Diagnostic and therapeutic evaluation of dilated cardiomyopathy in a Labrador dog: A case report

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Abstract
A case of six year old male Labrador dog weighing around 33 kg was presented to Teaching Veterinary Clinical Complex, Bikaner with the history of inappetance, lethargy, intermittent cough, respiratory distress and progressing ascites since three months. The dog was treated previously by a local veterinarian for liver failure and not much significant improvement was observed. On clinical examination revealed leghy, tachycardia and arrhythmia. On lateral chest radiograph revealed enlarged heart with Vertebral Heart Size (VHS) 12.12. Heart sound was gallop rhythm with third heart sound and lung sound was crackled on auscultation. No significant findings were detected on complete blood count and serum chemistry profiles. ECG showed wide P waves, Tall R waves, supraventricular QRS complexes and irregularly irregular R-R intervals with fast rhythms indicating clearly atrial fibrillation and intermittently ventricular premature complexes. The dog was treated with Furesomide @ 2 mg/kg BID, Pimobendan @ 0.25 mg/kg, PO, BID, Enalapril maleate @ 0.5 mg/kg BID and diltiazem (0.5 mg/kg, PO, BID). The dog had marked improvement and the medications are being continued lifelong with monthly reviews.

Keywords: Dilated cardiomyopathy, dog, tachycardia, atrial fibrillation

Introduction
Dilated cardiomyopathy (DCM) has been considered to be primarily an inherited disease, with higher prevalence in specific dog breeds (Dukes-McEwan et al., 2003; Sammarco, 2008). However, other causes of DCM include specific nutrient deficiencies (Freeman et al., 2001; Backus et al., 2003) and concurrent diseases, such as endocrine (Karlapudi et al., 2012; Janus et al., 2014) myocardiatis, and chronic tachycardia (Calvert et al., 1997). Dilated cardiomyopathy (DCM) is primary disease of the muscles of the heart, characterized by a progressive decline in the contractility of the ventricles, dilation of all the cardiac chambers, leading to congestive heart failure (CHF), arrhythmias, and death (Sisson et al., 1999). According to Fox et al. (1998), DCM is more common in middle age to old dog with male over dominance in ratio of 2:1 and recognized in several medium-size and giant breed dogs.

During the occult phase of DCM, physical examination of the cardiovascular system may be normal, but physical exam findings can reveal jugular pulsus, an irregular heart rhythm, weak pulses, pulse deficits, or a systolic murmur with a point of maximum intensity in the left sixth intercostal space (Guglielmini, 2003; Kahn, 2005). If CHF is present, crackles or muffled heart and lung sounds may be auscultated (Sisson et al., 2000). Thoracic radiographs can reveal generalized enlargement of the cardiac silhouette or more specifically enlargement in the area of the left atrium and left ventricle. Buchanan and Bucheler (1995) described a method for measuring the canine cardiac silhouette and involves measuring its long and short axes in a left lateral radiograph and comparing the sum of these measurements to the mid-thoracic vertebral bodies, to produce a unit less index called the vertebral heart score (VHS) and normal values for VHS as 9.7 ± 0.5 (mean ± SE) vertebrae in dogs. The vertebral heart size (VHS) was established to create a more objective way of diagnosing cardiomegaly via thoracic radiography (Nakayama et al., 2001). In addition, distention of the pulmonary veins, tracheal elevation, and pulmonary edema with or without pleural effusion or pleural effusion lines may also be present (Sisson et al., 2000). Echocardiography is necessary to definitively differentiate DCM from other cardiovascular diseases (Bonagura and Herring, 1985). Number of electrocardiographic abnormalities have been reported in dogs with DCM like wide and tall P waves, wide and tall QRS complexes, rhythm disturbances like SVT, Atrial Premature Contraction (APC), atrial fibrillation, Ventricular Premature Complex (VPC) and Ventricular...
Tachycardia (VT) having a very important both diagnostic and prognostic (Goodwin, 1998; Padalkar, 2012; Velhankar, 2013; Anil Kumar, 2013) [9, 16, 23, 1]. Electrocardiography (ECG) is widely used as a diagnostic tool for cardiac diseases in both - human and veterinary medicine. Electrocardiogram (ECG) evaluation can reveal supraventricular and ventricular arrhythmias, in addition to wide and tall P (indicative of atrial enlargement) and R (indicative of ventricular enlargement) waves (Meurs, 2003) [21]. It is important to note that a normal ECG does not rule out the presence of DCM, as most arrhythmias are intermittent and have high day-to-day variability (Spier and Meurs, 2004) [21]. Treatment of CHF, for dogs diagnosed with DCM, is similar to other CHF etiologies: decreasing preload and afterload while maintaining systemic blood pressure. In most cases, triple therapy is warranted for treating CHF. Triple therapy includes prescribing pimobendan (0.25 mg/kg by mouth, twice daily), a diuretic such as furosemide (typical starting dose is 2 mg/kg by mouth, twice daily), and an angiotensin-converting enzyme (ACE) inhibitor such as enalapril (0.25 to 0.5 mg/kg by mouth, twice daily (Plumb, 2018) and also use antiarrhythmic Diltiazem (0.5 mg/kg, PO, BID) (Yun-Hye et al., 2017) [24].

History, clinical findings and diagnosis
A case of six year old male Labrador dog weighing around 33 kg was presented to Teaching Veterinary Clinical Complex, Bikaner with the history of inappetance, lethargy, intermittent cough, respiratory distress and progressing ascites since three months.

On the physical examination, the dog had severe tachycardia, lethargy and arrhythmia. Clinical examination showed temperature 102.6°F, heart rate 160bpm, and respiration rate 50 breaths per minute. Heart sound was gallop rhythms with third heart sound and lung sound was crackled on auscultation. On lateral chest radiograph revealed enlarged heart with VHS score 12.12. The whole lung field on thoracic radiography was alveolar pattern and lungs lobe margins were disappeared.

Blood examinations showed parameters within the normal range: TEC- 6.44X106 cells/µl, TLC- 13.40 X 103 cells/µl, Hemoglobin-13.8g/dl, Platelet count-200X 103 cells/µl, and PCV-38%. Liver and kidney function tests were within normal range SGPT-33.07 IU/L and Creatinine 1.58mg/ dl respectively. No significant findings were observed in complete blood count and serum chemistry profiles. The result of ECG showed wide P waves, Tall R waves, supraventricular QRS complexes and irregularly irregular R-R intervals with fast rhythms, indicating clearly atrial fibrillation and intermittently ventricular premature complexes in the dog. Number of electrocardiographic abnormalities have been reported in dogs with DCM like wide and tall P waves, wide and tall QRS complexes, rhythm disturbances like SVT, Atrial Premature Contraction (APC), atrial fibrillation, Ventricular Premature Complex (VPC) and Ventricular Tachycardia (VT) having a very important both diagnostic and prognostic (Goodwin 1998; Padalkar, 2012; Velhankar 2013 and Anil Kumar 2013) [9, 16, 23, 1]. In this study successfully used ECG as a diagnostic modality for detection of rhythm disturbances and cardiac chamber enlargement in the dog suffering from DCM. The dog was treated with Furesomide @ 2 mg/kg BID administration to manage the pulmonary edema and ascites. As respiratory distress was improved and the dog could swallow food or water without difficult. The dog was treated by inodulator Pimobendan @ 0.25 mg/kg, PO, BID and Enalapril maleate @ 0.5 mg/kg BID were given for management of DCM. For management of the atrial fibrillation, we administrated antiarrhythmic Diltiazem (0.5 mg/kg, PO, BID) similarly to Yun-Hye et al., 2017 [24]. On 15th day after initial therapy, the dog had marked improvement and the medications are being continued lifelong with monthly reviews.

Therapy
Thoracocentesis and abdominocentesis were performed to remove a large volume of effusion from the pleural and abdominal space to alleviate respiratory distress. The dog was treated with Furesomide @ 2 mg/kg BID, pimobendan @ 0.25 mg/kg, PO, BID, Enalapril maleate @ 0.5 mg/kg BID and diltiazem (0.5 mg/kg, PO, BID). On 15th day after initial therapy, the clinical signs as well as the radiographic abnormalities were resolved. The dog had marked improvement and the medications are being continued lifelong with monthly reviews.

Conclusion
In the present study the clinical signs found were in accordance with Yun-Hye et al., 2017 [24]. Heart sound was gallop rhythms with third heart sound and lung sound was crackled on auscultation which were also reported by Sisson et al., 2000 [20] and Yun-Hye et al., 2017 [24]. Lateral chest radiograph revealed cardiomegaly with VHS score 12.12. Buchanan and Bucheler (1995) [4] developed a method for measuring canine heart size in radiographs, vertebral scale system, and reported the sum of the long and short axes of the heart expressed as vertebral heart size as 9.7±0.5 (means SE) in vertebrate dog. The whole lung field on thoracic radiography was alveolar pattern and lungs lobe margins were disappeared. In the present study no significant findings of complete blood count and serum chemistry profiles were in accordance with Yun-Hye et al., 2017 [24]. The result of ECG showed wide P waves, Tall R waves, supraventricular QRS complexes and irregularly irregular R-R intervals with fast rhythms, indicating clearly atrial fibrillation and intermittently ventricular premature complexes in the dog. Number of electrocardiographic abnormalities have been reported in dogs with DCM like wide and tall P waves, wide and tall QRS complexes, rhythm disturbances like SVT, Atrial Premature Contraction (APC), atrial fibrillation, Ventricular Premature Complex (VPC) and Ventricular Tachycardia (VT) having a very important both diagnostic and prognostic (Goodwin 1998; Padalkar, 2012; Velhankar 2013 and Anil Kumar 2013) [9, 16, 23, 1]. In this study successfully used ECG as a diagnostic modality for detection of rhythm disturbances and cardiac chamber enlargement in the dog suffering from DCM. The dog was treated with Furesomide @ 2 mg/kg BID administration to manage the pulmonary edema and ascites. As respiratory distress was improved and the dog could swallow food or water without difficult. The dog was treated by inodulator Pimobendan @ 0.25 mg/kg, PO, BID and Enalapril maleate @ 0.5 mg/kg BID were given for management of DCM. For management of the atrial fibrillation, we administrated antiarrhythmic Diltiazem (0.5 mg/kg, PO, BID) similarly to Yun-Hye et al., 2017 [24]. On 15th day after initial therapy, the dog had marked improvement and the medications are being continued lifelong with monthly reviews. Survival time of patient is known to be associated with cardiac-related clinical signs, echocardiographic or electrical abnormalities (Tidholm, 2006). Also, echocardiographic and electrical abnormalities were obvious, including chamber enlargement, systolic dysfunction, and atrial fibrillation. Medical therapy may provide significant improvement in lifespan and quality of life in affected dog.

References


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