



International Journal of Veterinary Sciences and Animal Husbandry



ISSN: 2456-2912

NAAS Rating (2026): 4.61

VET 2026; 11(8): 178-180

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www.veterinarypaper.com

Received: 13-06-2026

Accepted: 12-07-2026

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Successful management of dilated cardiomyopathy in a Labrador retriever: A case report on clinical evaluation and therapeutic approach

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DOI: <https://www.doi.org/10.22271/veterinary.2026.v11.i8c.3733>

Abstract

Background: Dilated cardiomyopathy (DCM) is a progressive cardiac condition commonly seen in Labrador Retrievers, characterized by impaired myocardial contractility and arrhythmias. This case presents a 10-year-old male Labrador retriever diagnosed with DCM, exhibiting clinical signs of lethargy, exercise intolerance, and respiratory distress.

Case Description: The dog was presented with severe respiratory distress, muscle wasting, abdominal distension, coughing, and decreased exercise tolerance. Physical examination revealed tachycardia, dyspnea, and ascites. A grade III systolic murmur was detected, indicative of mitral valve regurgitation. Laboratory findings showed mild anemia and marginal azotemia, and radiographs confirmed significant cardiomegaly (VHS 13.2). Echocardiography revealed severe mitral regurgitation, left ventricular dilation, reduced EF% (19.59%), and an elevated left atrium-to-aorta ratio (2.33). Electrocardiography showed atrial fibrillation with a heart rate of 180 bpm.

Diagnosis: The dog was diagnosed with dilated cardiomyopathy with moderate systolic dysfunction.

Clinical Management: The treatment plan included Pimobendan, Benazepril, diuretics, Diltiazem, Digoxin, taurine, L-carnitine, and a low-sodium diet to improve cardiac function and manage symptoms.

Outcome: Significant clinical improvement was noted, with echocardiographic EF% increase. The patient's heart rate and rhythm stabilized, and the overall quality of life improved significantly.

Keywords: Dilated Cardiomyopathy, Labrador retriever, Mitral Regurgitation, Atrial Fibrillation, Systolic Dysfunction, Echocardiography, Pimobendan, Cardiac Management, Canine Cardiology, and Veterinary Medicine.

Introduction

Dilated cardiomyopathy (DCM) is a prevalent cardiac disorder primarily affecting large and giant dog breeds, with Labrador Retrievers being among the most commonly predisposed. This condition is characterized by impaired myocardial contractility, progressive ventricular dilation, and arrhythmias, ultimately leading to systolic dysfunction and congestive heart failure. Clinical manifestations typically include exercise intolerance, lethargy, respiratory distress, coughing, and abdominal distension, reflecting the multisystemic nature of the disease (O'Grady & O'Sullivan, 2004; Meurs, 2019) ^[1, 2].

The etiology of DCM in Labrador Retrievers may involve genetic predisposition and dietary factors, including deficiencies in taurine and L-carnitine, both of which play critical roles in myocardial energy metabolism (Freeman *et al.*, 2001; Fascetti *et al.*, 2003) ^[3, 4]. Diagnostic evaluation typically combines advanced imaging modalities such as echocardiography and electrocardiography, along with laboratory findings, to assess cardiac structure, function, and rhythm disturbances (Tidholm *et al.*, 2001) ^[6].

Effective management of DCM requires a multidisciplinary approach, incorporating pharmacological agents to improve myocardial function, control arrhythmias, and manage congestive heart failure. While therapeutic advancements have improved outcomes, DCM remains a progressive and life-threatening condition in affected dogs (Fuentes *et al.*, 2020) ^[6]. This case study details the clinical presentation, diagnostic findings, therapeutic interventions,

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And outcomes of a 10-year-old male Labrador retriever diagnosed with DCM at the Raintree, emphasizing the importance of comprehensive care in managing this condition.

Case History and Clinical Observations

A 10-year-old male Labrador retriever was presented to the Raintree with a history of progressive lethargy, muscle wasting, decreased exercise tolerance, abdominal distension, coughing, and respiratory distress. These signs had reportedly worsened over the preceding weeks, significantly affecting the dog's quality of life. On physical examination, the dog exhibited tachycardia, dyspnea, and ascites, consistent with advanced cardiac dysfunction and possible congestive heart failure. Auscultation revealed a grade III systolic murmur (S1) at the left cardiac apex, strongly suggestive of mitral valve regurgitation. These findings, coupled with the dog's clinical history, indicated a severe cardiac condition necessitating further diagnostic evaluation.

Laboratory Findings

Hematobiochemistry results revealed mild anemia and marginal azotemia in the patient. The mild anemia, characterized by a decreased hemoglobin concentration and packed cell volume, is likely reflective of chronic disease and the systemic effects of cardiac dysfunction. The presence of marginal azotemia, indicated by an elevated blood urea nitrogen (BUN) and creatinine levels, further supports the hypothesis of impaired renal perfusion secondary to congestive heart failure. These findings are consistent with the progressive nature of dilated cardiomyopathy (DCM), which can lead to both cardiac and renal compromise in affected dogs. No significant abnormalities were noted in other biochemical parameters, such as total protein, albumin, or liver enzymes, suggesting the primary pathology is cardiac in nature.

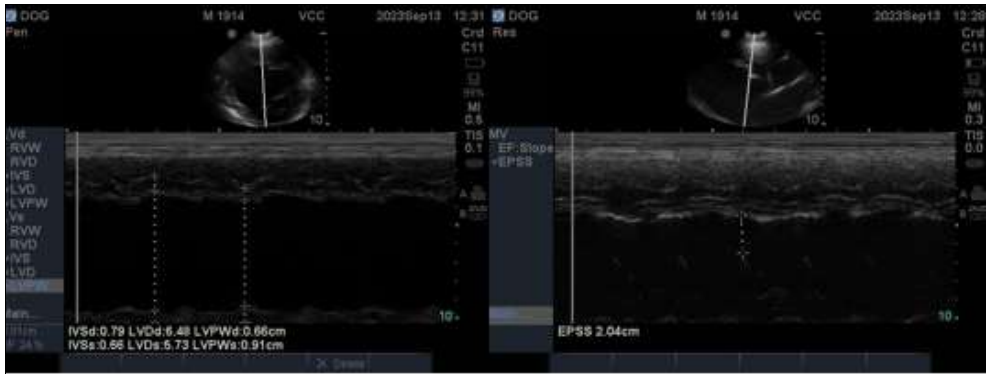
Table 1: Hematobiochemistry Parameters of the Patient

Parameter	Observed Value	Reference Range	Interpretation
Hemoglobin (g/dL)	10.5	12–18	Mild anemia
Packed Cell Volume (PCV, %)	30.5	37–55	Mild anemia
Blood Urea Nitrogen (BUN, mg/dL)	28	7–27	Marginal azotemia
Creatinine (mg/dL)	1.6	0.5–1.5	Marginal azotemia
Total Protein (g/dL)	6.5	5.4–7.5	Normal
Albumin (g/dL)	3.2	2.3–3.6	Normal
Alanine Aminotransferase (ALT, IU/L)	45	10–100	Normal
Alkaline Phosphatase (ALP, IU/L)	150	23–212	Normal

Radiographic Findings

Thoracic radiographs revealed marked cardiomegaly, with a vertebral heart score (VHS) of 13.2, which is well above the normal reference range for Labrador Retrievers. This finding is indicative of significant cardiac enlargement, consistent with the diagnosis of dilated cardiomyopathy (DCM). The enlarged heart is characteristic of ventricular dilation and impaired myocardial contractility, both of which are hallmarks of DCM. Additionally, signs of pulmonary congestion and mild pleural effusion were noted, which are commonly observed in dogs with advanced heart failure.

Echocardiography: Severe mitral regurgitation was detected, accompanied by marked left ventricular dilation. The left ventricular dimensions were significantly increased, with fractional shortening reduced to 19.59%, indicating moderate systolic dysfunction. Additionally, the left atrium-to-aorta ratio was elevated to 2.33, suggesting left atrial dilation, a common finding in dogs with chronic mitral valve disease and dilated cardiomyopathy. These echocardiographic findings corroborate the diagnosis of dilated cardiomyopathy with concurrent mitral valve regurgitation.



Electrocardiography: Atrial fibrillation was noted with a heart rate of 180 bpm. This arrhythmia is often associated with dilated cardiomyopathy and indicates electrical instability within the heart. Atrial fibrillation contributes to both the clinical signs of exercise intolerance and the increased risk of thromboembolic events in affected dogs.

Diagnosis

The final diagnosis for this patient was dilated cardiomyopathy (DCM) with moderate systolic dysfunction.

The clinical signs, laboratory findings, radiographic evidence, and special investigations including echocardiography and electrocardiography were all consistent with this diagnosis. The patient presented with significant cardiac enlargement, severe mitral regurgitation, impaired left ventricular contractility, and electrical disturbances, all of which are characteristic features of DCM. Additionally, moderate systolic dysfunction, as evidenced by a reduced fractional shortening of 19.59%, further supports the diagnosis.

Clinical Management and Follow-up Care

The treatment protocol for this patient was designed to address both the underlying dilated cardiomyopathy (DCM) and its associated complications. Pimobendan (0.25 mg/kg, q12h) was prescribed to improve myocardial contractility and reduce cardiac workload, helping to manage the heart failure symptoms. Benazepril (0.5 mg/kg, q12h), an ACE inhibitor, was administered to decrease afterload, thereby improving cardiac output and reducing further myocardial damage. Diuretic therapy with Furosemide and Spironolactone (2–4 mg/kg, TID) was introduced to manage pulmonary congestion and ascites, alleviating fluid retention and respiratory distress. Additionally, Diltiazem (1.5 mg/kg, TID) was used to control atrial fibrillation and maintain a controlled heart rate, while Digoxin (0.0025 mg/kg, BID) was prescribed to enhance myocardial contractility and assist with rate control in the presence of atrial fibrillation. Taurine and L-carnitine supplements were also included in the treatment plan to support myocardial function, as deficiencies in these nutrients are common in dogs with DCM. Furthermore, a low-sodium diet was recommended to minimize fluid retention and reduce the cardiac workload. Regular follow-up appointments were scheduled every two months, involving hematobiochemistry analysis, echocardiography, and electrocardiography to monitor the patient's response to therapy and adjust the treatment plan accordingly.

Outcome

The patient showed significant clinical improvement following the initiation of the treatment protocol. Echocardiographic follow-ups revealed a marked improvement in cardiac contractility, with fractional shortening increasing from 19.59% to 33.12%. Over the course of five months of therapy, the patient's heart rate and rhythm stabilized, and the overall quality of life improved substantially. The dog's clinical signs, including lethargy and respiratory distress, were alleviated, and exercise tolerance improved, reflecting the positive impact of the management plan. Regular follow-ups confirmed the stabilization of heart function and the effective control of arrhythmias. This case underscores the importance of early intervention and comprehensive management in improving the prognosis and quality of life for dogs with dilated cardiomyopathy.

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How to Cite This Article

Nanda PP, Verma A, Hetal H, Kelaiya J. Successful management of dilated cardiomyopathy in a Labrador retriever: A case report on clinical evaluation and therapeutic approach. *International Journal of Veterinary Sciences and Animal Husbandry.* 2026;11(7): 178-180.

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