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Short Communication

DIAGNOSTIC AND THERAPEUTIC MANAGEMENT OF IDIOPATHIC DILATED CARDIOMYOPATHY(DCM) IN ASSOCIATION WITH CARDIO-PULMONARY OEDEMA IN A LABRADOR RETRIEVER DOG

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SUMMARY

The aim of this case report is to document the complete diagnostic and therapeutic management of idiopathic dilated cardiomyopathy (DCM), associated with cardiopulmonary oedema and its prognosis in a Labrador Retriever dog. A 9-year-old male Labrador Retriever dog weighing 37 Kg was presented at VCC, Rajendranagar, with the history of exercise intolerance, severe respiratory distress, vigorous dry cough, abdominal sleeping posture with abducted elbows, dyspnoea at rest, insomnolence and lethargy. On complete physical and clinical examination of the animal, tachyarrhythmia, weak femoral pulse, dyspnoea at rest and abdominal breathing was observed.

Keywords: DCM, Dog, Exercise Intolerance, Dyspnoea

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Thoracic radiograph of lateral view and dorso-vental view documented an enlarged silhouette of heart (Cardiomegaly) with a Globose structure and mild cloudiness of the cranio-pulmonary lobe of the lungs, suggesting pulmonary edema (Fig. I). The VHS score recorded was 11.2., indicative of enlarged heart. The abdominal ultrasonography findings revealed free intra-abdominal fluid accumulation, indicative of ascites.

Echocardiography (2D-echo) was performed on the dog which revealed, bilateral atrial enlargement with delayed closure of the tricuspid valves, suggestive of right atrioventricular valvopathy (Fig. II). The left atrium to aorta ratio was calculated to be greater than 1.5 (recorded value=3.25), while M-mode recorded a shortening fraction (FS=12.2%) indicative of a decreased cardiac output. Pulse wave Doppler recorded moderate turbulence in the right atrial regions (Fig. III), while the Colour Doppler showed a mosaic pattern, suggestive of tricuspid valve regurgitation (Fig. IV). The electrocardiogram reports showed atrial fibrillation and intermittent ventricular premature contractions (VPCs). Low voltage QRS complexes were recorded, with few motion artifacts (Fig. V). The hematological and biochemical results were normal. Thyroxine and taurine serum concentrations were normal. Urinalysis results were unremarkable.

Based on the above findings, the case was diagnosed as dilated cardiomyopathy with right atrio-ventricular valvopathy and cardio-pulmonary edema. The treatment was initiated with Tab. Safeheart-5 @0.5 mg/Kg b.w.t OD p/o, Tab. Acceptor-5 @0.25-0.5 mg/Kg b.w.t OD/BID p/o, Tab. Lasilactone@2-4 mg/Kg b.w.t I/m and Tab. Carnisure-500. Strict sodium restricted diet was initiated with permanent exercise restriction. After 6 months of vigorous therapeutic management, the dog showed visible improvement with reduced abdominal distension and respiratory distress. Post-treatment thoracic radiograph documented visibly decreased cardiac size and absence of globoid structure (Fig. VI). 2D-Echo images recorded decreased diameters of the left atrium and aorta. Pulse wave doppler revealed reduced atrial fibrillation (Fig. VII).

Canine DCM is a multietiological progressive cardiac muscle disease of the canines, that impairs the myocardial contractibility and is characterized by dilation of the heart chambers (Borgarelli *et al.*, 2006). The etiological conditions vary from inherent causes, nutritional deficiencies to senility. The clinical signs and diagnostic parameters of the present case are in accordance with the studies. (Tidholm *et al.*, 2001). Pulmonary oedema being the most common complication and a sign of advanced DCM, as was noticed in the present case, is indicative of grave prognosis (Monnet *et al.*, 1995). The occurrence of Cardiopulmonary oedema and ascites as a complication of the right sided heart failure and tricuspid valvopathy corroborates with the studies of (Brloznik *et al.*, 2017). The treatment protocol of DCM includes a positive ionotropic vasodilator Pimobendan, ACE inhibitors, diuretics, and antiarrhythmics (McEwan *et al.*, 2003). The diagnostic and therapeutic management of DCM and its complications is henceforth evaluated in this study.

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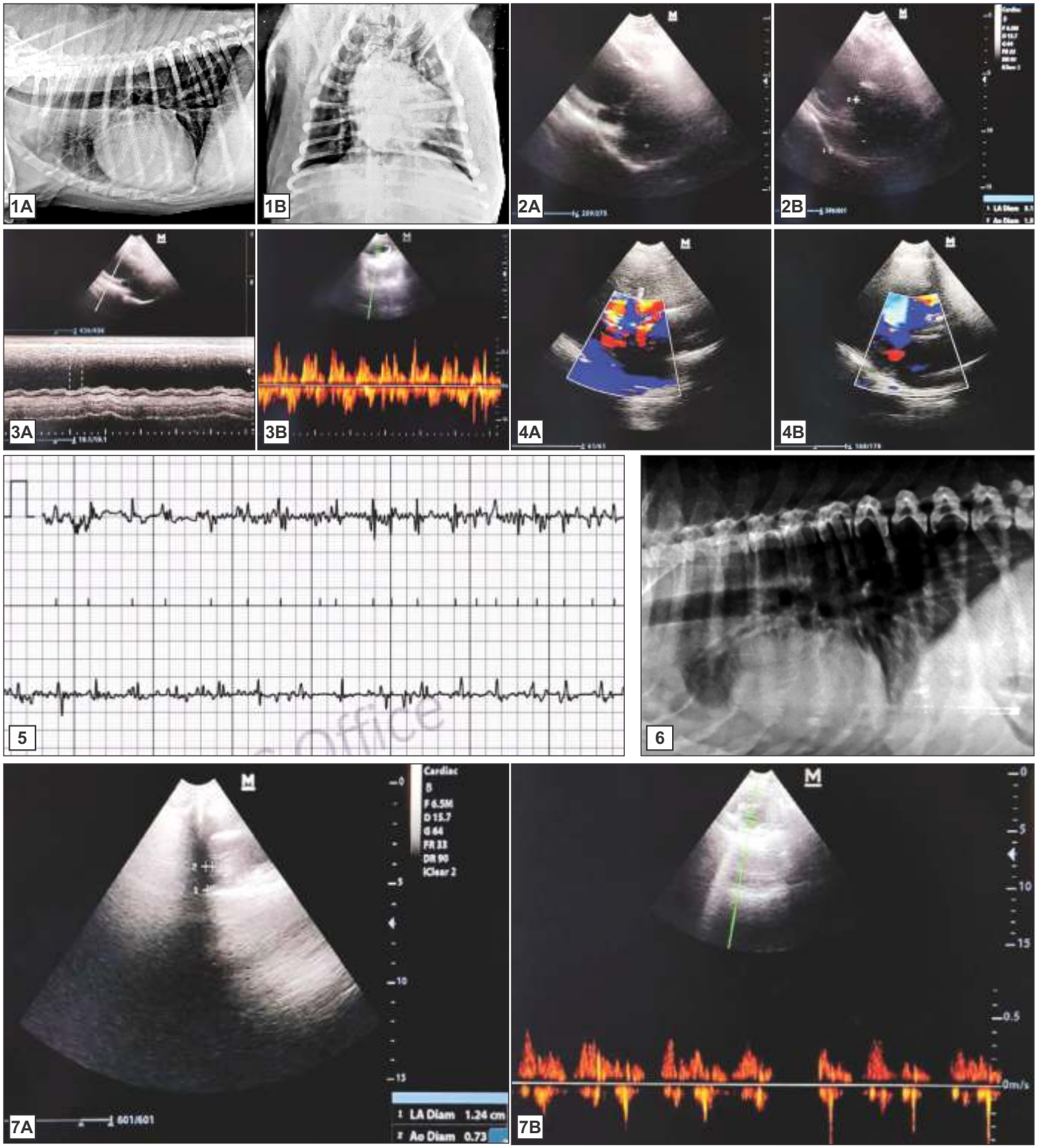

Fig. I. Lateral **(A)** and dorso-ventral **(B)** thoracic radiographs show cardiomegaly. A GLOBOID shaped heart is observed. The cranio-pulmonary lobe shows mild cloudiness, suggestive of pulmonary oedema. **Fig. II.** The 2D-Echo reveals chamber dilatation **(A)**. The Left atrial diameter and the Aortic diameter values were 5.18 cm and 1.59 cm respectively **(B)**. The left atrium to aorta diameter ratio recorded was 3.257 (more than the statistical values of its specific range). **Fig. III.** The M-mode echocardiographic images show an increased ventricular luminal width **(A)**. The Pulse-wave Doppler recorded atrial regurgitation with distinct atrial fibrillations **(B)**. **Fig. IV.** Color Doppler images documented mosaic patterns in the ventricles; indicative of turbulence associated with right atrio-ventricular valvopathy. **Fig. V.** ECG recordings at Lead II recorded atrial fibrillation and intermittent ventricular premature complexes (VPCs) at short intervals. (ECG documented in WPS Office format via Kardia). **Fig. VI.** The post therapeutic lateral thoracic radiographic image shows a reduced cardiac size. **Fig. VII.** The post therapeutic M-mode 2D-Echo images of the left atrial and aortic diameters measured 1.24 cm and 0.73 cm respectively. LA: A ratio calculated was 1.69 **(A)**. Pulse-wave doppler image reveals reduced atrial fibrillation and turbulence **(B)**.

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