# **Research Article**

## DIAGNOSIS AND THERAPEUTIC MANAGEMENT OF DILATED CARDIOMYOPATHY IN LABRADOR DOG

# E. VENKATESAKUMAR<sup>1\*</sup>, R.C. SUNDARA RAJAN<sup>2</sup>, P.A. ENBAVELAN<sup>3</sup>, R. RAMPRABHU<sup>4</sup> AND K. JEYARAJA<sup>5</sup>

<sup>1</sup>Department of Veterinary Clinical Medicine, Veterinary College and Research Institute, Namakkal, 637002, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India

<sup>2,3</sup>Department of Veterinary Medicine, Veterinary College and Research Institute, Tirunelveli, 627358, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India

<sup>4</sup>Veterinary Clinical Complex, Veterinary College and Research Institute, Tirunelveli, 627358, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India

<sup>5</sup>Department of Veterinary Clinical Medicine, Madras Veterinary College, Chennai, 600007, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India

\*Corresponding Author: Email - drvenkat75@gmail.com

Received: December 01, 2018; Revised: December 11, 2018; Accepted: December 12, 2018; Published: December 15, 2018

Abstract: Seven years old male Labrador dog was presented with cough, exercise intolerance, loss of appetite and bodyweight. Clinical examination showed moderate abdominal distention with loss of bodily condition, coughing and dyspnoea. Cardiac murmur, extended cardiac sounds and exaggerated breathing sounds over lung field were observed. Haematological parameters showed leukocytosis and neutrophilia. There was elevated creatinine and Blood urea nitrogen (BUN) and reduced total protein and albumin. Radiography of thorax showed cardiomegaly and pulmonary infiltration. Electrocardiography (ECG) revealed sinus arrhythmia and ST depression. Abdominal ultrasonography revealed moderate peritoneal effusion. Echocardiography showed dilated cardiomyopathy (DCM) with reduced cardiac output. Doppler echocardiography showed with regurgitation of mitral and tricuspid valves. The systolic blood pressure was 168 mm Hg. The animal was treated with Tab. Enrofloxacin @ 5 mg/kg PO for five days and Tab. Pimobendan @ 0.5 mg/kg divided dose PO, Tab. Furosemide @ 2 mg/kg PO and Tab. Enalapril @ 0.5 mg/kg PO. The animal showed improvement in feeding and reduction of cough. Periodical cardiac evaluation and continuous administration of medications were done. The animal showed marked improvement after the initiation of therapy.

**Keywords:** Dilated cardiomyopathy, Labrador, Echocardiography, Colour flow Doppler, Pimobendan

Citation: E. Venkatesakumar, et al., (2018) Diagnosis and Therapeutic Management of Dilated Cardiomyopathy in Labrador Dog. International Journal of Agriculture Sciences, ISSN: 0975-3710 & E-ISSN: 0975-9107, Volume 10, Issue 23, pp.- 7599-7601.

**Copyright:** Copyright©2018 E. Venkatesakumar, *et al.*, This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution and reproduction in any medium, provided the original author and source are credited.

Academic Editor / Reviewer: Dr P Thirunavukkarasu

#### Introduction

Dilated cardiomyopathy (DCM) is one of the most common cardiac diseases in canine and is characterized by ventricular enlargement, impaired myocardial function and tachyarrhythmias[1]. It occurs commonly in large breeds of dogs and is commonly idiopathic in nature. DCM is occurring due to various etiological factors like genetic factor, nutritional deficiencies, metabolic disorders, immunologic abnormalities, infectious and drug induced myocardial hypokinesis [2]. It is mostly an adult onset disease with a clinical presentation of exercise intolerance, weight loss, coughing, respiratory distress and ascites [3]. Higher prevalence of dilated cardiomyopathy in Labrador dogs was due to the mode of inheritance in the ancestral population [4]. The traditional therapeutic management of congestive heart failure due to DCM included use of angiotension converting enzyme inhibitors (ACE inhibitors), diuretics and positive ionotropes like digoxin or pimobendan [5]. The present study documented the diagnosis and therapeutic management of dilated cardiomyopathy in a Labrador dog.

### Case history and observations

A seven years old male Labrador dog weighing about 26 kg was presented to Small Animal Medical unit of Veterinary Clinical Complex, Veterinary College and Research Institute, Tirunelveli with the history of cough, exercise intolerance, loss of appetite and loss of bodyweight for the past one month. Clinical examination revealed moderate abdominal distention with loss of bodily condition, pink and moist mucous membrane, rectal temperature 39.2° C, pulse rate 106/min,

coughing and dyspnoea [Fig-1]. Auscultation of thorax revealed cardiac murmur, extended cardiac sounds and exaggerated breathing sounds over lung field. Haematological parameters showed leukocytosis (32000/cumm) and neutrophilia (82%). There was elevated creatinine (1.8 mg/dl) and Blood urea nitrogen (89 mg/dl) and reduced total protein (4.8 g/dl) and albumin (1.9 g/dl). Radiography of thorax showed increased vertebral heart score(12.5) indicating cardiomegaly and also with pulmonary infiltration [Fig-2].

Electrocardiography revealed sinus arrhythmia and ST depression. The systolic blood pressure was 168 mm Hg. Ultrasonography of abdomen and thorax was done using MyLabTM 40 VET Esaote. Abdominal ultrasonography (B-mode) was done using curvilinear probe with convex array with 5.0 MHz and revealed moderate peritoneal effusion. Echocardiography (M-mode)was done using cardiac probe with band phased array 2.5 MHz in right parasternal long axis view and short axis view at the level of aortic outflow tract and showed increased left ventricle internal diameter diastole (LVIDd- 55.9 mm) and left ventricle internal diameter systole (LVIDs-44.5 mm) indicating dilated cardiomyopathy and also showed reduction in ejection fraction (EF-41 %) and fractional shortening (FS-20 %) indicating reduced cardiac output [Fig-3].Left Atrium: Aorta ratio (LA/Ao) was 2.2. Doppler echocardiography showed mosaic pattern over left atrium and right atrium indicating regurgitation of mitral [Fig-4] and tricuspid valves [Fig-5], respectively.

||Bioinfo Publications|| 7599



Fig-1 Labrador dog with DCM showing respiratory distress and moderate abdominal distention



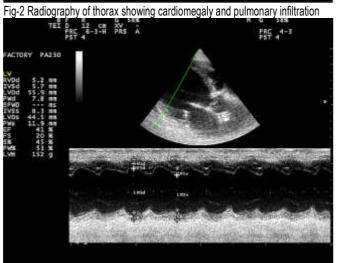


Fig-3 Echocardiography (right parasternal long axis view) showed DCM with Low cardiac output

## **Treatment and Discussion**

The animal was administered with Tab. Enrofloxacin @ 5 mg/kg PO for five days to check the infection of respiratory tract and Tab. Pimobendan @ 0.5 mg/kg bwtdivided dose twice daily PO, Tab. Furosemide @ 2 mg/kg bid PO and Tab. Enalapril @ 0.5 mg/kg bid PO for 15 days. The animal showed improvement in feeding and reduction of cough. The revaluation of cardiac status was performed once in a month for six months then once in three months. The animal showed improvement in feeding and reduction of cough and exercise intolerance. Dilated cardiomyopathy is mainly occurring in adult age.

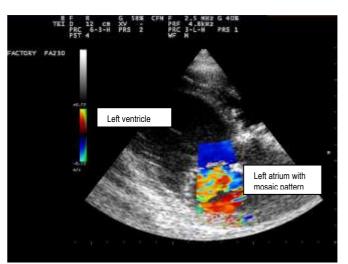


Fig-4 Doppler echocardiography showed mosaic pattern indicating mitral valve regurgitation

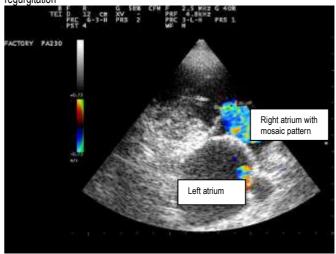


Fig-5 Right atrium showed mosaic pattern indicating tricuspid regurgitation

In the present study the male Labrador dog with seven years old was affected with DCM. Labrador dog with DCM had occurred with an average age of 6.68 ± 0.47 years and male dogs were affected more commonly than female dogs [4]. The clinical findings like cough, exercise intolerance, dyspnoea, cardiac murmur and abdominal effusion (Ascites) in the present study were in accordance with earlier researchers [4,6]. Dyspnoea in the present case might be due to pulmonary edema or infiltration subsequent to left sided failure and ascites due to right sided failure. Leukocytosis with neutrophilia in the present study was supported by earlier study[7] and contrasted by earlier researcher[6]. Increased leukocyte count might be attributed to concurrent bacterial infection of respiratory tract. So, antibiotic therapy with enrofloxacin was initiated for five days. There was significant decrease in the haemoglobin, packed cell volumes and red blood cell count in Labrador dogs with DCM[4]. Elevated BUN and creatinine in the present study might be due to reduced cardiac output resulting in prerenal azotaemia [4]. Laboratory abnormalities may reflect the effects of low cardiac output, congestion and neurohormonal activation [8]. Cardiomegaly in the present case was measured with increased Vertebral Heart Score (VHS) of 12.5 on radiography. VHS of 10.6 in health Labrador retrievers was recorded [9]. There was significant increase in the Vertebral Heart Score (12.5 ± 0.14) in DCM dogs compared to normal (10.78  $\pm$  0.03) and also reported pulmonary oedema and pleural effusion [4]. Sinus arrhythmia and ST depression observed in the present study was in accordance with early workers. ST depression was an indication of cardiomegaly. Atrial fibrillation, ventricular premature contraction and pulmonary oedema in few Labrador dogs with DCM was recorded [4, 10]. Atrial fibrillation was one of the most common arrhythmias in dogs with DCM [8].

Most of the dogs showed presence arrhythmia, abnormal amplitude and duration of P wave and QRS complex due to cardiac chamber enlargement due to DCM. However, the fluid accumulation either in pleural cavity or peritoneal cavity would reduce the amplitude of the QRS complex. Echocardiographic values of increased LA/Ao ratio, LVIDd and LVIDs and decreased ejection fraction (EF) and fractional shortening (FS)were indicating dilated cardiomyopathy with reduced cardiac output. The findings were concurring with earlier workers. Increased LA/Ao ratio  $(2.01\pm0.06)$ , LVIDd(cm)  $(5.84\pm0.13)$  and LVIDs (cm)  $(5.05\pm0.12)$  and decreased EF % (29.11±2.13) and FS % (13.45±0.73) were recorded in Labrador dogs with DCM[4]. LVIDd (mm) 37.58±1.05, LVIDs (mm) 23.98±0.97, EF(%) 65.50±2.15 and FS% 35.89±1.56 were recorded in normal Labrador dogs[11]. The range of left atrium to aortic root was between 0.8 and 1.2 in normal dogs [12]. Left ventricular internal diameter at end systole (LVIDs) and diastole (LVIDd) was very much useful in the assessment of cardiomyopathies in dogs [13]. Colour Doppler evaluation of DCM cases showed mitral insufficiency secondary to dilatation of mitral annulus. The annulus pulled apart, the mitral valve leaflets could not coaptate and not create a tight seal when the heart contracts leading to blood back flow across the valves Valvular insufficiency due to DCM contributed to the clinical signs of congestive heart failure [14]. In the present study both mitral and tricuspid valvular insufficiency was noticed due to DCM. Dogs with DCM had both mitral and tricuspid regurgitation which was appreciated as aliased regurgitated flow having mosaic pattern in atrium which was due to abnormal dilation of cardiac chambers and consequent leak in the atrioventricular valves [15]. Pimobendan is a benzimidazole-pyridazinone derivative with calcium sensitizer and phosphodiesterase III inhibitor effect leading to positive inotropy and vasodilator action on cardiac muscle. Treatment with pimobendan in congestive heart failure due to DCM was showing marked survival benefits in dogs. It is an approved drug in atrioventricular insufficiencies in dogs [5, 16]). In the present study, combination of pimobendan, enalapril and furosemide therapy showed very good support in the survival of a Labrador dog with DCM.

#### Summary

Seven years old Labrador dog was diagnosed as affected with dilated cardiomyopathy by clinical examination, ECG, radiography and echocardiography. The animal was successfully managed with pimobendan, enalapril and furosemide. The animal showed marked improvement.

**Application of research:** The methodology is useful in the management of dilated cardiomyopathy in dogs.

Research Category: Cardiology in dogs

**Acknowledgement / Funding:** Authors are thankful to Veterinary College and Research Institute, Tirunelveli, 627358, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India.

\*Principal Investigator or Chairperson of research: Dr E. Venkatesakumar University: Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu

Research project name or number: Clinical case report

Author Contributions: All authors equally contributed

**Author statement:** All authors read, reviewed, agreed and approved the final manuscript

Conflict of Interest: None declared

**Ethical approval:** Therapeutic intervention at Veterinary Clinical Complex, Veterinary College and Research Institute, Tirunelveli, 627358, Tamil Nadu Veterinary and Animal Sciences University, Chennai, 600051, Tamil Nadu, India. Ethical Committee Approval Number: Nil

#### References

- [1] Tidholm A., Haggstrom J., Borgarelli M. and Tarducci A. (2001) Vet J., 162. 92-107.
- [2] Janus I., Nowak A.N., Nowak M., Ciputa R., Gola M.K. and Paslawaka U. (2016) *BMC Vet research.*, 12, 3.
- [3] Kathryn M.M. (2005) Canine dilated cardiomyopathy insights into diagnosis and management. Proceeding of the NAVC North American Veterinary Conference, Florida, 19-122.
- [4] Jeyaraja K., Arun A., Hamsa Yamini S., Sesh P.S.L. and Nambi A.P. (2015) *International Journal of Advanced Research*, 3(12), 628 655.
- [5] Bowles, D. and Fry, D. 2011 Vetfolio-Compendium, 33(11), E1-E5.
- [6] Martin M.W.S., Stafford M.J., Strehlau G. and King J.N. (2010) Journal of Small Animal Practice, 51, 428–436.
- [7] Sesh P.S.L., Venkatesan P., Jeyaraja K., Chandrasekar M. and Pandiyan V. (2013) *International Journal of Advanced Veterinary Science and Technology*, 2(1), 47-51.
- [8] McEwan J.D., Borgarelli M., Tidholm A., Vollmar A.C. and Häggström J. (2003) *Journal of Veterinary Cardiology*, 5, 7-19.
- [9] Lamb C. R., Wikeley H., Boswoo, A. and Pfeiffer D.U. (2001) Veterinary Radiology and Ultrasound, 46(2), 122–130.
- [10] Thangapandiyan M., Mohanapriya T., Balachandran C., Jeyaraja K., Arulnadam K. and Sridhar R. (2016) *International Journal on Science, Environment and Technology*, 5(6), 4612-4617.
- [11] Gugjoo M.B., Hoque M., Saxena A. C., Shamsuz Zama M. M.and Dey S. (2014) *Iranian Journal of Veterinary Research*, 15(4), 341-346.
- [12] Bonagura J.D., O'Grady M.R. and Herring D.S. (1985) Vet Clin North Am Small AnimPract, 15(6), 1177-94.
- [13] Kittleson M.D. (1998) Primary myocardial disease leading to chronic myocardial failure. In: Kittleson, M.D. and Kienle, R.D. (Eds): Small Animal Cardiovascular Medicine. Mosby, St Louis, MO, USA, 319-346.
- [14] Boon J.A. (2011) Veterinary Echocardiograhy. 2<sup>nd</sup> ed. Wiley-Blackwell publication, Ames, Iowa, USA. 381-393.
- [15] Jeyaraja K., Hamsa Yamini S. and Thirunavakkarasu P.S. (2016) International Journal of Advanced Veterinary Science and Technology, 5(2), 256-265
- [16] Fuentes V.L. (2004) Vet Clin North Am Small AnimPrac, 34, 1145-1155.