-wave duration.

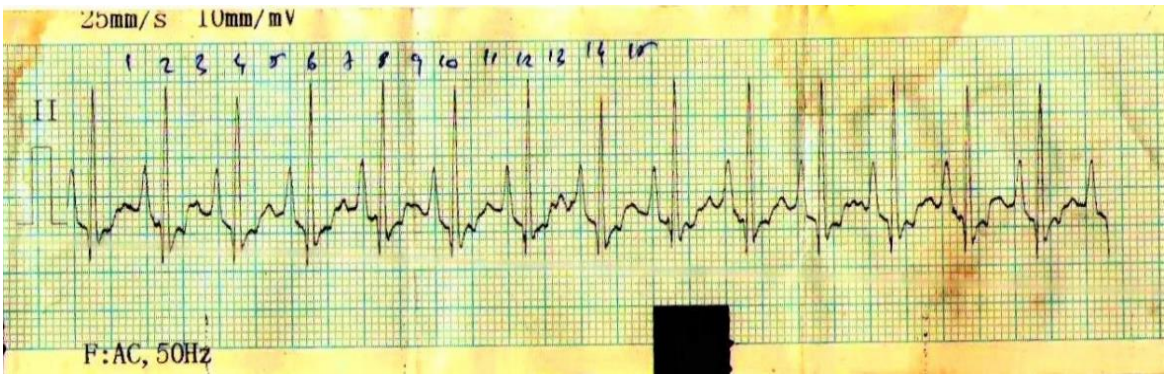


Fig 29: ECG-7 indicating increased amplitude of P wave with increased HR (Tachycardia)

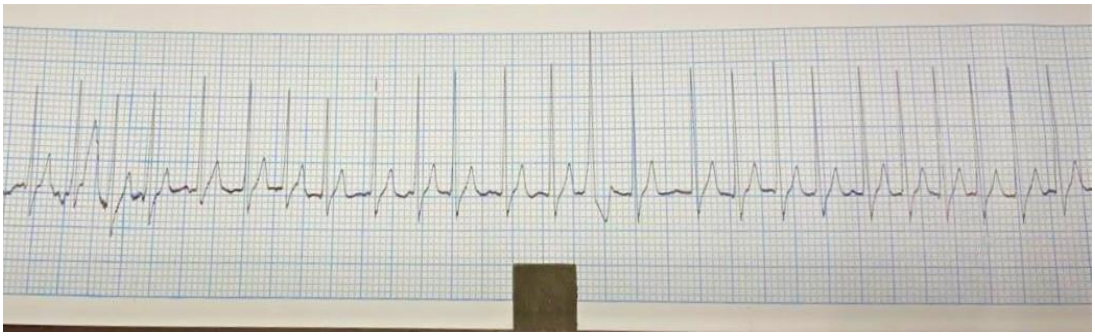


Fig 30: ECG-8 VPC, elevated T amplitude and S-T coving.

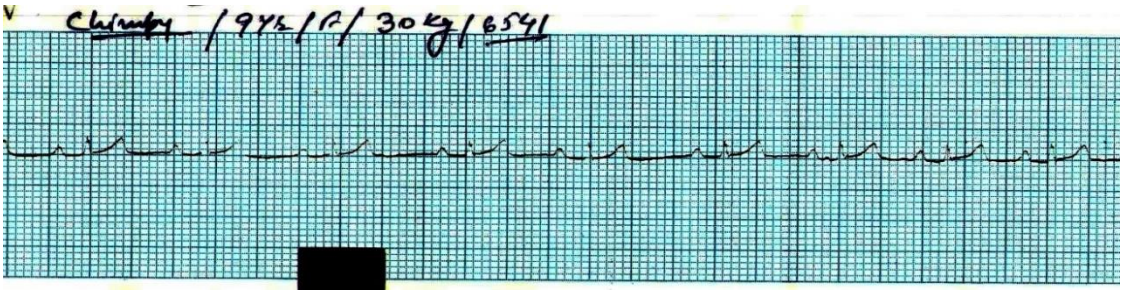


Fig 31: ECG-9 flattening of P-QRS-T wave.

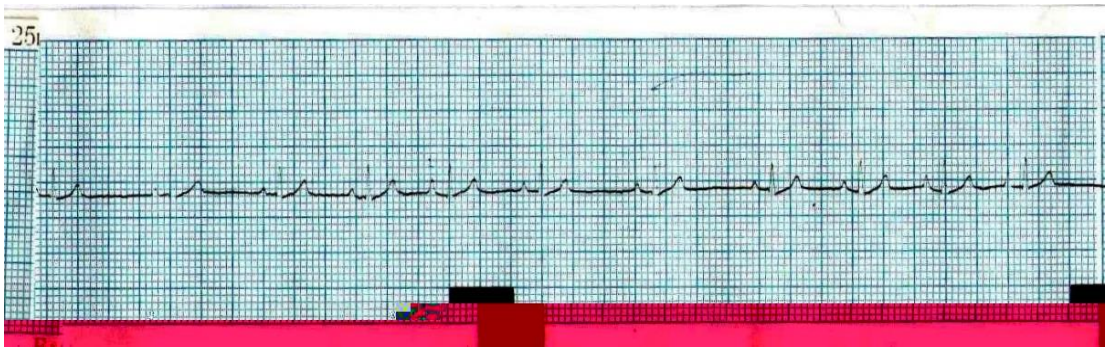


Fig 32: ECG-10 indicating flattening of P-QRS-T wave

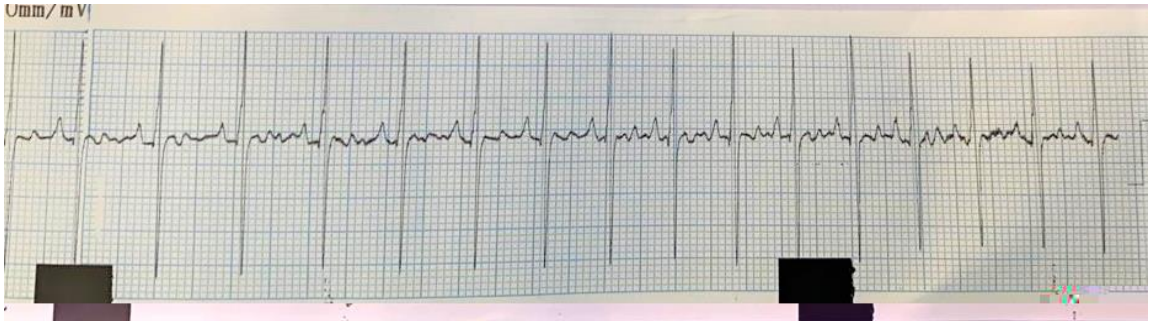


Fig 33: ECG-11 indicating increased (deepening) of S wave.

Thoracic Radiograph:

Thoracic Radiograph in the present study was one of the most important tools in the diagnosis of Cardiac disease in geriatric dogs. important information of cardiac problems like cardiomegaly chamber enlargement and the elite cardiac silhouette changes were assured by taking thoracic radiograph in lateral and ventrodorsal position and the result was interpreted in the tabular form and photographs. A perusal of the table and figure shows that cardiothoracic index in the present study was recorded up to 71.32 percent and VHS from 11 to 13 suggestive of cardiomegaly. The clock face analogue of the heart in lateral view suggestive of overall heart chamber enlargement (3), right ventricular enlargement (8), followed by left auricular and left ventricular enlargement in 4 cases each. (**Table no.24**)

Snap 4DX:

In the present study on 30 dogs snap 4DX test was done for evaluation of Heart worm (*Dirofilaria immitis*) and other haemoprotozoan disease and it was found that 4 geriatric dogs were suffering from heartworm disease and 17 were suffering from *ehrlichiosis* (*Ehrlichia canis)* 4 were found positive for anaplasmosis *(Anaplasma phagocytophilum).*

Cardiac troponin:

Cardiac troponin test was done in 30 geriatric dogs, 10 healthy and 20 suffering from congestive heart failure. The cardiac troponin test was found to be positive in 8 dogs suffering from congestive heart failure and cardiomegaly.

Table 24: Cardiac chamber enlargement of geriatric dog suspected for cardiopulmonary diseases

Left auricular enlargement	**4**
Right auricular enlargement	**1**
Left ventricular enlargement	**4**
Right ventricular enlargement	**8**
Overall	**3**

Fig 34: Cardiac chamber enlargement of geriatric dog suspected for cardiopulmonary diseases

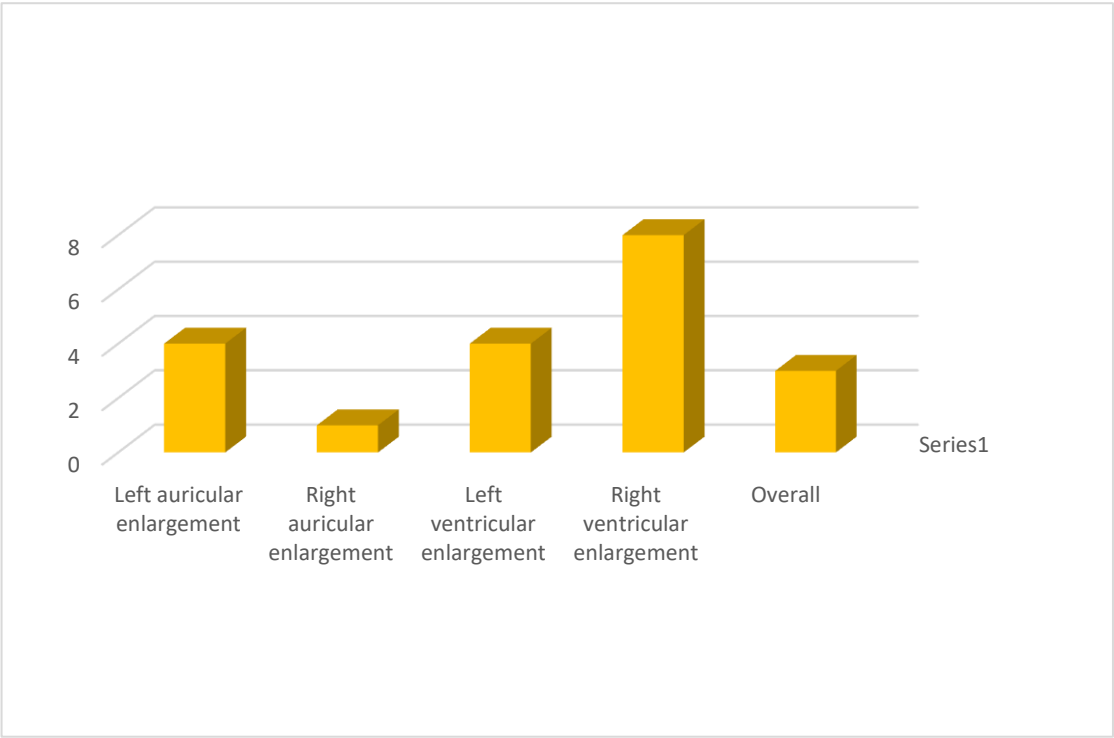

8
6
4
2
0
Left auricular enlargement
Right auricular enlargement
Left ventricular enlargement
Right ventricular enlargement
Overall
Series1

Fig 35: Lateral Radiograph of a dog showing left atrial enlargement

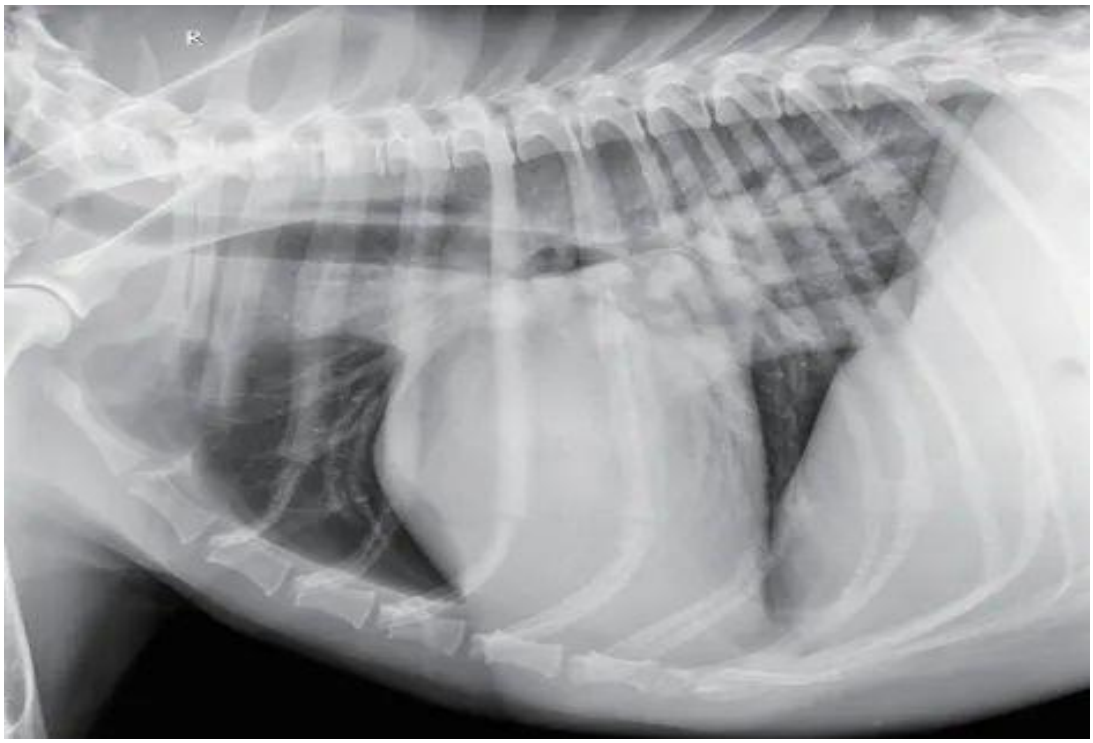


Fig 36: VD view of a dog showing left atrial enlargement

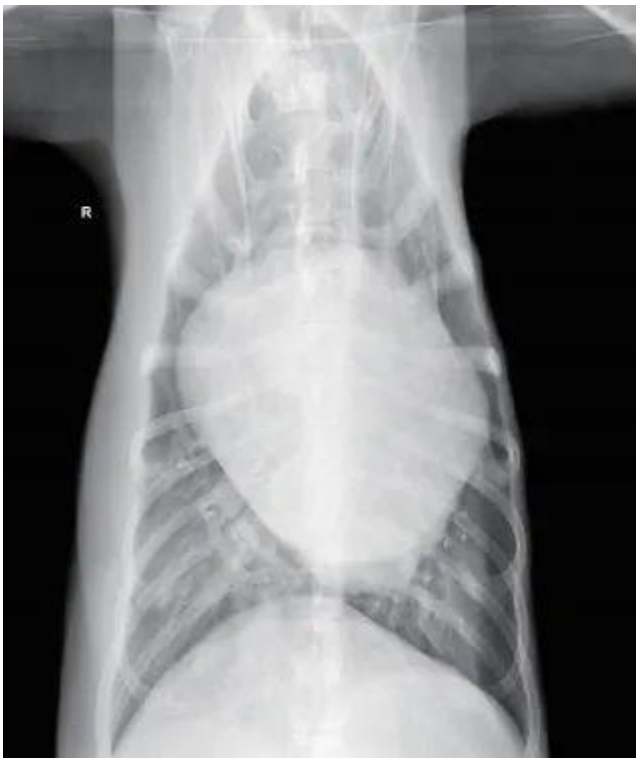


Fig 37: Lateral Radiograph of a dog showing left atrial enlargement

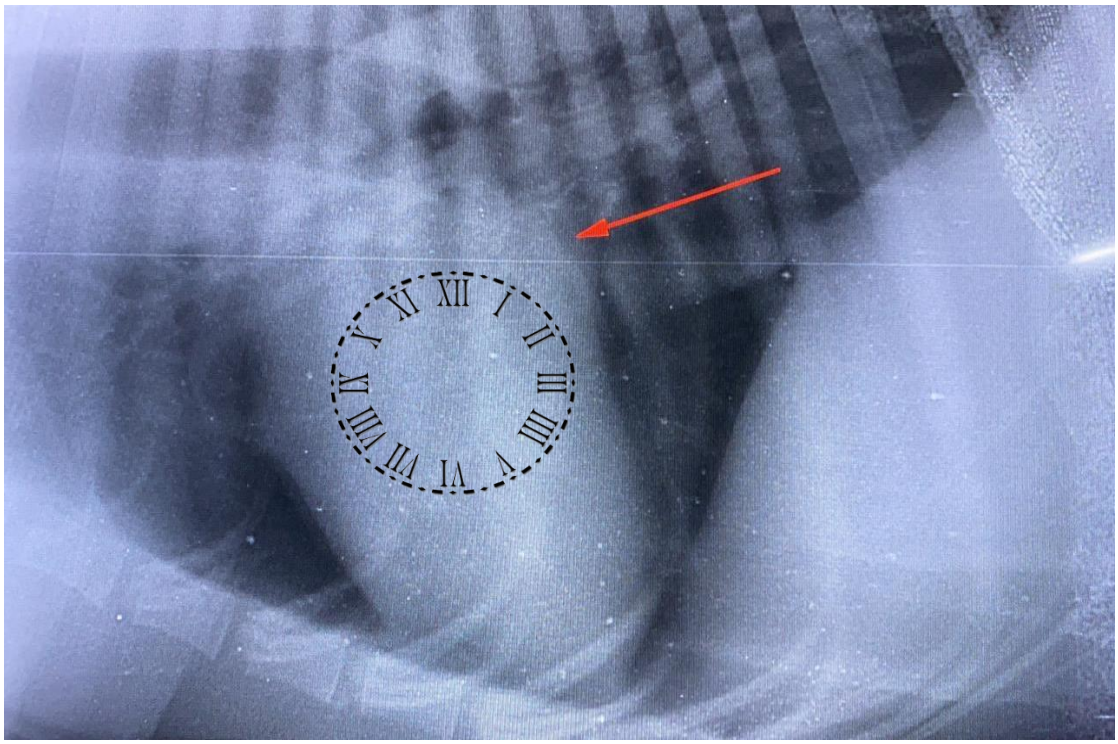
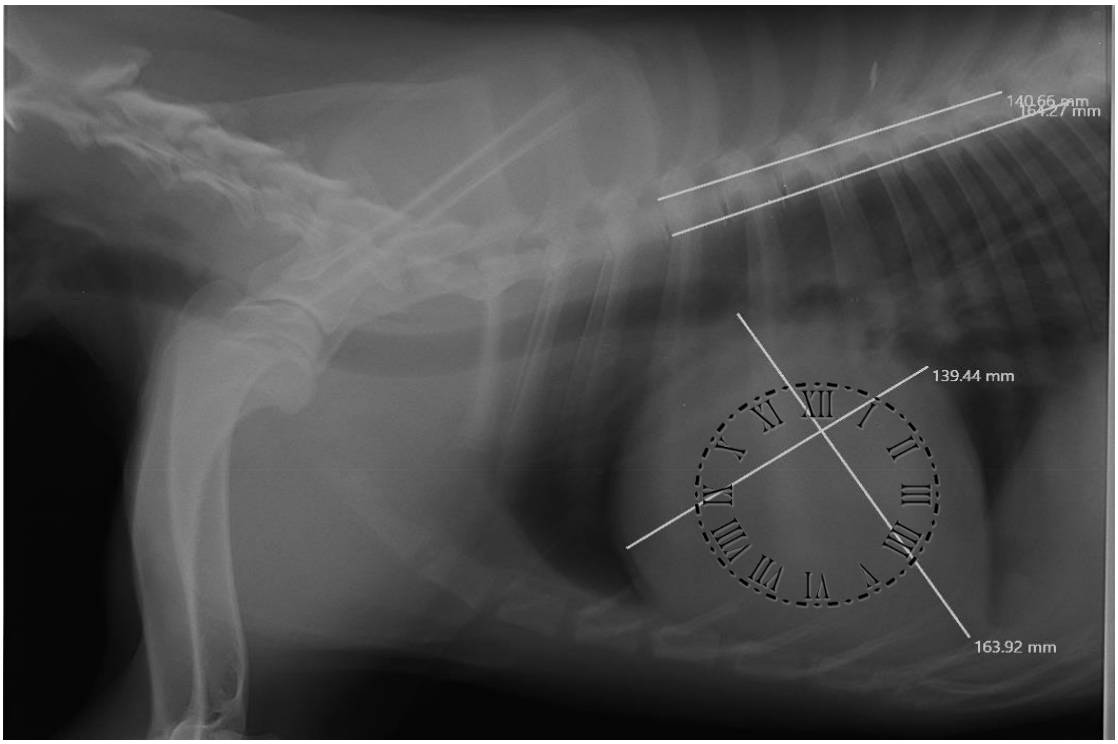


Fig 38: Lateral Radiograph of a dog showing overall enlargement of



heart

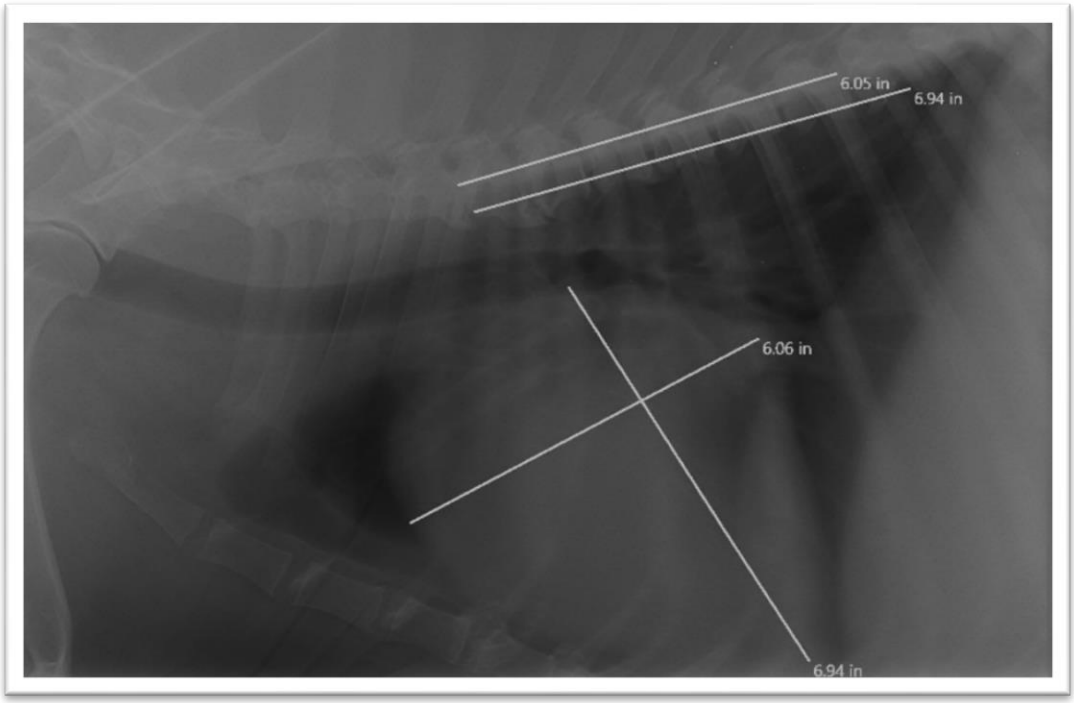


Fig 39: Lateral Radiograph of a dog showing overall enlargement of heart

Fig 40: DV of a dog showing overall enlargement of heart

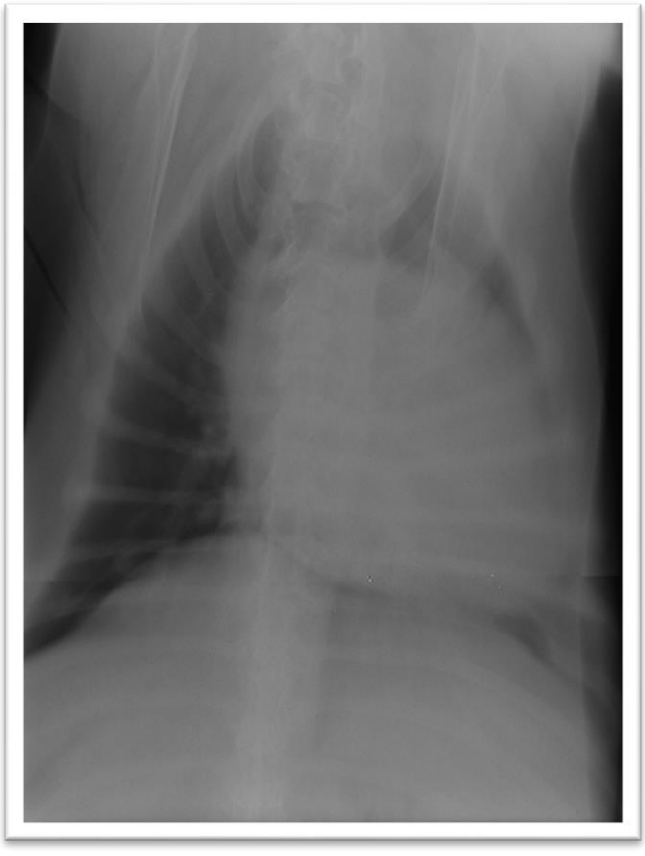
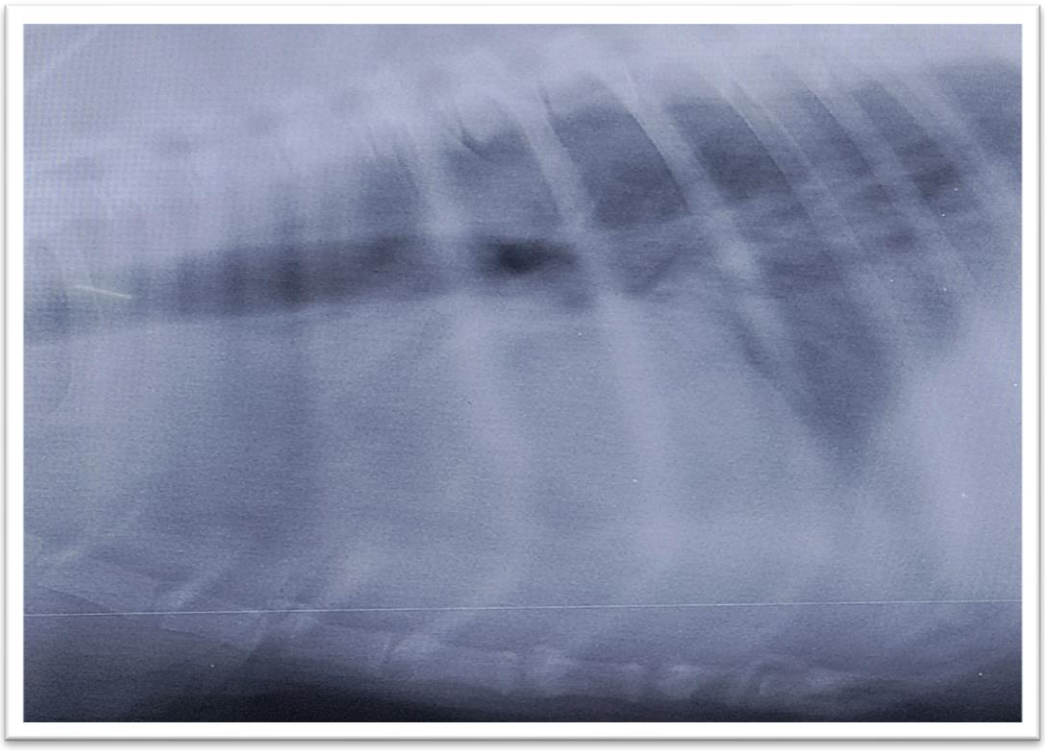


Fig 41 and 42: Lateral and DV view Radiograph of a dog showing Pleural Effusion



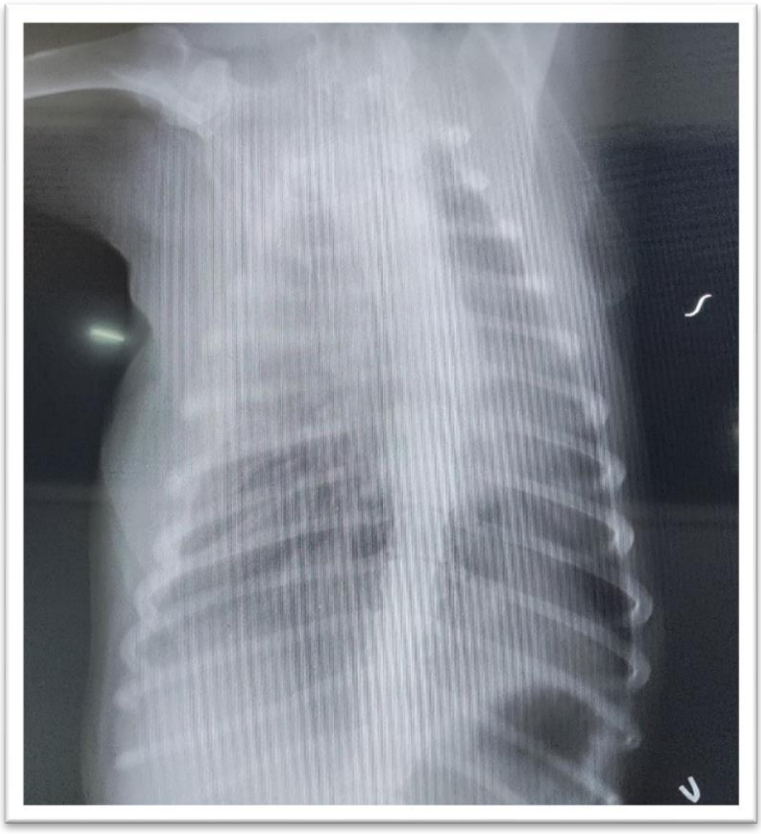


Fig 42

Fig 43: X-Ray (D-V View) showing Cardio-thoracic index 71.32%

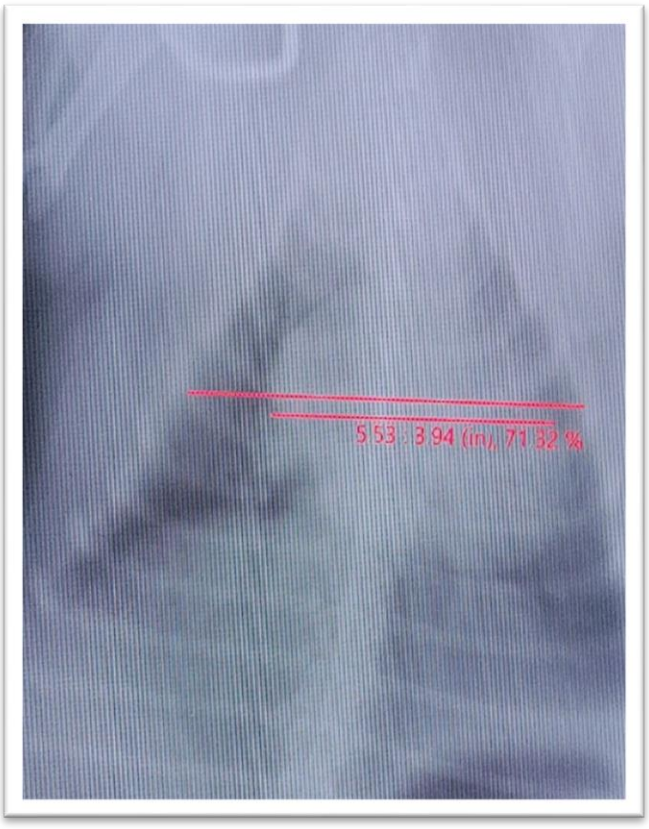


Fig 44: Dog with CHF, exhibiting signs of ascites and cachexia.

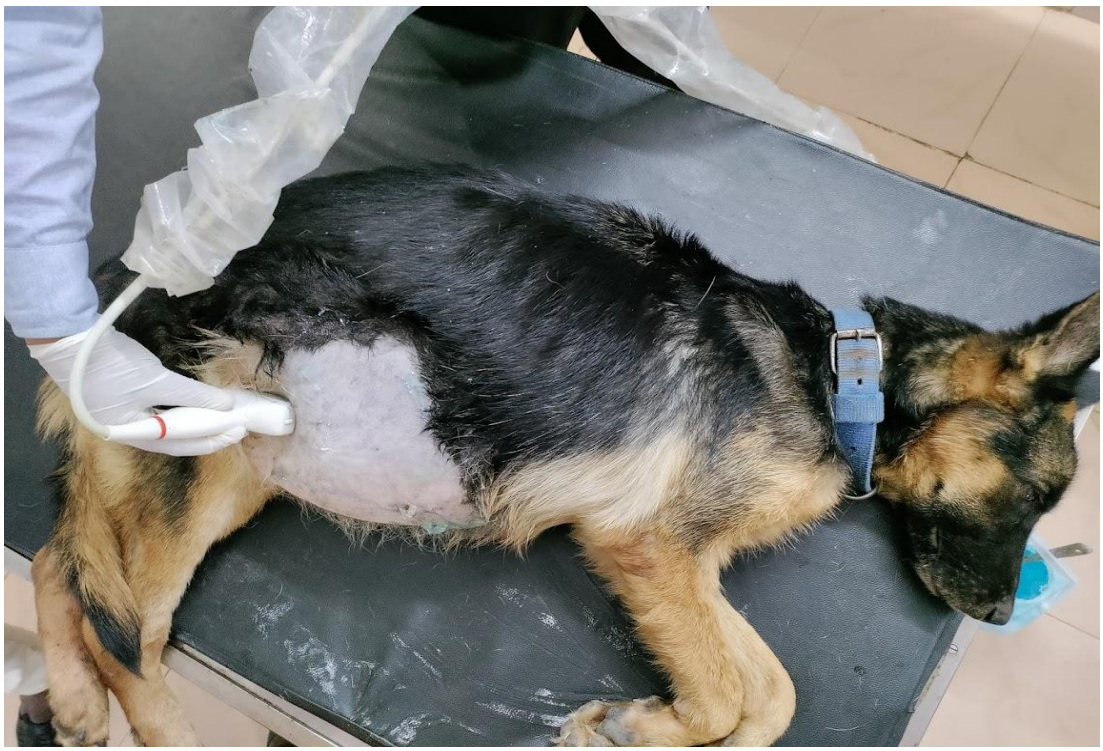
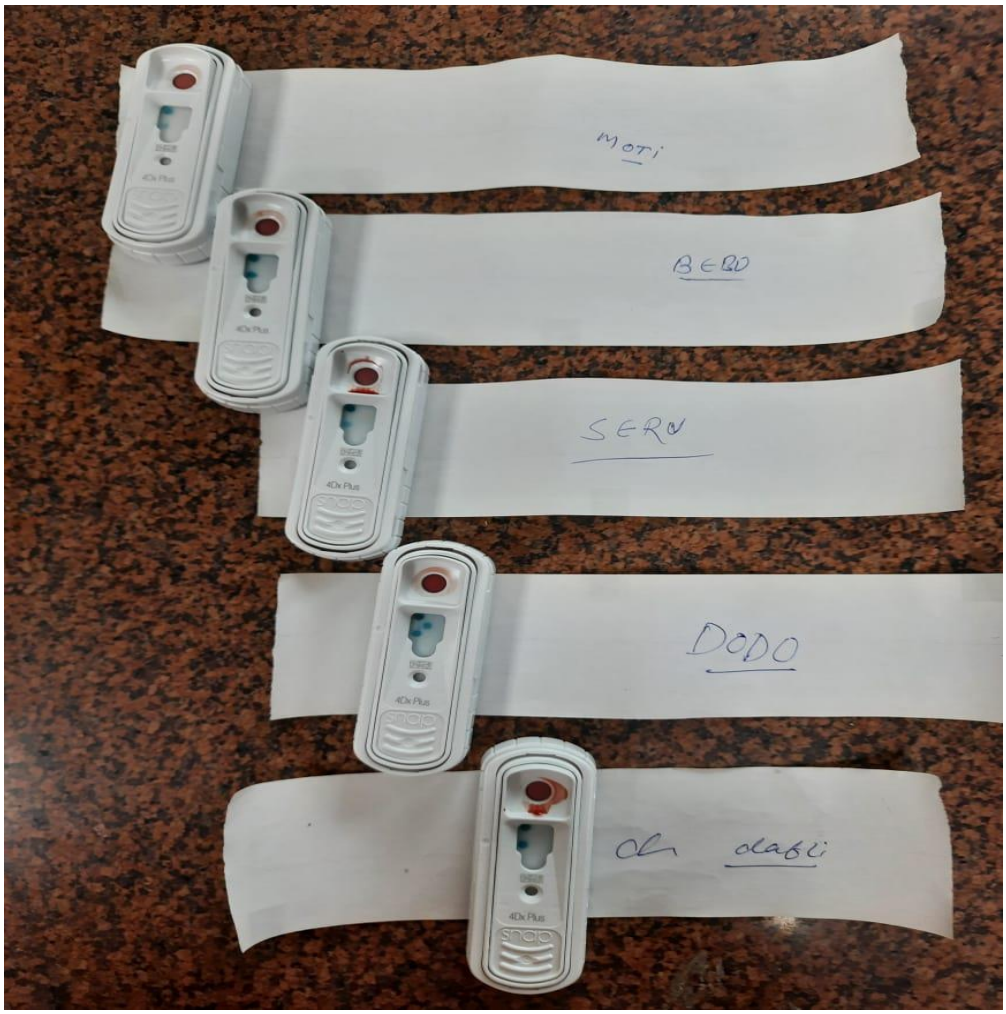


Fig 45: Cardiac troponin-I

Fig 46: Snap 4Dx



Discussion

During the period of research from February to September 2021 out of 7812 cases of dogs presented to clinical complex 1925 geriatric dogs were found. Out of 1925 cases of geriatric dogs 519 (27%) dogs were suspected to be found to suffer from cardiac diseases. Our findings are in corroboration with the findings of Atkinson *et al*. (2009) who reported 13.3% to 30% of cardiac diseases in geriatric dogs. Echelberg and seine (1996) reported 16.3% of cardiac diseases in geriatric dogs however Amar (2014) reported 6.25% of cardiac diseases in geriatric dogs. These differences may be due to change in geographical climatic and nutritional conditions in different parts of the world.

Rebecca (2008) mentioned that males are more prone to cardiac diseases in comparison to females. Similar results were found in our study in which 56% of males were found to be suffering from cardiac diseases. Our findings are also in agreement with the finding of Thirunavukkarasu (2019) who reported Labrador and Spitz were more

affected with cardiac diseases due to dilated cardiomyopathy (DCM) and mitral valve diseases (MVD) respectively and also older male dogs were found affected with Acquired Heart Diseases (AHDs). This may be due to more preferences of these breeds by the owner of Patna.

Feeding was the important findings in geriatric dogs and it was found that 17 dogs with cardio pulmonary signs were on commercial feed, while 22 were on mixed (commercial and homemade food) while the rest 11 where exclusively on homemade food, this may be due to low cost commercially available food, convenience and less time consuming (Carciofi 2006 and Vendramini 2020).

The Nutritional condition of dog suffering from cardiac disease were found cachectic while few dogs were very obese. It was observed that the geriatric dogs with cardio pulmonary dysfunction has lower body condition score (21). Cardiac cachexia in dogs suffering from cardiac disease may be due to weight loss which may be due to loss of amino acid from muscles to full fill the source of energy. The cardiac cachexia is reported by Devi *et al.* (2009) may be due to DCM, particularly with right side heart failure.

Mucous membrane examination was a significant finding in the evaluation of cardiopulmonary diseases. 2 geriatric dogs were found to be cyanotic with a capillary refill time of more than 3 to 5 seconds. Dryness (17) of mucous membrane with petechiae (09) was the important finding in screening. Physical examination of mucous membrane is in agreement with earlier reports of Chaudhary *et al.* (2008). Which may be due to anorexia, increased cytokines, renal diseases (Kumar *et al.* 2011) and may be due to haemoprotozoan diseases found in this area. Lymph node swelling were found in 21 geriatric dogs with cardiopulmonary signs. The present finding of lymph nodes swelling may be due to high ticks' infestation in dogs and presence of *E. Canis* and other haemoprotozoan diseases.

Epistaxis (6) were found in dogs under screenings while rails (6) whzeels (8) crackling (3) and dullness (4) were found in other dogs. Coughing (17) and respiratory distress (18) were the important findings in dogs with cardiopulmonary signs. The present findings concur with earlier reports of Martin *et al*. (2010) and Jeyraja *et al.* (2008)

Continuous labored breathing (9) occasional (7) and labored breathing during exercise (13) were the important findings in dogs with cardiopulmonary diseases. Nocturnal coughing (14) that is coughing during rest were represented from the dogs while ascites (16) was the important finding during the screening. Edema in limbs was found in 07 dogs and jugular vein pulsation in 14 dogs were present. The clinical sign of fluid accumulation in limbs and ascites in geriatric dog may be due to the low cardiac output in congestive heart failure (Martin *et al.* 2010 and Anju *et al.* 2011). Devi *et al.* (2009) in a study in Gujarat reported nocturnal coughing 2.5% and exercise intolerance 1.82 percent, hepato-jugular pulsation and swelling of abdomen as the prominent clinical symptom in geriatric dog.

Elevation of serum creatinine in geriatric dogs with congestive heart failure may be due to poor perfusion, arterial stenosis which reduces glomerular filtration, and thereby leading to accumulation of nitrogenous waste creatinine (Ettinger and Feldman 2005). The present finding is also in agreement with the finding of Jan *et al.* (2018) who reported an elevated level of creatinine up to 3.01 mg/dl and mean BUN 29.58 mg/dl in heart failure cases.

The present finding of the elevation of blood urea nitrogen agrees with the finding of Jan *et al.* (2018) who reported a mean BUN 29.58 mg/dl in heart failure cases. Renal congestion is a major contributing factor to renal dysfunction in heart failure. There is a co-existence of renal and cardiac dysfunction, where dysfunction in one organ induces acute or chronic dysfunction in the other (Deferrari 2020).

In the present study non-significant increase in total serum protein in the geriatric dogs with congestive heart failure was observed. Our findings are not in agreement with the findings of Martin *et al*. (2009 and 2010) which may be due to elevated level of globulin.

Low level of albumin was found in our study, similar findings was reported by Alexa *et al.* (2014) in elderly human patients with congestive heart failure which may be due to increased volume distribution, stasis in circulation and altered protein metabolism in the liver. Cachexia and malnutrition, diffuse inflammation, increased urinary loss and plasma volume expansion was reported to be cause of hypoalbuminemia (Lourenco *et al*. 2009, Zamora *et al*. 2012, Adlbrecht *et al*. 2008).

High globulin level is noticed in our study of congestive heart failure cases were also represented in human patients with congestive heart failure Li *et al.* (2018). Increased level of globulin could serve as a marker of chronic inflammation response and reflect cumulative exposure of various pro-inflammatory cytokines.

The geriatric dogs suffering from congestive heart failure have elevated bilirubin levels. Non-significant decrease in mean bilirubin level was observed in dogs on the 15th and 30th day of treatment. The elevated bilirubin is the common biochemical abnormalities in the congestive hepatopathy secondary to acute decompensated congestive heart failure (Chintanaboina *et al*. 2013).

The mean Level of CK-MB in geriatric dogs suffering from Cardiac disease was found to be elevated from a normal healthy control. The mean level of CK-MB was found to be 72.05 and 72.78 in both treatment groups. A significant reduction in CK-MB has been observed in both treatment groups on the 30th days of treatment with maximum improvement in the T-2 group. There are three types of creatinine kinase (CK) in which the CK-MB is found in the myocardium **(**Thrall 2004). The present findings corelates with findings of Schober *et al.* (1999) who represented elevated CK-MB in dogs suffering from congestive heart failure and dilated cardiomyopathy which may be due to myocardial injuries. A similar finding was also represented by Gavazza *et al.* 2020 the elevation in CK MB level may also be due to low cardiac output in congestive heart failure dogs (Dukes-Mc Ewan *et al.* 2003)

The low level of mean sodium was found in geriatric dogs with congestive heart failure. Improvement in the mean sodium level was found in the treatment group on 15 and 30 days of treatment. The present study was not per the findings of Dei cas *et al*. (1995) who described sodium within the normal range in the geriatric dog with congestive heart failure. However, the sodium in lower range was found, this might be due to reduced flow of blood and enhanced tubular reabsorption of sodium. Despite an increase in the total body sodium hypoproteinaemia might develop.

Elevated level of potassium was found in geriatric dog suffering from congestive heart failure. significant decrease in in the mean potassium level was observed in T-2 and T-3 group with maximum improvement in T-2 group. The present finding is in accordance with the finding of Macdonald and Struthers (2004) which stated that patients with congestive heart failure have moderate to severe renal insufficiency which

might results in moderate hypokalaemia. Knight (1995) also represented increased potassium bready arrhythmia in dogs suffering from congestive heart failure. Priyanka (2015) represented the similar finding in geriatric dog suffering from congestive heart failure she represented the mean potassium level of 6 m mole/litre.

The lower level of mean calcium level was found in geriatric dogs suffering from congestive heart failure. Improvement in the mean calcium level was observed in both treatment groups with the maximum in group T-2 which may be due to improvement in appetite decrease cardiac load and better cardiac function in congestive heart failure. No direct evidence of calcium deficiency and congestive heart failure was established but indeed the relationship between vitamin D deficiency and congestive heart failure in people Kraus *et al.* (2014).

Lower mean hemoglobin and PCV were recorded in a geriatric dog suffering from congestive heart failure and cardiomegaly. Non-significant decrease in hemoglobin and PCV compared to healthy geriatric dogs were recorded by Jan *et al*. (2018). Lower hemoglobin and PCV in the present study might be due to the high prevalence of E. Canis and anaplasma infection in the geriatric dog suffering from congestive heart failure and dilated cardiomyopathy. Acute infection with *E. canis* is a risk factor for myocardial injury severity of anaemia contribute to the pathophysiology of myocardial damage Diniz *et al.* (2008). As per Kittleson (1998) *E. canis* is considered a potential cause of cardiomyopathy.

Significantly lower mean platelets found in dogs suffering from cardiac diseases 0 days of treatment. improvement in the min platelets was found in both groups of treatment on the 30th day of treatment with maximum improvement in the mean platelets were observed, T-2 group. Lower platelets in dogs suffering from cardiac disease in the present study might be due to the presence of *E. canis* infection in about 56% of geriatric dogs suffering from congestive heart failure. Thrombocytopenia was found to be the important characteristic feature of the disease (Srikala *et al*. 2012, Bhardwaj 2013**).** The decreased lifespan of platelets and increased splenic slaughtering also led to thrombocytopenia (Harrus *et al.*1999). As per Diniz *et al.* (2008), *E. canis* infection was a risk factor for cardiac injury associated with severe anaemia. No direct evidence established between thrombocytopenia and congestive heart failure

The mean level up SAP and DAP were found to be elevated in dogs suffering from congestive heart failure. Significant decreases in SAP and DAP were observed in both treatment groups which may be due to decrease pressure overload on the heart, redistribution of blood, and decrease vascular resistance. A similar finding was found by Amar (2014) in both systolic and diastolic values in a geriatric dog suffering from heart disease. The present findings also agree with the finding of Merus *et al.* (2000). This may be due to increased pressure and volume load on the heart which leads to low cardiac output and impaired myocardial contractility (Martin 2009) however the present findings were not supported by James (2011) who could not find any significant difference in blood pressure between normal and DCM dogs.

Present finding of ECG in geriatric dogs (N= 50) suspected for Cardiac disease and the average weight of 25 to 35 kg shows that that mean heart rate as 103.75 beats per minute maximum 240 to a minimum of 60 beats per minute. Rao *et al.* (2008) and Tilley (1992), mentioned that heart rate of 70 to 160 beats per minute in normal healthy dogs. In DCM and congestive heart failure muscles fail to pump blood and blood flow is slowed down due to the heart's inability to contract and as a compensatory mechanism to the sympathetic nervous system, stimulation heart rate is increased (Aiello *et al.* 2016**,** and Durham 2017). The mean P amplitude was found in the normal range while the mean P duration was found to be increased from the healthy control. Increased P-wave duration in our present findings is one of the indications of atrial enlargement and indicating mitral valve insufficiency. However other parameters like double-peaked or notched P-wave, increased P duration, increased P-wave area, increased P-wave duration, and PR interval ratio increase in the ratio of the P wave duration to the PR segment. A prolonged P-wave would represent a criterion for the left atrial enlargement in both human and veterinary medicine. Macruz *et al.* (1958), Mirvis and Goldberger (2007), Zeng *et al.* (2003), Tilley (1992).

The mean PR interval in ECG finding was found to be 0.13 seconds which is although high but in the normal range. If the PR interval is greater than 0.14 sec, it is an indication of first-degree heart block (incomplete heart block). The mean QRS duration and R amplitude were within the normal range. The mean T amplitude and duration were found to be within the normal range, however, the Q wave deepening mean was found to be 0.56mv which is above the maximum limit of Q wave deepening, indicative of right ventricular enlargement (RVE). Represents the initial phase of ventricular depolarization

abnormal Q wave deepening in geriatric dogs indicate myocardial infarction, (Birnbaum *et al.* 1997). Deep and narrow Q waves have been observed in dogs affected with Duchenne's muscular dystrophy (Moise *et al.* 1991). The electrocardiogram finding in the present study and specific abnormality as detected in ECG findings were described by Hoque *et al.* (2019). This technique was widely used in dogs for monitoring heart rate, cardiac rhythm, conduction integrity, and in detecting cardiac abnormalities (Mukherjee *et al.* 2015), Tilley (1985). In the present findings, cardiac arrhythmia was found in some cases as described by Gugjoo *et al*. (2014). Variation in P-wave, R-wave and deep Q wave in the present finding were in corroboration with the findings of Pereira *et al*. (2004). Deep S-wave in the present study indicates right ventricular enlargement and is reported in pulmonary stenosis with severe right ventricular hypertrophy Fox (1999). Thus, the electrocardiographic changes observed in the present study were increase amplitude of T-wave, P-wave, deep Q and S-wave ventricular premature complexes low voltage QRS complex suggestive of cardio pathology

In the present study 8 cases of geriatric dogs suffering from cardiac disease was found to be positive for cardiac troponin I. The present finding is in conformity with the finding of Adams *et al.* (1994), Martin *et al.* (1996), and Collinson *et al.* (2001), who found that cardiac troponin I as a most specific indicator of myocardial damage in laboratory animals and human being. Sleeper *et al.* (2001) also found the same result in dogs and cats and established corelation with myocardial damage and blood cardiac troponin I level. This may also be due to *E. canis* infection as reported by Osathanon *et al.* (2013), Diniz *et al.* (2008). Our findings are in agreement with the findings of Diniz *et al.* (2008) who found elevated and positive cardiac troponin I in dogs suffering from canine ehrlichiosis. Similar cardiomyopathy in dogs was also reported by Bai *et al.* (2016), Lakkawar *et al.* (2003).

Thoracic radiography is the most important diagnostic tool for the diagnosis of cardiac diseases (Thrall 2007). Changes in cardiac size as observed in the present study were in conformity with the findings of Buchanan and Bücheler (1995). Hamlin (1999) cardiac changes in the present study however accurate diagnosis were sometimes difficult to assess Kittleson (1998), Lamb and Boswood (2002). The vertebral heart score (VHS) to measure the heart size in dogs with thoracic radiograph in the present finding is in conformity with Bodh *et al.* (2016) and Gugjoo *et al.* (2013). The normal range of VHS in dog was reported to vary from 8.7 to 10.7 (Lamb and Boswood 2002). Increased

VHS in the present study is in conformity with the findings of Lamb *et al*. (2001). Generalized heart enlargement (cardiomegaly) and left atrial enlargement in the present study were in conformity with the findings of mitral valve insufficiency Salguero *et al.* (2018). Cardiomegaly was also reported by Mc Ewan (2000) and Martin *et al.* (2010).

Snap 4DX plus rapid test kit were done to diagnose the case of heartworm and vector-borne disease in canine. The snap 4DX plus test kit detects the antibody for infection with multiple thick borne pathogens and canine heartworm antigens in a single assay. In the present study, this test detected *Dirofilaria immitis* (n = 4), *Borrelia burgdorferi* (n= 0), *Anaplasma phagocytophilum* (n = 4), and 17 dogs were suffering from *Ehrlichia canis* and *Ehrlichia ewingii*. Liu *et al.* (2018) found that snap 4DX was found to be 98% sensitive in detecting vector-borne disease in canines. *E. canis* infection in the present study would be responsible for cardiac problems in geriatric dogs. Similarly canine heartworm disease was also detected which may be responsible for the cardiomegaly and congestive heart failure. Heartworm disease was reported in the endemic area Bowman and Mannella (2011) and the snap 4Dx has been validated extensively for simultaneous detection of the antibody of *Anaplasma phagocytophilum*, *E. canis*, *E. ewingii*, *Borrelia burgdorferi* and *D. immitis* antigen. Chandrashekar *et al*. (2010).

In the present study the clinical cases in groups T2 and T3 shows sign of improvement between 15 to 30 days of treatment. After 30 days of treatment, clinical signs were minimized coughing and exercise intolerance decreases. The present finding is in agreement with earlier workers Mc Ewan (2000), Thomason *et al.* (2007), Atkinson *et al.* (2009) and Martin *et al.* (2010). Improvement in clinical condition was better observed in group T2 on pimobendan at the rate of 0.25 mg per kg body weight, ramipril at the rate of 0.125 mg per kg body weight, furosemide 2-6 mg per kg body weight, and spironolactone 1-2 mg per kg bodyweight, then group T3 on digoxin 0.125 mg per kg body weight benazepril 0.25- 0.5 mg per kg body weight and furosemide. Better and faster improvement in biochemical parameters like reduction blood urea nitrogen and creatinine, increase in sodium and decrease in potassium, improvement in total protein and albumin decrease in globulin and negative of cardiac troponin I and decrease in the CK-MB level were observed. The PCV hemoglobin and platelet count increases in both groups but faster and earlier improvements were noticed in geriatric group T2.

The improvement in clinical parameters may be due to a reduction in blood volume and ventricular diastole volume with furosemide, venodilation, and reduced renal sodium and water retention by decreasing aldosterone secretion action of ramipril, and the positive inotropic effect of pimobendan. Spironolactone in group T2 works in conjugation with furosemide diuretics and acts as the antifibrotic effect in the myocardium. Pimobendan is a novel inodilator, it works well in dilated cardiomyopathy and valvular heart disease in dogs. Pimobendan sensitizes the myocytes to calcium thus increasing the contractile strength of the cardiac muscle and at the same time increasing the oxygen consumption by the coronary artery dilation of the heart. our findings are in agreement with the findings of Martin (2003), Kanno *et al.* (2007), Thomason *et al*. (2007), Martin *et al*. (2010).

Summary and Conclusion

The study on radiographic and electrocardiographic changes with special reference to therapeutic evaluation of congestive heart failure in geriatric dog was conducted in the department of veterinary medicine and veterinary clinical Complex of Bihar Veterinary College, Bihar Animal Sciences University for a period of 6 month from February 2021 to September 2021 with the following objectives

- To evaluate the cardiac disease in geriatric dog with ECG and Radiography.
- To study the endogenous cardiac marker changes in congestive heart failure in dogs.
- To evaluate the therapeutic efficacy of positive inotropic, diuretics and ACE inhibitors in congestive heart failure in dogs by ECG, radiographic and endogenous markers.

Out of 1925 geriatric dogs presented to the clinical complex with 519 dogs were suspected for cardio pulmonary diseases. The detailed study of 50 geriatric cases were done on different parameters and 20 dog suffering from cardiomegaly with congestive heart failure were divided into two therapeutic groups 10 each and hematobiochemical, radiographic and, ECG findings were studied. Data collected from apparently healthy geriatric dogs on various parameters served as a control value in the study.

1. Clinical and the screening study reveals that Labrador and German Shepherd breeds ware highly susceptible for cardiac diseases.

2. Most of the dog in the age group of 7 to 9 we are found to suffer from cardiac disease and also the male was more susceptible.

3. Exercise intolerance and nocturnal coughing were the important history of cardiac disease in geriatric dogs.

4. Stiffness of gait, long standing condition, restlessness, juglar pulsation, change in PMI, abdominal respiration was the important clinical sign observed.

5. Laboured breathing, ascites and edema of legs were the other important clinical findings in dogs.

6. Elevated level of creatinine, blood urea nitrogen, total protein, globulin, potassium, bilirubin, SAP, DAP and CK-MB were found in dogs suffering from cardiac diseases.

7. Lower level of albumin, sodium, hemoglobin, PCV and, platelets were found in dogs suffering from cardiac diseases.

8. 40% of dogs were found to be positive for cardiac marker cardiac troponin I where as 13.3% and 56.6 % geriatric dog were found to be suffering from heartworm and *E. canis* infection.

9. ECG findings were important criteria for evaluation of dogs with cardiac diseases and it was found that deep Q-wave, arrhythmias, ST coving were the important findings.

10. Chest X-Ray also served as important diagnostic tools for cardiac disease and in the present study VHS from 11-13, cardio thoracic index of 71.32% were suggestive of cardiomegaly. In the cardiac chamber enlargement right ventricular enlargement, left ventricular enlargement and, overall cardiac enlargement where found.

11. In the therapeutic trial group T-2 on pimobendan, ramipril and spironolactone combination therapy acts effectively in controlling the congestive heart failure.

12. Thus it is concluded from this study that geriatric dogs with exercise intolerance, nocturnal coughing and, ascites must be evaluated for cardiac disease and therapeutic combination of pimobendan, ramipril, furosemide and spironolactone should be tried for the management of the disease.

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Title of M.V.Sc. thesis: *Radiographic and electrocardiographic changes with special reference to therapeutic evaluation of congestive heart failure in geriatric dogs.*