***Original Research Article***

**A COMPREHENSIVE APPROACH TO DIAGNOSING CANINE DILATED CARDIOMYOPATHY**

**ABSTRACT**

**Background:** Canine heart diseases are among the most prevalent silent threats in veterinary practice. Myocardial disease is a commonly acquired cardiac condition in dogs, with dilated cardiomyopathy (DCM) being a significant contributor to chronic illness and mortality. DCM is marked by weakened heart muscle function, ventricular enlargement, and frequent tachyarrhythmia. Early detection of DCM is crucial for timely treatment, which plays a vital role in ensuring the pet's survival. This study focuses on application of electrocardiography, radiography and echocardiography to diagnose dilated cardiomyopathy in dogs.

**Methods:** The study was conducted for a period of six months during which 4509 dogs were brought to Veterinary Clinical Complex, Jabalpur. Out of them, 109 dogs exhibiting clinical symptoms related to cardiac disorders such as ascites, obesity, abdominal distension, weight loss, persistent coughing, breathing difficulties, exercise intolerance, fainting or syncope, lethargy, swelling in the extremities, pale gums, and hind limb weakness were screened. Affected dogs were subjected to radiographic (thoracic), electrocardiographic and echocardiographic examination to diagnose the underlying cardiac disease. Complying to the score card followed, dogs suffering with cardiac diseases were categorized into two groups, viz. Group- I (dogs affected with DCM) and Group -II (dogs predisposed to develop DCM, in future).

**Result:** Amongst 109 dogs that underwent screening, 60 were found to have diverse cardiac disorders. Based on the diagnostic modalities used, six were diagnosed with dilated cardiomyopathy (DCM) however, remaining 49 dogs were evaluated to be at risk of developing DCM. Electrocardiographic examination revealed ST coving and fine atrial fibrillation along with significant increase in the vertebral heart score on thoracic radiograph was evident in all cases diagnosed to be of DCM. Dilated left ventricular lumen and enlarged left atrium were revealed by echocardiography in these six dogs. Various cardiac measurements were altered due to changes in both the structure and function of the heart, attributed to dilated cardiomyopathy.

**Key Words:** *Cardiomyopathy, DCM, Canine Cardiac disorder, Echocardiography*

1. **INTRODUCTION**

Cardiac disorders are considered as the second most frequently observed conditions following cancer (Sahoo *et al*., 2021). Canine myocardial disease is one of the most common forms of acquired cardiac anomaly in the dog (Kumar *et al.,* 2024). Amongst the myocardial diseases in dog, dilated cardiomyopathy is the most common form occurring but arrhythmogenic right ventricular cardiomyopathy, hypertrophic cardiomyopathy and myocarditis, among others, are also reported (Ettinger and Feldman, 2010). DCM is disease of unknown aetiology characterized by dilatation of one or both ventricles with severe impairment of systolic function in absence of other detectable cardiovascular disorder (Smith *et al.*, 2021). Several theories concerning genetic, nutritional, metabolic, inflammatory, infectious, drug or toxin induced myocardial diseases have been hypothecated (Kathryn, 2005). Metabolic disorders associated with DCM include hypothyroidism, diabetes mellitus and pheochromocytoma (Mavrogeni *et al*., 2015). The development of DCM appears to progress through 3 distinct phases. The 1st phase is characterized by a morphologically and electrically normal heart in an asymptomatic dog. The 2nd phase is characterized by evidence of a morphological (cardiac enlargement and reduced systolic dysfunction) or electrical (ventricular ectopy) derangement in an otherwise asymptomatic dog (occult or preclinical phase); and the 3rd phase is overt heart failure, characterized by clinical signs of forward or backward heart failure (Simpson *et al.*, 2021). The most observed symptoms of cardiomyopathy are exercise intolerance, dyspnoea, tachypnoea, and coughing (due to left sided heart failure), abdominal enlargement, jugular distension and pulsation (due to right sided heart failure), cold extremities, loss of weight, syncope, low-intensity systolic murmur, weak femoral arterial pulse and cyanosis (Parmar *et al.*, 2022).

DCM can be tentatively diagnosed by physical examination of the heart. A soft murmur consistent with mitral valve regurgitation and/or gallop rhythm (S3) may be auscultated at left apex. Tachyarrhythmia may be noted. Electrocardiographically, left atrial and ventricular enlargement, sinus tachycardia, atrial fibrillation, or ventricular tachyarrhythmias are diagnosed. Radiographic study reveals left atrial and ventricular enlargement with or without pulmonary venous distension and pulmonary oedema. Echocardiography is the diagnostic test of choice for canine DCM and is valuable in the detection of the occult disease (Yadav *et al.*, 2023).

Cardiac troponin I (cTnI) is a crucial biomarker for detecting myocardial injury and has been linked to dilated cardiomyopathy (DCM) in canines. Elevated levels of cTnI in dogs with DCM indicate ongoing myocardial cell damage and increased cardiac stress (Mukherjee, 2024). Studies suggest that higher cTnI concentrations correlate with disease severity, ventricular dysfunction, and poor prognosis in dogs with DCM (Akgul, 2023 and Romito, 2024). The elevation of cTnI may result from cardiac myocyte apoptosis, inflammation, oxidative stress, or ischemia, which are commonly associated with the progression of DCM. Additionally, measuring cTnI levels in canine patients can aid in early diagnosis, monitoring disease progression, and assessing treatment response.

Clinical management of the patients is aimed at controlling congestive heart failure, treating arrhythmias, preventing pulmonary and systemic emboli and often, at managing chest pain. Basic treatment for congestive heart failure resulting from dilated cardiomyopathy consists of ionotropic therapy, preload reduction and afterload reduction (Atkins *et al.,* 2009). It is important that every dog considered vulnerable to cardiac diseases should be examined for cardiac function during routine examination. A delay in diagnosis and treatment could result in a grave prognosis or life-threatening condition. The availability of echocardiographic facilities in veterinary practice has transformed cardiology by enhancing the diagnosis and treatment of cardiac diseases in dogs.

1. **MATERIALS AND METHODS**

A total of 109 dogs having clinical signs pertaining to cardiovascular disorders were screened out of 4509 canine cases registered at the Veterinary Clinical Complex, Nanaji Deshmukh Veterinary Science University, Jabalpur during a period of six months. The present study was undertaken to systematically detect dogs having dilated cardiomyopathy and other associated cardiac diseases by echocardiography in dogs and to correlate the echocardiographic findings with electrocardiographic and radiographic findings. A score card devised was used for categorically divide the subjects into dogs having DCM and the ones which are likely to develop DCM, in future.

The dogs first underwent an electrocardiographic examination to evaluate abnormalities in heart conductivity. Those showing deviations from normal conduction activity were further examined using echocardiography and radiography. Biochemical parameters including CK-MB, cardiac troponin and blood sugar were also recorded.

**Electrocardiographic examination**

Electrocardiographic recordings were obtained using Cardiart8408View® electrocardiography machine (BPL Limited) in a quiet, distraction-free environment. The dog was positioned in right lateral recumbency on an insulated surface, with its legs perpendicular to the body's long axis. After shaving the designated areas, a generous amount of coupling gel was applied and the electrodes were securely attached to the dog's skin. The ECG was recorded at a paper speed of 50 mm/sec. Prior to each recording, a one millivolt standardization pulse was applied. A prolong rhythm strip was obtained in lead II while four to five beats were captured for each lead. Standard guidelines given by Tilley and Smith (2016) were followed measuring complexes and intervals and also for interpretation of electrocardiograms.

**Radiographic examination**

Digital X-rays were taken to acquire orthogonal views of thoracic cavity and evaluate changes in the cardiac silhouette using a 100 mA X-ray machine and a CR system. The animal was positioned in left lateral recumbency and dorsal recumbency to capture left lateral and ventro-dorsal views, respectively. For these views, the sternum and spine were aligned in the same horizontal plane, and the front legs were extended cranially as far as possible. Exposure times and kilovoltage were adjusted based on the technique chart, and all radiographs were assessed empirically.

***Measurement of vertebral heart score (VHS)***

The evaluation of cardiac radiographs was based on the general principles outlined by Buchanan and Bucheler (1995). The vertebral heart score (VHS) assesses the heart in the lateral view relative to the dog's thoracic vertebrae. Measurements were conducted following the detailed procedure described by Sawhney *et al.* (2022). The maximum short axis (S) of the heart was measured at the widest point of the cardiac silhouette on a line perpendicular to the long axis (L) at the level of the caudal vena cava and compared this to the thoracic vertebrae, again starting from the 4th thoracic vertebra (T4). Vertebral Heart Score was calculated by using the formula: V.H.S. = L+S = No. of vertebrae traversed.

***Measurement of cardio-thoracic ratio (CTR)***

The Cardio-thoracic ratio (CTR) was determined by measuring the maximum width of the heart silhouette and comparing it to the distance between the thoracic walls on the same plane, following the methodology outlined by Mohammadyar *et al.* (2019).

**Echocardiographic examination**

The echocardiographic examination of the dogs was carried out to evaluate the structural and functional alterations. The animals were kept in minimal stress conditions, so that the evaluation would not be affected. The hair from the thoracic area were clipped, usually between 3rd - 5th inter-costal space on right side. Adequate amount of coupling gel was applied after clipping, for echocardiographic evaluation. Evaluation of all the dogs was performed by using sector transducer having frequency 2-4 MHz by using the ultrasonography machine. Assessment of the cardiac parameters was done by acquiring long axis view and short axis view along with colour Doppler for evaluation of regurgitation.

For the precise evaluation of cases, the score card given by McEwan (2010) (Table 01) was used to categorize the dogs in two categories. First category included the dogs suffering with dilated cardiomyopathy (DCM) (Score ≥ 6) and second category included the dogs that were at risk of developing dilated cardiomyopathy (Score ≤ 5), in future.

**Table 1. Score card for echocardiographic screening of dogs suffering with dilated cardiomyopathy**

|  |  |  |
| --- | --- | --- |
| **Criteria** | | **Score** |
| **Major criteria** | | 3 for each |
| Left ventricle (LV) dilatation | Systolic or diastolic LV diameter >95% confidence intervals from breed- specific reference range, or weight of do | 3 |
| Increased LV sphericity | LV diastolic length: diameter ratio < 1.65 | 3 |
| Reduced fractional shortening Or Reduced ejection fraction | <20-25% depending on breed-specific M-mode reference values  <50% based on 20 volume calculations | 3 |
| **Minor criteria** | | 1 for each |
| Arrhythmias strongly associated with breed (e.g. ventricular ectopy in Boxer, Dobermann) | | 1 |
| Atrial fibrillation | | 1 |
| Increased mitral E point to septal separation (M-mode) | | 1 |
| LV fractional shortening in equivocal range for breed | | 1 |
| Left or bi-atrial enlargement | | 1 |
| Pre-ejection period: ejection time ratio exceeding 95% confidence intervals (>0.4) | | 1 |
| Interpretation | |  |
| Diagnosis consistent with DCM | | Total score ≥6 |
| Recommend serial re-evaluation (e.g. annually) | | Total score≤5 |

**(McEwan, 2010)**

**Biochemical estimation**

Approximately 3 ml of blood was collected aseptically from cephalic or saphenous vein of animals suffering from dilated cardiomyopathy in clot activator vacutainer for biochemical analysis. The blood samples were assessed for the Creatinine kinase-MB and blood glucose using CHEM-5 plus semi-autoanalyzer and commercially available kits (Coral Clinical Systems, Tulip Diagnostic (India) Pvt. Ltd.). The Cardiac Troponin-I (cTnI) was estimated using Cardiac Troponin-I ELISA kit (Bioassay Technology Laboratory).

1. **RESULTS AND DISCUSSION**

**Incidence**

A total of 4509 cases of canine were registered at Veterinary Clinical Complex (V.C.C.) during the study period. The subjects presented with various symptoms of cardiac disease such as laboured breathing, exercise intolerance, distended abdomen, frequent coughing, dyspnoea, hind limb weakness, ascites, obesity and fainting or collapsing were 109 in total. On further examination, out of 109 cases only 60 (1.30%) cases were found to be of cardiac origin. Based on the score card of McEwan (2010), dilated cardiomyopathy was ascertained in total six dogs (0.13%) and 49(1.08%) were at risk of developing DCM.

**Clinical symptoms in dogs suffering with dilated cardiomyopathy**

The chief complaint by the owner associated with the dilated cardiomyopathy was distended abdomen, exercise intolerance, frequent or chronic coughing, hind limb oedema and weight loss. These symptoms were present either alone or in combination (Table 2).

**Table 02: Clinical symptoms observed in dogs suffering with dilated cardiomyopathy**

|  |  |
| --- | --- |
| **Symptom** | **No. of animals** |
| Distended abdomen | 05 |
| Exercise intolerance | 04 |
| Hind limb oedema | 03 |
| Chronic/frequent coughing | 03 |
| Weight loss | 01 |

Vishnurahav *et al.* (2018) also reported inappetence, abdominal enlargement, exercise intolerance, weakness, difficulty in respiration, syncope and vomiting as the major history and clinical signs in DCM. These clinical findings in DCM affected dogs were reported by various authors (Martin *et al*., 2010 and Palermo *et al*., 2011).

Considerable visible distension of abdomen (ascites) was noticed by owners as compared to other symptoms. Poor renal perfusion subsequently through series of events that lead to release of aldosterone, which acts on the distal convoluted tubule of the nephron, resulting in sodium and water retention, to further increase preload. Ultimately, excessive sodium and water retention results in oedema and effusions in the congestive heart failure setting (Njoroge and Teerlink, 2021). One common problem with heart failure is that, due to the heart's inefficient pumping ability, blood returning to the heart from the lungs tends to back up, producing pulmonary congestion which leads to coughing, exercise intolerance, syncope and weakness.

**Duration of symptoms in dogs suffering with dilated cardiomyopathy**

In the present study, the dogs were having variation in the duration of symptoms. 50.00% dogs were known to suffer from past 4-6 weeks whereas 33.33% dogs were having symptoms for more than 4 weeks and 16.67% was reported to have symptoms for more than 6 weeks. The presentation of cases in duration of 4-6 weeks might be due to late presentation of the cases to VCC due to owner negligence, unawareness or due to use of symptomatic treatment. It was also suspected that cases are underreported because animals are typically treated symptomatically and adequate diagnostic facilities are not available in various areas.

**Type of diet offered to dogs suffering with dilated cardiomyopathy**

On gathering information about the diet fed to the dogs suffering with dilated cardiomyopathy, three dogs were kept on vegetarian diet and commercial feed, whereas 2 dogs were kept on non-vegetarian diet and commercial feed, while 1 dog was fed vegetarian and non-vegetarian diet.

FDA (2019) conducted an investigation after a spike in cases of canine dilated cardiomyopathy (DCM) in dog breeds not previously known to have a genetic predisposition. It was found that the affected dog breeds were fed certain pet foods (labelled as "grain-free") which contained a high proportion of peas, lentils, other legume seeds (pulses), and/or potatoes in various forms as main ingredients. Hence a link between pet food and DCM was brought into consideration.

Dutton and Lopez-Alvarez (2018) and McCauley *et al.* (2020) indicated a nutritional deficiency in the amino acid taurine and carnitine as a contributing factor causing enlarged heart in canines. Taurine plays a role in regulating heartbeat and preventing calcium overload. Carnitine aids the muscle's ability to convert fatty acids into usable energy for muscle activity therefore inadequate presence of these two acids leads to strain on the heart muscle and cardiac malfunctioning. In the present investigation either of the two might be a reason to develop DCM.

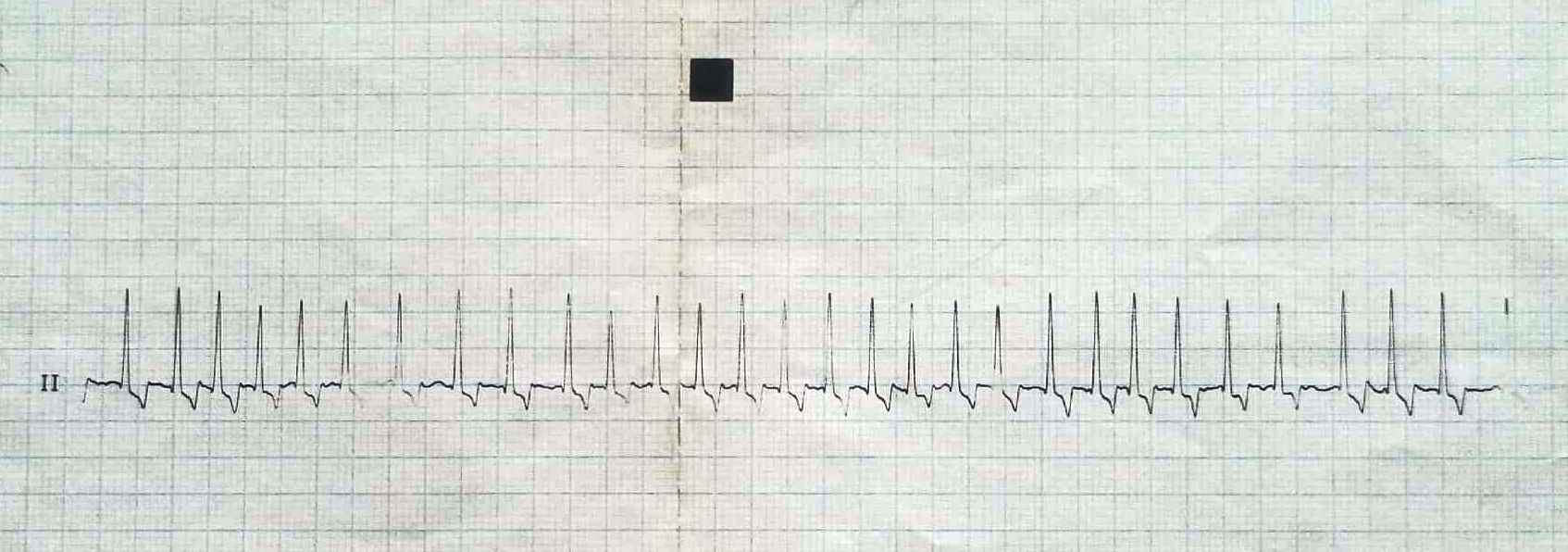
This may be due to the fact that dogs are basically carnivores and homemade vegetarian food may produce some deficiencies, metabolic and energy disturbances which may further lead to DCM. Mostly the pet owners feed the same food they consume themselves and this dietary shift can predispose dogs to cardiac diseases, as it does in humans.

**Electrocardiographic evaluation in dogs suffering with dilated cardiomyopathy**

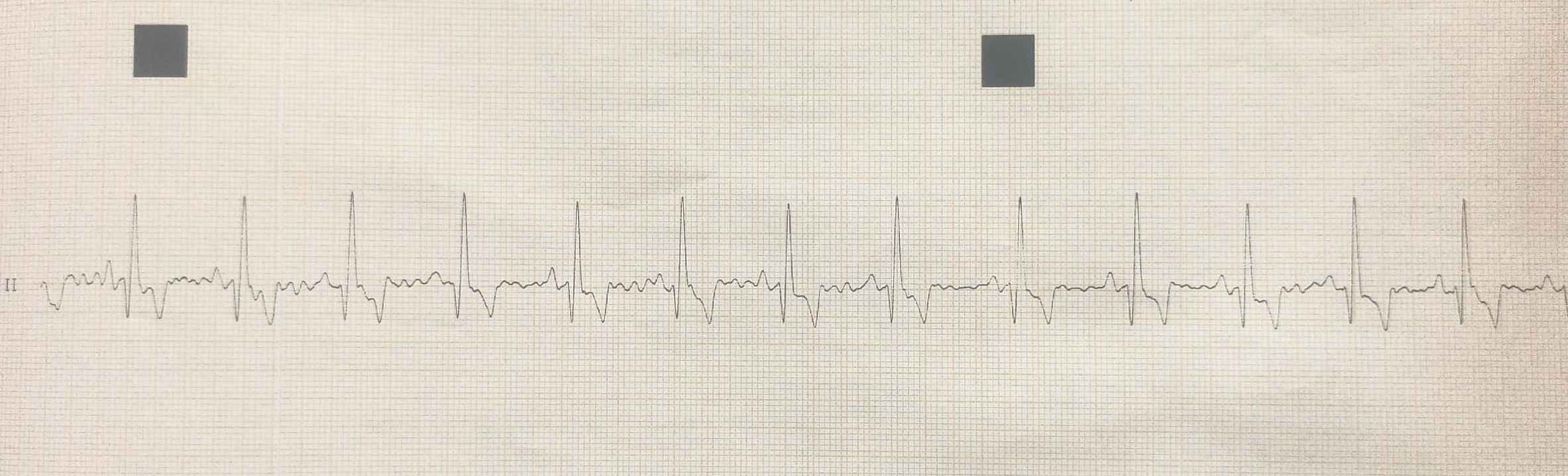
The electrocardiographic findings of dogs suffering with dilated cardiomyopathy are depicted in Table 03 and Figure 01. Ingole (2019) reported that all the dogs in DCM group had ST coving, which is suggestive of left ventricular enlargement. Similarly, Padalkar (2012) and Velhankar (2013) also reported ST coving in the ECG of DCM affected dogs with left ventricular enlargement. These findings are in conformity with the observations of the present study.

**Table 3. Electrocardiographic findings in dogs suffering with dilated cardiomyopathy**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Case no.** | **1** | **2** | **3** | **4** | **5** | **6** |
| **P wave amplitude (mV)** | 0.30 | 0.20 | 0.40 | 0.30 | 0.00 | 0.00 |
| **P wave duration (sec)** | 0.03 | 0.02 | 0.04 | 0.03 | 0.00 | 0.00 |
| **PR interval (sec)** | 0.08 | 0.06 | 0.09 | 0.07 | 0.00 | 0.00 |
| **R wave amplitude (mV)** | 2.70 | 2.80 | 2.60 | 2.70 | 2.20 | 1.90 |
| **QRS duration (sec)** | 0.04 | 0.05 | 0.06 | 0.04 | 0.05 | 0.06 |
| **T wave amplitude (mV)** | 0.78 | 0.92 | 0.80 | 0.85 | 0.46 | 0.35 |
| **R/4** | 0.68 | 0.70 | 0.65 | 0.68 | 0.55 | 0.48 |
| **ECG Findings** | ST coving | ST coving | ST coving | ST coving | Fine atrial fibrillation | Fine atrial fibrillation |



**a.**



**b.**

**Fig. 1. Electrocardiogram showing (a) ST coving (b) Atrial fibrillation (50mm/sec and 1mV)**

**Radiographic evaluation in dogs suffering with dilated cardiomyopathy**

The radiographic findings of dogs suffering with dilated cardiomyopathy are described in Table 04 and 05 and Figure 02. The trachea was displaced dorsally in 4 cases (66.67%). Cardiac silhouette appeared as taller and wider than the normal in all the dogs. In all the cases of dilated cardiomyopathy, the vertebral heart score ranged between 10.9-14.5 vertebrae, which was much higher than that of normal dogs, indicated cardiomegaly. Interstitial pattern was present in all cases (mild: 66.66%, moderate: 16.66% and severe: 16.66%). Alveolar pattern (mild) was present in 50% cases. Pulmonary edema was present at hilar region in 100% cases.

**Table 04: Vertebral heart score (VHS) in dogs suffering with dilated cardiomyopathy**

|  |  |  |  |
| --- | --- | --- | --- |
| **Case No.** | **No. of vertebrae traversed by short axis (S)** | **No. of vertebrae traversed by long axis (L)** | **Vertebral heart score**  **(L+S)** |
| 1 | 05.70 | 05.20 | 10.90 |
| 2 | 08.40 | 06.10 | 14.50 |
| 3 | 07.50 | 06.10 | 13.60 |
| 4 | 06.10 | 05.80 | 11.90 |
| 5 | 07.10 | 05.10 | 12.20 |
| 6 | 07.90 | 05.10 | 13.00 |

Cardiothoracic ratio was evaluated in all the cases and the values were above 0.50 which is suggestive of cardiomegaly. The values of CTR ranged between 0.55 to 0.80. Ernst *et al.* (2001) demonstrated that the CTR is an important predictor of mortality in patients with dilated cardiomyopathy, even when compared to the echocardiographic exam, considered the gold standard in hemodynamic and myocardial function assessment.

**Table 5. Cardiothoracic ratio (CTR) in dogs suffering with dilated cardiomyopathy**

|  |  |  |  |
| --- | --- | --- | --- |
| **Case No.** | **Cardiac silhouette diameter (a)** | **Chest dimension at widest region (b)** | **Cardiothoracic ratio (a/b)** |
| 1 | 3.88 | 6.56 | 0.59 |
| 2 | 5.89 | 7.28 | 0.80 |
| 3 | 5.59 | 8.33 | 0.67 |
| 4 | 5.12 | 7.65 | 0.66 |
| 5 | 4.04 | 7.28 | 0.55 |
| 6 | 4.95 | 8.16 | 0.60 |

|  |  |
| --- | --- |
| Right lateral radiograph showing vertebral heart score (VHS) (\*Tracheal displacement ⚫ Interstitial pattern**)**  \* | Ventro-dorsal radiograph showing cardiothoracic ratio |

**Fig. 2: Radiographic observations in dogs suffering with dilated cardiomyopathy**

Azevedo *et al.* (2016) reported that CTR is intimately related to the volume of the left ventricle and CTR values above 0.5 in humans are pathological. This study is in agreement with Kumar (2019) who also reported increased CTR in dogs suffering with DCM (Mean ± SE- 53.86±2.15).

Hoque *et al*. (2019) reported that the VHS measurements tend to increase in dogs with cardiac disease thus gives an idea about ongoing disease. It was concluded that there is a fair correlation between VHS measurements and a variety of other indices of cardiac chamber enlargement, including end-systolic and end-diastolic ventricular diameters, which is in accordance with the present study.

Ingole (2019) reported upward deviation of trachea in 93.33% cases. Also, the dogs were having varying degree of congestion, edema of pulmonary parenchyma with its severity proportional to cardiac enlargement or valvular regurgitation. Similar findings were reported by Kittleson (1998) and Vollmar (2000). Present study is in agreement with the observations of these authors however in partial disagreement that pericardial effusion was not diagnosed, radiographically. The pericardial effusion was detected only during echocardiographic examination. The probable reason to fail to diagnose pericardial effusion on radiographic examination could be due to lesser quantity of pericardial fluid accumulated in the pericardial sac around the heart and thereby imparting the same tissue density as that of myocardium on x–ray image.

These observations i.e. increased VHS along with congestion of lungs, pulmonary edema and elevation of trachea were also in consonance with Martin *et al*. (2009). Meurs *et al*. (2001) observed pulmonary edema, generalized cardiac enlargement, left atrial enlargement and pleural effusion as the common radiographic abnormalities with DCM.

Previous authors (Devi *et al.,* 2009 and Velhankar, 2013) reported signs of pulmonary congestion in left sided heart failure while pulmonary edema or pleural effusions, ascites and pedal edema in right sided heart failure cases. This enlargement in the cardiac silhouette is due to the left ventricular enlargement and in some case due to combined enlargement of atria and ventricles both.

**Echocardiographic examination in dogs suffering with dilated cardiomyopathy**

The echocardiographic parameters recorded in the present study are depicted in the table 06 and figure 3. Upon echocardiographic examination, dilated left ventricular lumen and enlarged left atrium was observed in dogs suffering with dilated cardiomyopathy. The interatrial septum displaced towards the right side was observed in three cases. In all the cases fractional shortening, stroke volume and ejection fraction reduced markedly and EPSS increased considerably. Two cases (2nd and 3rd) showed mitral valve regurgitation on colour Doppler echocardiography which was depicted as mosaic pattern at the time of closing of mitral valve (Fig. 03). In M-mode or duplex ultrasound images in 33.33% (2/6) cases the interventricular septum was contracting but the left ventricular free wall showed very less contraction whereas in 66.67% (4) cases both the walls (IVS and LVPW) showed less contraction.

**Table 6. Echocardiographic parameters of clinical cases suffering with dilated cardiomyopathy**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Case No.** | **LVIDd (mm)** | **LVIDs (mm)** | **FS (%)** | **EDV (ml)** | **ESV (ml)** | **SV (ml)** | **EF (%)** | **LAD (mm)** | **EPSS (mm)** |
| 1 | 62.10 | 56.20 | 09.50 | 194.69 | 154.92 | 39.77 | 20.42 | 51.75 | 08.62 |
| 2 | 67.10 | 53.90 | 19.67 | 232.13 | 140.70 | 91.43 | 22.57 | 67.90 | 10.90 |
| 3 | 60.30 | 53.30 | 11.60 | 182.06 | 137.11 | 44.95 | 24.68 | 52.40 | 07.05 |
| 4 | 61.20 | 59.40 | 02.94 | 188.32 | 175.90 | 12.42 | 06.59 | 75.65 | 09.40 |
| 5 | 59.90 | 48.90 | 18.36 | 179.24 | 112.25 | 66.99 | 37.37 | 45.40 | 09.00 |
| 6 | 46.70 | 35.40 | 24.19 | 100.83 | 52.27 | 48.56 | 48.16 | 46.05 | 10.40 |

Ingole (2019) reported that the mean values of LVIDd and LVIDs in the dogs suffering with DCM were 4.99 ±0.18 cm and 4.34 ±0.18 cm, respectively. Similar findings indicating that the dogs having DCM suffered from left ventricular enlargement both during diastole and systole were reported by various authors (Padalkar, 2012 and Rath *et al.*, 2024) which is in consonance with the present study.

|  |  |  |
| --- | --- | --- |
| B-mode right parasternal long axis view indicating left atrial and left ventricular dilation | Simultaneous B-mode and M-mode echocardiogram showing dilated left ventricle | Colour Doppler echocardiogram indicating mitral valve regurgitation depicted as mosaic pattern |

**Fig. 3: Echocardiographic observations in dogs suffering with dilated cardiomyopathy**

Ingole (2019) reported that the EF and FS percentage (13% to 42% and 6% to 21%) in DCM affected dogs were decreased below the reference ranges, respectively. These observations were in consonance with the present study which clearly indicate that all dogs in this group had compromised left ventricular systolic function adversely affecting cardiac output and myocardial contractility. Similar observations have been reported by previous researchers in their studies on dilated cardiomyopathy in dogs (Velhankar, 2013; Padalkar, 2012 and Matli and Rao, 2022).

Significant increase in the values of EPSS in dogs suffering from DCM reported in previous studies (McEwan *et al.,* 2003; Khangal, 2017 and Ingole, 2019) are in agreement of the present study. Ingole (2019) reported the mean left atrial diameter in DCM group (3.83 ±0.16 cm). Similar observation was also reported by Padalkar (2012) and Velhankar (2013).

The increase in the left ventricular internal diameter is attributed to be due to the dilation of the left ventricle and this increased lumen caused decreased contractility and hence decreased ejection fraction and fractional shortening. The mitral valve regurgitation was due to the increase in the distance between the two leaflets as the left ventricle dilated, considerably.

**Biochemical estimation of dogs suffering with dilated cardiomyopathy**

The biochemical parameters *viz.* CK-MB ranged from 6.80 to 24.20 IU/L and blood sugar ranged from 102 mg/dL to 110 mg/dL in this study were found to be within the normal physiological range in all the dogs affected with dilated cardiomyopathy.

In contrast to the present study, Vishnurahav *et al.* (2017) and Kumar (2019) reported that CK-MB was found significantly elevated in dogs with cardiomyopathy. The values of CK-MB in the normal physiological range may be due to the fact that CK-MB levels also return to baseline within 48 hours so late diagnosis of injury is not possible with this marker, when there is late presentation of the clinical cases (Sagar *et al.,* 2021).

The result of qualitative estimation of cardiac troponin-I was observed negative in all dogs suffering with DCM in our study. In partial disagreement to the present study, Kumar (2012) reported that cardiac troponin-I (qualitative) was found positive in 8.1% cases and Kumar (2019) reported elevated levels of Troponin-I in all the cases suffering from DCM. Wells and Sleeper (2008) reported that the half-life of troponin and its complex in the circulation is about 2 hours. The levels remain elevated for 7–10 days (cTnI) and 10–14 days (cTnT). Hence, the negative cardiac troponin-I test might be due to low sensitivity or delayed presentation of cases. Also, lower sensitivity of qualitative Troponin-I in present study could be attributed to its high detection limits (0.50 ng/ml for Troponin-I).

**Category I: Dogs diagnosed with dilated cardiomyopathy**

Confirmed six dogs diagnosed for dilated cardiomyopathy were treated symptomatically using ACE inhibitors, cardiac glycosides and diuretics (Borgarelli *et al.,* 2001). Initially all animals responded to treatment and started to show alleviation in clinical signs. Although two cases collapsed within one week of diagnosis and the rest of the cases survived till the study period. Palermo *et al.* (2011*)* reported that the prognosis was worse in dogs with LV dilation compared to dogs with a normal LV and ventricular arrhythmias.

**Category II: Dogs at risk of developing dilated cardiomyopathy**

Out of 54 dogs that were not suffering with dilated cardiomyopathy, 49 dogs were evaluated to be at risk as they were having various arrhythmias, left atrial dilation or bi-atrial dilation (Table 07 and Figure 04). The owners of these dogs were advised for the bi-annual or annual echocardiographic assessment of the cardiac function. Three dogs were found to be suffering with hypertrophic cardiomyopathy (HCM) and remaining two dogs with pericardial effusion (PE). These dogs were treated as per the condition encountered.

**Table 7****. Distribution of dogs at risk of developing dilated cardiomyopathy**

|  |  |  |
| --- | --- | --- |
| **Condition encountered** | **No. of dogs** | **Per cent** |
| Arrhythmia | 42 | 85.71 |
| Left atrial or bi-atrial enlargement | 07 | 14.28 |
| Total | 49 | 100.00 |

|  |  |
| --- | --- |
| Simultaneous B-mode and M-mode echocardiogram showing arrhythmia | B-mode right parasternal long axis view showing left atrial enlargement |
| Simultaneous B-mode and M-mode echocardiogram showing hypertrophic cardiomyopathy | Simultaneous B-mode and M-mode echocardiogram showing pericardial effusion depicted as black line |

**Figure 04: Echocardiographic observations in of developing dilated cardiomyopathy**

1. **CONCLUSION**

The cumulative prevalence of cardiac disorders over a span of six months was documented to be 1.30% within the confines of Jabalpur city. During initial assessments, cardiac arrhythmias emerged as the predominant anomaly identified, succeeded by left or bi-atrial enlargement and dilated cardiomyopathy (DCM). Subsequent evaluation utilizing a scoring system confirmed the presence of dilated cardiomyopathy in six canines, while an additional 49 were identified as being at risk for its future development. Echocardiography was established as the primary imaging modality for the evaluation of dilated cardiomyopathy (DCM) and the screening of various cardiac pathologies. The left ventricular ejection fraction, fractional shortening, and left ventricular internal diameter during both systole and diastole were identified as critical parameters for the diagnosis of dilated cardiomyopathy. Colour Doppler echocardiography proves essential for the identification of functional valve abnormalities, such as regurgitation. An elevated vertebral heart score (VHS) was also determined to be a consistent finding in canines afflicted with DCM. Biochemical markers, including cardiac troponin-I and CKMB, are instrumental in the early detection of myocardial damage during the initial phases of the disease. Dilated cardiomyopathy is characterized by its progressive nature; consequently, early detection of alterations is paramount to avert its future progression. As a silent affliction, dilated cardiomyopathy arises in dogs due to numerous etiological factors, resulting in impaired left ventricular function, subsequent alterations in other cardiac compartments, and ultimately culminating in congestive heart failure and death. These cardiac modifications have systemic implications due to alterations in hemodynamics and consequently the oxygenation of essential organs. Therefore, a comprehensive assessment utilizing electrocardiography, radiography, and echocardiography is imperative to elucidate the underlying causes of hemodynamic disturbances attributed to cardiac disorders, at various healthcare facilities as promptly as feasible. The likelihood of a favorable therapeutic outcome is significantly contingent upon the early and precise diagnosis of the prevailing cardiac condition.

1. **CONSENT**

All authors declare that written informed consent was obtained from the patient’s owner for publication of the research. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

1. **ETHICAL APPROVAL**

The research described in the study was conducted in compliance with the ethical standards and guidelines of the Institutional Animal Ethics Committee (IAEC) and due permission was received from the ethical committee of the University.

1. **DISCLAIMER (ARTIFICIAL INTELLIGENCE)**

Authors hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

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