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Management of Dilated Cardiomyopathy in a Labrador

Khushi Sharma, K.Satish Kumar, Manaswini, M., Ashwini, S., Zeenath Fathima and Rachana, T.*
Department of Veterinary Medicine,
College of Veterinary Science, Rajendranagar
PVNRTVU, Hyderabad-30

Abstract

Dilated cardiomyopathy is the most common form of cardiomyopathy in canines which is characterized by progressive ventricular dilation and loss of myocardial contractility. Four year old, male Labrador was presented with complaint of tachypnea, activity intolerance, inability to walk and inappetence for a period of 15 days to Veterinary Clinical Complex, CVSc., Rajendranagar, Hyderabad. ECG showed deep Q wave and elevated R-wave with VPCs which were also confirmed on 24 hour Holter. Elevated urea, uric acid and SDMA levels were noticed. Thoracic radiograph and echocardiography revealed biventricular dilation, globoid appearance of heart, systolic dysfunction with reduced ejection indices, thus diagnosing the case as DCM. The dog was treated with benazepril pimobendan and furosemide and there was significant clinical improvement after one month of therapy.

Keywords: Dilated Cardiomyopathy, Labrador, diagnostic imaging

Introduction

Dilated cardiomyopathy (DCM) is a progressive myocardial disease in dogs, characterised by ventricular dilation, impaired systolic function, and reduced cardiac contractility, ultimately leading to congestive heart failure and increased risk of sudden cardiac death (Freeman & Rush, 2022). The etiology of canine DCM is multifactorial. Clinically, affected dogs may remain asymptomatic for an extended period before developing signs such as exercise intolerance, respiratory distress, syncope, and ascites (Tidholm & Häggström, 2023). Early detection is crucial, as a significant proportion of cases present with acute heart failure requiring immediate intervention, and sudden cardiac death remains a major concern, especially in predisposed breeds (Tidholm & Häggström, 2023). In the present case report, successful management of dilated cardiomyopathy (DCM) in a dog is reported.

Case history and observation

Four year-old male Labrador Retriever was presented with complaint of dyspnea, reduced exercise tolerance, lethargy and inappetence for the past two weeks. Thoracic radiographs revealed cardiomegaly, pulmonary congestion and edema, tracheal elevation, increased sternal contact, with an increased vertebral heart score of 13.5 (Fig.1). ECG revealed a deep Q wave (0.8 mV), ventricular premature complexes, and an elevated R wave amplitude (3 mV), findings consistent with biventricular enlargement whereas, continuous ECG monitoring using a Holter device revealed sinus arrhythmia, episodes of sinus bradycardia and sinus tachycardia, alterations in P wave morphology, and intermittent pauses lasting up to 2.3 seconds (Fig 2-3). Haematobiochemical analysis indicated normal hepatic function, elevated blood urea (107.5 mg/dL), blood urea nitrogen (BUN, 50.3 mg/dL), and symmetric dimethylarginine (SDMA, 15.2 µg/dL). NT-pro BNP assay demonstrated a markedly elevated concentration (5391.4 pmol/L), indicative of significant cardiac stress. Two-dimensional B-mode long-axis imaging revealed marked dilation of both the left and right cardiac chambers, as well as thinning of the inter-ventricular septum. In the short-axis view, the left ventricle appeared globoid with a dilated lumen. There was increased left atrial-to-aortic root ratio (LA/Ao) of 2.20, consistent with left atrial enlargement. Fractional shortening (FS) of 12% (reference range: 33.6–38.93%) and an ejection fraction (EF) of 12% (reference range: 55–70%) were observed indicative of severe systolic dysfunction. Based on these findings, along with echocardiographic evidence of severe cardiac chamber dilation and systolic dysfunction, it was diagnosed with overt dilated cardiomyopathy.

Treatment and discussion

The dog was administered with benazepril @ 0.5 mg/kg body weight orally twice daily), pimobendan

*Corresponding author: drsatish.ksk@gmail.com

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(@ 0.3 mg/kg body weight, orally twice daily), furosemide (@ 2 mg/kg body weight, orally twice daily), and levocarnitine was prescribed for a duration of one month. After one month of therapy, dog was re-evaluated. Electrocardiographic assessment revealed resolution of ventricular premature complexes and normalization of the Q wave and thoracic radiographs showed with no evidence of pulmonary congestion or edema. Echocardiographic parameters, including ejection fraction (EF) and fractional shortening (FS), showed modest improvement. Clinical improvement in the dog was observed.

Dilated cardiomyopathy (DCM) is one of the common acquired cardiac disease in dogs, accounting for approximately 10% of all canine cardiac diagnoses (Freid *et al*., 2021). The existence of an occult phase, during which affected dogs may exhibit minimal or no overt clinical signs despite progressive myocardial dysfunction, further complicates timely diagnosis (Calvert et al., 1997). This highlights the importance of routine cardiac screening including echocardiography, electrocardiography, and biomarker analysis in at-risk populations for early detection and intervention (Freid *et al*., 2021). Once diagnosed, dogs with DCM require lifelong therapy with a combination of medications such as inodilators (e.g., pimobendan), diuretics (e.g., furosemide), and ACE inhibitors (e.g., benazepril or enalapril) to manage symptoms, delay disease progression, and improve quality of life (Keene *et al.,* 2019). Recent advances in pharmacotherapy, including the introduction of angiotensin receptor-neprilysin inhibitors (ARNi), have shown promise in improving cardiac function and renal hemodynamics in experimental and early clinical studies, suggesting a potential future role in the management of canine DCM (Porciello et al., 2022).

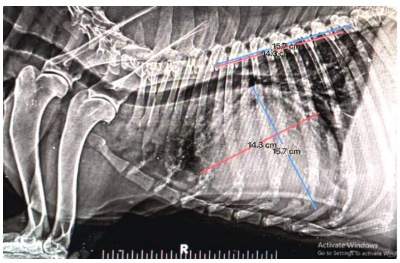

Fig. 1: Right lateral radiograph showing enlarged cardiac silhouette and VHS of 13.5.

Fig. 2: Electrocardiography (ECG) tracing depicting deep Q wave (circle), ventricular premature complexes (arrows) and elevated R

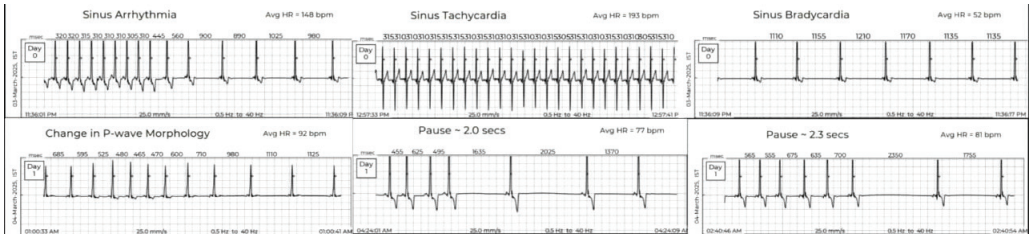

Fig. 3 : Holter ECG- sinus arrhythmia, episodes of sinus bradycardia and sinus tachycardia, alterations in P wave morphology, and intermittent pauses lasting up to 2.3 seconds

Figure 4: B-mode short axis view-dilation of left ventricle, thinning of inter-ventricular septum (IVS). M-mode echocardiograph- dilated left ventricle lumen and reduced EF

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